The Role of Sleep Disturbance in Anxiety-Related Symptoms

By

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CHAPTER I

Introduction

Despite the fact that approximately one third of the human life is spent sleeping, research has only recently begun to elucidate the nature of sleep as an important physiological process with physical, cognitive, and emotional benefits. Recent research suggests that sleep serves a restorative function, including neocortical maintenance and clearance of metabolic waste from the brain (Siegel, 2005; Xie et al., 2013). Likewise, longer self-reported sleep duration is linked to increased functional connectivity between the ventromedial prefrontal cortex and the amygdala (Killgore, 2013). Sleep is also implicated in cognitive processing, and a large body of research points to the beneficial impact of sleep on learning and memory (Diekelmann & Born, 2010; Inostroza & Born, 2013; Mander, Santhanam, Saletin, & Walker, 2011). These findings highlight the importance of sleep for neurocognitive function.

In conjunction with the evidence of the beneficial effects of good sleep, a much larger body of literature has examined the detrimental impact of poor sleep by delineating the negative downstream consequences for physiological, cognitive, and affective function. Numerous studies have highlighted the detrimental effects of sleep disturbance on physiological processes across multiple systems, including dysregulated endocrine function in stress (Omisade Buxton, & Rusak, 2010) and feeding (Spiegel, Tasali, Penev, & Van Cauter, 2004) systems, as well as impaired immune system function (Dinges, Douglas, Hamaraman, Zaugg, & Kapoor, 1995). Similarly, sleep disturbance is implicated in impaired cognitive function, including problems

with decision making (Harrison & Horne, 2000), memory (Chee & Choo, 2004; Goel, Rao, Durmer, & Dinges, 2009), attention (Horowitz, Cade, Wolfe, & Czeisler, 2003; Muto et al., 2012), and inhibition (Drummond, Paulus, & Tapert, 2006; Renn & Cote, 2013). Finally, sleep disturbance is linked to dsyregulated affective function, including increased subjective stress to low-stress tasks (Minkel et al., 2012) and problems with emotion regulation (Mauss, Troy, & LeBourgeois, 2013).

Further, considerable evidence has linked sleep disturbance to most forms of psychopathology (Benca, Obermeyer, Thisted, & Gillin, 1992; Roth et al., 2006). Sleep disturbance has been suggested as a potential transdiagnostic factor due to its links with multiple systems related to affective function, including the serotonin, dopamine, and circadian systems (Harvey, Murray, Chandler, & Soehner, 2011). While the majority of extant research has focused on the link between sleep disturbance and depression (Armitage, 2007), a growing body of research has implicated sleep disturbance in anxiety-related disorders (Cox & Olatunji, 2016), and recent research suggests that sleep disturbance predicts the development of an anxiety disorder (Batterham et al., 2012; Neckelman et al., 2007). Indeed, evidence of objective and subjective sleep disturbance has been found in multiple anxiety-related disorders, including generalized anxiety disorder (GAD; Alfano, Reynolds, Scott, Dahl, & Mellman, 2013; Berger et al., 2009), panic disorder (Hoge et al., 2011; Sloan et al., 1999), obsessive-compulsive disorder (Nota, Coles, & Sharkey, 2015), and posttraumatic stress disorder (Straus, Drummond, Nappi, Jenkins, & Norman, 2015; Calhoun et al., 2007).

Despite the links between sleep disturbance and anxiety-related disorders, as well as the evidence for the negative consequences of sleep loss across multiple systems, limited research has attempted to elucidate specific mechanisms by which sleep disturbance may confer risk for

the development of anxiety-related symptoms and disorders. Further, anxiety-related disorders and depression are highly comorbid (Kessler et al., 2003), and extant research is equivocal on whether the link between sleep disturbance and anxiety-related disorders is accounted for by depression. The purpose of the present study is to address these gaps in the literature by examining the unique link between sleep disturbance and anxiety-related symptoms and testing a potential mechanism by which sleep disturbance may result in increased anxiety-related symptoms. Study one examines the relationship between sleep disturbance and obsessive-compulsive symptoms in a nationally representative sample and tests whether depression accounts for this relationship. Study two examines whether decreased executive function accounts for the relationship between sleep disturbance and increased maladaptive repetitive thought, including worry and rumination.

CHAPTER II

Study 1: Sleep disturbance and obsessive-compulsive symptoms: Results from the National Comorbidity Survey Replication

1.1 Introduction

Obsessive-compulsive disorder (OCD) is a debilitating disorder characterized by obsessions, or repetitive intrusive, distressing thoughts, and compulsions, or repetitive behaviors performed in order to reduce the distress caused by the obsession (American Psychiatric Association, 2013). While lifetime prevalence estimates indicate that OCD is relatively rare (2.3%), results from a nationally representative sample indicate that approximately 28.2% of the population report experiencing obsessive-compulsive symptoms (OCS) in their lifetime (Ruscio et al., 2010). Due to the debilitating nature of the disorder, research efforts have increasingly focused on delineating processes that may maintain OCD. Although the majority of extant research examining sleep disturbance in anxiety disorders has focused on posttraumatic stress disorder (see Babson & Feldner, 2011 for a review), a small body of research suggests that sleep disturbance may also contribute to obsessive-compulsive disorder (OCD) (Nota et al., 2015; Paterson et al., 2013).

Although relatively few studies have objectively measured sleep in those with OCD, extant research indicates that individuals with OCD exhibit multiple disturbances in sleep compared to healthy controls, including decreased total sleep time (Alfano & Kim, 2011; Insel et al., 1982; Rapoport et al., 1981; Voderholzer et al., 2007), increased wake after sleep onset

(Alfano & Kim, 2011; Insel et al., 1982; Volderholzer et al., 2007), and decreased sleep efficiency (Hohagen et al., 1994; Rapoport et al., 1981; Voderholzer et al., 2007). Similarly, self-reported sleep disturbance has been linked to OCS in clinical and unselected samples. Among children with OCD, subjective sleep problems are associated with OCS severity (Storch et al., 2008), and sleep disturbance is linked to poor OCD treatment outcome (Ivarsson & Skarphedinsson, 2015). Likewise, among unselected samples, increased OCS are linked to delayed sleep timing (Nota & Coles, 2015) and subjective insomnia severity (Raines et al., 2015). Further, one recent study found that subjective insomnia severity is uniquely linked to obsessions, and this relationship is not accounted for by depression (Timpano et al., 2014).

