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Does Cognitive–Behavioral Therapy for PTSD Improve Perceived Health and Sleep Impairment?

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There is a paucity of empirical study about the effects of evidence-based psychotherapy for posttraumatic stress disorder (PTSD) on concurrent health concerns including sleep impairment. This study compares the differential effects of cognitive processing therapy (CPT) and prolonged exposure (PE) on health-related concerns and sleep impairment within a PTSD sample of female, adult rape survivors (N = 108). Results showed that participants in both treatments reported lower health-related concerns over treatment and follow-up, but there were relatively more improvements in the CPT condition. Examination of sleep quality indicated significant improvement in both CPT and PE across treatment and follow-up and no significant differences between treatments. These results are discussed with regard to the different mechanisms thought to underlie the treatments and future innovations in PTSD treatment.

A growing literature implicates traumatic stress in adverse health outcomes when measured by biological and physiological indices, self-report, and mortality rates (Friedman & Schnurr, 1995; Green & Kimerling, 2004). Environmental and behavioral factors unique to traumatized individuals complicate the relationship between exposure to trauma and poor health status. A second factor contributing to negative health consequences within traumatized individuals is the significant sleep impairment observed in this population. A minimally studied area is whether treatment of trauma-related sequelae, specifically posttraumatic stress disorder (PTSD), also ameliorates concerns about physical health. This directly compares the effects of two evidence-based, trauma-focused psychotherapies, cognitive processing therapy (CPT; Resick & Schnicke, 1992, 1993) and prolonged exposure (PE; Foa, Hearst, Dancu, Hembree, & Jaycox, 1994), on both health-related concerns and sleep impairment, within female sexual assault survivors suffering from PTSD.

Posttraumatic stress disorder and poor health-related outcomes have been consistently linked in cross-sectional research. Several large-scale studies conducted with combat veterans have compared those with and without PTSD, and found that veterans with PTSD report significantly more chronic health conditions and generally poorer perception of physical health than their non-PTSD counterparts (Barrett et al., 2002, Kulka et al., 1990; Schnurr & Jankowski, 1999; Schnurr et al., 2000), even when controlling for behaviors known to independently contribute to poor health outcomes such as smoking, alcohol use, deployment status, military status, and demographics (Barrett et al., 2002; Schnurr & Spiro, 1999). Although less research has been conducted in non-veteran populations, specific medical conditions such as irritable bowel syndrome (Irwin, Falsetti, Lydiard, & Ballenger, 1996) have been associated with higher rates of trauma exposure and PTSD. However, it is unclear whether this incidence of trauma is directly related to the medical disorder or merely part of a larger, more

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diffuse symptom presentation and/or tendency to amplify physical sensations, report health-related concerns, and seek treatment (Blanchard, 2000).

Difficulty in sleep onset and maintenance is the most frequently reported symptom of the 17 symptoms of PTSD (Glaubman, Mikulincer, Poral, Wasserman, & Birger, 1990; Inman, Silver, & Doghramji, 1990). Ohayon and Shapiro (2000) report that as many as 70% of PTSD trauma survivors suffer sleep impairment. Sleep loss has also been shown to have a deleterious effect on overall health and well-being. For example, Clum, Nishith, and Resick (2001) found that sleep impairment accounted for a significant portion of the variance in physical health complaints even after controlling for other PTSD symptoms and depression. Thus, it follows that the significant and chronic sleep loss commonly endorsed in PTSD survivors may contribute to additional negative health ramifications (e.g., pain disorders, asthma, hypertension, gynecological conditions, poor reproductive health, etc.; see Green & Kimerling, 2004 for review).

