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The Case for Investigating a Bidirectional Association between Insomnia Symptoms and Eating Disorder Pathology

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Abstract

Many people with eating disorders (EDs) report symptoms of insomnia (i.e., frequent difficulty falling asleep, staying asleep, and/or early morning wakening) and sleep problems have been linked to alterations in eating behaviors; however, mechanisms of these bidirectional associations remain poorly understood and under researched. This is a problem because higher insomnia symptom severity is a risk factor for the onset and perpetuation of anxiety, mood, trauma, substance use disorders and, potentially, ED symptoms. Furthermore, insomnia symptoms may hinder recovery and increase relapse rates following successful psychotherapy. In this paper, we describe potential mechanisms underlying bidirectional associations between insomnia and eating psychopathology that may contribute to the etiology and maintenance of both disorders. We suggest novel directions for future research to characterize the association between dysregulated sleep and ED symptoms and to evaluate impacts of insomnia symptoms on relapse and recovery for people with co-occurring pathology. Finally, we discuss options for testing the incorporation of existing evidence-based treatments for insomnia disorder (e.g., Cognitive-Behavioral Therapy for Insomnia) with ED care. Overall, insomnia symptoms present a promising intervention point for ED treatment that has not been systematically tested, yet would be highly feasible to address in routine clinical care.

Keywords

Eating disorders; insomnia disorder; sleep

Emerging research suggests that insomnia symptoms are common in people with eating disorders (EDs) (e.g., Allison et al., 2016; Goel et al., 2020; KyungRan et al., 2010; Lombardo et al., 2014; Padez-Vieira & Afonso, 2016). To date, the majority of research on insomnia symptoms and EDs has focused on the prevalence and nature of sleep disturbance (e.g., delayed sleep onset, alterations in sleep architecture), with comparatively less focus on mechanisms (see reviews in Allison et al., 2016; Cooper et al., 2020; Lauer & Krieg, 2004). Although mechanisms linking sleep and eating behaviors have been studied in non-clinical

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and animal samples, the literature in people with EDs has been limited by smaller sample sizes and there is great opportunity for growth in this field of study. Thus, this review proposes a translational approach and offers applications for how it may apply to ED presentations, highlighting areas where more information is needed. To do so, we briefly explain the neurobiology of insomnia, highlight potential mechanisms linking insomnia and ED symptoms, and describe hypothesized pathways for insomnia to influence treatment outcomes. Finally, we describe several directions to guide future research on insomnia and eating pathology.

Understanding Mechanisms of Sleep in Insomnia

The psychobiologic processes controlling good sleep include two mechanisms: circadian processes (Process C) and homeostatic processes (Process S) (Perlis et al., 2011). In normal sleep, circadian processes contribute to varying levels of wakefulness and sleepiness throughout the day. Specifically, most individuals are alert in the morning after awakening until the “post-lunch dip” in the afternoon, after which alertness increases until it peaks around 7–9 PM in the evening. Following this point, alertness decreases and individuals become primed for sleep in the late evening. In terms of Process S, the homeostatic process (“sleep drive”) is directly related to time awake in a linear fashion, such that the longer an individual is awake, the stronger the sleep drive (i.e., the individual becomes more sleepy). When these processes are functioning in conjunction, they facilitate appropriate sleep onset and maintenance.

For individuals with insomnia disorder, these processes become interrupted, leading to difficulties with sleep onset and maintenance that occur three or more days per week for at least three months (American Psychiatric Association, 2013). Of note, although insomnia symptoms are among the diagnostic criteria for other psychological disorders, insomnia disorder is a distinct diagnosis (Harvey, 2001). People with insomnia disorder often engage in coping behaviors that dysregulate the circadian and homeostatic processes and maintain dysregulated sleep (Perlis et al., 2011). For example, they may “sleep in” to make up for poor sleep, which has the paradoxical effect of subsequently causing increased difficulty falling or staying asleep due to inability to build sleep drive, and disrupts circadian rhythms. Other common maladaptive behaviors include going to bed before one is actually sleepy to ensure “enough time” to fall asleep and napping to compensate for a night of poor sleep.