The available evidence suggests that the presence of sleep disturbance may compound the severity and impairment of OCD (Ivarsson & Skarphedinsson, 2015; Robinson et al., 1998). However, no study to date has assessed this relationship in a nationally representative sample. Such a study is critical in order to assess whether the link between sleep disturbance and OCS is limited to specific subgroups (i.e., undergraduate students; individuals with OCD) or whether this relationship is present in the general population. Evidence for links between sleep disturbance and OCS in the general population may point to a unique role of sleep disturbance in the development of OCS in clinical and subclinical individuals. Such a role may provide evidence for the utility of incorporating sleep-related interventions into a more comprehensive approach to the treatment of OCD. The current study addresses this limitation in the extant research by assessing the link between subjective sleep disturbance and OCS in the National Comorbidity Survey Replication (NCS-R) and the role of depression in this relationship. It was hypothesized that individuals with sleep disturbance would report increased OCS compared to individuals without sleep disturbance. A careful review of the literature suggests that many of

the sleep disturbances associated with OCS are also characteristic of depression. Although some research suggests that the link between subjective sleep disturbance and symptoms of OCD is not accounted for by depression (Timpano et al., 2014), others have found that depression may explain this relationship (Diaz-Roman et al., 2015). Given the inconsistent findings in the literature, the present study also examined the extent to which sleep disturbance severity is associated with OCS severity when controlling for depression.

1.2. Materials and Methods

1.2.1 Sample

The NCS-R is a nationally representative survey of English-speaking adults (18 or older) residing in the United States who participated in in-person interviews in their homes between February 2001 and April 2003 (see Kessler et al., 2004 for a detailed description of survey procedure). All survey participants completed Part I of the survey, which assessed for core psychological disorders (n = 9282), and a subset of participants completed Part II, which assessed for additional disorders and clinical correlates, including insomnia (n = 5692). Of the Part II participants, a random subsample (n = 2073) were administered the module assessing OCD. Of the participants who were administered the OCD module, 2 refused to respond, which yielded a final sample of n = 2071 participants included in analysis. Informed consent was obtained from all participants.

The sample was 50.2% female with a mean age of 45.22 years (SE = .57) ranging from 18 to 94 years. The racial composition of the sample was as follows: Black (12.5%), Hispanic (11.4%), White (72.1%), Other (4.0%). The majority of the participants were married (56.2%), while 19.9% were separated, widowed, or divorced, and 23.8% were never married. The years of

education obtained by the sample were as follows: 0-11 years (17.1%), 12 years (32.3%), 13-15 years (26.2%), more than 16 years (24.4%).

1.2.2 Measures

OCS. OCD was assessed with the World Health Organization Composite International Diagnostic Interview 3.0 (CIDI 3.0; Kessler & Ustun, 2004). Due to a skip logic error discovered following survey administration (Ruscio et al., 2010), only the first 9 items of the OCD module were examined in the present study. Participants completed 9 yes/no items regarding 9 subtypes of OCD (contamination, checking, ordering, hoarding, sexual/religious, moral, harming, illness, other) in response to the following prompt: "Did you ever have a period in your life lasting two weeks or longer when most days you experienced any of the following unpleasant thoughts, images, or impulses, or repeated behaviors that you felt compelled to do?" An OCS severity score was created by summing the responses on these 9 items.

Sleep Disturbance. Sleep disturbance was assessed in the Chronic Conditions section of the CIDI 3.0 with 4 yes/no items each measuring problems with sleep initiation, sleep maintenance, early morning awakening, and daytime sleepiness in response to the following prompt: "Did you have a period lasting two weeks or longer in the past 12 months when you had any of the following problems with your sleep?" Participants endorsing one or more sleep problem were coded as presenting with sleep disturbance, while participants endorsing no sleep problem were coded as having no sleep disturbance. Further, a sleep disturbance severity score (0-4) was created by summing the responses on these 4 items.

Depression. Depression was assessed with the CIDI 3.0 (Kessler & Ustun, 2004). In the present study, depression was defined as experiencing a major depressive episode in the past 12 months.

1.2.3 Data Analytic Strategy

All analyses were conducted in SPSS 20.0 using the Part II sample weighting. First, an independent samples t-test was conducted to assess whether individuals who report sleep disturbance report increased OCS severity compared to individuals who do not report sleep disturbance. Cohen's *d* was calculated using G*Power 3.1 (Faul et al., 2009). Second, an analysis of covariance (ANCOVA) was conducted to assess whether sleep disturbance severity is associated with OCS severity when controlling for depression. Depression was included in the model because depression is often comorbid with OCD (Ruscio et al., 2010) and sleep disturbance (Soehner et al., 2014). Cohen's *f* was calculated using G*Power 3.1 (Faul et al., 2009).

1.3. Results

1.3.1 Associations Between Study Variables and Frequency of Relevant Disorders

As shown in Table 1.1, sleep disturbance severity and major depressive episode in the past 12 months were significantly associated with presence of any lifetime OCS, while presence of sleep disturbance was not significantly associated with presence of lifetime OCS. Further, 42.5% of participants reported the presence of sleep disturbance in the past 12 months, 28.2% reported having experienced OCS in their lifetime, and 7.7% met criteria for a major depressive episode in the past 12 months. Presence of additional anxiety disorders and substance use disorders in the past 12 months are shown in Table 1.2.

