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Augmenting Cognitive Processing Therapy to Improve Sleep Impairment in PTSD: A Randomized Controlled Trial

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Objective: Despite the success of empirically supported treatments for posttraumatic stress disorder (PTSD), sleep impairment frequently remains refractory after treatment. This single-site, randomized controlled trial examined the effectiveness of sleep-directed hypnosis as a complement to an empirically supported psychotherapy for PTSD (cognitive processing therapy [CPT]). ***Method:*** Participants completed either 3 weeks of hypnosis (n = 52) or a symptom monitoring control condition (n = 56) before beginning standard CPT. Multilevel modeling was used to investigate differential patterns of change to determine whether hypnosis resulted in improvements in sleep, PTSD, and depression. An intervening variable approach was then used to determine whether improvements in sleep achieved during hypnosis augmented change in PTSD and depression during CPT. ***Results:*** After the initial phase of treatment (hypnosis or symptom monitoring), the hypnosis condition showed significantly greater improvement than the control condition in sleep and depression, but not PTSD. After CPT, both conditions demonstrated significant improvement in sleep and PTSD; however, the hypnosis condition demonstrated greater improvement in depressive symptoms. As sleep improved, there were corresponding improvements in PTSD and depression, with a stronger relationship between sleep and PTSD. ***Conclusion:*** Hypnosis was effective in improving sleep impairment, but those improvements did not augment gains in PTSD recovery during the trauma-focused intervention.

What is the public health significance of this article?
This study suggests that hypnosis may be a viable treatment option in a stepped-care approach for treating sleep impairment in individuals suffering from PTSD.

Keywords: PTSD, sleep impairment, insomnia, cognitive processing therapy, hypnosis

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Consistently, clinical practice guidelines identify trauma-focused interventions, such as prolonged exposure (Foa & Rothbaum, 1998) and cognitive processing therapy (CPT; Resick, Monson, & Chard, 2010), as effective treatments for posttraumatic stress disorder (PTSD; see Forbes et al., 2010, for a review). Despite the demonstrated efficacy of these (and other) psychosocial interventions for PTSD, improvement is not universal across patients, nor is recovery equitable across symptoms (Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008; Spoormaker & Montgomery, 2008). The augmentation of existing evidence-based

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protocols with adjunctive interventions may enhance outcomes, particularly if interventions target difficult-to-treat features of PTSD, such as trauma-related sleep impairment. Difficulty in sleep onset and maintenance is perhaps the most frequently reported symptom of PTSD (Germain, 2013), with up to 70% of survivors diagnosed with PTSD reporting clinically significant sleep impairment (Ohayon & Shapiro, 2000).

An empirically derived theory of the relation between PTSD and sleep is lacking; however, it is clear that sleep disturbance following trauma is predictive of the development and maintenance of PTSD (Harvey, Jones, & Schmidt, 2003). Theoretically, trauma-related sleep impairment may be biologically adaptive and related to hypervigilance, such that sleep may become disrupted by the perception that vigilance must be maintained at night (Barlow, 2002). The presumed need for constant vigilance should extinguish over time in the absence of threat. However, in the case of PTSD, hypervigilance is maintained and individuals may intentionally stay awake despite exhaustion or experience significant worry about danger during the night. The disruptive experience of nightmares and association of sleep-related trauma cues (e.g., darkness) with danger may further disrupt sleep through the development of fear of sleep (DeViva, Zayfert, & Mellman, 2004). Trauma-related sleep impairment may then evolve into an independent dysfunction, complicating general distress and decreasing ability to cope with trauma cues during the daytime, thereby exacerbating PTSD symptoms (Krakow, Hollifield, et al., 2001).

Although potential overlap across domains of sleep architecture exists between nightmares and sleep impairment, descriptively and neurobiologically, the two are considered distinct symptoms in the pathophysiology of PTSD and may warrant specified interventions. Polysomnographic study, patient self-report, electroencephalography, and sleep neuroimaging methodology have identified both REM and non-REM (NREM) abnormalities in PTSD sleep disturbances (Germain, 2013). Nightmares, specifically, have historically been conceptualized as occurring during REM sleep, while insomnia is considered a result of NREM sleep disruptions. However, trauma-related nightmares in PTSD have been shown to occur in NREM sleep (van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984), and, further, not all distressed midsleep awakenings in PTSD are associated with nightmares (Krakow et al., 2000).

Historically, sleep impairment in PTSD has rarely received specific primary intervention (Krakow, Johnston, et al., 2001), perhaps due to the prior conceptualization of sleep impairment as a secondary symptom of PTSD as opposed to a core feature of the disorder. Spoormaker and Montgomery (2008) concluded that the accumulation of evidence supports sleep disturbance in PTSD as a core feature of the disorder, thereby warranting specific intervention. Consistent with this conceptualization, sleep impairment may not remit, despite improvement, following PTSD treatment (Babson & Feldner, 2010). One study found that although sleep significantly improved following cognitive–behavioral therapy (CBT) for PTSD, 70% of the participants reported significant sleep problems at posttreatment and improvements were not maintained after 6 months (Belleville, Guay, & Marchand, 2011). In a comparison of CPT and prolonged exposure, both interventions resulted in significant improvements in sleep; however, sleep impairment remained above clinical cutoffs (Galovski, Monson, Bruce, & Resick, 2009).

Interventions targeting PTSD-related sleep disruptions have shown improvements in both sleep and posttraumatic distress. Imagery rehearsal therapies (IRTs) are some of the most well-supported interventions for nightmares and are described as a "cognitive-imagery technique that directly targets nightmares of various types" (Moore & Krakow, 2010, p. 233). Variations in IRT protocols are significant, stemming from a variety of theories (Casement & Swanson, 2012) and differing in number of sessions, session length, amount of trauma exposure (Davis, 2009; Forbes, Phelps, & McHugh, 2001; Forbes et al., 2003; Long et al., 2011), extent of psychoeducation and sleep hygiene (Moore & Krakow, 2009), and the inclusion of CBT for insomnia (CBT-I; Ulmer, Edinger, & Calhoun, 2011). A recent meta-analytic review (Casement & Swanson, 2012) has suggested that IRTs show large effect sizes for nightmare frequency, sleep quality, and PTSD symptoms. Given the variability in treatment protocols, authors specifically noted the need for further research to identify components of the various IRT protocols necessary for PTSD symptom remediation. A second meta-analysis, including 20 studies testing both IRTs and interventions using imaginal confrontation with nightmare contents (ICNC), found large effects for nightmare frequency and PTSD and moderate effects for sleep quality and depression (Hansen, Höfling, Kröner-Borowik, Stangier, & Steil, 2013). Overall, interventions for trauma-related nightmares show promise; however, studies have not examined whether improvements in PTSD and depressive symptoms are a direct result of elements of the intervention or a result of the sleep improvement. Interpretation of results of IRT studies is complicated because distress levels typically remained in the clinical range, sleep worsened for some, participants were often in concurrent PTSD treatments, control groups were lacking, and dropout was often high or not reported (Harvey et al., 2003; Schoenfeld, DeViva, & Manber, 2012).