For those who do not meet full criteria for insomnia disorder, insomnia symptoms can often still cause clinically significant distress and impairment, including poorer quality of life and increased mental health symptoms (LeBlanc et al., 2007), suggesting the importance of examining the range of possible insomnia symptoms. Even one night of poor sleep (Short, Allan, et al., 2017) and/or acute insomnia can cause difficulties with cognitive-affective functioning, impact physiology, and cause significant distress and problems for daytime functioning (Ellis et al., 2012). However, chronic insomnia appears to be even more risky for the development of longer-term problems, such as suicide risk and mental health symptoms (Nadorff et al., 2013; Ohayon & Roth, 2003), potentially including EDs. This is likely because the impact of insomnia is cumulative over time, as maladaptive sleep-related

behaviors become more ingrained and the impact of poor sleep on one's physiology becomes chronic (e.g., et al., 2013).

It is likely that there is a bidirectional association between sleep and eating processes, such that eating pathology disrupts sleep and dysregulation in sleep influences eating behaviors (Linnaranta et al., 2020) and that this association is particularly problematic for people with EDs who may experience exacerbation and maintenance of ED pathology via dysregulated sleep. In the following paragraphs, we will discuss promising preliminary mechanisms underlying this bidirectional association (Figure 1). We propose a model in which acute symptoms of insomnia exacerbate problematic eating behaviors and ED behaviors cause alterations in sleep. Over time, this positive feedback loop results in cognitive, physiological, and behavioral changes that may further entrench individuals in EDs and result in insomnia disorder. This review should be taken as a starting point for inquiry and is not intended to encompass all theories of how insomnia and EDs may be related.

Potential Mechanisms Underlying Bidirectional Associations between ED Pathology and Insomnia Symptoms

ED Behaviors and Cognitions Disrupt Sleep Processes

Common ED behaviors may interfere with sleep processes; however, more research is needed to establish these claims as the literature has primarily examined eating behaviors in people without EDs, rather than clinical samples, and acute effects of poor sleep on behavior. For example, one study in a non-clinical sample found that vigorous exercise shortly before bedtime increases sleep-onset latency and decreases sleep efficiency (Stutz et al., 2019), which may be of particular relevance to individuals who exercise due to shape or weight concerns. Sleep may also be a means to avoid eating, aversive emotions, or distressing situations. For instance, people may nap or sleep in to avoid needing to eat meals or confronting stressors. This is problematic, because daytime napping decreases sleep load and results in less drive to fall asleep at appropriate bedtimes. Finally, binge-eating episodes, which are common in evenings (Schreiber-Gregory et al., 2013), may disrupt sleep cycles by delaying bedtime or interfering with sleep onset or quality due to increased arousal, fullness, or digestive processes. Similarly, restricting one's eating may lead to difficulties with sleep onset due to hunger, or may facilitate sleep onset (including at unwanted times during the day) due to fatigue and malnutrition.

People with night-eating syndrome (NES), an ED characterized by recurrent eating after waking in the night or excessive eating after the nighttime meal (American Psychiatric Association, 2013), are at a higher risk for insomnia disorder (Vander Wal, 2012). Although not adapted as diagnostic criteria for DSM 5, insomnia occurring four days per week was proposed as a component of the diagnosis (Allison et al., 2010). NES has been proposed to function as a peripheral oscillator, causing delayed sleep phase (Kandeger et al., 2018). It is possible that over time, NES could entrench sleep habits that maintain and perpetuate insomnia, even in the absence of NES symptoms.

In terms of cognitive features of EDs, Harvey's cognitive model of insomnia (Harvey, 2002) proposes that daytime negative affect is a core maintenance factor for insomnia.

Specifically, individuals with insomnia have excessive negative affect and negatively toned cognitions during the day that contribute to increased arousal and trouble sleeping at night. Such cognitive and emotional patterns may be particularly problematic during the pre-sleep period, when individuals with insomnia often engage in negative repetitive thought. Indeed, those with insomnia often attribute their sleep difficulties with the inability to suppress pre-sleep cognitive activity (Harvey, 2003). Individuals with EDs frequently report high levels of worry and rumination (Prefit et al., 2019; Startup et al., 2013) and elevated negative affect is a risk factor for EDs (Stice et al., 2017). Thus, it is possible that global and/or ED-specific repetitive negative thought in the pre-sleep period contributes to the development and maintenance of insomnia in those with EDs. It is also possible that affective disorders commonly comorbid with EDs such as depression and anxiety could mediate associations between ED symptoms and insomnia. Indeed, cross-sectional studies have found that anxiety and depression symptoms mediated the association between insomnia and binge eating (Kenny et al., 2018) and between insomnia and ED psychopathology (Goel et al., 2020).