1.3.2 OCS Severity in Participants With and Without Sleep Disturbance

Results of a t-test revealed that participants who reported sleep disturbance reported significantly increased OCS severity ($M = 0.82$, $SE = 0.06$) compared to participants who reported no sleep disturbance ($M = 0.31$, $SE = 0.03$), $t(42) = 8.73$, $d = 10.93$ $p < 0.001$.

1.3.3 Association Between Sleep Disturbance and OCS Severity

As shown in Table 1.3, results of an ANCOVA revealed that sleep disturbance severity was significantly associated with OCS severity when controlling for depression, F (1, 42) = 47.80, $p < 0.001$. The total model was significant, F (2, 41) = 70.71, $f = .36$, $p < 0.001$, and depression and sleep disturbance severity were significantly associated with OCS severity (see Table 2).

Additional ANCOVA models were analyzed to test the association between sleep disturbance severity and OCS severity when controlling for other anxiety-related disorders. In all models, sleep disturbance severity remained significantly associated with OCS severity (see Table 1.4).

1.4. Discussion

The present study examined the relationship between sleep disturbance and OCS in a nationally representative sample. The results of this study indicate that individuals with sleep disturbance report increased OCS compared to individuals without sleep disturbance. This finding is consistent with previous research linking sleep disturbance with increased symptoms of psychopathology (Tkachenko et al., 2014), including OCS (Raines et al., 2015). Importantly, these findings suggest that the link between sleep disturbance and OCS is not limited to clinical-

level insomnia, but rather that even minor sleep disturbance may increase the likelihood of experiencing OCS in a diverse and representative sample.

The present study also found that sleep disturbance severity is associated with OCS severity, even when controlling for depression. This result is consistent with extant research linking insomnia symptoms to OCS when controlling for depression (Timpano et al., 2014) and extends this research by replicating this result in a nationally representative sample. Symptoms of depression and OCD often co-occur. However, the results of the present study indicate that increased sleep disturbance severity is linked to increased levels of OCS, and this relationship is not accounted for by co-occurring depression. The present study also extends previous research in showing that the link between increased sleep disturbance and increased levels of OCS is also not accounted for by co-occurring anxiety disorder diagnoses. This finding reinforces the notion that the link between sleep disturbance and OCS is unlikely to be a mere artifact of general negative affect.

Although these findings indicate a potentially unique link between sleep disturbance and the severity of OCS, the specific mechanism(s) that may account for this relationship is unclear. It may be the case that the adverse downstream effects of sleep loss, such as impaired executive function (Harrison & Horne, 2000), dysregulated diurnal cortisol (Wright et al., 2015), and deficits in systems related to emotional function (Harvey et al., 2011), may increase the likelihood of experiencing symptoms of OCD. Given the cross-sectional nature of the present study, an alternative interpretation may be that repetitive intrusive and distressing thoughts contribute to sleep disturbance by interfering with the initiation and maintenance of sleep. Indeed, extant research indicating high comorbidity between Delayed Phased Sleep Disorder (DPSD) and OCD suggests that sleep disturbance in OCD may be a function of delayed sleep

onset (Lange et al., 2012; Turner et al., 2007). Future research is necessary to elucidate mechanisms that may explain why sleep disturbance is linked to OCS.

The results of this study indicate that sleep disturbance is related to OCS severity, and this finding suggests that addressing sleep disturbance may be useful in the treatment of OCD. Indeed, recent research indicates that persistent sleep problems following OCD treatment are associated with worse treatment outcome (Ivarsson & Skarphedinsson, 2015). However, the assessment of OCS in the present study should not be confused with the assessment of OCD per se. Although the representative sample consisted of individuals with a diagnosis of OCD, the majority of the participants were in the nonclinical range. Despite this study limitation, it is important to note that taxometric research has shown that OCS are present to a greater or lesser extent in all individuals (Olatunji et al., 2008). Accordingly, the present findings in a nationally representative population are likely to be generalizable to understanding the etiology, maintenance, and treatment of OCD.

The results of this study are the first to find that individuals with sleep disturbance report increased OCS and that sleep disturbance severity is linked to OCS severity, even when controlling for depression, in a nationally representative population. However, these findings must be considered within the context of additional study limitations. First, sleep disturbance was measured subjectively in this study. As the majority of previous research indicating sleep disturbance in OCD utilized objective sleep measurement, future research is necessary to replicate these findings with objective assessments of sleep disturbance. Similarly, the assessment of OCS in the present study was not comprehensive. Additional research utilizing a more thorough assessment of OCS severity is necessary to replicate these findings. Furthermore, the cross-sectional and correlational nature of the present study limits the ability to make

inferences about direction or causality. Future research employing longitudinal designs and experimental manipulation of sleep is necessary to compliment the present findings. The present results offer evidence of a unique link between sleep disturbance and anxiety-related symptoms in a nationally representative sample. However, these findings do not speak to potential mechanisms that may account for this relationship. To address this limitation, study two was conducted to test executive function as a candidate mechanism in the relationship between sleep disturbance and anxiety-related symptoms.

CHAPTER III

Study Two: Linking sleep disturbance and maladaptive repetitive thought: The role of executive function

2.1 Introduction

Sleep disturbance is consistently linked to impaired emotional function, and sleep restriction and poor sleep quality are implicated in emotion regulation deficits in healthy adults and adolescents (Baum et al., 2014; Mauss, Troy, & LeBourgeois, 2013). Maladaptive repetitive thought, or excessively perseverating on one's negative experiences and emotions (Ehring & Watkins, 2008), is an unconstructive emotion regulation strategy linked to multiple forms of psychopathology (Aldao & Nolen-Hoeksema, 2010), and recent research has found that maladaptive repetitive thought is also associated with sleep disturbance (Nota & Coles, 2014; Pillai & Drake, 2015). More specifically, worry, or repetitive thought about negative future events (Borkovec, Ray, & Stober, 1998), has been linked to sleep problems in healthy and clinical populations (Kertz & Woodruff-Borden, 2011; O'Kearney & Pech, 2014). In healthy adolescents, sleep problems predict catastrophic worry one year later (Danielsson, Harvey, MacDonald, Jansson-Frojmark, & Linton, 2013), and sleep deprivation increases the perceived threat level of worries (Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Further, a recent study of sleep and worry in individuals with generalized anxiety disorder (GAD) found that sleep quality predicted subsequent worry frequency and duration, and the frequency of evening worry predicted subsequent sleep quality (Thielsch et al., 2015).