Studies assessing CBT specifically targeting insomnia (CBT-I) have also shown improvements in PTSD and sleep impairment, but have notable methodological limitations. CBT-I consists of a number of elements that may differ across protocols, including stimulus control, sleep restriction, sleep hygiene, cognitive restructuring, and relaxation. DeViva, Zayfert, Pigeon, and Mellman (2005) addressed *refractory* sleep impairment in PTSD by implementing CBT-I with five PTSD treatment responders. Results showed improvement in sleep time, sleep onset latency, wake time after sleep onset, and sleep efficiency, but scores did not drop below clinically significant cutoffs. In a larger randomized controlled trial (RCT), Talbot et al. (2014) examined an 8-week course of CBT-I without a nightmare-targeted component. CBT-I participants demonstrated improvements in sleep, with some variation depending on the assessment tool. However, study participants also received concurrent PTSD therapy. Therefore it is unknown whether the CBT-I intervention or resultant sleep benefits influenced PTSD or depressive symptoms.

Sleep impairment occurs in the context of a host of mood and anxiety disorders that often co-occur with PTSD. Polysomnographic sleep characteristics observed in individuals with PTSD are similar to those observed in individuals with major depressive disorder (Germain, 2013; Harvey et al., 2003). Sleep disturbance has been consistently linked with major depressive disorder, such that approximately 75% of depressed patients in clinical samples report significant sleep impairment (Tsuno, Besset, & Ritchie,

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2005). Staner (2010) reported that antidepressant drugs and use of CBT for depression were unrelated to the effects on complaints of insomnia. Insomnia has been identified as a predictor of depression and also has been shown to persist despite remission of depression. The specific relation between sleep impairment and depression in the context of PTSD has received less attention and warrants further investigation.

Present Study

The present study used an RCT design to evaluate the effectiveness of CPT preceded by either three weekly sessions of sleep-directed hypnosis (hypCPT condition) or three weeks of daily sleep and symptom monitoring (ssmCPT condition). Hypnosis provides deep relaxation to establish adaptive associations with environmental stimuli, thereby facilitating sleep onset and maintenance. Findings from case reports (Kennedy, 2002; Modlin, 2002; Scholz & Ott, 2000; Stanton, 1999) and one clinical trial (Stanton, 1989) have supported the effectiveness of hypnosis for sleep impairment in nontrauma populations. Further, hypnosis is a brief, nontrauma specific intervention that requires minimal therapist training. In the Stanton trial, hypnosis participants showed a 50% improvement in sleep after Session 2 and no differences in improvements were seen at the end of Session 3 compared with the end of treatment at Session 4. These results suggest that three weekly sessions of hypnosis are sufficient to achieve significant improvements in sleep, particularly sleep onset latency.

During the sleep intervention phase of this study, we hypothesized that hypCPT participants would demonstrate significant improvement in sleep (but not PTSD, nightmares, or depressive symptoms) compared with ssmCPT participants. The hypnosis intervention was specifically designed to target sleep onset and participants were instructed to practice autohypnosis before bedtime. Thus, we hypothesized that hypnosis would reduce sleep latency (time to fall asleep), increase global sleep quality, and increase overall sleep duration. Sleep in older adults (Cordi, Hirsiger, Mérillat, & Rasch, 2015) and in a nonclinical sample (Cordi, Schlarb, & Rasch, 2014) has been successfully "deepened" with hypnosis in two clinical trials showing increases in slow wave sleep by 57% and 81%, respectively. In the nonclinical sample, time spent awake was reduced by 67%. We did not hypothesize that hypnosis would directly target PTSD nightmares or related disruptions. Finally, we hypothesized that the relaxation strategies taught through the course of hypnosis would have a differential and beneficial effect on trauma-related sleep impairment.

We also hypothesized that improvements in sleep realized during hypnosis would augment CPT, such that PTSD symptom reduction (during CPT) would be greater in the hypCPT condition relative to the ssmCPT condition. Finally, given the similarities noted in sleep disturbance between depression and PTSD, we hypothesized that improvements in sleep during hypnosis would augment improvements in depression (during CPT) in hypCPT compared with the ssmCPT condition. We did not hypothesize that hypnosis would be a potent enough intervention to reduce PTSD or depression during the sleep-directed hypnosis portion of the intervention.

Method

Participants

A total of 108 female interpersonal assault survivors were enrolled in this single-site RCT. Participants were recruited from the St. Louis metropolitan area through flyers, advertisements, referrals, and word-of-mouth. Eligible participants satisfied the following inclusion criteria: female gender, diagnosis of PTSD secondary to sexual or physical assault, and clinically significant sleep impairment as indicated by a severity score of 3 or more on the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990) sleep impairment symptom (item D-1). Participants had to be at least 3 months posttrauma at initial assessment and stable on any psychotropic medication for at least 1 month. Participants could continue existing medications but were asked to keep medication usage stable. Exclusion criteria included psychosis, mental retardation, active suicidality, parasuicidality, or current drug or alcohol dependence. Individuals currently in an abusive relationship or being stalked were excluded. Participants could have received prior therapy, with the exception of CPT, and could receive concurrent therapy provided that it was not trauma or sleep focused. Before randomization, participants were asked to maintain the following standards throughout treatment: limit alcohol consumption to 14 servings per week with no more than five servings a day; limit caffeine consumption to 500 mg/day and to refrain from caffeine after 6:00 p.m.; and try not to vary bed and rise times by more than 1 hr. Sleep hygiene was not reviewed or revisited as part of the hypnosis intervention.