There is speculation that trauma-related insomnia may not remit with evidence-based psychosocial and psychopharmacological PTSD treatment despite more global improvement in PTSD severity, and sleep impairment has been argued to be the most refractory symptom of the PTSD symptom constellation (Cooper & Clum, 1989; Zayfert & DeViva, 2004). A handful of studies have been conducted using psychotherapeutic insomnia interventions to specifically target sleep impairment in trauma populations. These studies are limited in their use of single case studies and the lack of random treatment assignment or controls. For example, Krakow and colleagues (2001) used a cognitive–behavioral therapy (CBT) designed for insomnia combined with imagery rehearsal specifically targeting nightmares in a female sample of crime victims. Significant improvements were found in overall PTSD symptoms, overall sleep quality, and frequency of nightmares. Improving upon their earlier work by using a controlled design, these investigators report the intervention as superior to a waitlist control group on similar dimensions (Krakow et al., 2001).

In their review of the status of sleep in PTSD, Singareddy and Balon (2002) identified several uncontrolled studies testing pharmacological modalities, such as trazodone, nefazodone, and fluvoxamine, specifically targeting sleep in PTSD samples. They concluded that these medications can be helpful in addressing sleep deficits in PTSD, but add the caveat that the evidence to support this assertion is scarce. More recently, two controlled trials have emerged suggesting that prazosin may be particularly effective in reducing trauma-related nightmares known to significantly interfere with sleep (Raskind et al., 2007; Taylor et al., 2008). Small sample size, lack of long-term follow-up, and lack of comparison to normative data in these studies preclude further discussion of these promising results. Overall, despite improvements reported, sleep deficits typically remained in the clinical range even after a course of CBT or pharmacotherapy specifically targeting sleep.

Little is known about the effects of evidence-based psychotherapy for PTSD on general health-related concerns and sleep impairment. Rauch et al. (2008) compared PE and prolonged exposure with cognitive restructuring (PE/CR) to a waitlist control to determine changes with treatment of PTSD in reported physical health difficulties, discomfort associated with those difficulties, and general social functioning among PTSD-positive female survivors of sexual assault. Health-related concerns were measured by a modified version of the Pennebaker Inventory of Limbic Languidness (PILL; Pennebaker, 1982). Using analyses of variance and last observation carried forward techniques for missing data, they found that the frequency of reported health problems significantly decreased in both active treatment groups, but did not change in the waitlist control group. Decreases in PTSD and depressive symptoms accounted for a significant portion of the variance in perceived health concerns reductions. Physical health discomfort did not improve in either active condition.

We used data from a randomized controlled trial (RCT) comparing PE and CPT in a sample of women who had rape-related PTSD (Resick, Nishith, Weaver, Astin, & Feuer, 2002) to compare the health-related treatment effects of each intervention. Both treatments were efficacious in decreasing PTSD symptoms and primary comorbid mental health conditions associated with PTSD (i.e., depression, general anxiety). Given the minimal documented effects of PTSD treatment on physical health concerns and sleep impairment, as well as the research design of the parent RCT in which participants did not continue to be assessed after dropout, we analyzed only those participants who completed a course of CPT or PE. Thus, our primary research question investigated whether a full course of evidence-based treatment for PTSD could improve health-related concerns and sleep impairment. Given the minimal available literature on this topic, no specific hypotheses regarding differential effects between the two active treatments were formulated.

METHOD

Participants

This study includes 108 female sexual assault victims suffering from PTSD aged 18 and over, who completed pre- and posttreatment assessments and a full course of CPT ($n = 54$) or PE ($n = 54$) within the larger study conducted by Resick et al. (2002). Sexual assault was defined as a completed experience of rape (oral, anal, or vaginal) in childhood or adulthood. All participants were at least 3-months posttrauma and stabilized on medication for at least one month prior to treatment. Participants were instructed to remain stable on medication throughout the study. Exclusion criteria included current psychosis, mania, current drug or alcohol dependence, current severe suicidality, and illiteracy. Participants could not currently be in an abusive relationship, peritraumatic situation, or being stalked. In the case of marital rape, the participant was

Table 1. Demographics Across Treatment Condition

Demographic characteristics and comorbid disorders	Cognitive processing therapy ($n = 54$)		Prolonged exposure ($n = 54$)	
	n	%	*n*	%
Non-White race[a]	11	19	6	12
Married/partnered	15	28	15	28
Income ≤ 20,000	17	50	15	40
Current MDD	20	43	23	52
Lifetime MDD	40	89	36	88
Current PD	4	9	8	18
Lifetime PD	9	21	10	23

Note. Data are given as number (percentage) unless otherwise indicated. MDD = Major depressive disorder; PD = panic disorder.
[a]Race of non-White participants was 14.8% African American and .9% Asian.

required to have been out of the relationship for at least 6 months. All women met *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; American Psychiatric Association [APA], 1994) criteria for PTSD secondary to their rape as measured by the Clinician-Administered PTSD Scale (CAPS: Blake, Weathers, Nagy, Kaloupek, Charney et al., 1998).