Although the majority of extant research on sleep disturbance and repetitive thought has been focused on worry (Pillai & Drake, 2015), rumination, or engaging in negative thoughts related to perceived threats to the self or distressing experiences (Trapnell & Campbell, 1999), is also linked to poor sleep in healthy and clinical populations (Borders, Rothman, & McAndrew, 2014; Slavish & Graham-Engeland, 2015). In healthy individuals, poor sleep quality is associated with rumination after controlling for negative affect (Thomsen, Mehlsen, Christensen, & Zachariae, 2003), and rumination following a stressful event predicts increased sleep onset latency (Zoccola, Dickerson, & Lam, 2009) and decreased sleep quality (Guastella & Moulds, 2007). Similar patterns are found in clinical populations; for example, recent research indicates that sleep and rumination contribute to posttraumatic stress disorder (PTSD)- in veterans, sleep problems mediate the relationship between rumination and symptoms of PTSD (Borders et al., 2014). Taken together, these findings indicate a link between sleep and maladaptive repetitive thought, such as worry and rumination, in healthy and clinical populations, including those with anxiety-related disorders.

Despite increasing evidence supporting a relationship between sleep and maladaptive repetitive thought, the mechanism that may explain this link is unclear. One candidate mechanism is executive function. Executive function, which encompasses higher-order cognitive processes, such as memory, attention, and inhibition, that activate, integrate, and manage downstream functions, may be particularly sensitive to the effects of sleep loss (Jones & Harrison, 2001; Nilsson et al., 2005). For example, sleep deprivation leads to impairment in the consolidation of episodic (Goel, Rao, Durmer, & Dinges, 2009) and working memory (Chee & Choo, 2004). Sleep deprivation also diminishes attention (Muto et al., 2012), including divided (Drummond, Gillin, & Brown, 2001) and selective attention (Horowitz, Cade, Wolfe, &

Czeisler, 2003). Likewise, fMRI studies indicate an increased recruitment of cognitive resources to accomplish attention tasks following sleep deprivation (Drummond et al., 2001). Sleep disturbance is also linked to inhibitory deficits- response inhibition, or the ability to withhold an automatic response, is diminished following sleep deprivation (Drummond, Paulus, & Tapert, 2006), and sleep restriction leads to decreased inhibitory control of risk-taking behavior (Rossa, Smith, Allan, & Sullivan, 2014).

The relationship between sleep disturbance and impaired executive function is well-established, and the effects of sleep loss on executive function may have negative downstream consequences for emotional processes. For example, one night of sleep deprivation results in increased amygdala reactivity to negative stimuli and decreased functional connectivity between the amygdala and the medial-prefrontal cortex (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). This finding suggests that sleep loss impairs frontal lobe capabilities and may diminish top-down executive control of the amygdala. In turn, the unregulated amygdala shows heightened responding, which may express as dysregulated emotion and the repetitive thinking that is characteristic of many emotional disorders. Indeed, decreased executive function is linked to maladaptive repetitive thought (Segerstrom, Roach, Evans, Schipper, & Darville, 2010), and specific dimensions of executive function are linked to worry and rumination. For example, induced attentional bias to threat stimuli predicts subsequent engagement in worry (Hirsch et al., 2011), and high levels of worry are associated with inefficient filtering of threat-related memories (Stout, Shackman, Johnson, & Larson, 2015). Similarly, rumination is linked to deficits in working memory in healthy individuals and decreased ability to inhibit irrelevant information in those with depression (Zetsche, D'Avanzato, & Joormann, 2012), and cognitive inhibition has been proposed as a key impairment underlying engagement in rumination

(Joorman, 2010). These findings suggest that deficits in executive function may diminish cognitive control over emotional processes, which may lead to increased engagement in worry and rumination.

Although the respective relationships between sleep, executive function, and the maladaptive repetitive thought that characterizes many anxiety-related disorders is well-established, no study to date has assessed the role of executive function in the relationship between sleep disturbance and maladaptive repetitive thought. Accordingly, the present study employed structural equation modeling (SEM) to examine executive function as a potential intervening variable in the relationship between sleep problems and maladaptive repetitive thought, specifically worry and rumination. It was predicted that increased sleep disturbance would be associated with increased levels of maladaptive repetitive thought, and this relationship would be accounted for by increased impairments in executive function. However, to enhance specificity, it is critical to control for as many relevant confounds as is practical. Such relevant confounds include nonspecific third variables that may be related to sleep disturbance and maladaptive repetitive thought. Rather than executive function specifically, an alternative hypothesis posits that sleep disturbance is linked to worry and rumination due to the non-specific association between sleep disturbance and general distress. Indeed, aspects of distress, such as depression, stress, and distress tolerance, are broadly associated with sleep disturbance and maladaptive repetitive thought (Buysse et al., 2008; Minkel et al., 2014; Huang, Szabo, & Han, 2009; Morrison & O'Connor, 2005). Accordingly, general distress was included as a rival factor in the model to assess whether executive function accounted for the relationship between sleep disturbance and maladaptive repetitive thought above and beyond the effect of general distress.

2.2 Method

2.2.1 Participants

A total of 341 participants (81.5% female) comprised the present sample. The mean age of the participants was 33.56 years (*SD* = 13.64), ranging from 18 to 66 years. The ethnicity composition was as follows: African American (n = 26; 7.6%), Asian (n = 23; 6.8%), Caucasian (n = 271; 79.7%), Hispanic/Latino (n = 15; 4.4%), Other (n = 5; 1.5%).