Measures

Clinician-Administered PTSD Scale. CAPS (Blake et al., 1990) is a 22-item scale that assesses the presence of PTSD. The CAPS contains separate 5-point frequency and intensity rating scales (0–4) for each *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* (*DSM–IV*; American Psychiatric Association, 1994) PTSD symptom. Items are summed to form a total score, with higher scores indicating greater severity (range: 0−136). The CAPS has demonstrated excellent reliability and validity (Weathers, Keane, & Davidson, 2001). Internal consistency for the 17 PTSD symptom items in the current study was good ($\alpha = .79$). This study used the total score minus the Sleep Impairment item, which was analyzed separately. The Sleep Impairment item in Cluster D (hyperarousal) was used as the study's measure of trauma-related sleep impairment (hypothesized to improve over the course of hypnosis) and the Nightmare item in cluster B (reexperiencing) was used to assess change in nightmares.

Interrater reliability on CAPS interviews. Initial reliability was established with new diagnostic interviewers by using training tapes and having initial interviews supervised and rated. After initial reliability was established, interviewers had audiotapes reviewed by senior project staff on an ongoing basis. Interviewers attended weekly group meetings to review diagnostic conceptualizations and to reconcile conflicting diagnostic decisions. Interrater reliability was conducted for a random sample of 24 CAPS interviews. CAPS cluster scores and total scores all achieved high reliability: CAPS kappa (current diagnosis) of 1.00; CAPS Pearson

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r of .96 (total score), .92 (Cluster B), .92 (Cluster C), and .90 (Cluster D).

Beck Depression Inventory−II. The Beck Depression Inventory−II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item measure that assesses depressive symptoms corresponding with the *DSM–IV* (American Psychiatric Association, 1994) criteria. Items are rated on a 4-point scale. Total scores are obtained by summing individual items and range from 0–63. The following cutoff score guidelines have been provided: 0–13 (*minimal*), 14–19 (*mild*), 20–28 (*moderate*), and 26–63 (*severe*). The BDI has well-established reliability and validity (Beck et al., 1996). Internal consistency was high in this study (α = .89).

Pittsburgh Sleep Quality Index. The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) is a self-rated questionnaire that assesses sleep over a 1-month interval. Nineteen items generate seven component scores, ranging from 0 (*no difficulty*) to 3 (*severe difficulty*), and are summed to yield a global score, with higher scores indicating poorer sleep (range: 0–21). Consistent with hypotheses, this study assessed global sleep as a measure of overall sleep quality, the Sleep Latency (time to fall asleep) subscale, and the Sleep Duration subscale (total amount of sleep obtained). Previous validation studies have suggested that global score of 5 or more indicates sleep disturbance (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). In this study, the PSQI had a Cronbach's alpha of .41. Further inspection suggested that internal consistency may have been adversely impacted by the medication component score (comprised of a single item). Internal consistency increased to an acceptable range (α = .57) with the medication component omitted. Weak correlation between the medication component score and PSQI global score has been observed in prior research (Spira et al., 2011).

Insomnia Severity Index. The Insomnia Severity Index (ISI; Morin, 1993) is a seven-item measure assessing insomnia symptoms and related distress in the past month. Items evaluate the severity of difficulties with sleep onset, sleep maintenance, early morning awakening, sleep dissatisfaction, interference of sleep problems with daytime functioning, noticeability of sleep difficulties by others, and distress caused by sleep difficulties. A 5-point Likert scale, ranging from 0 (*none*) to 4 (*very severe*), is used for each item. Total scores range from 0−28 and are interpreted according to the following cutoffs: absence of insomnia (0–7), subthreshold insomnia (8–14), moderate insomnia (15–21), and severe insomnia (22–28). The ISI has previously demonstrated adequate psychometric properties (Bastien, Vallières, & Morin, 2001) and showed good internal consistency for this study (α = .83).

Procedure

All study procedures were approved by the institutional review board at the University of Missouri−St. Louis. On completion of a telephone screen, participants were consented to the study and completed a baseline assessment consisting of clinician-administered and self-report measures of PTSD, depressive symptoms, and sleep impairment. Using a 1:1 allocation ratio, a computer-generated randomization sequence randomized eligible participants to the ssmCPT condition (n = 56) or the hypCPT condition (n = 52). A status check assessment was conducted at the conclusion of the initial 3-week sleep intervention phase. Participants were also assessed 2 weeks after completing CPT (posttreatment assessment) and 3 months after completing CPT (follow-up assessment). Consistent with intention-to-treat (ITT) principles, treatment dropouts were invited back for assessments. Participants were compensated modestly for assessments. Status check, posttreatment, and follow-up assessments were conducted by independent raters blind to treatment condition and completion status.

Therapists and Training

Therapists were two master's-level and two doctoral-level providers trained and supervised in the administration of sleep-directed hypnosis and CPT by the principal investigator. The CPT and hypnosis manuals and relevant readings were provided in the training. Therapists demonstrated adequate adherence and competence in the delivery of CPT with two training cases prior to commencing treatment with study participants. After randomization, participants were matched to a therapist based on availability. Throughout the study, therapists attended weekly 2-hr group supervisions with the principal investigator. All treatment sessions were video-recorded and portions of sessions were regularly reviewed in supervision.

Adherence and competence. Expert CPT clinicians not otherwise affiliated with the study reviewed and rated treatment sessions for adherence and competence. Treatment elements were rated for presence or absence (adherence) and for quality (competence), ranging from 1 (*not satisfactory*) to 7 (*excellent*). Raters coded a total of 61 sessions (8.6% of the total 710 sessions conducted, including removed participants). Additionally, a second independent rater coded 14 (23.0%) of these sessions to ensure reliability among independent raters. Interrater agreement across sessions was acceptable on both the presence of session elements (κ = .65) and the rating of session elements (r = .46). Using a single coding from each session, 96.8% of the unique session elements were judged to be present and 87.7% of present items were judged satisfactory or higher. The average rating of present elements across therapists was 4.51 out of 7. Regarding essential but not unique items (e.g., warmth, efficient structuring of session time), 93.8% of nonunique elements were judged to be present. Coders rated 92.6% of present elements judged as satisfactory or higher, with an average rating of 4.64 out of 7.

