ANXIETY SENSITIVITYAS A MODERATOR OF THE RELATIONSHIP BETWEEN DYSFUNCTIONAL BELIEFS ABOUT SLEEP AND SLEEP QUALITY

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ABSTRACT

Anxiety sensitivity, a construct defined by fear of anxiety and the feared consequences of anxiety, has been shown to play a role in the maintenance of insomnia. However, some researchers hypothesize that anxiety sensitivity must interact with some specific cognitive vulnerability to produce poor sleep quality. Additionally, the majority of studies examine only subjective measures of sleep quality, despite discrepancies between subjective report and objective measures such as polysomnography or actigraphy. The current study investigated anxiety sensitivity as a moderator of dysfunctional beliefs about sleep and sleep quality, measured both subjectively and objectively, in a sample of 145 participants recruited from the American University community. While an interaction does exist between anxiety sensitivity and dysfunctional beliefs about sleep, it appears that individuals scoring lower in anxiety sensitivity experience objectively worse sleep quality if they also score higher in dysfunctional beliefs about sleep. Individuals scoring higher in anxiety sensitivity appear protected from these effects. Subjective sleep quality varied independently as a function of anxiety sensitivity, but no interaction with dysfunctional beliefs about sleep was shown.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF ILLUSTRATIONS

Figure

INTRODUCTION

Background

Inadequate sleep from insomnia is a prevalent problem with numerous consequences to both physical and mental health (Kyle, Morgan, & Espie, 2010; Harvey & Tang, 2011). Insomnia often co-occurs with anxiety and mood disorders, and anxiety plays an integral role in the genesis and maintenance of insomnia (Wright et al., 2011). Anxiety sensitivity, which is related to but distinct from trait anxiety, is a construct defined by fear of anxiety and the feared consequences of anxiety. Anxiety sensitivity has been shown to have a role in the maintenance of insomnia (Vincent & Walker, 2001). Although anxiety and anxiety sensitivity are important predictors of sleep processes, some researchers have suggested that anxiety must interact with specific cognitions about sleep to lead to insomnia (Babson, Trainor, Bunaciu, & Feldner, 2008). The purpose of this study is to determine if anxiety sensitivity moderates the relationship between dysfunctional beliefs about sleep and sleep quality, measured both subjectively and objectively. Most of the research on sleep and anxiety has used subjective, self-report measures to assess sleep quality, but the use of objective measures such as polysomnography and actigraphy has shown some discrepancies between perceived sleep deficits and true insomnia (Benca, 2005). Thus, in the present study we assess both subjective and objective (actigraphy) indices of sleep quality.

Although all of the precise functions of sleep remain unclear, research indicates that it plays a vital role in central nervous system restoration, memory consolidation, and affect regulation (Lee & Douglas, 2010). Thus, it is important for both normal motor and cognitive function. Unfortunately, many people report experiencing either occasional or chronic sleep disturbance. Of all sleep complaints, insomnia (defined as the perception of inadequate,

insufficient, or non-restorative sleep) is the most common, with lifetime prevalence estimates ranging from 30 to 35%. The lifetime prevalence of chronic insomnia is estimated to be between 10 and 15% (Breslau, Roth, Rosenthal, & Andreski, 1995). Thus, insomnia affects a sizeable portion of the general population, and these rates increase for patients in primary care settings and for people presenting with psychiatric disorders (Simon & VonKorff, 1997).

Research has shown that deficits in sleep are associated with a number of negative outcomes. Insomnia, for example, has a negative impact on health related quality of life, including vitality, energy, and other aspects of mental, social, and physical functioning (Kyle et al., 2010). Furthermore, insomnia often predicts the development of mental health conditions such as depression, anxiety, and substance use disorders (Wright et al., 2011; Breslau et al., 1996). It should be noted that insomnia as a disorder is characterized not only by nighttime sleep difficulty, but also daytime disturbances such as fatigue or sleepiness, mood disturbances, and cognitive difficulties.

Insomnia Predicts Anxiety and Depression

Individuals with insomnia (without comorbid psychiatric disorders) typically present with symptoms of depression and anxiety that are greater than those of good sleeper controls, but less severe than those in individuals with diagnosed mood and anxiety disorders (Buysse et al., 2007). Longitudinal studies reveal that insomnia signals an increased risk for new episodes of any psychiatric disorder as assessed six months later, especially depressive disorders. This led to the theory that insomnia could be an early symptom of major depressive disorder or insomnia might play a causal role in the formation of depressive disorder (Breslau et al., 1996). Studying insomnia in isolation from other disorders may not provide a complete picture, as 60% of

individuals with insomnia symptoms meet criteria for another primary diagnosis (Maroti, Folkeson, Jansson-Frojmark, & Linton, 2011).