2.2.2 Procedure

Recruitment for the study included utilization of ResearchMatch, a national health volunteer registry that was created by several academic institutions and supported by the U.S. National Institutes of Health as part of the Clinical Translational Science Award (CTSA) program. ResearchMatch has a large population of volunteers who have consented to be contacted by researchers about health studies for which they may be eligible. Healthy (no reported conditions) ResearchMatch volunteers who lived within 15 miles of Vanderbilt University, or in 11 randomly selected states (Arkansas, Connecticut, Idaho, Illinois, Kansas, New Hampshire, New Jersey, North Carolina, North Dakota, Oklahoma, or South Dakota) were contacted with information on how to participate. Vanderbilt University undergraduates in psychology courses were also recruited and received course credit for participating. Participants received a link to complete an online battery of questionnaires and were informed that they would have the opportunity to enter their name into a drawing for a $100 gift card. Previous research suggests that online administration of self-report scales has several advantages over paper and pencil methods, including reduced burden on the participant (Buchanan & Smith, 1999), and studies comparing online to paper and pencil administration indicate that these

methods are typically psychometrically comparable (Coles, Cook, & Blake, 2007; Zlomke, 2009). Study data were collected and managed using REDCap (Research Electronic Data Capture) hosted at Vanderbilt University (Harris et al., 2009). REDCap is a secure, web-based application designed to support data capture for research studies and is supported by UL1 TR000445 from NCATS/NIH. Review and approval for this study and all procedures was obtained from the Vanderbilt University Institutional Review Board, and informed consent was obtained from all individual participants included in the study.

2.2.3 Measures

Attentional Control Scale (ACS; Derryberry & Reed, 2002). The ACS is a 20-item self-report measure of the ability to control attention and consists of two subscales: shifting, or the ability to disengage attention from one task and reallocate it to a new task, and focusing, or the ability to maintain attention on a particular task, despite potential distractors. Items on the ACS are rated on a Likert scale from 1 (*almost never*) to 4 (*always*), and higher scores indicate better attentional control. In the present sample, the ACS Focusing and Shifting subscales had adequate internal consistency ($\alpha = .83$ and $\alpha = .80$, respectively).

Depression, Anxiety, and Stress Scales- Short Form (DASS; Lovibond & Lovibond, 1995). The DASS is a 21-item self-report measure of symptoms of depression, anxiety, and stress over the past week. Only the Depression[1] and Stress subscales were used in the present study (13 items). Items on the DASS are rated on a Likert scale from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*), and higher scores indicate higher degrees

[1] Due to an error in survey development, data was not obtained for one item of the DASS Depression subscale (item 2, "I felt that I had nothing to look forward to"). The Depression subscale was retained given that the remaining items were depression-relevant and evidenced adequate internal consistency.

of stress and depression. In the present sample, the Depression and Stress subscales had adequate internal consistency (α = .89 and α = .89, respectively).

Distress Tolerance Scale (DTS; Simons & Gaher, 2005). The DTS is a 15-item self-report measure of the ability to experience and tolerate negative emotions. Items on the DTS are rated on a Likert scale from 1 (*strongly agree*) to 5 (*strongly disagree*), and higher scores indicate a higher tolerance for distressing emotions and experiences. The DTS had good internal consistency in the present sample (α = .94).

Insomnia Severity Index (ISI; Bastien, Vallieres, & Morin, 2001). The ISI is a 7-item self-report measure of insomnia symptoms over the past two weeks and is used to detect cases of insomnia and assess treatment response. Items on the ISI are rated on a Likert scale from 1 (*none*) to 4 (*very severe*), and higher scores indicate a higher severity of insomnia. The ISI had adequate internal consistency in the present sample (α = .88). Although sleep quality can be a difficult process to self-report (Plessow, Kiesel, Petzold, & Kirschbaum, 2011), the assessment of subjective perception of sleep is important, as subjective sleep complaints are linked to dsyregulated emotional processes (Takano, Iijima, & Tanno, 2012) and are critical for diagnosing insomnia (Buysse, 2008).

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is a 19-item self-report measure of sleep quality and sleep disturbance over the past month. Items include reporting sleep variables such as typical number of hours slept each night and typical bedtime, as well as rating sleep problems on a Likert scale from 0 (*not during the past month*) to 3 (*three or more times a week*). The PSQI has 7 component scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep

disturbances, use of sleep medication, and daytime dysfunction), and higher scores indicate more severe sleep problems. A 3-factor model (Perceived Sleep Quality, Sleep Efficiency, and Daily Disturbances) can be created by adding the subjective sleep quality, sleep latency, and sleeping medication use components to create a Perceived Sleep Quality factor, the sleep duration and sleep efficiency components to create a Sleep Efficiency factor, and the sleep disturbances and daytime dysfunction components to create a Daily Disturbances factor (Cole et al., 2006). The PSQI had low internal consistency in the present sample ($\alpha = .54$).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item self-report measure of one's tendency to worry. Items are rated on a Likert scale from 1 (*not at all typical of me*) to 5 (*very typical of me*), and higher scores indicate an increased tendency to worry. The PSWQ had good internal consistency in the present sample ($\alpha = .95$).

Rumination-Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999). The RRQ is a 24-item self-report measure of self-attentiveness used to distinguish rumination from reflection. Only the 12-item rumination subscale was used in the present study. Items are rated on a Likert scale from 1 (*strongly disagree*) to 5 (*strongly agree*), and higher scores indicate an increased tendency to ruminate. The RRQ rumination subscale had good internal consistency in the present sample ($\alpha = .94$).

Webexec (WEX; Buchanan et al., 2010). The WEX is a 6-item self-report measure of problems related to broad executive function, including inhibition, attention, and task coordination, specifically designed for online administration. Items are rated on a Likert scale from 1 (*no problems experienced*) to 4 (*a great many problems experienced*), and higher scores

indicate more difficulties with executive function. The WEX had adequate internal consistency in the present sample (α = .87).