Treatment Conditions

Sleep and symptom monitoring plus CPT. Participation in ssmCPT began with 3 weeks of daily monitoring of PTSD, depressive symptoms, and sleep. SsmCPT participants completed weekly phone checks to ensure that they did not need emergency care and to assess symptoms. Participants were encouraged to call if they wanted to talk to their therapist. Thus, the symptom monitoring condition served to control for the passage of time, perceived social support, and expectancy of improvement. Prior studies have shown that symptom monitoring alone can have a therapeutic benefit (e.g., Galovski, Blain, Mott, Elwood, & Houle, 2012). Thus, this condition also controlled for any effect of monitoring in the active treatment condition (hypCPT).

After the 3 weeks of daily symptom monitoring, ssmCPT participants received CPT (see Resick et al., 2010, for a complete

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protocol). CPT was administered individually and consisted of 12 weekly, 60-min sessions. CPT is predominantly a cognitive therapy in which patients are taught to identify, question, challenge, and replace faulty assumptions and thoughts about the traumatic event and its implications in their current lives. While the patients engage with the traumatic memory, they are able to allow natural affect to run its course. CPT first targets specific, inaccurate interpretations of the trauma itself and then targets current and future maladaptive and inaccurate beliefs about world, self, and others across a number of belief systems typically disrupted after experiencing a traumatic event.

Hypnosis plus CPT. Participants assigned to the hypCPT condition received 3 weekly, 60-min sessions of sleep-directed hypnosis and monitored symptoms similarly to the ssmCPT group, prior to receiving CPT. The hypnosis intervention specifically targeted sleep impairment; no trauma-related material was included. A number of factors drove the decision to choose hypnosis as the sleep intervention. To adequately assess the added and specific benefit of sleep restoration prior to PTSD therapy, we first sought to reduce the potential for overlap in therapeutic elements used in cognitive behaviorally−oriented therapies. Second, we wanted the intervention to be as brief as possible while still effective in improving sleep so as not to substantially delay initiation of trauma-focused treatment. Third, we wanted an adjunctive therapy for sleep that did not require a significant level of additional specialization for the therapist. The hypnosis was scripted, thus the protocol was easily learned because the content was precisely provided for the therapist. Likewise, the script contained no trauma information, so trauma-related distress as an interruption to the palliative process of the hypnosis and sleep restoration was minimized. As such, hypnosis should have presented minimal increased risk of premature dropout. Dream therapy is not always well-tolerated by PTSD survivors potentiating increased risk in already high dropout sample (Schoenfeld et al., 2012). Finally, there has long been a call for additional research on alternative−complementary therapies for sleep (McCurry, Logsdon, Teri, & Vitiello, 2007; Montgomery & Dennis, 2004; National Institutes of Health, 2005). Hypnosis was suitable as a brief, effective, intervention that is easily standardized and learned, and designed to specifically target sleep impairment.

Hypnosis was conducted in accordance with an unpublished manual consisting of standardized scripts developed for this study (available on request). Following the precedent of Wright and Wright (1987), the hypnosis intervention was designed to achieve two goals. The first was to target sleep onset difficulty by reducing stimulation, decreasing sleep anxiety, and increasing the readiness to become relaxed and embrace sleep. In the first session, the therapist provided brief psychoeducation about the negative effects of sleep loss. Then hypnosis began by inducing a hypnotic trance using an eye fixation technique, which was then deepened using progressive muscle relaxation. The hypnosis script also includes guided imagery to enhance relaxation, ego-strengthening statements designed to increase efficacy and confidence related to sleep and relaxation capabilities, and suggestions for self-hypnosis and relaxation. The second goal of the intervention targeted midsleep awakening and the inability to fall back to sleep. Using a transition technique involving cues to help the subject move back into a sleep state on recognition of midsleep awakening, it was suggested that midsleep awakening is normal and peaceful, and that it is enjoyable and safe to slip back into a state of restfulness. Thus, the trance targeted arousal level such that the anxiety involved in midsleep awakening was recognized, but not mobilized. The mental activity involved in midsleep awakening was redirected into calming, familiar imagery. Sessions 2 and 3 began by problem solving any difficulties with home practicing and a repeat of hypnosis exercise. Thus the hypnosis content of Sessions 2 and 3 was nearly identical to Session 1 in an effort to cement skills and foster corrective information. Participants received an audiotape of the hypnosis conducted in session and were instructed to practice daily before bed. Following the 3 hypnosis sessions, twelve 60-min sessions of CPT were conducted on a weekly basis. The CPT portion of the therapy was identical in both conditions.

Analytic Strategy

We applied a multilevel approach to analyzing longitudinal data (Singer & Willett, 2003) to address the study hypotheses, using the NLME package from R (Pinheiro & Bates, 2000; Pinheiro, Bates, DebRoy, & Sarkar, 2012). Although a number of models/steps were assessed to arrive at a final conclusion, the ultimate model included a determination of the pattern of change for the modeled outcome, and whether the treatment groups (ssmCPT vs. hypCPT) differed in that change experience while simultaneously controlling for years of education. The models of change often had linear, quadratic, and cubic components (e.g., initial rate of change followed by some leveling and/or acceleration in that rate of change).

To address the effect size differences in change between assessment intervals, we assessed the difference in rates of change between the study conditions, treating them as independent samples (Feingold, 2009). Assessment intervals consisted of a baseline assessment (Interval 0), status check (after the sleep intervention phase; Interval 1), a posttreatment assessment (after the conclusion of the CPT; Interval 2), and a 3-month follow-up assessment (3 months after the conclusion of CPT; Interval 3).

Finally, we adopted an intervening variable approach coupled with the multilevel models of change to investigate whether hypnosis corresponded with improvements in PTSD and depression, via its impact on sleep symptoms (Krull & MacKinnon, 2001; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002).

Results

Participants

In all, 181 women were assessed for eligibility (see Figure 1). Of these, 62 did not meet study criteria and another 11 individuals did not complete the assessment. The majority of ineligible cases (56%) were excluded due to being PTSD negative. Of the 108 women randomized, 16 were excluded from analysis (ssmCPT n = 8; hypCPT n = 8), by design, for meeting exclusion criteria subsequent to eligibility (e.g., medication changes, n = 5). The ITT study sample (N = 92) included all randomized participants, except the 16 removed due to the later presence of exclusionary criteria.

