Change in Sleep Symptoms across Cognitive Processing Therapy and Prolonged Exposure: A Longitudinal Perspective

Cassidy A. Gutner[a,b], **Melynda D. Casement**[a,b], **Karina Stavitsky Gilbert**[c], and **Patricia A. Resick**[a,b]

[a]National Center for Posttraumatic Stress Disorder, VA Boston Healthcare System

[b]Boston University School of Medicine, Boston, MA

[c]VA Boston Healthcare System, Boston, MA

Abstract

Sleep disturbance is a core component in posttraumatic stress disorder (PTSD). Although cognitive-behavioral treatments for PTSD reduce the severity of sleep symptoms, they do not lead to complete remission. The present study examines the impact of Cognitive Processing Therapy (CPT) and Prolonged Exposure (PE) on subjective measures of sleep disturbance from treatment randomization through long-term follow-up (LTFU). Participants were 171 female rape victims with PTSD who were randomly assigned to CPT, PE, or Minimal Attention (MA). After 6-weeks, the MA group was randomized to CPT or PE. Sleep symptoms were assessed at baseline, post-MA, post-treatment, 3-months, 9-months and LTFU using the Pittsburgh Sleep Quality Index (PSQI) and nightmare and insomnia items from the Clinician Administered PTSD Scale. Change in sleep during MA, from pre- to post-treatment for CPT and PE, and from post-treatment through LTFU was assessed using piecewise hierarchical linear modeling with the intent-to-treat sample. Controlling for medication, sleep improved during CPT and PE compared to MA, and treatment gains were maintained through LTFU. CPT and PE were equally efficacious and improvements persist over LTFU, yet, neither produced remission of sleep disturbance. Overall, sleep symptoms do not remit and may warrant sleep-specific treatments.

Keywords

Posttraumatic Stress Disorder; Trauma; Sleep; Follow-up

The prominence of sleep disturbance in posttraumatic stress disorder (PTSD) has been widely documented, and includes both insomnia symptoms and nightmares in its diagnostic criteria (Spoormaker & Montgomery, 2008). Over 70% of individuals with PTSD report posttraumatic nightmares (Leskin, Woodward, Young, & Sheikh, 2002), with more reporting significant difficulty maintaining sleep (Neylan et al., 1998). Sleep disturbances predict both PTSD onset and severity (Koren, Arnon, Lavie, & Klein, 2002; Mellman, David, Bustamante, Fins, & Esposito, 2001), further highlighting the significant role of sleep in PTSD. Sleep disturbances are associated with negative outcomes such as increased suicidal ideation (Nishith, Resick, & Mueser, 2001), neurocognitive deficits (Drummond, Paulus, & Tapert, 2006), and increased anxiety (Babson, Feldner, Trainor, & Smith, 2009).

Poor sleep may not only maintain elevated base levels of anxiety, but may also hinder the natural recovery from PTSD (Babson & Feldner, 2010; van der Helm & Walker, 2009).

Sleep disturbance may compromise response to empirically supported treatments for PTSD because restorative sleep is necessary for both consolidation of emotional memories (see, Stickgold & Walker, 2007, for review) and generalization of fear extinction (e.g., Germain, Buysse, & Nofzinger, 2008; Pace-Schott et al., 2009). Several studies demonstrate that treatment for PTSD improves nightmares, insomnia, and perceived sleep quality, yet sleep symptoms remain at clinically significant levels even with clinically significant PTSD improvement (Belleville, Guay, & Marchand, 2010; Galovski, Monson, Bruce, & Resick, 2009; Zayfert & DeViva, 2004). Research has suggested that these residual sleep disturbances often remains prominent and highly distressing, notably impacting daily functioning (e.g., Clum et al., 2001; Kramer et al., 2003). These results have led some to suggest that PTSD-related sleep disturbances should be conceptualized as separate sleep disorders rather than symptoms of PTSD (Harvey, 2008; Spoormaker & Montgomery, 2008).

Cognitive Processing Therapy (CPT; Resick & Schnicke, 1993) and Prolonged Exposure (PE; Foa, Hearst, Dancu, Hembree, & Jaycox, 1994) are the gold standard PTSD treatments, and utilize different mechanisms of change for symptom reduction (Gallagher & Resick, 2012). CPT is founded in cognitive theory and relies on modification of distorted beliefs and cognitive processing of emotional information for symptom reduction. Extensive research has highlighted the impact of sleep disturbance on cognitive processing (e.g., Walker, 2010), which is thought to be a mechanism of change in CPT. Sleep appears to be critical in the processing of emotional experiences, and sleep loss significantly disrupts affective learning (Holland & Lewis, 2007; van der Helm & Walker, 2009; Wagner, Hallschmid, Rasch, & Born, 2006). Furthermore, cognitive factors such as worry impact subjective sleep quality (e.g., Takano, Iijima, & Tanno, 2012). The impact of cognitions and the dependency of emotional learning on sleep underscore the crucial role of sleep for PTSD treatments, such as CPT that rely on emotional processing and new learning for recovery.

