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Sleep disturbance as a predictor of affective functioning and symptom severity among individuals with PTSD: An ecological momentary assessment study

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ABSTRACT

Recent research has highlighted the etiological role of sleep disturbance in posttraumatic stress disorder (PTSD); however it is currently unknown how daily changes in sleep are associated with next-day PTSD symptoms. Furthermore, sleep is critical for maintaining appropriate affect, leading some to hypothesize that affective dysfunction may account for the link between sleep disturbances and PTSD symptoms. Thus, the current study tested the relationship between sleep disturbances, affective valence, and PTSD symptoms utilizing an ecological momentary assessment (EMA) design among individuals with PTSD (n=30) who participated in 4 EMA-based assessments daily over 8 days. Multilevel modeling indicated that, after accounting for prior evening's PTSD symptoms, poor sleep quality and reduced sleep efficiency were associated with increased PTSD symptoms and negative affect. Furthermore, results supported the indirect effect of poor sleep quality on elevated PTSD symptoms through increased negative affect in the morning. Findings add to the body of research demonstrating the negative impact of poor sleep for individuals with PTSD by indicating that daily variations in sleep can affect next-day PTSD symptoms, and identifying negative affect as a mechanism of this relationship.

Recent research has transformed the way we view sleep disturbances in the context of mental illness from a common yet mechanistically unimportant symptom to an independent comorbid diagnosis that is potentially an etiological factor in various conditions (Harvey, 2008). This is due to evidence suggesting insomnia prospectively predicts a variety of psychological disorders (Breslau, Roth, Rosenthal, & Andreski, 1996; Johnson, Roth, & Breslau, 2006; Sivertsen et al., 2014; Taylor, Lichstein, & Durrence, 2003), and by emerging findings that sleep is critical for mood regulation and appropriate emotional responding (Goldstein & Walker, 2014). Considering this evidence, insomnia may affect psychological conditions via impaired affective functioning (Harvey, 2008). This area of research is exciting because of the potential to leverage sleep medicine to improve quality of life for individuals with mental illness. Indeed, cognitive behavioral therapy for insomnia (CBT-I) decreases symptoms of mood and anxiety disorders (Belleville, Cousineau, Levrier, & St-Pierre-Delorme, 2011; Manber et al., 2008).

One disorder receiving abundant attention due to its close ties with sleep disturbance is posttraumatic stress disorder (PTSD), an impairing condition characterized by the inability to recover from a stress reaction following an extreme stressor (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Individuals with PTSD experience a range of symptoms, including re-experiencing, avoidance, negative alterations in cognition and mood, and alterations in arousal (American Psychiatric Association, 2013). Those with PTSD and comorbid sleep disturbance have more severe presentations of PTSD, including increased symptom severity (Short, Babson, Boden, & Bonn-Miller, 2014a; Short, Raines, Oglesby, Zvolensky, & Schmidt, 2014b), functional impairment (DeViva, Zayfert, & Mellman, 2004), substance use, depression, and self-injurious behavior (Krakow et al., 2000; Nishith, Resick, & Mueser, 2001; Short et al., 2015). Furthermore, prospective studies find that sleep disturbance following trauma predicts PTSD diagnosis (Koren, Arnon, Lavie, & Klein, 2002; Mellman, Pigeon, Nowell, & Nolan, 2007), as well as other posttrauma psychological disorders (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010). Finally, sleep problems do

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not remit spontaneously or through PTSD treatment, and are associated with ongoing PTSD symptoms (Wright et al., 2011). These lines of evidence suggest sleep disturbances may play an etiological role in PTSD symptoms.

Sleep-dependent affective processes may be important in explaining how sleep disturbance affects PTSD symptoms (Germain, 2013; Goldstein & Walker, 2014). Sleep loss is associated with increases in depressive (Caldwell, Caldwell, Brown, & Smith, 2004; Fredriksen, Rhodes, Reddy, & Way, 2004) and anxiety symptoms (Sagaspe et al., 2006; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). This is often paired with decreases in positive affect (Paterson et al., 2011; Talbot et al., 2010). These impairments in affective responding may be one mechanism explaining why sleep disturbances are associated with increased PTSD symptoms (Germain, 2013; Goldstein & Walker, 2014). Poor sleep can be associated with next day affective impairment, such as unstable mood that is easily disrupted by mild stressors (Short et al., 2016). This increased negative affect could exacerbate re-experiencing symptoms, which may be more upsetting to the individual, and lead to increased avoidance of trauma reminders. Furthermore, poor sleep can exacerbate negative alterations in cognition, mood, and arousal in PTSD, maintaining those symptoms as well.