Finally, prolonged starvation likely impacts sleep processes through the dysregulation of orexin receptors. Orexins are neuropeptides that are hypothesized to increase during starvation to promote wakefulness and food-searching behavior (e.g., Willie et al., 2001). Indeed, increased orexin-A has been linked to poorer sleep in people with and without anorexia nervosa (Sauchelli et al., 2016). One explanation is that in individuals without malnutrition, glucose and leptin appropriately suppress orexins; however, in the case of chronic starvation, when glucose and leptin levels are altered, orexin neuronal firing becomes dysregulated, resulting in increased wakefulness.

Dysregulated Sleep Processes Contribute to ED Behaviors

Sleep dysregulation can also alter eating behaviors, thereby exacerbating ED symptoms. For instance, acute sleep deprivation is associated with an increased tendency to crave (Greer et al., 2013) and consume (De Leon & Hanlon, 2020) high-calorie foods in non-clinical samples. Furthermore, chronic insomnia has been associated with dysregulated levels of leptin and ghrelin (e.g., Motivala et al., 2009; Spiegel et al., 2004; Taheri et al., 2004), which are neuropeptides that regulate hunger and satiety and influence food consumption. Shorter sleep duration has been associated with increased weight gain in non-clinical samples (Lyytikäinen et al., 2011; Patel & Hu, 2008), although this finding has yet to be replicated with a sample with EDs. However, it is possible that people who experience such weight gain will then attempt to use unhealthy weight control behaviors, entering into a cycle of ED psychopathology. Further, sleep-related maladaptive behaviors could also contribute to ED symptoms. For example, individuals with insomnia may be awake when most others would be asleep, and get out of bed to eat as a way to cope with their insomnia. Overall, mechanisms linking insomnia to specific ED behaviors remain primarily hypothetical and understudied at this time and more research in this area is needed. Effects of Insomnia on ED Treatment Response and Recovery

Treatment Response

Understanding insomnia symptoms in EDs is important because insomnia-related behaviors may influence treatment outcomes. First, treatments such as Enhanced Cognitive Behavior Therapy (CBT-E) require clients to maintain a schedule of regular eating (Fairburn, 2008); however, disruptions in sleep may hamper the ability to follow this schedule. For example, sleeping in or daytime napping may cause clients to miss necessary feeding times. Furthermore, insomnia is associated with both objective and subjective impairments in cognitive performance, particularly related to negative emotional tasks (Goldstein & Walker, 2014; Wardle-Pinkston et al., 2019). Thus, clients with insomnia symptoms may experience impairment that interferes with their ability to engage with treatment, particularly tasks that are more effortful or distressing. In the case of ED treatment, this could include exposures, regular eating, and ED behaviors abstention. Indeed, individuals with insomnia have poorer distress tolerance and increased negative affect during frustrating or distressing tasks (Short et al., 2016). Finally, insomnia symptoms are risk factors for common co-occurring diagnoses with EDs (Hertenstein et al., 2019), such as mood, anxiety, and substance-use disorders, which could complicate treatment outcomes. Research on the notion that insomnia may be a risk factor for EDs is sparse. However, one study found that disturbed sleep predicted higher ED severity among ED inpatients after six months of treatment. This effect was mediated through an indirect effect of increased depressive symptoms (Lombardo et al., 2015).

Relapse and Recovery

Insomnia symptoms may increase relapse rates for psychological disorders after successful treatment (e.g., Babson et al., 2013; Chen et al., 2017; Manber et al., 2008; Ohayon & Roth, 2003; Short, Mathes, et al., 2017). As of yet, it is unknown if residual insomnia symptoms pose the same risks to sustained ED recovery following treatment, which is a critical question given the elevated rates of relapse in EDs. As insomnia is a separate disorder with its own maintenance factors, although treating comorbid disorders may reduce insomnia symptoms, residual insomnia is the norm after treatment for depression, anxiety, and posttraumatic stress (Belleville et al., 2010). This is likely because although CBT may reduce cognitive processes associated with poorer sleep (e.g., rumination, worry, increased negative affect), it does not typically address maintaining behaviors (e.g., extended time in bed, poor sleep hygiene). This is problematic because research in anxiety and mood disorders has found that insomnia symptoms are a risk factor for relapse after treatment of these conditions (e.g., Chen et al., 2017; Manber et al., 2008; Ohayon & Roth, 2003). Furthermore, insomnia symptoms are associated with a return to maladaptive behaviors, such as cannabis use (Babson et al., 2013) and tobacco use (Short, Mathes, et al., 2017) following quit attempts. Consequently, it is important to establish if insomnia symptoms similarly effect abstinence from ED behaviors following treatment. Innovative Directions for Research