Demographic and clinical characteristics of the 108 women in each treatment condition are reported in Table 1. The majority of participants were White (90%) and single (including divorced, separated, or widowed) at the time of intake (72%). The mean age of each group at the time of intake was 33 years ($SD = 10$) and the mean education of each group was 15 ($SD = 2$). The mean number of months since the index assault was 112 ($SD = 114$) in the CPT group and 108 ($SD = 90$) in the PE group. Pearson chi-square analyses and two-tailed between group t tests conducted to test pretreatment differences between the CPT and PE conditions on the demographic characteristics revealed no significant differences in any of the demographics.

Examination of several comorbid conditions at intake indicated that participants reported high rates of comorbid psychiatric disorders as diagnosed by the Structured Clinical Interview for DSM-IV (SCID-P) (First, Spitzer, Gibbon, & Williams, 1997). Table 1 includes the comorbidity by treatment condition. Almost half (47%) of the participants met criteria for major depressive disorder (MDD), while 88% had suffered from at least one past MDD episode. Almost half of the sample (49%) likewise met criteria for lifetime diagnoses of alcohol dependence. Panic disorder (PD) was present in the population to a lesser extent, with 14% of the participants meeting criteria for a current diagnosis and 22% for a past diagnosis. Chi-square analyses indicated no differences between the CPT and PE conditions on MDD or PD history, but the PE condition had a higher rate of lifetime alcohol dependence than the CPT condition, χ^2 (1, $N = 89$) = 4.05, $p < .05$). The CPT and PE conditions did not differ at baseline on the dimensional measure of depressive symptoms nor on PTSD severity.

Measures

The SCID-P (First et al., 1997) is a clinician interview designed to be consistent with diagnostic criteria from the *DSM-IV* (APA, 1994), and was used to assess the following Axis I disorders: mood, substance abuse/dependence, and panic. The SCID-P's psychometric properties are well-established (Williams et al., 1992). As reported in Resick et al. (2002), interrater reliability on current and lifetime diagnoses of depression, and alcohol and substance abuse revealed a range of kappa values from .80 to 1.00 for the parent study.

The Clinician-Administered PTSD Scale (Blake et al., 1998) is a 22-item, semistructured interview administered by a trained clinician and used to assess for the presence or absence of lifetime and current PTSD. The CAPS contains separate 5-point frequency and intensity rating scales (0 to 4) for symptoms consistent with a diagnosis of PTSD in the *DSM-IV*. The CAPS also has items that rate social and occupational functioning, global PTSD symptom severity, and the validity of the participant's responses. The scale has been shown to have excellent psychometric properties (Blake et al., 1995). As reported in Resick et al. (2002), interrater reliability using the total PTSD score revealed a kappa value of .97 for the parent study.

The Pennebaker Inventory of Limbic Languidness (Pennebaker, 1982) is a 54-item, 5-point Likert scale measurement of the frequency of occurrence of common physical symptoms and sensations (e.g., coughing, back pains, headaches). Responses, ranging from *Never or almost never* to *More than once every week* are summed to obtain a total score, with higher scores indicating greater symptomatology. There are two methods of scoring: summing items and binary presence of symptoms/sensations. Using the summing technique, scores on the PILL range from 0–216, with normative data resulting in a mean score of 59 with a standard deviation of 25. Both internal consistency ($\alpha = .91$) and test-retest reliability have been found to be high

($r = .83$; Pennebaker, 1982). Validity of the PILL has been supported by data showing that its scores are positively associated with the frequency of physician and health-center visits, use of aspirin, health-related work absences (Pennebaker, 1982) and other measures of health complaints (Watson & Pennebaker, 1989). Moreover, the PILL has been shown to share a significant amount of variance with measures of negative affect, including physiological components of anxiety and depression (O'Brien, Atchison, Gremillion, Waxenberg, & Robinson, 2008).

