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Association between sleep, childhood trauma and psychosis-like experiences

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ABSTRACT

Psychosis-like experiences (PLEs), or attenuated positive symptoms of psychosis, present along a severity continuum and have been associated with distressing thoughts and impairments in functioning. Although knowledge of the clinical importance of PLEs is expanding, risk factors for their expression are still poorly understood. Sleep disturbances are one known factor that exacerbate PLEs expression and distress, and trauma exposure is associated with occurrence of PLEs, as well as increased risk of later sleep difficulties. This study examined the joint influences of sleep and trauma on PLEs in an undergraduate sample. Self-report questionnaires on presence and distress of PLEs, sleep problems, and occurrence of previous traumatic experiences were completed by participants ($N = 409$). In order to determine the unique impact of sleep on PLEs, three sets of predictors: sociodemographic, psychosocial (including trauma), and sleep were entered in steps into a hierarchical multiple regression model. In the final model, specific sleep domains uniquely predicted PLEs, while previous trauma exposure, which was a significant predictor when entered in step two with other psychosocial variables, was no longer a significant predictor. Results suggest the possibility that disruptions in sleep following or occurring alongside a traumatic experience may somehow contribute to, or exacerbate the presence of PLEs.

1. Introduction

Psychosis-like experiences (PLEs) occur in the general population, at times causing distress or impairment. Research indicates that experiencing a previous traumatic event may predispose individuals for expression of PLEs (Read et al., 2005). In addition, disruption in sleep, a frequent consequence of trauma, also has demonstrated associations with increases in PLEs (Reeve et al., 2015). There is little understanding; however, of possible associated risk factors, symptom overlap, or possible underlying mechanisms within and between these three factors.

1.1. Psychosis-like experiences

Attenuated positive symptoms of psychosis, or PLEs, are present in roughly 8% of the general population (Van Os et al., 2009). Individuals experiencing PLEs share demographic, etiological, and psychopathological risk factors with those experiencing psychotic disorders (Linscott and Van Os, 2013), suggesting that a better understanding of PLEs will allow a more complete picture of the psychosis spectrum. PLEs present along a severity continuum. Some are mild and transient, not leading to distress or impairment, others are more severe and persistent but do not progress to psychosis (Yung et al., 2009), whereas still others precede the onset of clinical psychosis (Dominguez et al., 2010). Regardless of their association with later psychosis, it remains important to elucidate factors related to PLEs as they have been associated with impairments in functioning (Yung et al., 2005; Kelleher et al., 2014), help-seeking behavior (DeVylder et al., 2014a, 2014b, 2014c), psychiatric diagnoses (Kelleher et al., 2012; DeVylder et al., 2014a, 2014b, 2014c), and thoughts or actions of self-harm (Kelleher et al., 2013; DeVylder et al., 2015). These patterns have been observed in both community and college-ascertained samples. Individuals with PLEs are further at a greater risk to experience comorbid disturbances in mood (Calkins

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et al., 2014), and higher levels of substance use (Kelleher and Cannon, 2011), and both mood and substance use has the potential to exasperate presence of PLEs (Krabbendam et al., 2005; Barkus et al., 2006). Yet, despite a growing understanding of the clinical and conceptual importance of PLEs, the joint contributions of environmental and biobehavioral risk factors for their expression are less clear.

1.2. Sleep disruption and PLEs

Sleep disturbance is one factor that appears to increase the risk for the expression of PLEs. A range of sleep problems are observed across the psychosis spectrum (Cohrs, 2008; Lunsford-Avery and Mittal, 2013; Koyanagi and Stickley, 2015; Davies et al., 2017; Poe et al., 2017), including among clinical and community samples of adults (Oh et al., 2016) and youth experiencing PLEs (Nishida et al., 2008; Oshima et al., 2010; Lee et al., 2012; Fisher et al., 2014; Jeppesen et al., 2015; Reeve et al., 2015; Taylor et al., 2015; Thompson et al., 2015). Such associations between sleep disturbance and PLEs presentation are consistent in international studies and across multiple cultural contexts (Koyanagi and Stickley, 2015; Oh et al., 2016). Several specific sleep disorders have been associated with PLEs, including insomnia, fragmented sleep, night anxiety, movement at night, and sensations at night (definitions of specific sleep disruptions are outlined in Table 1) (Kaneita et al., 2006; Oh et al., 2016; Andorko et al., 2017). These sleep disturbances are known to impact stress tolerance, immunological functioning, and cognitive functioning, as well as to exacerbate socioemotional distress (Hofstetter et al., 2005; Bromundt et al., 2011; Waters et al., 2011; Kelly and El-Sheikh, 2014; Poe et al., 2017), all of which are considered to play roles in the etiology of psychosis (Walker et al., 2008; Bergink et al., 2014; Bora and Murray, 2014). Polysomnographic and brain imaging studies further indicate that neural structures regulating sleep (thalamus, cortical gray matter) are also affected in individuals furthest along the psychosis continuum (i.e., schizophrenia) (Lunsford-Avery and Mittal, 2013).