An initial question for ED and sleep research is to characterize how sleep and ED behaviors interact with each other. This could be accomplished by combining wearable devices that track sleep/wake and physical activity with ecological momentary assessment of ED behaviors and subjective perceptions of sleep. Ideally, this would include mechanistic

assessments of variables such as cognitive functioning, affect, and emotion dysregulation. This method could test questions such as if poor sleep is a risk factor for ED behaviors the next day or if binge eating at night results in dysregulated sleep. Furthermore, it could be used to evaluate if facets of sleep disturbance (e.g., insomnia, circadian dysfunction, inadequate sleep) are associated with specific ED symptoms. Several recent studies highlight such approaches to evaluating these questions. For example, one study utilized ecological momentary assessment along with actigraphy among youth with overweight/obesity, and found that longer sleep duration one night is associated with reduced consumption of solid fats, alcohol, and added sugar (Goldschmidt et al., 2020). Another study found that good sleep quality attenuated the impact of stress on unhealthy food consumption among Chinese workers (Liu et al., 2017), suggesting a more complex model of the impact of sleep on eating behaviors. Results from these types of analyses would allow researchers to identify potential points of intervention and could suggest timing of treatment interventions for sleep and eating behaviors.

At present, research examining actigraphy and sleep in EDs has primarily focused on characterizing the nature of sleep-wake cycles in small samples of people with BED (e.g., Linnaranta et al., 2020; Roveda et al., 2018; Tzischinsky et al., 2000; Tzischinsky & Latzer, 2006), with additional studies in AN (Latzer et al., 2001) and BN (Latzer et al., 1999). Although these methods have allowed for increased understanding of sleep-wake patterns, there is an urgent need to incorporate an assessment of ED behaviors to evaluate mechanisms of the association between sleep and eating behavior in EDs.

Experimental manipulations of sleep or ED behaviors within laboratory settings would also be valuable. For example, sleep deprivation, restriction, or extension paradigms could be used to examine whether sleep impacts engagement in ED behaviors, and whether this effect may be mediated by dysregulation of hormones related to appetite or satiety or other mechanisms (Irwin et al., 2016). Studies using partial sleep deprivation paradigms have found that partial sleep deprivation may decrease the consumption of nutritious foods (e.g., containing fiber) and increase daily snacks among young adults with and without BED symptoms (Cerolini et al., 2018). Furthermore, partial sleep deprivation increased food intake among healthy men (Hogenkamp et al., 2013) and good sleepers (Lombardo et al., 2020) highlighting the importance of assessing potential moderators of the relationship between sleep and eating behaviors. Another promising direction would be to examine the impact of experimental sleep deprivation on neural circuits related to ED behaviors. For example, Greer and colleagues (2013) found that sleep deprivation is associated with decreased activity in higher-order cortical brain regions and excess subcortical limbic reactivity, potentially resulting in the selection of foods high in weight gain potential. Alternatively, manipulations of ED behaviors (e.g., test meals) could be conducted prior to lab-based sleep studies to determine how such behaviors impact sleep and its architecture.

It is unknown how sleep problems influence treatment trajectories. First, it would be useful to establish if insomnia symptoms are associated with poorer treatment response (e.g., Lombardo et al., 2015). Second, there is limited knowledge about how ED treatments affect sleep behaviors. Although CBT tends to reduce insomnia severity over the course of treatment, a significant proportion of people do not experience full recovery from

insomnia symptoms (e.g., Cousineau et al., 2016; Mason & Harvey, 2014). At present, no evidence-based treatment for EDs incorporates core principles of behavioral interventions for insomnia, such as stimulus control and sleep restriction and titration. Evaluating the impact of existing ED treatments on insomnia will determine the necessity of incorporating insomnia-specific interventions into clinical practice. Finally, it will be important to establish if residual insomnia symptoms are risk factors for relapse for people treated for EDs. To answer these questions, researchers could longitudinally track insomnia symptoms during and following treatment discharge using measures such as the Insomnia Severity Index (Morin et al., 2011), which is a brief seven-item measure with an empirically established cutoff for probable insomnia disorder.