PE is hypothesized to facilitate recovery of PTSD through extinction learning during imaginal and *in vivo* exposures. Research has documented that re-experiencing symptoms from trauma result from impaired extinction learning (e.g., Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007; Milad et al., 2009; Orr et al., 2000), a primary mechanism of change in PE. Sleep deprivation, which often results from the cumulative effects of the hyperarousal symptoms of PTSD, has also been demonstrated to cause significant impairment in extinction learning (e.g., Pace-Schott et al., 2009). Given the evidence that sleep promotes generalization of extinction memory (Pace-Schott et al., 2009), it is important to consider the impact of poor sleep on treatment outcome in individuals with PTSD that are being treated with PE.

Both CPT and PE demonstrate comparable treatment results on PTSD (Resick, Nishith, Weaver, Astin, & Feuer, 2002) utilizing different treatment mechanisms (Gallagher & Resick, 2012), and sleep disturbance plays a unique role in both cognitive processing and extinction-based learning. The interference of sleep disturbance on mechanisms implemented in CPT and PE highlights the need to better understand the impact of sleep during treatment, to effectively target and minimize interference in PTSD recovery. Despite the evidence for the link of PTSD-related hyperarousal and significant sleep disturbance in PTSD, few trauma-focused treatment studies have examined the impact of these treatments on sleep, and fewer studies have used validated sleep measures to do so (Nappi, Drummond, & Hall, 2011). Furthermore, these studies look at the impact of treatment using a short-term follow-up (up to 1-year), which may not provide information on the full process that is

impacting the intricate interplay between sleep and PTSD. To better understand the potential impact of poor sleep on PTSD treatment, and to help delineate the best treatment course (e.g., treat sleep first, last, or simultaneously), additional information is needed using validated measures of sleep and PTSD symptoms, with a long-term follow-up (LTFU) to provide clarity to the course of these symptoms over time.

Although we have initial evidence that overall sleep improves with both PE and CPT at 9-month follow-up (Galovski et al., 2009), understanding the impact on specific symptoms related to insomnia and longitudinally would provide further guidance for treatment of sleep disturbances in PTSD. This paper builds on a previous study (Galovski et al., 2009) by examining sleep data before it has been transformed into ordinal scale scores. Examining sleep efficiency (SE), sleep onset latency (SOL), total sleep time (TST), and sleep quality (SQ) as continuous rather than ordinal measures may be more sensitive and more useful to sleep clinicians who often use the continuous measures for diagnosis and treatment delivery. We also use insomnia and nightmare items from the PTSD measure, the Clinician Administered PTSD Scale (CAPS), to further investigate the role of sleep from pre-treatment through LTFU.

The primary aim of this paper is to examine the impact of CPT and PE on subjective sleep symptoms (SE, SOL, TST, and SQ) through a LTFU. Given current literature on reduction of depression through treatment of PTSD (Aderka, Foa, Applebaum, Shafran, & Gilboa-Schechtman, 2011; Liverant, Suvak, Pineles, & Resick, 2012), and the central feature of sleep disturbance in PTSD, we hypothesize that sleep symptoms will improve over the course of PTSD treatment. Based on previous research (Resick, Williams, Suvak, Monson, & Gradus, 2012), we expect that treatment gains related to sleep symptom change will also be maintained at LTFU. To our knowledge, this is the first study to examine sleep outcomes more than a year after PTSD treatment.

Methods

Participants

Participants were female rape victims with PTSD who enrolled in a randomized clinical trial that examined the relative efficacy of CPT, PE and Minimal Attention (MA; see, Resick et al., 2002, for more detailed information on sample including inclusion/exclusion criteria). Of the 181 women randomized to treatment, 10 were terminated for meeting exclusion criteria such as experiencing new violence (i.e., no longer 3-months posttrauma), medication change, and substance dependence relapse. The intent to treat (ITT) sample consisted of 171 women, of which 13 never came to the first session, and 37 dropped out of treatment. There were 81 women who completed either CPT or PE and 41 completed the MA condition. Of those originally randomized to MA, 13 completed CPT and 14 completed PE after completing MA. There were no significant differences in the demographic characteristics of participants randomized to CPT, PE, or MA. The ITT sample had a mean age of 31.99 (SD = 9.98), and a mean of 14.36 (SD = 2.34) years of education. The sample was 71.6% Caucasian, 25.4% African American, and 3.0% other races. Participants had severe PTSD at baseline (mean CAPS total score = 74.13, SD = 19.39).