Additionally, acute episodes of sleep deprivation have been linked with sudden onset of PLEs in individuals with no prior history of such experiences (Orzeł-Gryglewska, 2010; Petrovsky et al., 2014). In these occurrences, PLEs rarely persist following the regaining of typical sleep hours, and are likely caused by decreases in metabolism of glucose within the prefrontal cortex, a known consequence of continuous states of wakefulness (Thomas et al., 2000). The prefrontal cortex is a neural region with significant associations to PLEs and schizophrenia (Hill et al., 2004). Given such pervasive relations between sleep and PLEs, sleep disturbance is increasingly recognized as a core pathophysiological feature of the psychosis spectrum (Yates, 2016).

Table 1
Brief definitions of sleep subscales.

Subscale	Definition
Insomnia	
Initial insomnia	Difficulty initiating sleep in the beginning of the night.
Fragmented sleep	Difficulty staying asleep.
Night anxiety	Excessive worrying while trying to initiate sleep.
Light sleep	Easily awoken during the night.
Lassitude	
Non-restorative sleep	Feeling that sleep has been insufficiently refreshing.
Excessive sleep	Sleeping more than recommended (>12 h).
Fatigue	Excessive sleepiness, or low energy during the day.
Parasomnia	
Nightmares	Unpleasant, frightening, or disturbing dreams.
Movement at night	Recurrent movement (kicking, jerking, flailing) while asleep.
Sensations at night	Unpleasant or uncomfortable feelings in limbs at night (restless legs).
Circadian rhythm	
Irregular schedule	Varying sleep and wake times.

1.3. Trauma and PLEs

Given the proposition that the neurobiological consequences of sleep dysfunction are related to abnormalities observed in the psychosis spectrum, a useful extension of this work could be to identify shared, mechanistically plausible risk factors for the two phenomena. One such factor is childhood trauma exposure. Prevalence estimates indicate that the majority of adults (89.7%) have experienced exposure to some sort of traumatic event (Kilpatrick et al., 2013). Longitudinal, cross-sectional, and meta analytic studies indicate that physical abuse, unwanted sexual experiences, exposure to domestic violence, and bully and police victimization are associated with the occurrence and severity of PLEs (Read et al., 2005; Spauwen et al., 2006; Schreier et al., 2009; Fisher et al., 2013; Gibson et al., 2016; Grivel et al., 2017). Research suggests that trauma exposure is related to PLEs by virtue of its tendency to disrupt cognitive, emotional, and stress-regulatory systems (Morrison et al., 2003; Van Os et al., 2009), all of which are also affected by sleep dysfunction. Consistent with this possibility, researchers have found that high levels of stress sensitivity (Gibson et al., 2016) and anxiety (Freeman and Fowler, 2009) – both common consequences of trauma (Cicchetti and Toth, 2005) – mediate the relation between childhood trauma exposure and PLEs. Like sleep dysfunction, childhood trauma exposure is also associated with abnormalities in immunological and cognitive functioning (Gibson et al., 2016).

1.4. Trauma, sleep disruption and distress

A history of acute or chronic trauma exposure is associated with an increased risk of later sleep difficulties (Lavie, 2001; Singh and Kenney, 2013). Sleep is generally impacted both immediately following a trauma and in the long-term (Sadeh, 1996), with affected sleep domains including nightmares, fragmented sleep, initial insomnia, fatigue, sensations at night, light sleep, and night anxiety (Lavie, 2001; Germain et al., 2008; Spilsbury et al., 2014; Ho et al., 2016). These effects on normal sleep processes are considered one of the most frequent and distressing complaints following a traumatic event (Nappi et al., 2012). In addition, longitudinal studies indicate that sleep disruption, prior to and following trauma exposure leads to exacerbation of subsequent trauma-related distress (Koren et al., 2002), specifically for those with pre-existing insomnia or demonstrating less resiliency overall (Gehrman et al., 2013; Seelig et al., 2016). Further, nightmares can serve as an indicator of the degree to which a stressful event becomes psychologically traumatic (Thompson et al., 2015). Ultimately, the direction of this signal is unclear, however, as it may be that sleep disturbances after a psychological trauma exacerbate the traumatic response, or that the severity of the trauma leads to both sleep disturbances as well as a more severe response to trauma (Koren et al., 2002).

In addition to speculating on causal relations between sleep disturbances and trauma, Thompson et al. (2015) report a higher prevalence of psychotic experiences at age 18 in individuals who reported nightmares at age 12. The authors suggest that psychosis and psychological trauma may share the similar mechanisms of affective network dysfunction. Further, in order to better understand the relations between all three variables, Thompson and colleagues called for future research elucidating the links between trauma, psychosis, and sleep disturbances. Despite the interrelations of sleep disturbances to both trauma exposure and PLEs, however, no study to date has examined these domains together. This remains an important gap in the literature, given the degree of overlap and potential for shared mechanisms in the development and exacerbation of PLEs.

1.5. Current study

The present study sought to examine the joint influences of childhood trauma and sleep problems on the severity of PLEs in a sample of undergraduate students, a young-adult sample at peak age to develop

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