Conversely, many other disorders may be poorly understood if considered separate from insomnia. Approximately 61% of panic disorder patients and 44% of GAD patients also have insomnia (Lee & Douglas, 2010). Research reveals that insomnia and other psychiatric disorders cannot be regarded as separate entities, but rather as enmeshed pathologies. One study examined the effects of psychotherapy for depression in patients with comorbid depression, multiple sclerosis, and insomnia. They found that patients with persistent insomnia after psychotherapy were nearly six times more likely to continue to meet criteria for major depressive disorder at post treatment and over three times more likely to have clinically elevated anxiety. Similarly, Maroti et al. (2011) showed that CBT for insomnia (CBT-I) not only produced reliable and durable improvements in sleep patterns and the subjective experience of sleep in patients with primary insomnia, but also reduced anxiety and depression symptoms for individuals suffering from insomnia comorbid with anxiety and depressive disorders. Researchers have also found that insomnia assessed in soldiers six months pre-deployment predicts the occurrence of depression and PTSD at twelve months post-deployment (Wright et al., 2011). Thus research suggests that insomnia may precipitate other psychiatric disorders, such as anxiety disorders and depression, and is a comorbid diagnosis that often plays an important role in maintaining co-occurring disorders. The lack of sleep in those with insomnia is thought to prevent normal, sleep-dependent emotional memory processing and puts individuals at risk for the development of mood and anxiety disorders. Consequently, screening people for sleep disturbance helps identify people at risk for developing mood and anxiety disorders.

Cognitive Mediation of Sleep Problems

A cognitive theory of chronic insomnia outlined by Harvey (2002) lists five key processes that function both at night and during the day to increase anxiety and maintain insomnia: worry, selective attention and monitoring, misperception of sleep and daytime deficits, unhelpful beliefs about sleep, and safety behaviors. Each component serves to reinforce worry, which over time can lead to insomnia. In the model, worry leads directly to arousal and distress (Harvey, 2002). For example, anxiety occurring upon entering bed is associated with self-reported delayed sleep onset, which may further increase anxiety, creating a cycle which leads to insomnia (Babson et al., 2008). This theory implicates both anxiety and cognitions as key factors in insomnia.

Cognitions about sleep such as faulty beliefs and attentional biases are particularly important in perpetuating insomnia. For instance, some may believe that inadequate sleep will severely impair their daytime functioning, and thus will be overly attentive to any indication of impaired functioning, attributing any impairments to sleep deficits. Others may have unrealistic beliefs about how much sleep they need and design safety behaviors accordingly, which are intended to make sure they get enough sleep, but ultimately cause more anxiety and worry (Morin, Vallieres, & Ivers, 2007). Not surprisingly, cognitive behavior therapy for insomnia has proven to be the most effective treatment of the disorder (Morin, Celecchi, Stone, Sood, & Brink, 1999). Consequently, Morin developed the Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS), which has been used clinically to help identify cognitions which can later be targeted in CBT (Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). Reductions in DBAS scores post-treatment are highly correlated with improvements in sleep efficiency measured subjectively by daily sleep diaries and objectively by polysomnography (Morin, Blais, & Savard, 2002).

However, while CBT-I may help patients achieve more regular sleep, some studies have shown that it does not help self-reported daytime disturbances attributed to insomnia or psychological well-being (Chambers & Keller, 1993; Morin et al., 1999b).

Anxiety Sensitivity

Within this area of research, anxiety sensitivity has proven to be an important concept. Anxiety sensitivity is a construct that refers to fear of anxiety and the feared consequences of anxiety (Reiss, Peterson, Gursky, & McNally, 1986 from Vincent & Walker, 2001). Some of the consequences of anxiety include physical distress, public embarrassment, and fear of mental incapacitation. Consequently, these three feared anxiety outcomes correspond to the three subscales of the Anxiety Sensitivity Index (ASI): fear of mental incapacitation (ASI-M), physical distress (ASI-P), and social concerns (ASI-S).