Measures

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989)—The PSQI is a self-report inventory designed to measure retrospective sleep quality and disturbances over a 1-month interval. The PSQI assesses several domains, including sleep latency, duration of sleep, frequency and severity of specific sleep-related problems, and daytime function. The PSQI has excellent psychometric properties, and is

commonly used in assessment and treatment studies of insomnia (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). PSQI total scores greater than five indicate clinically significant sleep disturbance (Buysse et al., 1989). Five sleep outcomes were derived from the PSQI, including: 1) SOL in minutes, 1) TST in hours, 3) SE (ratio of total sleep time to the amount of time in bed), 4) SQ (item 6; ordinal scale ranging from 0 to 3), and 5) the total PSQI score. Sleep medication use, which was included as a covariate in analyses to control for the effects of sleep medication use on sleep outcomes, was assessed using PSQI item 7. Low values for the total score, SOL, SQ, insomnia, and nightmares indicate better sleep. Low values for TST and SE indicate worse sleep.

Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995)—The CAPS is a diagnostic interview for PTSD diagnosis and severity. The CAPS yields a total severity score, computed by summing the symptom frequency and intensity scores, separately rated on 0 (*low*) to 4 (*high*) scales, for all 17 items. It has been found to have excellent psychometric properties (Blake et al., 1995; Weathers, Keane, & Davidson, 2001). CAPS diagnosis was computed according to the scoring manual. Frequency of one or more plus intensity scores of 2 or greater on any given item are considered clinically significant for diagnosis. In addition to total severity score, we calculated sleep outcomes derived from the CAPS from insomnia severity (D1 sum scores) and nightmare severity (B2 sum scores).

Procedure

CPT and PE were conducted over 6 weeks, with twice-weekly sessions, and in MA women were told they would be provided with therapy in 6 weeks and the interviewer called them every 2 weeks (see, Resick et al., 2002, for more information). After the delay, the MA participants were randomized into CPT and PE if they did not improve during MA and still wanted treatment. Sleep measures were collected prior to treatment randomization (pre-treatment or pre-MA), after completion of MA for participants originally assigned to this condition (post-MA), and after treatment completion for those originally assigned to CPT and PE and those assigned to active treatment following MA (immediately post-treatment, and at 3-month, 9-month and LTFU an average of 6 years later (*SD* = 1.22; Resick, et al., 2012). Given that the LTFU was not part of the original study design, LTFU varied between 5–10 years. See Resick et al. (2012) for information on the LTFU study.

Results

Data Analytic Strategy

Multilevel modeling was performed to assess the impact of treatment condition, CPT and PE, on sleep outcomes from pre-treatment through 6-year follow-up. Multilevel models estimate treatment effects using all available data, even when some cases are missing data at some assessment points due to attrition or other factors. Data were modeled using Hierarchical Linear and Non-Linear Modeling (HLM; Raudenbush, Bryk, & Congdon, 2004) software using full maximum likelihood estimation.

Seven models were evaluated, each examining the effect of treatment condition (CPT, PE) and time interval (months prior to treatment for participants assigned to the MA condition, months in treatment, months post-treatment) on a different dependent measure (i.e., SOL, TST, SE, SQ, total PSQI score, CAPS insomnia severity, and CAPS nightmare severity). Piecewise modeling was performed to allow for different symptom trajectories (slopes) from pre-MA to post-ma/pre-treatment for those assigned to the MA condition, from pre-treatment to post-treatment for all participants, and from post-treatment through LTFU for all participants (Singer & Willett, 2003). To this end, three time variables were incorporated into each model: a) number of months prior to treatment initiation (coded as "-1.5" for the

pre-MA assessment and "0" from the start of treatment through LTFU), b) number of months in treatment (coded as "0" at the pre-MA and pre-treatment, and "1.5" for the post-treatment through LTFU), and c) number of months post-treatment (coded as "0" for the pre-MA, pre-treatment and post-treatment, and "3", "9", and "60" for the 3 month, 9 month, and LTFU, respectively). The latter represents Treatment × Time interactions testing the effect of treatment condition on the degree of symptom change during MA, treatment, and follow-up periods. Random effects were estimated for the intercept, slope during treatment, and slope during follow-up. Sleep medication use was centered around the grand mean and included as a covariate. All of the analyses were conducted with the ITT sample.

Treatment Study Results

Table 1 includes the mean, standard deviation, and sample size by outcome measure, study assessment and treatment group. Table 2 includes the parameter estimates and effect sizes for each multilevel model. Results indicate that sleep improved during treatment and this decrease in sleep symptoms did not vary by treatment condition when controlling for sleep medication. Specifically, both CPT and PE had large effects on insomnia severity and nightmare severity (Cohen's $d > .80$; $p < .001$). CPT and PE also had moderate effects on perceived SQ and total PSQI score (Cohen's $d > .50$; $p < .001$), and small but statistically significant effects on SOL, TST, and SE (Cohen's $d > .20$; $p < .001$). Sleep symptoms changed very little between the pre-MA and pre-treatment (Cohen's $d < .20$; $p > .05$ for all dependent measures), and between post-treatment and LTFU for CPT and PE (Cohen's $d < .20$; $p > .05$ for all dependent measures).[1]