2.2.4 Data Analytic Approach

Structural equation modeling (SEM) was employed using Mplus version 7.11 (Muthén & Muthén, 2010) to examine the relationship between sleep disturbance, executive function, maladaptive repetitive thought, and distress. Prior to examining the structural models, we used confirmatory factor analysis (CFA) to identify a well-fitting measurement model. The four latent factors of interest were specified with the following observed indicators: (1) Sleep Disturbance: the ISI total score, the PSQI perceived sleep quality factor score, and the PSQI daily disturbances factor score; (2) Executive Function: the ACS focusing subscale score, the ACS shifting subscale score, and the WEX total score; (3) Maladaptive Repetitive Thought: the PSWQ total score and the RRQ rumination subscale score; and (4) Distress: the DASS depression subscale score, the DASS stress subscale score, and the DTS total score. The variance of each factor was fixed to 1.00 to provide a metric for these latent constructs (Byrne, 1989).

The Robust Maximum Likelihood (MLR; Yuan, & Bentler, 2000) estimator was used for all analyses. The MLR estimator uses a pseudomaximum likelihood (PML) asymptotic covariance matrix, which has been found to eliminate biases in parameter estimate in the event of unequal probability sampling (Asparouhov, 2005). Model fit was examined via the following fit indices: Comparative Fit Index (CFI), Tucker-Lewis index (TLI; Tucker & Lewis, 1973), Standardized Root Mean Square Residual (SRMR), and the Root Mean Square Error of Approximation (RMSEA). Cut-offs for good fit included the following: CFI and TLI > .95 (Hu & Bentler, 1999), RMSEA < .08 (Browne & Cudeck, 1993) and SRMR < .08 (Hu & Bentler,

1999). Significance of the SEM parameters was tested by converting their unstandardized parameter estimate into a z-statistic (by dividing it by their respective standard errors); *z* values were then considered significant (at the $p < .01$ level) if greater than 2.58.

To examine whether Executive Function and Distress accounts for the association between Sleep Disturbance and Maladaptive Repetitive Thought, we used a combination of tests as recommended by Mackinnon and colleagues (2002). First, we examined the Baron and Kenny (1986) approach which requires four conditions to be met to support the presence of significant intervening variables: (1) the independent variable (i.e., Sleep Disturbance) is significantly associated with the outcome variable (i.e., Maladaptive Repetitive Thought); (2) the independent variable (i.e., Sleep Disturbance) is significantly associated with the proposed intervening variables (i.e., Executive Function and Distress); (3) the proposed intervening variables are significantly associated with the dependent variable (i.e., Maladaptive Repetitive Thought) while controlling for the independent variable (i.e., Sleep Disturbance); and (4) the significant relationship between the independent variable and the dependent variable becomes non-significant once the proposed intervening variables are included in the model. Second, we examined the significance of the indirect pathways through both intervening variables, simultaneously. We also compared the following models (using the Chi-Square Difference Test for the Satorra-Bentler Scaled Chi-Square; Satorra, & Bentler, 1994): (a) the model whereby the direct effect (c') path was constrained to zero and (b) the model whereby the direct effect (c') path was freely estimated. If chi-square does *not* significantly degrade when testing the constrained model relative to the freely estimated model, then this would support the notion that the intervening variables fully account for the association between Sleep Disturbance and

Maladaptive Repetitive Thought (i.e., that the direct path drops to 0 upon including the intervening variables in the model).

2.3 Results

2.3.1 Measurement Model

Table 2.1 contains the descriptive statistics (means and standard deviations) and correlations for all observed indicators of the four latent factors. The measurement model (relating the four latent factors Sleep Disturbance, Executive Function, Distress, Maladaptive Repetitive Thought) may be seen in Figure 2.1. Results revealed that all factor loadings were significant ($p < .05$), and all correlations between factors were significant ($p < .01$), with the exception of the correlation between ACS Shifting and PSQI Perceived Sleep Disturbance. The measurement model also demonstrated an adequate fit to the data [χ^2 (38) = 119.76, $p < .01$; CFI = .955; TLI = .934; RMSEA = .079; SRMR = .055].

2.3.2 Structural Model

The Baron and Kenny (1986) approach supported the hypothesis that Executive Function and Distress account for the relationship between Sleep Disturbance and Maladaptive Repetitive Thought (see Figure 2.2). First, Sleep Disturbance significantly predicted Maladaptive Repetitive Thought ($\zeta = .50$, SE = .060, $z = 8.41$, $p < .01$); second, the independent variable (i.e., Sleep Disturbance) significantly predicted the Executive Function intervening variable ($\zeta = -.34$, SE = .072, $z = -4.68$, $p < .01$) and Distress intervening variable ($\zeta = .63$, SE = .057, $z = 10.97$, $p < .01$); third, both the proposed intervening variables significantly predicted the dependent variable (i.e., Maladaptive Repetitive Thought) while controlling for the independent variable (i.e., Sleep Disturbance); specifically, while Sleep Disturbance was included in the model, the Executive

Function intervening variable significantly predicted Maladaptive Repetitive Thought (ζ = -.36, SE = .069, z = -5.24, p < .01), and the Distress intervening variable significantly predicted Maladaptive Repetitive Thought (ζ = .79, SE=.075, z = 10.53, p < .01); lastly, (4) the significant relationship between the independent variable and the dependent variable (ζ = .50, SE = .060, z = 8.41, p < .01) became non-significant once the proposed intervening variables were included in the model (ζ = -.11, SE = .067, z = -1.67, *ns*).

Second, we examined the significance of the indirect (intervening) pathways through the two proposed intervening variables and both pathways were significant (see Figure 2). Specifically, the indirect pathway through the Executive Function intervening variable was significant (ζ = .12, SE = .037, z = 3.33, p < .01). Similarly, the indirect pathway through the Distress intervening variable was also significant (ζ = .49, SE = .071, z = 7.00, p < .01).