The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1988) is a 19-item self-report measure that assesses sleep quality and disturbance over the past month. The 19 individual items produce a global sleep quality score ranging from 0 to 21 and the following seven component scores, which range from 0 (*no difficulty*) to 3 (*severe difficulty*): subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medications, and daytime dysfunction. The scale has demonstrated good psychometric qualities (Carpenter & Andrykowski, 1998). Buysse and colleagues (1988) provide normative data on the PSQI. Specifically, the global PSQI score of >5 was found to distinguish poor sleepers from good sleepers with a diagnostic sensitivity of 89.6% and a specificity of 86.5%.

Procedure

A full description of the recruitment procedures and interventions is described in the primary outcome paper (Resick et al., 2002). In brief, participants were assessed prior to treatment, 2 weeks following the conclusion of treatment, and 9 months posttreatment. All assessments were conducted by trained, advanced doctoral candidates who were blind to study condition. Both treatments consisted of biweekly sessions for a total of 13 hours of therapy over a 6-week period. The first session of PE was one hour with each subsequent session totaling 90 minutes. To equate the two conditions on therapy time, 30 minutes were added to Sessions 4 and 5 of CPT to provide the therapist additional time to process the written trauma narratives. All therapy was conducted by clinical psychologists trained in both CPT and PE. Treatment adherence and competence was evaluated by independent raters.

Briefly, CPT is predominantly a cognitive therapy for PTSD. The therapy begins by offering psychoeducation about the theory of PTSD and the rationale for treatment. Throughout therapy, the client and therapist identify distorted thoughts (assimilated and overaccommodated cognitions) and faulty interpretations of the event. Through the use of Socratic questioning, complemented by worksheets and practice assignments, the client is taught to monitor, challenge, and eventually modify distorted thoughts and arrive at alternative, more accurate statements. At the conclusion of Session 3, the client is asked to write a detailed account of the trauma, read it on a daily basis, and read it aloud to the therapist in Session 4. The therapist assists the client in processing emotions and challenging erroneous beliefs about the client's role in the event. The account is written for a second time and the process is repeated in Session 5. Throughout the remainder of the sessions, cognitive therapy continues first targeting cognitions about the index event and then moving into challenging more generalized, global beliefs around issues involving safety, trust, power/control, esteem, and intimacy.

Prolonged exposure is primarily an exposure-based intervention and includes the following four components: psychoeducation (conducted primarily in Sessions 1 and 2), breathing retraining, behavioral exposures, and imaginal exposures. In Session 2, a hierarchy of in vivo exposures is developed and the first in vivo practice assignment is given. The content of the remaining sessions is largely devoted to repeated imaginal exposures aimed at reducing avoidance and extinguishing conditioned negative emotional responses. The therapist assists the client in processing the emotion elicited during the exposures with nondirective statements. The client experiences further exposure by listening to tape recordings of the session at home between sessions along with the behavioral exercises.

Data Analysis

Hierarchical linear modeling (Raudenbush, Bryk, Cheong, Congdon, & du Toit, 2004) was used to examine the effect of CPT and PE on the physical health complaints (PILL) and sleep problems (PSQI) over time. Hierarchical linear modeling was used because the models estimate individual differences in the trajectory of change over time, and can accommodate missing data (Singer & Willett, 2003). Total PILL, total PSQI, and individual subscales of the PSQI were the dependent variables of interest. Consistent with prior research (Monson et al., 2006) and recommendations in modeling the time variable (Singer & Willet, 2003), assessment period was log transformed to account for greater changes over the course of treatment compared with changes from posttreatment to 9-month follow-up.