Discussion

Our study assessed the long-term impact of CPT and PE on sleep disturbance. Previous research has demonstrated that individuals who received CPT and PE have significant improvements in PTSD through post-treatment and LTFU (Resick et al., 2002; Resick et al., 2012). To understand the influence of these gold standard PTSD treatments on sleep disturbance, we examined the specific impact of treatment on sleep disturbance in an ITT sample and demonstrated significant improvement and maintenance of gains through the LTFU when controlling for sleep medication. This lends further support to the notion that sleep disturbance can be significantly decreased through treatment of the primary diagnosis, PTSD, and maintained over time. Significant decreases in sleep disturbance were demonstrated across a wide range of qualitative and quantitative variables, indicating that cognitive behavior therapies (CBT) for PTSD target various aspects of sleep. Despite the significant decrease in symptoms, it is important to note that similar to past research (e.g., Zayfert & DeViva, 2004), sleep disturbance did not reach the point of remission and remained symptomatic following treatment for PTSD.

Previous reports on this sample showed that PTSD symptom improvement was maintained at LTFU (Resick et al., 2012), and our study indicated that sleep disturbance remains at clinical levels at LTFU. Taken together, the role of sleep disturbance in response to PTSD treatment may not be as directly linked with respect to poor sleep interfering with emotional processing (Walker, 2010) or generalization of extinction learning (Pace-Schott et al., 2009) required for symptom reduction in these treatments. In this light, full remission of sleep disturbance was not necessary to achieve a clinically significant treatment response for PTSD, or to maintain these gains years after treatment completion. Poor sleep hygiene may become an independent problem that must be treated separately. It is, however, important to acknowledge the potential role of improved sleep disturbance on treatment response because

---

[1]The same pattern of results was demonstrated when analyses were run with treatment completers.

sleep plays a crucial role in learning and memory (Holland & Lewis, 2007; Wagner et al., 2006) and the improvement in sleep may be needed for consolidation of emotional memories and extinction learning necessary for response to these interventions. Given this interplay, additional sleep treatment before or after PTSD treatment may further enhance treatment gains by reducing or eliminating sleep interference in emotional processing and extinction learning in CPT and PE respectively. Future research is needed to investigate the impact of sleep specific treatments, such as CBT for insomnia (CBT-I; Perlis, Jungquist, Smith, & Posner, 2005), on PTSD symptoms at follow-up. It is also important to note that these remaining sleep disturbances have the potential for impacting daily functioning (Clum et al., 2001; Kramer et al., 2003), and it is essential to clinically address these residual symptoms to further improve quality of life.

Given the unique mechanisms of change for CPT and PE (Gallagher & Resick, 2012), and the important role of sleep in both cognitive processing and extinction learning, studying the interplay between sleep disturbance and these mechanisms may explain some of the clinical difficulty treating PTSD that can result in non-response (Bradley, Greene, Russ, Dutra, & Westen, 2005). Our findings support the importance of monitoring sleep disturbance in the treatment of PTSD, because lack of sleep improvement has the potential to interfere with treatment gains. Furthermore, in the presence of treatment gains in this sample (see Resick et al., 2012), sleep does not improve to the point of remission (39.05% reached the point of remission on the PSQI at posttreatment and 40.37% at LTFU), and further sleep treatment could potentially improve daily functioning and facilitate the recovery from PTSD that occurs after the completion of active treatment. As a result, it would be beneficial for both clinicians and researchers to simultaneously monitor sleep in the context of PTSD treatment, because effective treatments for insomnia symptoms (e.g., CBT-I) and nightmares (e.g., Krakow & Zadra, 2006) exist. Future studies should investigate the impact of variable length CPT (Galovski, Blain, Mott, Elwood, & Houle, 2012) and PE on sleep to examine potential benefits of additional sessions. Additionally, studying predictors of sleep improvements would be informative to sleep and PTSD literature.

Future research should address limitations of this study by conducting similar PTSD clinical trials with LTFU that appropriately address sleep throughout the treatment. Providing sleep measures at weekly increments and at follow-up assessments, as well as weekly sleep diaries, would provide greater insight into the interplay of PTSD and sleep disturbance. The addition of objective measures of sleep disturbance, such as actigraphy, would further clarify the role of sleep disturbance in treatment of PTSD and eliminate some of the limitations of relying on only PSQI items or CAPS sleep items as used in the current study. Use of other self-report sleep measures and daily diaries would also eliminate concerns around limitations of the PSQI. Investigation of the full range of sleep disturbance both subjectively and objectively to address the 24-hour impact of symptoms in PTSD would provide further insight into the relationship between sleep and PTSD and impact of CBT for PTSD on a larger spectrum of sleep symptoms. The addition of more objective measures throughout treatment would help to further elucidate the relationship between change or improvement in sleep symptoms and the improvement in PTSD symptoms and would provide further insight into the association between poor sleep, extinction learning and treatment outcome.