We then conducted the chi-square difference test between the constrained model (whereby the direct path was constrained to 0) and the freed model (whereby the direct path was freely estimated). The Scaled Difference of the Satorra-Bentler Scaled Chi-Square between the constrained model [χ2(40) = 196.45, scaling factor = 1.0726] and the freed model [χ 2(39) = 193.48, scaling factor = 1.0734] was 2.85, *ns*. The non-significance of this difference supports the notion that the intervening variables fully account for the association between Sleep Disturbance and Maladaptive Repetitive Thought.

Together, the causal link test, the test of indirect effects via the mediators, and the Scaled Difference of the Satorra-Bentler Scaled Chi-Square test supported the proposition that Executive Function and Distress fully account for the relationship between Sleep Disturbance and Maladaptive Repetitive Thought.

2.3.3 Gender Differences of the Intervening Effect.

To test for gender differences in the extent to which intervening variables fully account for the association between Sleep Disturbance and Maladaptive Repetitive Thought, we re-conducted all analyses based on the female-only subsample (n = 278) and the male-only subsample (n = 63). Results were identical in the female-only and male-only subsamples, including both intervening variables being associated with significant indirect effects in both subsamples.

Further, in the male-only sample, the measurement model fit well (i.e., CFI = .961, TLI = .943, SRMR = .057, RMSEA = .078), and the Scaled Difference of the Satorra-Bentler Scaled Chi-Square between the constrained model [χ2(40) = 76.12, scaling factor = 0.98] and the freed model [χ 2(39) = 75.71, scaling factor = 0.98] was 0.42, ns. Similarly, in the female-only sample, the measurement model fit well (i.e., CFI = .953, TLI = .932, SRMR = .061, RMSEA = .08), and the Scaled Difference of the Satorra-Bentler Scaled Chi-Square between the constrained model [χ2(40) = 163.88, scaling factor = 1.0774] and the freed model [χ 2(39) = 160.56, scaling factor = 1.0753] was 2.86, ns. These results thus support the notion that Executive Function and Distress fully account for the association between Sleep Disturbance and Maladaptive Repetitive Thought in both males and females.

2.4 Discussion

The present study employed SEM to examine the relationship between sleep disturbance, executive function, and maladaptive repetitive thought in a healthy sample. Examination of the measurement model revealed significant associations between a latent sleep disturbance variable which consisted of indicators of insomnia severity, perceived sleep quality, and daily

disturbances related to sleep and a latent maladaptive repetitive thought variable that consisted of worry and rumination. This finding is consistent with extant research linking worry and rumination to disrupted sleep in nonclinical populations (Pillai, Steenburg, Ciesla, Roth, & Drake, 2014; Takano et al., 2012). Likewise, this finding is consistent with extant research on anxiety-related disorders, as sleep problems are often observed in disorders characterized by maladaptive repetitive thought, such as PTSD and GAD (Marcks, Weisberg, Edelen, & Keller, 2010). In conjunction with previous research, the results of this study indicate a role of disturbed sleep in maladaptive repetitive thought, such as worry and rumination.

The present study also revealed a significant relationship between the latent sleep disturbance variable and a latent executive function variable that consisted of attentional control of focusing and shifting and general problems with broad executive function, including attention, inhibition, and planning. These results are also consistent with existing research linking sleep loss to impaired executive function (Van Dongen, Maislin, Mullington, & Dinges, 2003). Recent research has found that sleep deprivation decreases activation of and functional connectivity in the prefrontal cortex, which may explain the specificity of cognitive deficits in higher-order processes, such as inhibition and attention, following sleep loss (Ma, Dinges, Basner, & Rao, 2015; Verweij et al., 2014). Furthermore, the latent executive function variable was also associated with the latent maladaptive repetitive thought variable. This finding compliments prior research that links deficits in executive function to maladaptive repetitive thought (Davis & Nolen-Hoeksema, 2000; Segerstrom et al., 2010) and suggests that decreased executive function may diminish the ability to inhibit maladaptive repetitive thought, such as worry and rumination.

Although disrupted sleep has been linked to excessive worry and rumination (Pillai et al., 2014; Takano et al., 2012), the mechanism that may account for this association remains unclear.

Drawing from the available literature, it was hypothesized that deficits in executive function may account for the association between sleep disturbance and excessive worry and rumination. Consistent with this hypothesis, the structural model in the present study indicated that the latent executive function variable partially accounted for the association between the latent sleep disturbance variable and the latent maladaptive repetitive thought variable. In conjunction with extant research, these results suggest that sleep disturbance may impair executive function, which may then lead to increased engagement in maladaptive repetitive thought. It may be the case that the decreased activation of and functional connectivity within brain regions associated with executive function observed following sleep loss (Ma et al., 2014, Verweij et al., 2014) may contribute to a diminished ability to inhibit engagement in maladaptive repetitive thought. More research is necessary to determine the temporal sequence and neural underpinnings of these processes, and future research should employ behavioral methods to further delineate the pathway between disturbed sleep and maladaptive repetitive thought.

To assess the specificity of executive function in the relationship between sleep disturbance and maladaptive repetitive thought, it is critical to control for as many relevant confounds as is practical. Such confounds should include nonspecific third variables that may be related to sleep disturbance and maladaptive repetitive thought (Smits, Julian, Rosenfield, & Powers, 2012). Accordingly, the present study included a latent distress variable that consisted of depression, stress, and distress tolerance as a rival nonspecific mediator to executive function, as both executive function and distress are linked to maladaptive repetitive thought (e.g., Allan, Macatee, Norr, & Schmidt, 2014; Olatunji, Broman-Fulks, Bergman, Green, & Zlomke, 2010; Segerstrom, Tsao, Alden, & Craske, 2000). Including distress in the structural model allowed for the determination of the extent to which executive function was related to maladaptive repetitive

thought over and above its relation with general distress. In the model predicting maladaptive repetitive thought, executive function contributed to the indirect effect above and beyond general distress. The finding that the relationship between sleep problems and maladaptive repetitive thought is partially accounted for by executive function extends previous research and provides preliminary evidence to suggest that this link is not due to nonspecific distress alone.