Demographics. Randomization was largely successful. There were no demographic differences between the ssmCPT and the hypCPT conditions, except with respect to years of education (ssmCPT: M = 13.50 years, SD = 3.19; hypCPT: M = 14.77

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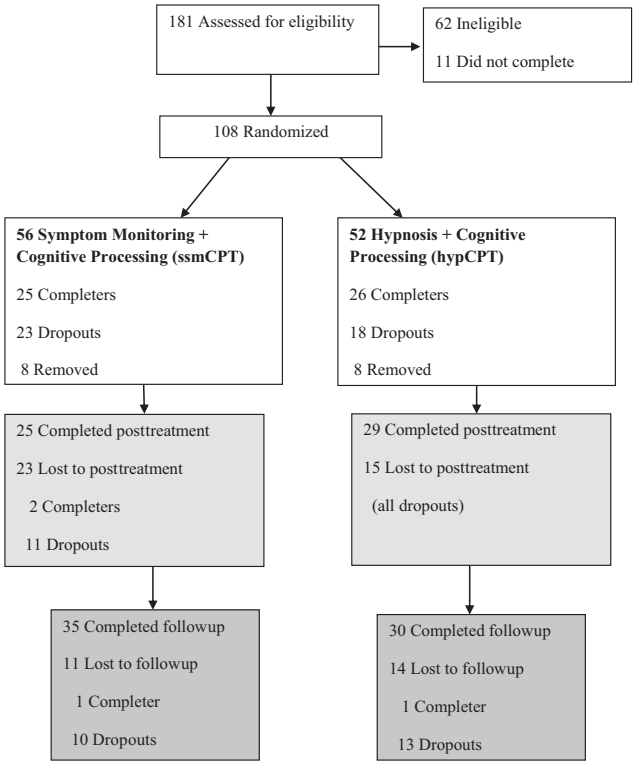

Figure 1. Flow of participants through the study protocol.

years, $SD = 2.46$; $p = .034$). Participant age ranged from 18−70 years ($M = 36.87$ years, $SD = 11.80$). Participants described themselves as predominantly Black (50%) or White (50%), with 20% of the sample identifying as multiracial. A total of 3% described themselves as Hispanic. Most of the sample was single (54%), 15% were married or living with someone, and 30% were separated, divorced, or widowed. The majority reported at least some post high school education (50%), with a mean of 14.16 years of education ($SD = 2.89$), and 74% reported an annual income of $20,000 or less.

Trauma history. Within the ITT sample, there were no differences between treatment groups on type of index event, time since trauma, or lifetime trauma history. Participants reported a complex trauma history, with lifetime endorsements of child sexual abuse by 71%, child physical abuse by 58%, adult sexual assault by 63%, adult criminal victimization by 32%, and domestic violence by 56% of the sample. Participants identified a worst event for the assessment of PTSD and initial treatment focus. Index events were 39% child sexual abuse, 11% child physical abuse, 26% adult sexual assault, and 24% adult physical assault. Time since the index event ranged from 3 months to 53.60 years ($M = 16.33$ years, $SD = 14.32$).

Study retention. Of the 92 ITT study participants, 41 dropped out of their initial treatment condition (hypCPT: $n = 18$ [40.9%]; ssmCPT: $n = 23$ [47.9%]). Dropout did not differ significantly across conditions, $\chi^2(1, N = 92) = 0.46$, $p = .499$. Figure 1 shows dropout across conditions. Consistent with the larger literature, dropouts were younger ($p = .037$) and had lower household income ($p < .004$) than completers. No significant differences were found between treatment completers and dropouts on PTSD or depressive symptom severity, PTSD-related sleep impairment, sleep latency, sleep medication usage, global sleep quality, or insomnia. However, there were significant baseline differences between these groups on sleep duration ($p = .046$). Reasons for dropout were largely unknown ($n = 26$). Most individuals ($n = 15$) who gave a reason for dropout cited ongoing psychosocial stressors (e.g., serious stressor, logistics, debilitating medical illness). Finally, dropout differed significantly by therapist, such that one of the four therapists had 71% dropout. This therapist's attrition rate significantly contributed to the overall dropout rate.

Practice compliance. Participants practiced autohypnosis an average of 4.7 times each week during the sleep intervention phase. Their practice increased in frequency as the weeks progressed. During Week 1, clients practiced an average of 1.57 times ($SD = 3.82$); during Week 2, they practiced an average of 5.83 times ($SD = 4.37$); and during Week 3, they practiced an average of 7.29 times ($SD = 6.13$). During CPT, homework completion and perceived helpfulness of assignments was tracked on a session-by-session basis. HypCPT and ssmCPT participants were similar with respect to homework completion ($t = -1.63$, $p > .05$) and perceived helpfulness of homework ($t = -1.34$, $p > .05$).

Treatment Outcomes

Sleep intervention outcomes. The effect of the hypnosis on sleep impairment was first evaluated during the sleep intervention phase of treatment. Participants randomized to the hypCPT condition showed statistically significant change (and medium effect sizes) relative to the ssmCPT condition from Interval 0 to Interval 1 (baseline to status check), on several outcomes. Specifically, four measures of sleep, including trauma-related sleep impairment (CAPS), Sleep Latency (subscale of PSQI), global sleep (total PSQI), and insomnia (ISI) showed differential change during the sleep intervention phase (ps $< .05$) in the expected direction. Sleep Duration (subscale of PSQI) and nightmares (CAPS Nightmare item) showed no significant moderation of treatment condition. The hypCPT condition demonstrated greater reduction than the ssmCPT condition in depressive symptoms (BDI-II) over the sleep intervention phase ($p < .05$); however, PTSD (CAPS) did not differ significantly ($p > .1$) between study conditions in their change between these assessment intervals. Tables 1 and 2 displays the means, standard deviations, and effect sizes for all outcomes.

Clinical significance of sleep outcomes. To provide a more robust metric of clinically significant change in sleep improvements, we calculated the percentage of participants in each condition who showed a decrease of at least 1 *SD* on the total PSQI or a decrease of at least 8 points on the ISI. Forty-seven percent of hypCPT participants and 25% of ssmCPT participants decreased at least 1 *SD* on the PSQI during hypnosis (range: 3–9 points). Improvements on the ISI showed similar trends such that 38% of the hypCPT and 17% of the ssmCPT condition improved by at least 8 points. When calculated between baseline and post-CPT assessment intervals (mean decrease on PSQI total was 4.53 points [$SD = 4.40$]), 56% of hypCPT participants and 50% of ssmCPT participants improved by at least 4 points on the PSQI. Likewise, 50% of hypCPT and 43% of ssmCPT participants improved by at least 8 points on the ISI between baseline and post-CPT intervals.