Additionally, the current study included a sample of female participant with circumscribed index traumas, and given the associations between sleep and gender (Mong et al., 2011) a follow-up study that includes men and a variety of traumatic experiences, would provide more information on the generalizability of these results. Finally, it was not possible to determine whether sleep symptoms improved as a result of PTSD treatment, or as a result of improvements in PTSD symptoms and depression, due to the high correlation between

improvements in sleep and improvements in PTSD and depression (for further discussion of misuse of analyses of covariance, see Miller & Chapman, 2001). Research demonstrates that subjective report of sleep quantity is related to a variety of psychological factors including depression, anxiety, and poor coping resources (Fernandez-Mendoza et al., 2011). The reciprocal relationships between sleep problems and psychiatric disorders should be examined in future studies.

Acknowledgments

References

Aderka IM, Foa EB, Applebaum E, Shafran N, Gilboa-Schechtman E. Direction of influence between posttraumatic and depressive symptoms during prolonged exposure therapy among children and adolescents. Journal of Consulting and Clinical Psychology. 2011; 79(3):421–425. [PubMed: 21480695]

Babson KA, Feldner MT. Temporal relations between sleep problems and both traumatic event exposure and PTSD: A critical review of the empirical literature. Journal of Anxiety Disorders. 2010; 24(1):1–15. [PubMed: 19716676]

Babson KA, Feldner MT, Trainor CD, Smith RC. An experimental investigation of the effects of acute sleep deprivation on panic-relevant biological challenge responding. Behavior Therapy. 2009; 40(3):239–250. [PubMed: 19647525]

Belleville G, Guay S, Marchand A. Persistence of sleep disturbances following cognitive-behavior therapy for posttraumatic stress disorder. Journal of Psychosomatic Research. 2010; 70(4):318–327. [PubMed: 21414451]

Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, Keane TM. The development of a Clinician-Administered PTSD Scale. Journal of Traumatic Stress. 1995; 8(1):75–90. [PubMed: 7712061]

Blechert J, Michael T, Vriends N, Margraf J, Wilhelm FH. Fear conditioning in posttraumatic stress disorder: evidence for delayed extinction of autonomic, experiential, and behavioural responses. Behaviour Research and Therapy. 2007; 45(9):2019–2033. [PubMed: 17442266]

Bradley R, Greene J, Russ E, Dutra L, Westen D. A multidimensional meta-analysis of psychotherapy for PTSD. American Journal of Psychiatry. 2005; 162(2):214–227. [PubMed: 15677582]

Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. Sleep. 2006; 29(9):1155–1173. [PubMed: 17040003]

Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. Psychiatry Research. 1989; 28(2):193–213. [PubMed: 2748771]

Clum GA, Nishith P, Resick PA. Trauma-related sleep disturbance and self-reported physical health symptoms in treatment-seeking female rape victims. Journal of Nervous and Mental Disorders. 2001; 189(9):618–622.

Drummond SP, Paulus MP, Tapert SF. Effects of two nights sleep deprivation and two nights recovery sleep on response inhibition. Journal of Sleep Research. 2006; 15(3):261–265. [PubMed: 16911028]

Fernandez-Mendoza J, Calhoun SL, Bixler EO, Karataraki M, Liao D, Vela-Bueno A, Vgontzas AN. Sleep misperception and chronic insomnia in the general population: role of objective sleep duration and psychological profiles. Psychosomatic Medicine. 2011; 73(1):88–97. [PubMed: 20978224]

Foa, EB.; Hearst, DE.; Dancu, CV.; Hembree, EA.; Jaycox, LH. Prolonged exposure (PE) manual. Medical College of Pennsylvania; Eastern Pennsylvania Institute; 1994. Unpublished manuscript.

Gallagher MW, Resick PA. Mechanisms of change in Cognitive Processing Therapy and Prolonged Exposure Therapy for PTSD: Preliminary evidence for the differential effects of hopelessness and habituation. Cognitive Therapy and Research. 2012; 36(6):750–755.

Galovski TE, Blain LM, Mott JM, Elwood L, Houle T. Manualized therapy for PTSD: Flexing the structure of cognitive processing therapy. Journal of Consulting and Clinical Psychology. 2012; 80(6):968–981. [PubMed: 23106761]

Galovski TE, Monson C, Bruce SE, Resick PA. Does cognitive-behavioral therapy for PTSD improve perceived health and sleep impairment? Journal of Traumatic Stress. 2009; 22(3):197–204. [PubMed: 19466746]

Germain A, Buysse DJ, Nofzinger E. Sleep-specific mechanisms underlying posttraumatic stress disorder: Integrative review and neurobiological hypotheses. Sleep Medicine Reviews. 2008; 12(3):185–195. [PubMed: 17997114]

Harvey AG. Insomnia, psychiatric disorders, and the transdiagnostic perspective. Current Directions in Psychological Sciences. 2008; 17(5):299–303.