If insomnia symptoms are a risk factor for poorer treatment response or increased relapse rates, then research into the treatment of insomnia in people with EDs holds great promise. Treatment staging research could evaluate whether it is beneficial to treat insomnia symptoms prior to, concurrently with, or following ED treatment. Fortunately, there are evidence-based treatments for insomnia that are well validated, relatively brief, and easy to administer even by non-professionals. For example, CBT for Insomnia (CBT-I) demonstrates strong evidence for reducing insomnia symptoms and maintaining treatment gains (van der Zweerde et al., 2019; van Straten et al., 2018). Additionally, it is a highly flexible treatment option and can be delivered using individual, guided self-help, or group formats; through telehealth, online, or in-person; and in periods as short as four weeks. Consequently, it is a highly promising treatment for integration with existing ED care. Although there have yet to be any published trials of evidence-based treatment of insomnia disorder in people with eating pathology, a recently published protocol using a single-case experimental design to treat residual insomnia symptoms following treatment for an ED is a promising first step towards addressing questions about the efficacy of treating insomnia in this population (Christensen et al., n.d.). Finally, more research on the associations between insomnia and EDs could reveal novel treatment targets to improve treatment outcomes for both disorders.

Conclusion

Overall, although researchers have evaluated insomnia as a transdiagnostic risk and maintenance factor for psychopathology, it remains understudied in eating pathology. Innovative research is necessary to characterize the bidirectional association between sleep and eating behaviors and to identify the risks posed by dysregulated sleep on ED treatment outcomes and relapse rates. With this knowledge, ED treatment developers can learn how to best incorporate evidence-based interventions for insomnia and improve recovery from EDs.

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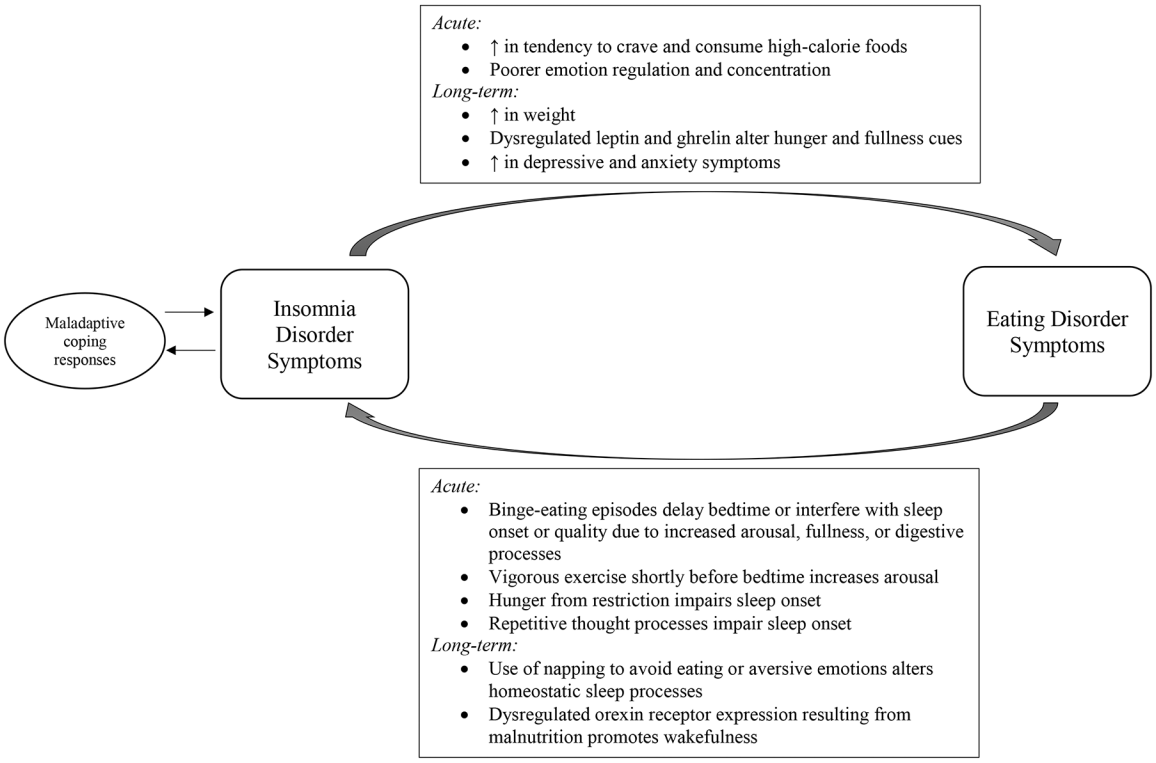

Figure 1: Model of Mechanisms Underlying Bidirectional Association between Insomnia and Eating Disorder Symptoms.

Acute symptoms of insomnia may exacerbate problematic eating behaviors and eating disorder behaviors may cause alterations in day-to-day sleep patterns. Over time, this positive feedback loop results in cognitive, physiological and behavioral changes that may further entrench individuals in eating disorders and result in insomnia disorder.