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Table 1
Means (Standard Deviations) for Treatment Outcomes by Condition and Assessment Interval

	ssmCPT assessment interval				hypCPT assessment interval			
	0	1	2	3	0	1	2	3
Measure	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
CAPS Sleep Impairment item	6.79 (1.11)	6.36 (1.46)	3.44 (3.12)	3.46 (3.35)	7.18 (1.45)	5.06 (2.71)	3.52 (3.27)	2.83 (3.36)
PSQI Sleep Latency	2.13 (0.93)	2.27 (0.92)	1.72 (1.06)	1.68 (1.04)	2.59 (0.77)	2.06 (0.89)	1.79 (0.94)	1.33 (1.15)
PSQI Sleep Duration	2.04 (0.81)	1.88 (0.88)	1.24 (1.05)	1.26 (1.14)	2.19 (0.77)	1.76 (0.92)	1.21 (1.05)	1.30 (1.02)
PSQI total	12.72 (3.16)	11.68 (2.72)	8.48 (4.26)	8.50 (4.02)	14.41 (3.06)	11.24 (3.88)	8.93 (4.60)	9.20 (5.34)
ISI total	18.24 (5.86)	16.19 (4.95)	9.70 (7.39)	10.36 (7.73)	19.00 (5.39)	13.52 (7.34)	10.62 (8.20)	11.31 (9.37)
CAPS Nightmare item	3.85 (2.60)	2.95 (2.79)	1.16 (2.21)	1.79 (2.83)	4.07 (2.94)	2.89 (2.82)	1.14 (2.07)	1.37 (2.25)
CAPS total[a]	69.23 (15.74)	57.75 (14.80)	23.40 (22.45)	27.63 (26.91)	72.48 (15.01)	54.81 (20.05)	22.66 (17.77)	27.87 (27.26)
BDI-II total	31.17 (9.84)	29.44 (14.31)	14.92 (16.60)	14.71 (13.11)	33.50 (11.07)	24.08 (14.93)	11.14 (12.05)	14.63 (15.75)

Note. Assessment intervals are labeled: 0 = baseline; 1 = status check (after sleep intervention); 2 = posttreatment (after cognitive processing therapy); 3 = 3-month follow-up. BDI-II = Beck Depression Inventory−II; CAPS = Clinician Administered PTSD Scale; hypCPT = hypnosis plus cognitive processing therapy; ISI = Insomnia Severity Index; PSQI = Pittsburgh Sleep Quality Index; ssmCPT = sleep and symptom monitoring plus cognitive processing therapy.
[a] CAPS total score without sleep item (D-1).

CPT outcomes. The four measures of sleep that demonstrated differential effects during the sleep intervention phase (CAPS Sleep item, Sleep Latency [PSQI subscale], total PSQI, and ISI) were then assessed from Interval 0 to Interval 3 (baseline to follow-up) in order to assess overall change patterns in sleep across the intervention. The trajectories (fitted values) of these variables for the hypCPT and ssmCPT conditions are displayed in Figure 2. Results from the change models (i.e., HLM analyses where treatment was assessed as a moderator of the change trajectory) indicated that participants in the hypCPT condition experienced significantly different change patterns than those in the ssmCPT condition across all four measures of sleep ($ps < .05$). The nature of the change is displayed in Figure 2 as well as in the means shown in Table 1. Further examination of the change patterns suggested that, although participants in the hypCPT condition improved more on these variables during the sleep intervention phase, the pattern was essentially reversed during CPT. In other words, the ssmCPT participants (who showed less sleep improvement than hypCPT participants after the sleep intervention phase) caught up to their hypCPT counterparts and realized equivalent gains by the end of CPT in the absence of a specific sleep intervention. Examination of effect sizes per treatment phase in Table 2 further demonstrates the significantly greater improvement for the hypCPT participants during hypnosis, but the reverse during CPT. Treatment gains were equally maintained for both groups at follow-up with only sleep latency showing a moderate effect size in favor of hypCPT.

Table 2
Effect Sizes Comparing Change Differences for hypCPT and SsmCPT

	Assessment interval			
	1–0	2–1	3–2	3–0
Measure	ES	ES	ES	ES
CAPS Sleep Impairment item	−1.08	1.43	−0.21	0.01
PSQI Sleep Latency	−0.83	0.30	−0.45	−1.13
PSQI total	−0.71	0.58	0.05	−0.37
ISI total	−0.67	0.92	−0.01	−0.08
CAPS Nightmare item	−0.06	−0.07	−0.07	−0.10
CAPS total[a]	−0.45	0.72	0.10	−0.33
BDI-II total	−0.68	0.15	0.30	−0.03

Note. The reference group is ssmCPT. Assessment intervals are labeled: 0 = baseline; 1 = status check (after sleep intervention); 2 = posttreatment (after cognitive processing therapy); 3 = 3-month follow-up. BDI-II = Beck Depression Inventory−II; CAPS = Clinician Administered PTSD Scale; hypCPT = hypnosis plus cognitive processing therapy; ISI = Insomnia Severity Index; PSQI = Pittsburgh Sleep Quality Index; ssmCPT = sleep and symptom monitoring plus cognitive processing therapy.
[a] CAPS total score without sleep item (D-1).

Overall treatment moderation models for PTSD and depressive symptoms. We then examined treatment condition as a moderator of change on PTSD (CAPS) and depression (BDI-II) to assess differences across conditions on overall trajectories of change on these primary symptoms. The corresponding means, standard deviations, and effect sizes are shown in Tables 1 and 2. There was no significant interaction between groups on overall change in PTSD when considering the full model estimate ($p > .1$). Both groups exhibited statistically significant change in PTSD symptoms ($p < .001$), with the hypCPT group decreasing 42.63 points on the CAPS between baseline and posttreatment and the ssmCPT group decreasing 48.23 points. Thus, CPT was successful in significantly reducing PTSD symptoms. Overall, decreases in symptoms of depression across the treatment were also demonstrated, with both groups realizing significant change on the BDI-II from baseline to posttreatment (hypCPT: 20.64-point reduction; ssmCPT: 15.08-point reduction). The rate of change differed significantly by condition on depression symptoms. For the BDI-II, the quadratic term (in the orthogonally coded model) significantly interacted with treatment condition ($t = 2.42$, $p = .02$), indicating that the change experience in BDI-II differed for those in the hypCPT and the ssmCPT conditions. The relation between the phases of treatment and the different rates of change across conditions is further explored in the next section.