It would be consistent within Harvey's cognitive theory of insomnia that anxiety sensitivity would amplify the worry, distress, and selective attention and monitoring which can lead to chronic insomnia (Harvey, 2002). For example, a person higher in anxiety sensitivity would be more attentive to physiological and mental processes when trying to fall asleep. They are likely to start to worry about the consequences of their sleep worries. Should they experience difficulty falling asleep, their sensitivity will trigger a cycle of distress and anxiety about this inability to fall asleep. Therefore, people higher in anxiety sensitivity would likely experience greater rates of insomnia. One of the first studies investigating the role of anxiety sensitivity in insomnia looked at anxiety sensitivity as a predictor of fear of sleeplessness. They found that anxiety sensitivity was not related to fear of sleeplessness, but greater fear of mental incapacitation (ASI-M) was associated with greater sleep-related impairment as measured by Morin's five-item Sleep Impairment Index (SII). However, anxiety sensitivity was also not

related to more disturbed sleep as assessed by the PSQI. Thus, those who fear cognitive symptoms of sleep loss may be more likely to report daytime impairment due to sleep loss even when sleep is actually quite normal. This effect was present even when controlling for worry and psychiatric comorbidity. The authors further conclude that anxiety sensitivity may explain why some insomniacs treated with CBT show objective improvements in sleep efficiency but still report sleep disturbances. In other words, sleep may improve with treatment, but the underlying fear of mental incapacitation or lack of cognitive control remains (Vincent & Walker, 2001).

Anxiety sensitivity is also predictive of sleep onset latency, another measure used to assess sleep quality, in patients with panic disorder when controlling for other variables including panic symptoms (Hoge et al., 2011). Other research has shown that anxiety sensitivity is associated with sleep problems in children (as assessed by the SSR), especially the mental incapacitation component, which still significantly predicted sleep problems on the sleep problem scale for children after controlling for anxiety. Such problems include difficulty falling asleep, having nightmares, and sleeping too little (Gregory & Eley, 2005).

Another study looked at the effect of the relationships between anxiety sensitivity, sleep anticipatory anxiety, and sleep onset latency in particular. Independently, the physical and mental incapacitation subscales of the Sleep Anticipatory Anxiety Questionnaire (SAAQ) and the physical subscale of the ASI were positively correlated with sleep onset latency. They found a small but significant moderating relationship between the physical distress subscale of the ASI and the physical component of the SAAQ in predicting latency. In their conclusions, the authors suggest that anxiety sensitivity must interact with a specific anxiety about sleep to cause sleep disturbance (Babson et al., 2008). This would suggest that anxiety sensitivity acts as a moderator between certain faulty cognitions about sleep and sleep disturbances. For instance, if a person

high in anxiety sensitivity holds the faulty belief that they must get nine hours of sleep in order to function normally the next day, they might constantly monitor their sleep quality in an attempt to ensure that they obtain "sufficient" sleep. However, if their sleep is disturbed, they will believe that their lack of sleep will affect them the next day. This will cause anxiety which could prevent them from falling back asleep or falling asleep in the first place.

Neuroticism and other measures of anxiety of depression may also predict poor sleep quality. In a sample of junior high school students, individuals scoring high in Neuroticism on the NEO tended to go to bed later and experienced shorter sleep duration, different sleep habits, more sleep problems, and impaired daytime function in comparison to individuals with low scores on neuroticism (Gau, 2000). Patients with insomnia also exhibit higher risk of neurotic depression, rumination, chronic anxiey, inhibited emotions, and an inability to discharge anger outwardly (Kales et al., 1983). Anxiety sensitivity is a distinct, albeit related, construct to many of these measures. Proper control of such variables is necessary to ensure valid analyses.

Subjective vs. Objective Measurement

Although each of the above studies is an important contribution to our understanding of anxiety, cognitions, and sleep, it should be noted that none of these studies used objective measures of sleep such as polysomnography or actigraphy. Thus, it is unclear whether these findings using subjective measures would generalize to objective measures. The advent of polysomnography and actigraphy allows researchers to measure some aspects of sleep objectively, which is necessary for a complete understanding of sleep disorders (Borkovec, 1982). A consistent finding in such research reveals objective measures of sleep do not always correlate well with the patient's experience of insomnia (Benca, 2005). This finding conveys one of Borkevec's caveats in research on insomnia: insomnia is primarily a subjective phenomenon.