Holland P, Lewis PA. Emotional memory: Selective enhancement by sleep. Current Biology. 2007; 17(5):R179–R181. [PubMed: 17339020]

Koren D, Arnon I, Lavie P, Klein E. Sleep complaints as early predictors of posttraumatic stress disorder: A 1-year prospective study of injured survivors of motor vehicle accidents. American Journal of Psychiatry. 2002; 159(5):855–857. [PubMed: 11986142]

Krakow B, Zadra A. Clinical management of chronic nightmares: Imagery rehearsal therapy. Behavioral Sleep Medicine. 2006; 4(1):45–70. [PubMed: 16390284]

Kramer TL, Booth BM, Han X, Williams DK. Service utilization and outcomes in medically ill veterans with posttraumatic stress and depressive disorders. Journal of Traumatic Stress. 2003; 16(3):211–219. [PubMed: 12816332]

Leskin GA, Woodward SH, Young HE, Sheikh JI. Effects of comorbid diagnoses on sleep disturbance in PTSD. Journal of Psychiatric Research. 2002; 36(6):449–452. [PubMed: 12393315]

Liverant GI, Suvak MK, Pineles SL, Resick PA. Changes in posttraumatic stress disorder and depressive symptoms during cognitive processing therapy: Evidence for concurrent change. Journal of Consulting and Clinical Psychology. 2012; 80(6):957–967. [PubMed: 23067427]

Mellman TA, David D, Bustamante V, Fins AI, Esposito K. Predictors of post-traumatic stress disorder following severe injury. Depression and Anxiety. 2001; 14(4):226–231. [PubMed: 11754130]

Milad MR, Pitman RK, Ellis CB, Gold AL, Shin LM, Lasko NB, Rauch SL. Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. Biological Psychiatry. 2009; 66(12):1075–1082. [PubMed: 19748076]

Miller GA, Chapman JP. Misunderstanding analysis of covariance. Journal of Abnormal Psychology. 2001; 110(1):40–48. [PubMed: 11261398]

Mong JA, Baker FC, Mahoney MM, Paul KN, Schwartz MD, Semba K, Silver R. Sleep, rhythms, and the endocrine brain: influence of sex and gonadal hormones. Journal of Neuroscience. 2011; 31(45):16107–16116. [PubMed: 22072663]

Nappi CM, Drummond SP, Hall JM. Treating nightmares and insomnia in posttraumatic stress disorder: A review of current evidence. Neuropharmacology. 2011; 62(2):576–585. [PubMed: 21396945]

Neylan TC, Marmar CR, Metzler TJ, Weiss DS, Zatzick DF, Delucchi KL, Schoenfeld FB. Sleep disturbances in the Vietnam generation: Findings from a nationally representative sample of male Vietnam Veterans. American Journal of Psychiatry. 1998; 155(7):929–933. [PubMed: 9659859]

Nishith P, Resick PA, Mueser KT. Sleep difficulties and alcohol use motives in female rape victims with posttraumatic stress disorder. Journal of Traumatic Stress. 2001; 14(3):469–479. [PubMed: 11534879]

Orr SP, Metzger LJ, Lasko NB, Macklin ML, Peri T, Pitman RK. De novo conditioning in trauma-exposed individuals with and without posttraumatic stress disorder. Journal of Abnormal Psychology. 2000; 109(2):290–298. [PubMed: 10895567]

Pace-Schott EF, Milad MR, Orr SP, Rauch SL, Stickgold R, Pitman RK. Sleep promotes generalization of extinction of conditioned fear. Sleep. 2009; 32(1):19–26. [PubMed: 19189775]

Perlis, ML.; Jungquist, C.; Smith, MT.; Posner, D. Cognitive behavioral treatment of insomnia: A session-by-session guide. New York: Springer; 2005.

Raudenbush, SW.; Bryk, AS.; Congdon, R. HLM 6 for Windows [computer software]. Skokie, IL: Scientific Software International, Inc; 2004.

Resick PA, Nishith P, Weaver TL, Astin MC, Feuer CA. A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. Journal of Consulting and Clinical Psychology. 2002; 70(4):867–879. [PubMed: 12182270]

Resick PA, Williams LF, Suvak MK, Monson CM, Gradus JL. Long-term outcomes of cognitive–behavioral treatments for Posttraumatic Stress Disorder among female rape survivors. Journal of Consulting and Clinical Psychology. 2012; 80(2):201–210. [PubMed: 22182261]

Resick PA, Schnicke MK. Cognitive processing therapy for sexual assault victims. Journal of Consulting and Clinical Psychology. 1992; 60(5):748–756. [PubMed: 1401390]

Resick, PA.; Schnicke, MK. Cognitive processing therapy for rape victims: A treatment manual. Sage Publications, Inc; 1993.