Augmentation of PTSD and depression outcomes by sleep improvement. We took an intervening variable approach (Krull & MacKinnon, 2001; MacKinnon et al., 2002) to assess the effect

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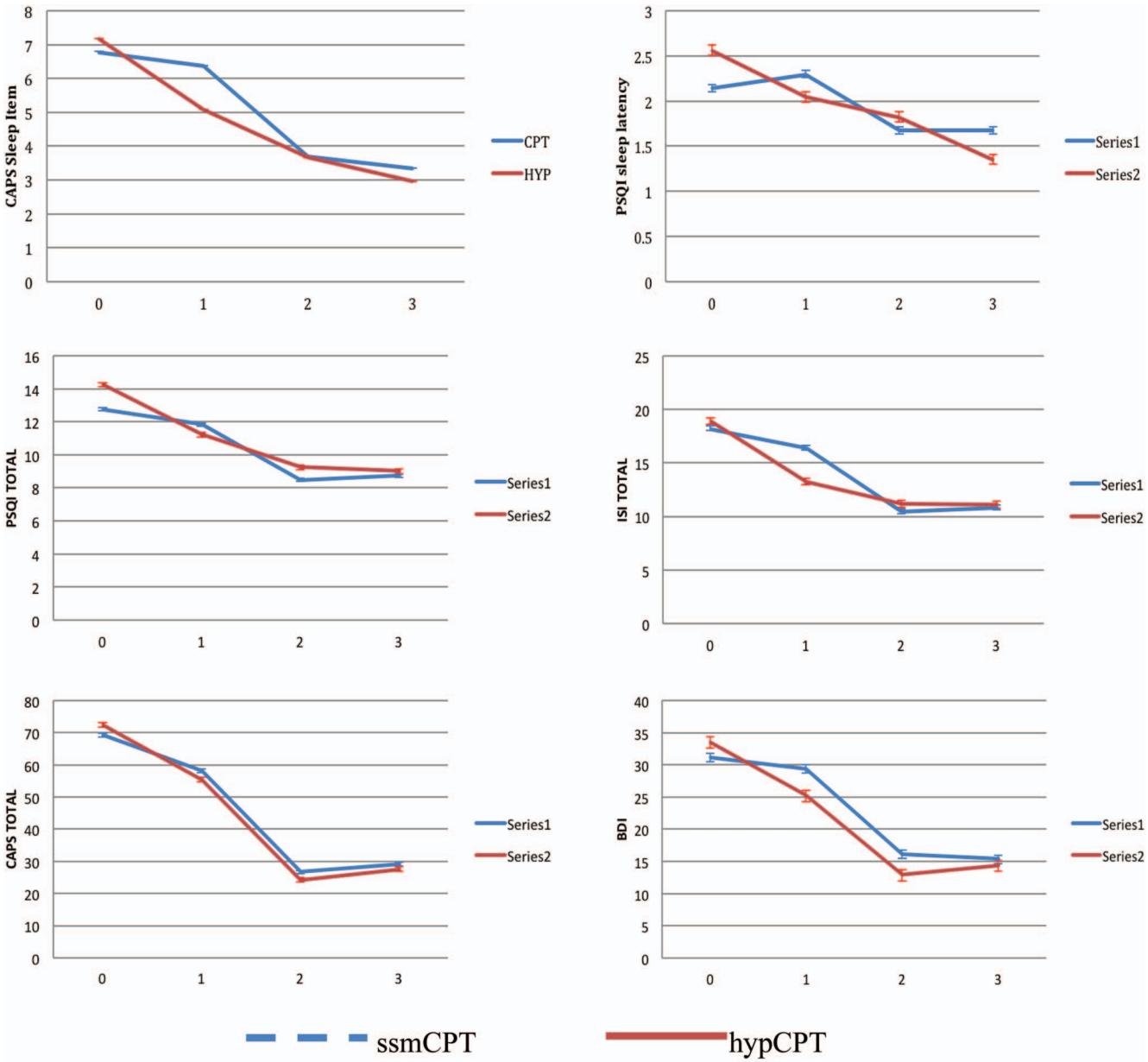

Figure 2. Sleep and other symptom changes over the study assessment intervals. Assessment intervals: baseline assessment (Interval 0), status check (after the sleep intervention phase; Interval 1), a posttreatment assessment (after the conclusion of the CPT; Interval 2), and a 3-month follow-up assessment (3 months after the conclusion of CPT; Interval 3). CPT = cognitive processing therapy. hypCPT = hypnosis plus cognitive processing therapy; ssmCPT = sleep and symptom monitoring plus cognitive processing therapy. See the online article for the color version of this figure.

of treatment condition (hypCPT vs. ssmCPT) on sleep outcomes (established above) on the primary outcomes (CAPS total and BDI-II). We illustrate the modeled change (fitted values) in the primary outcomes in Figure 2. From a modeling standpoint, we were interested in the correspondence in sleep improvements with improvements in primary outcomes as differentiated by treatment condition. Because the PSQI and ISI are both time-varying, their significance would indicate the covariance with CAPS total or BDI-II, respectively. That is, as scores on the PSQI or ISI improve with hypnosis, then controlling for the change (time) and hypnosis, so would changes in CAPS total or BDI-II scores, depending on the model.

For CAPS total with PSQI as intervening variable, the relation (controlling for time and treatment condition) was $b = 2.71$ ($p < .01$). That is, as PSQI changed by 1 unit, CAPS total was expected to change by 2.71 units. For CAPS total with ISI as the intervening variable, the relation was $b = 1.31$ ($p < .01$). For BDI-II with PSQI as the intervening variable, the relation was $b = 1.58$ ($p <$

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.01). For BDI-II with ISI as the intervening variable, the relation was $b = .83$ ($p < .01$). However, it should be noted that for CAPS total, the initial total effect of $Y = X$ (i.e., treatment condition differences in change trajectories) was not significant. This could be due to limited effect size in the overall relation or other patterns in the intervening chain of events (see MacKinnon, Krull, & Lockwood, 2000). As sleep improved, there were corresponding improvements in PTSD and depression. The direct effect of the sleep intervention on PTSD was less pronounced than that on depression.