People vary widely in the amount of sleep that they apparently require to feel rested and perform well during the day (Borkevec, 1982). Many, but not all, insomniacs magnify both sleep and wake aspects of their sleep problems when reporting on these in a retrospective, global manner. In other words, they tend to overestimate sleep onset latency and underestimate total sleep time (Harvey & Tang, 2011). This misperception is not due problems related to estimating time in general, to the variability of the night-to-night sleep and wake experiences of individuals who have insomnia, to psychological variables, or to the length of time being estimated (Fichten, Creti, Amsel, Bailes, & Libman, 2005).

Some research appears to indicate that the presence of sleep misperception itself has detrimental effects. Insomnia patients who misperceive sleep score higher on the Stanford Sleepiness Scale (SSS) than "true" insomniacs. Greater misperception is associated with worse cognitive function and greater cognitive disability. This does not preclude the existence of real sleep deficits. Some people complain of poor sleep quality yet have objectively normal sleep. Others actually experience an objectively measured sleep deficit. Sleep misperception occurs in insomniacs with objectively "normal" sleep duration but not in those with objective sleep deficits (Fernandez-Mendoza et al., 2011). In other words, people who misperceive their sleep report more daytime sleepiness than people who actually experience sleep deficits. Furthermore, it has been suggested that patients who think they are sleeping less than they are when they actually experience normal sleep times are at risk of getting trapped into becoming progressively more absorbed by and anxious about their sleep problem, potentially causing true sleep deficits (Harvey & Tang, 2011). So, it is important when studying sleep to consider the relative contributions of both objective sleep quality and perceived (subjective) sleep quality.

Finally, most studies of anxiety sensitivity and sleep disturbance have focused on specific populations such as patients with panic disorder, children, and clinical samples. This further limits the generalizability of their results. Using a nonclinical sample could help elucidate the impact of anxiety sensitivity on sleep within the general public. Given the high rates of sleep disturbance in the population at large, it is important to understand the interplay between anxiety and sleep in nonclinical populations as well as clinical ones.

Overview and Hypotheses

In the current study, we aimed to test whether anxiety sensitivity acts as a moderator of the relationship between dysfunctional beliefs about sleep and sleep disturbances including both sleep onset latency and sleep efficiency. We used both subjective and objective measures of sleep variables. At an initial visit to the laboratory, participants completed measures of dysfunctional beliefs and attitudes about sleep (DBAS-16) and anxiety sensitivity (ASI-3). Then, over the course of a week, participants completed daily sleep diaries which included a subjective measure of sleep quality and duration. During the week they also wore an actigraph, a wrist-watch like device which monitors movement and light which was used as an objective measure of sleep, including both sleep onset latency and sleep efficiency.

We hypothesized that i) the ASI-3 would be independently correlated with subjective but not objective measures of sleep disturbance; ii) the DBAS-16 would be independently correlated with subjective and objective measures of sleep disturbance and; iii) anxiety sensitivity will moderate the relationship between dysfunctional beliefs about sleep and both sleep onset latency and sleep efficiency (measured both subjectively and objectively by actigraphy).

METHOD

Participants

The sample consisted of 145 individuals recruited from the American University community including both students and normal adults. Participants were recruited through psychology courses, recruitment posters placed in the psychology department, and online through American University's daily newsletter to students, faculty, and staff. We excluded participants who were under 18 years of age. We discarded 8 participants' data. One participant wore the actigraph on his ankle rather than his wrist. One subject did not provide enough useable data between his actigraph usage and surveys per a predetermined criterion of 4 matched days of surveys and actigraph use. The data of three subjects was discarded because they recorded incorrect subject ID numbers, making it impossible to match their actigraphy to their survey data. The data of three more participants was discarded because their survey responses for time in bed and time out of bed were in complete disagreement with their actigraphy. The final sample included 137 individuals (83 women, 54 men). Within the sample, 72.3% identified as Caucasian, 13.1% as Asian, 9.5% as African American, and 5.1% identified as another race. Participants ranged in age from 18 to 40 (M=20.19, SD=3.179). Participants were compensated with a combination of course credit and/or cash.

Procedure

At an initial visit to the laboratory, participants completed the DBAS-16, the ASI-3, and answer questions regarding demographic information and diagnosed sleep disorders. Participants also completed several other measures relevant to a larger study. A research assistant then familiarized participants with an actigraph wristwatch device which had been programmed to record continuously for a week. Participants were asked to wear the device continuously for the

next week except when the actigraph might be damaged by water or impact, or when wearing the device would make the participant uncomfortable.