As an initial step in modeling changes in physical health and sleep problems, we analyzed sleep medication use according to the PSQI to determine if there were baseline differences in sleep medication use and/or changes in medications over assessment period by condition. Assessment period was included as a Level 1 predictor and treatment condition was included as a Level 2 predictor in this analysis. As reported below, there were differences at baseline and across assessment by condition in sleep medication use. Consequently, to control for these effects, sleep medication use across assessment was included as a time-varying covariate at Level 1 in subsequent analyses of the PILL total score and PSQI total and subscale scores, along with assessment period. Treatment condition was included as a Level 2 predictor. The effect size difference between the conditions are presented as partial rs, and described according to Cohen's (1992) descriptions of strength. In addition to hierarchical linear modeling results, we present the

overall means for the global sleep scores (PSQI) at all three time points for the entire sample. As discussed above, a global score >5 on the PSQI indicates clinically significant sleep impairment. Our sample ($N = 107$) began treatment in the clinically significant range ($M = 10.96$; $SD = 4.20$), realized improvement over time, but remained in the clinically significant range at posttreatment ($M = 7.48$, $SD = 4.09$) and at the 9-month follow-up assessment interval ($n = 74$; $M = 7.08$; $SD = 3.83$).

RESULTS

There were significant differences in sleep medication use at baseline by treatment condition, with significantly more sleep medication use in the PE versus CPT condition, $t(106) = 2.77$, $p < .01$. There was also a significant time by treatment condition interaction, with significantly more decreases in sleep medication use over time in PE compared with CPT, $t(106) = 2.95$, $p < .01$. Thus, baseline sleep medication use and changes in sleep medication use across assessment were controlled in subsequent analyses.

As shown in Table 2, there were no differences by condition at baseline assessment on the PILL when PSQI sleep medication use was included in the model. Changes in sleep medications on the PSQI were significantly associated with changes on the PILL, with more medication use associated with more health problems. However, there were no differences by condition in the association between sleep medication changes and changes on the PILL. As shown in Figure 1, there were significant improvements on the PILL in both conditions across assessments; however, there were significantly more improvements for participants in the CPT versus PE condition across assessment, with a moderate effect size difference (partial $r = .27$).

Also shown in Table 2, there were no differences by condition in total PSQI or PSQI subscale scores at baseline assessment when PSQI sleep medication use was included in the model. Changes in sleep medications were significantly associated with changes in total PSQI and most all of the PSQI subscale scores; increases in medication use were associated with less sleep improvements. Exceptions to this pattern were found on sleep duration and daytime dysfunction, where sleep medication changes were not associated with changes in these problems. There were no differences by condition in the association between sleep medication changes and changes in sleep problems. There were significant improvements in total PSQI and PSQI subscale scores across assessment, but no significant differences by treatment condition. The effect size differences between the conditions were small across these subscales (partial rs = .02–.08).

DISCUSSION

The full course of the evidence-based treatments, CPT and PE, were successful in reducing health-related concerns and sleep dysfunction in a sample of female sexual assault survivors. These findings support previous research indicating that PTSD symptomatology is highly related to health-related factors (Schnurr & Green, 2004), and domains of functioning beyond PTSD symptoms, including health-related concerns (Galovski, Sobel, Phipps & Resick, 2005). There was an advantage of CPT over PE in reducing concerns related to physical health. The effect size advantage was of a moderate size.

Table 2. Hierarchical Linear Modeling Results for Sleep and Physical Health Outcomes for Prolonged Exposure and Cognitive Processing Therapy