Discussion

To date, no trial has specifically tested the benefit of reducing sleep impairment *prior* to the commencement of an evidence-based therapy for PTSD. This specific ordering of interventions is necessary to assess whether improvements in sleep augment overall outcomes in PTSD and comorbid depressive symptomatology. We administered a sleep-directed hypnosis intervention prior to CPT to test the ability of hypnosis to successfully treat sleep impairment in the context of PTSD, and then to determine whether improvements in sleep augmented gains in PTSD and depressive symptoms during CPT.

Our results indicated that sleep-directed hypnosis was effective in treating sleep impairment occurring within the context of PTSD in female interpersonal assault survivors. Specifically, the large effect sizes in sleep latency suggested that, as expected, hypnosis was successful in reducing the amount of time necessary to fall asleep. It was also anticipated that total hours of sleep would show a differential effect in favor of the hypnosis group, as had been previously observed in a nonclinical sample (Cordi et al., 2014). However, the treatment conditions did not differ significantly in hours of sleep or in trauma-related nightmares, the latter of which was not intentionally targeted by the brief hypnosis intervention. It is possible that the experience of trauma-related nightmares may have introduced significant alarm and/or fear during sleeping hours, rendering the hypnosis training ineffectual to restore sleep. Trauma-related sleep impairment also evidenced differential improvement in favor of hypnosis. This symptom maps most consistently onto global sleep impairment and general insomnia, providing further evidence that participants were consistently reporting improvement in overall sleep impairment. The observed low internal consistency on the PSQI sleep measure should again be noted. However, this appears to be largely due to the medication component score with acceptable values on the components relevant to study hypotheses. It is also important to note that approximately half of the hypnosis participants improved on global sleep during hypnosis between $1-3$ *SD* or evidenced clinically significant improvement on the ISI. Although these gains are substantial and superior to the control condition, still, half of the individuals treated with hypnosis did not realize clinically significant gains over the course of the 3 weeks.

We did not hypothesize that the hypnosis would directly target PTSD or depression. Interestingly, we found that PTSD symptoms (with sleep impairment removed) did, in fact, decrease during hypnosis. However, PTSD severity also decreased during the control condition, with no significant interaction. Thus, it is impossible to attribute the decreases in PTSD to the hypnosis. This is in contrast to Krakow, Johnston, et al.'s (2001) conclusion that the targeted treatment of sleep is associated with improvements in PTSD. Perhaps the improvements in psychological distress observed in the Krakow study may have been direct effects of the intervention versus an effect of sleep improvement.

Depression significantly decreased during hypnosis for the hyp-CPT condition only. It is unknown whether the hypnosis itself directly influenced depression or whether the benefits of sleep improvement caused the palliative effect. The content of the hypnosis intervention was standardized and did not include any components consistent with elements of therapy typically contained in evidence-based practices for depression, suggesting that it is unlikely that the hypnosis intervention itself influenced depressive symptoms. More likely, improved sleep may have played a causal role in improvement in mood. Previous research found that adding CBT-I to antidepressants in patients suffering from insomnia and depression results in better treatment outcomes than medications alone (Manber et al., 2008), suggesting that reducing sleep impairment independently will have palliative effects on depression. The relation between depression and sleep impairment within the context of PTSD is less understood. Our results suggest that, in relieving sleep impairment, one might expect to see improvements in mood or depressive symptoms, even in the context of PTSD.

We hypothesized that improvements in sleep would augment the effects of CPT such that hypnosis participants would show greater improvement in PTSD. Although participants' sleep impairment improved during the hypnosis intervention, these improvements did not then hasten recovery from PTSD during CPT. A small effect size favoring the hypnosis group at the follow-up assessment interval indicated a differential effect of hypnosis on overall PTSD recovery, but the rates of change across both phases of the intervention were nearly identical. Contrarily, the improvements in depression observed during the hypnosis phase of the study were maintained throughout the study. In other words, participants in hypnosis appeared to receive a "jump start" on relief from depression and control participants never quite caught up by the end of CPT. These results suggest some specificity in the relation between sleep impairment and depression compared with that of sleep impairment and PTSD symptoms.

Zayfert and DeViva (2004) observed that, in treatments targeting PTSD, sleep impairment may not remit despite more global improvement in PTSD. In the current study, control participants realized the same gains in sleep impairment as the hypnosis participants by the end of CPT, hence, the large effect sizes observed in pre-CPT to post-CPT change in sleep (in favor of the control condition). Considering effect sizes from baseline to the follow-up interval, only sleep latency showed a large effect in favor of hypnosis, with global sleep showing a moderate effect. This sample initially presented with severe sleep impairment and, overall, this symptom significantly improved. However, despite these gains, mean scores hovered around clinical cutoffs at posttreatment and follow-up. Nightmares did, however, significantly improve and fell below clinical cutoffs at posttreatment, and remained low at follow-up. Hypnosis did not appear to influence nightmares, but the symptom remediated during CPT for both groups.

This study is not without limitations. The sample size presents statistical power limitations, perhaps compounded by similarities in interventions and attrition. It is difficult to interpret this attrition rate because it is the highest of any CPT trial conducted at this site, including a trial that was conducted concurrently. There was

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clearly a therapist effect that accounted for a significant portion of the dropout rate. It is possible that the sheer length of time in the trial may have increased attrition. Unlike most sleep trials in PTSD samples (Casement & Swanson, 2012; Hansen et al., 2013), this trial required all participants to meet criteria for severe sleep impairment. Perhaps severe sleep impairment is a risk factor for dropout. Similarly, this study is limited in the fact that it included only female survivors of interpersonal trauma. This choice was made due to the prevalence of interpersonal violence within the study site's trauma population. Although, the sleep intervention did, in fact, result in a differential effect for the hypnosis group, it should be noted that global sleep scores remained in the clinically significant range at the posthypnosis assessment without much additional improvement after CPT. Scores on the ISI and CAPS Sleep item dropped below clinical cutoffs to the subthreshold range. Perhaps a more potent intervention such as CBT-I would have been even more successful in improving sleep, which then may have parlayed into more profound effects on PTSD outcomes. That said, hypnosis is easily accessible for clinicians, is easily learned by patients, and was completed in less than half the time of a standard course of CBT-I. Hypnosis may be suitable for patients who need relief from sleep deprivation in order to begin trauma-focused therapy.

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