Singer, JD.; Willett, JB. Applied longitudinal data analysis: Modeling change and event occurrence. USA: Oxford University Press; 2003.

Spoormaker VI, Montgomery P. Disturbed sleep in post-traumatic stress disorder: Secondary symptom or core feature? Sleep Medicine Reviews. 2008; 12(3):169–184. [PubMed: 18424196]

Stickgold R, Walker MP. Sleep-dependent memory consolidation and reconsolidation. Sleep Medicine. 2007; 8(4):331–343. [PubMed: 17470412]

Takano K, Iijima Y, Tanno Y. Repetitive thought and self-reported sleep disturbance. Behavior Therapy. 2012; 43(4):779–789. [PubMed: 23046780]

van der Helm E, Walker MP. Overnight Therapy? The role of sleep in emotional brain processing. Psychological Bulletin. 2009; 135(5):731–748. [PubMed: 19702380]

Wagner U, Hallschmid M, Rasch B, Born J. Brief sleep after learning keeps emotional memories alive for years. Biological Psychiatry. 2006; 60(7):788–790. [PubMed: 16806090]

Walker MP. Sleep, memory and emotion. Progress in Brain Research. 2010; 185:49–68. [PubMed: 21075233]

Weathers FW, Keane TM, Davidson JR. Clinician-administered PTSD scale: A review of the first ten years of research. Depression and Anxiety. 2001; 13(3):132–156. [PubMed: 11387733]

Zayfert C, DeViva JC. Residual insomnia following cognitive behavioral therapy for PTSD. Journal of Traumatic Stress. 2004; 17(1):69–73. [PubMed: 15027796]

Highlights

- Sleep improved during Cognitive Processing Therapy and Prolonged Exposure.

- Treatment gains were maintained through long term follow-up.

- Neither treatment produced remission of sleep disturbance.

Table 1

Means, standard deviations, and sample sizes for sleep outcomes in the CPT and PE samples

Outcome	CPT			PE		
	M	*SD*	*n*	*M*	*SD*	*n*
CAPS D1						
Pre-MA	5.81	1.94	21	5.12	2.60	26
Post-MA	5.61	1.91	18	5.09	2.24	22
Pre-treatment	5.71	2.27	80	5.54	2.42	84
Post-treatment	2.85	2.88	55	2.15	2.72	55
3 month follow-up	2.56	2.96	50	3.12	3.03	51
9 month follow-up	2.39	2.64	41	2.67	2.80	39
Long-term follow-up	2.14	3.05	63	3.03	3.31	63
CAPS B2						
Pre-MA	4.52	2.20	21	4.73	1.46	26
Post-MA	4.67	1.46	18	4.86	2.03	22
Pre-treatment	5.24	1.66	80	5.14	1.88	84
Post-treatment	1.44	1.76	55	1.85	2.38	55
3 month follow-up	2.08	1.99	50	2.35	2.15	51
9 month follow-up	2.29	1.95	41	1.72	2.13	39
Long-term follow-up	2.44	2.04	63	1.73	2.09	63
PSQI SOL						
Pre-MA	31.92	23.76	13	45.19	36.97	18
Post-MA	37.67	31.62	15	39.90	31.51	24
Pre-treatment	48.95	48.68	77	44.77	32.25	85
Post-treatment	28.94	28.65	54	30.44	33.56	54
3 month follow-up	35.54	37.69	27	19.06	16.98	16
9 month follow-up	30.41	35.78	39	21.53	19.12	39
Long-term follow-up	37.73	44.31	56	32.64	26.25	56
PSQI TST						
Pre-MA	6.42	2.39	13	5.64	1.48	18
Post-MA	6.35	2.25	15	5.80	1.48	25

Outcome	CPT			PE		
	M	*SD*	*n*	*M*	*SD*	*n*
Pre-treatment	5.73	1.72	76	5.79	1.76	85
Post-treatment	6.56	1.34	54	6.65	1.50	54
3 month follow-up	6.49	1.32	27	6.80	1.12	16
9 month follow-up	6.49	1.37	39	6.80	1.39	39
Long-term follow-up	6.35	1.48	56	6.45	1.81	56
PSQI SE						
Pre-MA	77.13	12.15	13	80.92	14.71	17
Post-MA	81.54	15.10	15	80.60	16.37	25
Pre-treatment	77.16	18.56	76	78.30	19.62	84
Post-treatment	84.27	13.88	54	87.50	17.19	54
3 month follow-up	84.60	13.80	27	86.89	11.69	16
9 month follow-up	86.78	13.53	38	88.78	12.60	39
Long-term follow-up	87.58	19.85	55	85.69	18.19	56
PSQI SQ						
Pre-MA	1.77	0.73	13	1.89	0.68	18
Post-MA	1.93	0.80	15	1.72	0.84	25
Pre-treatment	1.94	0.85	77	1.89	0.78	87
Post-treatment	1.11	0.72	54	1.19	0.85	54
3 month follow-up	1.30	0.78	27	0.94	0.77	16
9 month follow-up	1.08	0.74	39	1.00	0.73	39
Long-term follow-up	1.19	0.91	57	1.36	0.94	56
PSQI total						
Pre-MA	10.54	4.33	13	10.80	3.08	15
Post-MA	10.40	4.50	15	10.48	3.72	23
Pre-treatment	10.79	4.05	73	11.37	4.33	81
Post-treatment	7.25	3.62	51	7.34	4.55	53
3 month follow-up	7.22	4.48	27	5.63	3.22	16
9 month follow-up	7.08	4.35	38	6.55	3.52	38
Long-term follow-up	7.51	4.34	55	8.20	5.07	54