	Global sleep		Sleep quality		Sleep latency		Sleep duration		Habitual sleep efficiency		Sleep disturbance		Daytime dysfunction		Total PILL	
	B	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*
Baseline																
Intercept	9.1***	0.5	1.6***	0.1	.7***	0.2	1.6***	0.2	0.8***	0.2	1.7***	0.1	1.8***	0.1	129.8***	4.5
Condition[a]	0.0	0.7	−0.1	0.2	−0.1	0.2	0.1	0.2	0.3	0.2	−0.1	0.1	−0.1	0.2	−3.9	6.2
Sleep medication changes																
Intercept	1.8***	0.2	0.1*	0.1	0.2 **	0.1	0.1	0.1	0.3***	0.1	0.1*	0.0	0.1	0.1	5.6***	1.7
Condition[a]	0.5	0.4	0.2	0.1	0.1	0.1	0.1	0.1	−0.1	0.1	0.1	0.1	0.1	0.1	1.5	2.5
Change over time by condition																
Intercept	−3.6***	0.5	−0.8***	0.1	−0.7***	0.1	−0.6***	0.2	−0.5*	0.2	−0.4 ***	0.1	−0.8***	0.1	−13.3***	2.9
Condition[a]	0.3	0.7	0.0	0.2	0.1	0.2	0.1	0.2	−0.1	0.2	0.0	0.1	0.1	0.2	−11.6**	4.1

Note. PILL = Pennebaker's Inventory of Limbic Languidness.
[a]Prolonged exposure was the reference condition.
$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

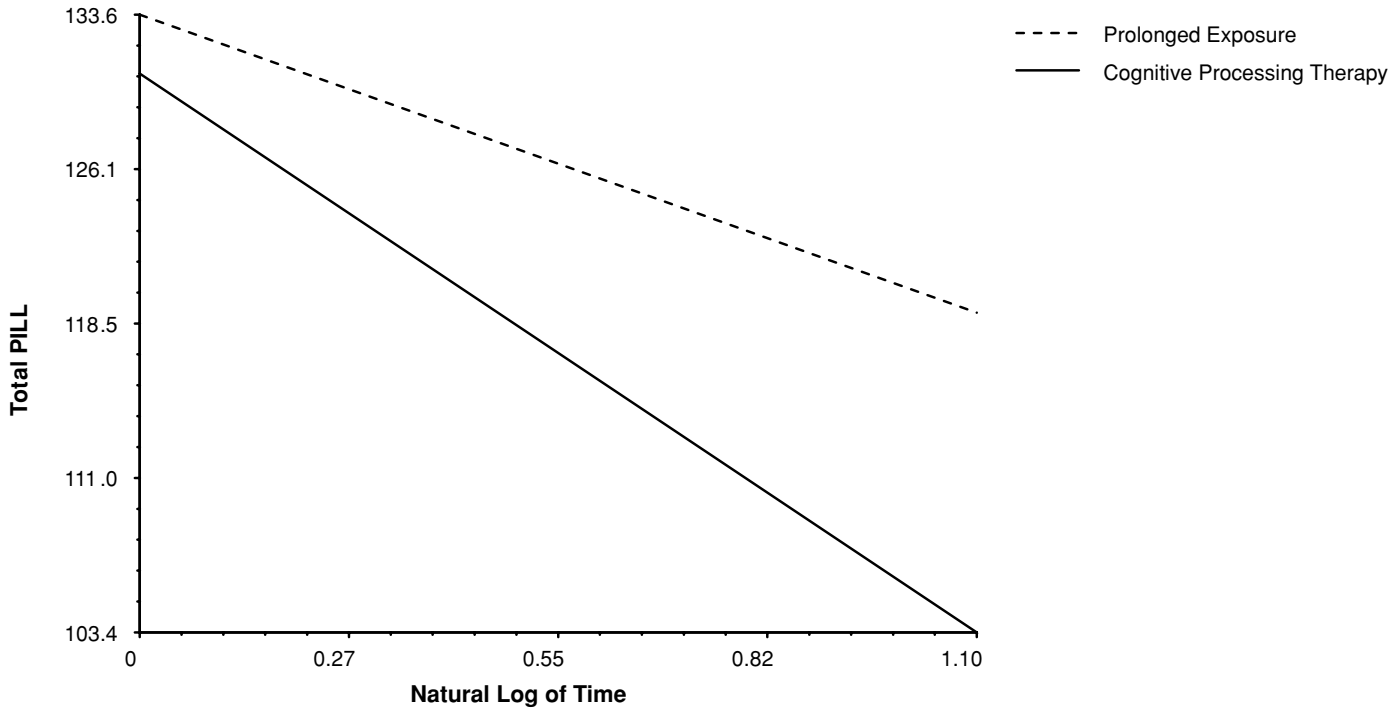

Figure 1. Changes in total PILL (Pennebaker Inventory of Limbic Languidness) scores over time by condition.

