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# Change in sleep symptoms across Cognitive Processing Therapy and Prolonged Exposure: A longitudinal perspective

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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.

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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, Nishith, & Resick, 2001; Kramer, Booth, Han, & Williams, 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,

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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 (van der Helm & Walker, 2009; Holland & Lewis, 2007; 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, 2) 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