Note. CAPS items D1 and B2 assess the severity of insomnia and nightmares, respectively. CPT=cognitive processing therapy; PE= prolonged exposure; SE=sleep efficiency; SOL= sleep onset latency; SQ= sleep quality; TST= total sleep time; tx= treatment.

Table 2

Parameter estimates and effect sizes for multilevel models of sleep in the CPT and PE intent-to-treat samples

Measure	CPT			PE			CPT-PE
	β	SE	d	β	SE	d	d
CAPS D1							
Intercept	5.76	0.27	2.44	5.41	0.38	2.29	0.15
Wait-list slope	−0.28	0.45	−0.12	0.14	0.60	0.06	−0.18
Treatment slope	−1.98	0.25	−0.84	−2.02	0.35	−0.85	0.02
Post-treatment slope	−0.01	0.01	0.00	0.01	0.01	0.00	−0.01
Medication intercept	0.46	0.11	2.31				
CAPS B2							
Intercept	5.29	0.21	2.95	5.16	0.29	2.88	0.07
Wait-list slope	0.45	0.34	0.25	0.15	0.45	0.08	0.17
Treatment slope	−2.34	0.18	−1.30	−2.11	0.26	−1.18	−0.13
Post-treatment slope	0.01	0.01	0.01	−0.00	0.01	0.00	0.01
Medication intercept	−0.04	0.08	−0.02				
PSQI SOL							
Intercept	49.94	4.42	1.27	43.33	6.13	1.11	0.17
Wait-list slope	5.60	4.66	0.14	−2.48	6.17	−0.06	0.21
Treatment slope	−11.29	2.87	−0.29	−8.97	4.03	−0.23	−0.06
Post-treatment slope	0.09	0.08	0.00	0.03	0.11	0.00	0.00
Medication intercept	6.60	1.26	0.17				
PSQI TST							
Intercept	5.73	0.20	3.31	5.83	0.27	3.37	−0.06
Wait-list slope	−0.03	0.21	−0.02	0.08	0.27	0.05	−0.06
Treatment slope	0.52	0.12	0.30	0.51	0.16	0.29	0.01
Post-treatment slope	0.00	0.00	0.00	−0.00	0.01	0.00	0.00
Medication intercept	−0.13	0.06	−0.08				
PSQI SE							
Intercept	76.75	2.00	4.37	79.25	2.78	4.51	−0.14
Wait-list slope	2.93	2.58	0.17	−1.24	3.44	−0.07	0.24

Measure	CPT			PE			CPT-PE
	β	SE	d	β	SE	d	d
Treatment slope	5.03	1.37	0.29	5.14	1.92	0.29	−0.01
Post-treatment slope	0.05	0.05	0.00	−0.01	0.07	0.00	0.00
Medication intercept	−4.54	0.65	−0.26				
PSQI SQ							
Intercept	1.95	0.09	2.42	1.85	0.13	2.29	0.12
Wait-list slope	0.06	0.12	0.07	−0.03	0.16	−0.04	0.11
Treatment slope	−0.52	0.07	−0.64	−0.47	0.10	−0.58	−0.06
Post-treatment slope	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Medication intercept	0.14	0.03	0.18	0.00			
PSQI total							
Intercept	10.91	0.39	3.25	10.74	0.54	3.20	0.05
Wait-list slope	−0.31	0.48	−0.09	0.06	0.66	0.02	−0.11
Treatment slope	−2.23	0.31	−0.67	−2.25	0.43	−0.67	0.01
Post-treatment slope	0.01	0.01	0.00	0.00	0.01	0.00	0.00
Medication intercept	1.92	0.13	0.61				

Note. All multilevel regression analyses were based on the intention to treat sample (CPT *n* = 83, PE *n* = 88; waitlist CPT *n* = 21, waitlist PE *n* = 26). Effects of sleep medications are for the combined sample (*n* = 171). CAPS items D1 and B2 assess the severity of insomnia and nightmares, respectively. CPT=cognitive processing therapy; *d* =Cohen's *d* (measure of effect size); PE= prolonged exposure; SE= sleep efficiency; SOL= sleep onset latency; SQ=sleep quality; TST= total sleep time.