The differential effect supporting CPT may be explained, in part, by the emphasis on cognitive restructuring contained in the CPT intervention. Perceived physical health status may be considered a part of a larger cognitive process that serves as a common risk factor for PTSD and health problems can emerge secondary to a trauma and thereby is perhaps more efficiently targeted by a cognitive therapy. It is noteworthy that, as depicted in Figure 1, the CPT group's average score at posttreatment was 103 and the PE group's average score was 119. Thus, participants' overall health-related concerns after completion of therapy remained at least two standard deviations above the normal population's average score. It is feasible that living without PTSD may continue to positively influence change in health-related concerns beyond the scope of the 9-month follow-up assessment included in this article.

With respect to sleep dysfunction, the results on the PSQI likewise showed that both interventions were effective in reducing sleep disturbances that are so commonly reported in traumatized populations. We controlled for initial differences in sleep medication use and changes over time in use in the analyses, because the PE group reported significantly higher use of sleep medications prior to the commencement of treatment compared with CPT and reduced medication use more than CPT over time. This pattern is likely the result of regression to the mean over time in the PE group, because there were no significant changes in use in the CPT condition. It is interesting to note that increased sleep medication use was associated with poorer, not better, sleep over time in both conditions. This finding argues against the notion that sleep medications accounted for the treatment effects of CPT and PE on perceived health and sleep. It also raises the question of whether sleep medication may actually interfere with the efficacy of trauma-focused interventions for PTSD. There is some support for this speculation in the literature suggesting that benzodiazepines may interfere with trauma-focused interventions (Davidson, 2004; Friedman, 2003). Sleep medications may exert the same sedating effect causing a similar interference. Future studies with data to answer these questions are needed.

Consistent with previous trials, it is also noteworthy that the entire sample, irrespective of treatment condition, did not reach "good sleeper" status or, put another way, did not return to normal sleep functioning despite treatment gains. DeViva and colleagues (2005) added an element of CBT specifically targeting insomnia in a sample of five PTSD treatment responders whose trauma-related insomnia remained refractory after PTSD-directed CBT. Results showed significant improvement in self-recorded sleep time, sleep onset latency, wake time after sleep onset, and sleep efficiency. However, similar to the current study, scores on measures of sleep quality did not drop below clinically significant cutoffs. Sleep impairment remains one of the more treatment-refractory symptoms within the PTSD

constellation and warrants further attention and, perhaps, specific intervention. One possible solution may be to augment trauma-focused therapies such as CPT and PE with an intervention specifically designed to target sleep administered prior to the trauma-focused therapy. Reducing sleep impairment prior to focusing on trauma content may enhance an individual's ability to engage in the trauma therapy. Future research may also particularly investigate the role of trauma-related nightmares in overall sleep disturbance within a PTSD-positive sample.

This study only included women who reported a sexual assault as their index traumatic event. Thus, it is unclear whether these results would be generalizable to men or to individuals suffering from PTSD secondary to other types of trauma. Interpretation of the results of this study is somewhat limited in the lack of a no-treatment comparison condition. Although the parent study did include such a condition, sleep impairment and health-related concerns were not assessed within the attention control condition in an effort to minimize participant burden. Thus, it cannot be determined from these data that demonstrated improvements are not entirely due to factors such as repeated assessment, instrumentation, or clinician attention. In the absence of such a control group, minimal differential treatment outcomes were expected given the comparison of two active, efficacious experimental conditions. Also, given the chronic nature of sleep impairment in this population, longer term follow-up is needed to assess the larger impact on sleep functioning. Despite these limitations, results suggest that effectively treating PTSD also appears to significantly improve specific quality of life concerns such as physical symptoms and overall sleep quality. It remains to be seen whether modifying these demonstrably efficacious interventions for PTSD to specifically target health concerns and sleep dysfunction may restore sleep to normal levels in survivors of trauma.

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