To test the hypothesis that sleep disturbances predict increases in daytime PTSD symptoms via increases in negative affect, the current study used an ecological momentary assessment (EMA) design. Throughout the current manuscript, we refer to insomnia symptoms and nightmares collectively as "sleep disturbance." However, it is important to note that insomnia and nightmare disorders are considered to be comorbid disorders with their own unique etiology and prognosis, rather than simply symptoms of PTSD (Harvey, 2001). Yet, we are unable to formally diagnose these disorders in the current paper, hence utilizing the term "sleep disturbance." First, we hypothesized that sleep disturbances (i.e., sleep efficiency, duration, quality, and nightmare frequency) would be associated with increased next day PTSD symptoms after covarying for the prior evening's PTSD symptoms. Second, we hypothesized sleep disturbances would be associated with increased next day negative affect, decreased positive affect, and more negatively valenced affect ratios after covarying for the prior evening's PTSD symptoms. Third, we hypothesized that the effect of sleep disturbances on next day mid-morning and afternoon PTSD symptoms would be mediated by increases in morning negative affect, after covarying for the prior evening's PTSD symptoms. Additionally, we examined positive affect as an alternative mediator, given lack of theoretical basis for decreased positive affect alone as an etiological factor. Fourth, we hypothesized our results would hold when only examining the unique symptom clusters of PTSD (i.e., re-experiencing and avoidance), excluding the symptoms that could be construed as general negative affectivity (i.e., alterations in cognition and mood, alterations in arousal and reactivity). Fifth, consistent with recent research (Alvaro, Roberts, & Harris, 2013), we hypothesized there would be a bidirectional relationship, such that daytime elevated PTSD symptoms would negatively influence sleep.

1. Method

1.1. Participants

We recruited 30 participants diagnosed with PTSD according to the Structured Clinical Interview for DSM-V (SCID; First, Williams, Karg, & Spitzer, 2015). Participants were at least 18 years of age and were recruited from the local community (n=24, 80.0%) and the university's undergraduate student research pool (n=6, 20.0%). Participants were screened via phone or the university's research pool using the PTSD Checklist (Weathers, Litz, Herman, Huska, & Keane, 1993), and then scheduled for a baseline appointment, in which PTSD diagnoses were confirmed. Participants were required to have a diagnosis of PTSD and own a smartphone with access to texting and the internet to participate in the EMAs. Exclusion criteria included being diagnosed with a psychotic disorder not stable on psychiatric medication, and a diagnosis of severe substance use disorder.

Participants were by majority female (61.3%) with ages ranging from 18 to 60 (M=38.03, SD=15.14). The majority of the sample identified as White (64.5%), followed by Black (29.0%), and American Indian/Alaskan Native (3.2%). Participants reported the following as their most bothersome trauma: sexual assault (43.3%), non-sexual assault (16.6%), other (e.g., witnessing a death, mass grave sites; 13.3%), serious accident (10.0%), combat (6.7%), imprisonment (6.7%), while one participant did not select a most bothersome event on the PCL (3.3%). At baseline, participants scored an average on 39.28 on the PCL-5, with specific symptom cluster averages as follows: re-experiencing (12.17), avoidance (5.79), alterations in cognition and mood (9.90), and alterations in arousal and reactivity (11.41). The majority (63%) reported receiving psychopharmacological treatment. Most participants had chronic PTSD as time since traumatic event ranged from several months to 50 years (M = 9.18 years). In addition, 93.3% of the participants met criteria for at least one comorbid disorder. Specifically, 80% of the sample met criteria for an anxiety disorder, followed by mood disorder (73.3%), obsessive compulsive or related disorder (26.7%), substance use disorder (13.3%), and eating disorder (6.7%). PTSD was the primary diagnosis for the majority of participants (73.3%).

1.2. Procedure

1.2.1. Baseline appointment

Written informed consent was obtained upon arrival to the laboratory. Then, the PTSD module of the SCID was used to confirm PTSD diagnostic status (First et al., 2015). Participants not meeting criteria for PTSD were dismissed. All other participants continued by completing the remaining SCID modules and a battery of self-report questionnaires. Next, participants were asked for their normal sleep schedule to optimize assessment time points. Individuals were then instructed how to participate in the remainder of the study, and received a sample link and practiced filling out measures on their smartphone with a research assistant to ensure their understanding. Specifically, participants filled out the PTSD section with the experimenter to ensure they understood the questions and the time period they referred to. Participants were encouraged to call the laboratory with any questions regarding the EMA questionnaires. The EMA questionnaires took less than 5 min to complete on average (typically 2–3 min). EMAs were time stamped and checked for temporal accuracy. To reduce participant burden, we used brief versions of all measures used and ensured each time point took less than 5 min to complete. Participants were also compensated for their time.

1.2.2. EMA period

Starting the day after each individual's baseline appointment, participants received 4 text messages daily linking to surveys to complete their assessments over the next 8 days. Data regarding average wake and sleep times collected during the baseline appointment were used to determine an appropriate assessment schedule. Participants received text messages on a quasi-random schedule, with one survey upon waking, one in the mid-morning, one in the afternoon, and one in the evening before bedtime (see Fig. 1). Text messages contained a link to the participant's appropriate assessment. If the participant did not complete the survey after receiving a reminder and 60 min had elapsed, that time point was considered missed (5.3% of assessments). Participants' survey responses were time stamped and reviewed to ensure temporal