The present study builds on previous research linking sleep disturbance to worry and rumination by highlighting a potential mechanism that may explain *why* sleep problems are related to maladaptive repetitive thought. More specifically, the present study highlights deficits in executive function as a potential mechanism by which sleep disturbance may confer risk for increased engagement in maladaptive repetitive thought. However, inferences based on these findings must consider the study limitations. First, the use of self-reported healthy participants limits the ability to generalize these findings to clinical populations. Further, sleep and executive function are two processes that can be difficult to self-report, particularly when a deficit is present (Plessow et al., 2011; Vanable, Aikens, Tadimeti, Caruana-Montaldo, & Mendelson, 2000); therefore, future research employing objective and/or behavioral measures is necessary to provide further support for the present results. Similarly, the relatively low internal consistency of the PSQI in the present sample prevents definitive inferences from being drawn from the present findings. The low reliability may be partly due to the diversity of the sample, as well as the inherent difficulty of retrospectively self-reporting sleep (Baker, Maloney, & Driver, 1999). Future research along these lines may benefit from utilizing ecological momentary assessment, as this method may enhance the reliability of sleep assessment. Finally, the correlational nature of the data limits the ability to make any causal inferences. It has been well-documented that estimates of inherently longitudinal mediational effects (sleep disturbance → executive function

→ repetitive thoughts) based on cross-sectional data are often incomplete (Cole & Maxwell, 2003); therefore, the sequential relationship in this model remains unclear. However, the present results provide preliminary evidence for the role of executive function as a candidate mechanism that may link sleep disturbance and maladaptive repetitive thought and suggest that future research incorporating longitudinal assessment and experimental manipulations of the study variables is needed to compliment this study.

Despite these limitations, the present study may have implications for anxiety-related disorders. The present findings indicate that the negative downstream consequences of perceived sleep disturbance on maladaptive repetitive thought may be partly due to disrupted executive function rather than general distress alone. Based on this finding, addressing subjective sleep disturbance may aid in the treatment of maladaptive repetitive thought. As anxiety-related disorders such as PTSD and GAD are characterized by difficulties with worry, rumination, and sleep problems, treating the latter may positively impact the former (Belleville, Cousineau, Levrier, & St-Pierre-Delorme, 2011). Further, these results suggest that interventions aimed at enhancing executive function may be useful for the reduction of maladaptive repetitive thought in individuals with sleep disturbance (Cohen, Mor, & Henik, 2015; Diamond & Lee, 2011). Future research addressing the downstream effects of sleep disturbance on cognitive and emotional function is necessary to further explore this pathway and delineate the role of executive function in the relationship between sleep and maladaptive repetitive thought.

CHAPTER IV

Overall conclusions

The present study examined the unique relationship between sleep disturbance and anxiety-related symptoms, as well as one potential mechanism by which sleep disturbance may contribute to anxiety-related symptoms. Study one found that sleep disturbance was linked to increased OCS symptoms in a nationally representative sample, even when controlling for depression. Study two found that executive function partially accounted for the relationship between sleep disturbance and maladaptive repetitive thought over and above the effect of general distress. Taken together, these results suggest that sleep disturbance is uniquely linked to anxiety-related symptoms, including obsessions, compulsions, worry, and rumination, and this relationship is not accounted for by depression or depressive symptoms. Further, the present findings indicate that executive function may be one mechanism by which sleep disturbance contributes to anxiety-related symptoms.

The results of these studies offer preliminary evidence for sleep disturbance as a transdiagnostic process that is linked to multiple anxiety-related symptoms that characterize various anxiety-related disorders. The evidence for links between sleep disturbance and OCS, worry, and rumination suggest that sleep disturbance may be an important factor in the presentation of disorders characterized by these processes, such as OCD and GAD. Additional research assessing the links between sleep disturbance and anxiety-related symptoms in clinical

populations is necessary to better understand how sleep disturbance may contribute to anxiety-related disorders

The results of these studies also indicate that the link between sleep disturbance and anxiety-related symptoms is not accounted for by depression or depressive symptoms. The apparent specificity of this relationship is noteworthy, given the high rates of comorbidity between anxiety and mood disorders (Kessler et al., 2003) and depression and sleep disturbance (Armitage, 2007). The evidence for a unique link between sleep disturbance and anxiety-related symptoms suggests that treatments for anxiety-related disorders may benefit from addressing sleep disturbance specifically. Additional research is necessary to replicate these findings in clinical populations.

Finally, the results of study two offer preliminary evidence for the role of executive function in the relationship between sleep disturbance and anxiety-related symptoms. Given the evidence for the negative downstream consequences of sleep loss on cognitive (Harrison & Horne, 2000) and emotional function (Minkel et al., 2012), it may be the case that sleep disturbance leads to deficits in executive function, which results in impaired ability to control or regulate anxiety-related symptoms. Additional research is necessary to replicate this finding in other anxiety-related symptoms, such as OCS, and in clinical populations.

Taken together, the results of the present study suggest a unique relationship between sleep disturbance and anxiety-related symptoms that is not accounted for by depression and highlight decreased executive function as one potential mechanism by which sleep disturbance may confer risk for the development of anxiety-related symptoms. Future research utilizing longitudinal methods is necessary to move beyond simply associating sleep disturbance and

executive function deficits with anxiety-related symptoms to determine whether sleep disturbance and consequent impaired executive function cause increased anxiety-related symptoms in the short-term, and potentially anxiety-related disorders over time. Further, future studies utilizing multi-method approaches that employ objective sleep measures and behavioral measures of executive function and anxiety-related symptoms are necessary to fully elucidate the relationships between these variables. Finally, research examining sleep disturbance and executive function in individuals with anxiety-related disorders is necessary to determine whether the present findings generalize to clinical populations.

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