Over the course of the week, while participants were wearing their actigraphs, they completed short, daily surveys twice a day (one in the morning and one in the evening) which they received through automated emails at 4:00 a.m. and 8:00 p.m. each day. Participants completed the morning survey within an hour of when they woke up in the morning and the evening survey within an hour of when they went to sleep at night. The daily surveys contained the Carney Sleep Log as well as other questions relevant to a larger study.

At the end of the week, participants returned to the lab to complete a short survey relevant to a larger study, during which time the data from their actigraph was downloaded, analyzed, and saved. The sleep times reported by the participants in the daily surveys each morning were inputted into the ActiLife program. Participants who were interested in their sleep patterns were allowed to view their actigraph data. Participants were then compensated for their participation.

Measures

Initial Measures

Dysfunctional Beliefs and Attitudes about Sleep-16. The DBAS-16 is an abbreviated version of the DBAS which assesses sleep-disruptive cognitions which can help maintain insomnia in which responders indicate on an 11-point scale (0=”Strongly Disagree” to 10=”Strongly Agree) the degree to which certain sleep related statements apply to them. Sample questions of the DBAS-16 include “I need at least 8 hours of sleep to feel refreshed and function well during the day” and “After a poor night’s sleep, I know that it will interfere with my daily activities the next day.” The psychometric properties of this abbreviation have been shown to be

adequate and similar to the original 30-item DBAS (Morin, Vallieres, & Ivers, 2007). Crohnbach's alpha for our sample was calculated at α=0.840.

Anxiety Sensitivity Index-3. The ASI-3 is an 18-item measure on which respondents indicate on a five-point Likert scale (0="Not at all like me" to 4="Very much like me") the degree to which they are concerned about the possible negative consequences of anxiety and anxiety symptoms. The ASI-3 is composed of three subscales (physical concerns, cognitive concerns, and social concerns) which are summed to give a total score. Sample questions include "When I cannot keep my mind on a task, I worry that I might be going crazy", "I worry that other people will notice my anxiety", and "When I notice my heart skipping a beat, I worry that there is something seriously wrong with me." The ASI-3 has been shown to have sufficient discriminant, convergent, and criterion related validity and reliability (Taylor et al., 2007). Crohnbach's alpha for our sample was calculated at α=0.865.

NEO-Neuroticism. The neuroticism scale from the NEO-Five Factor Inventory is composed of 12 questions which responders indicate on a 5-point scale ("Strongly Disagree" to "Strongly Agree") the degree to which certain statements apply to them. Sample statements include "I am not a worrier", "Sometimes I feel completely worthless", and "I often get angry at the way people treat me." Two week retest reliability of the NEO-FFI is uniformly high and the test shows adequate internal consistency (McCrae & Costa, 2002). Crohnbach's alpha for our sample was calculated at α=0.846.

Mood and Anxiety Questionnaire – Short Form. The MASQ-Short Form is a 62-item measure which assesses symptoms of depression and anxiety which commonly occur in mood and anxiety disorders. Responders indicate the degree to which they experienced certain symptoms over the past week on a 5-point scale ("Not at all" to "Extremely"). The questionnaire

is comprised of four subscales: non-specific anxious symptoms, anxious arousal symptoms, non-specific depressed symptoms, anhedonic depressed symptoms. Sample symptoms include "Felt faint," "Was proud of myself (reverse scored)," and "Was disappointed in myself" (Watson, Weber, & Clark, 1995). The MASQ-Short Form has been shown to have high convergent validity with the original MASQ as well as comparably high internal consistency and construct validity (Wardenaar et al., 2010). Crohnbach's alpha for our sample was calculated at α=0.925.

Daily Measures

Carney Sleep Log. The Carney Sleep Log is a self-report sleep journal in which respondents record items such as their time in bed, sleep onset latency, nighttime awakenings, wake time, daytime naps, and daytime alcohol and caffeine intake. The log provides sleep and wake times that will be used in analyzing the actigraph data as well as subjective measures of sleep onset latency and sleep efficiency, which can be compared to the objective measures obtained by the actigraph. The Carney Sleep Log is divided into morning and nighttime relevant questions.

Stanford Sleepiness Scale. The SSS is a simple self-rated measure of sleepiness on a 7-point scale (1="Feeling active, vital, alert, or wide awake" to 7="No longer fighting sleep, sleep onset soon; having dream-like thoughts"). The SSS has been shown to be sensitive to sleep deprivation and partial recovery effects (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). Participants will complete the SSS as part of both their morning and evening sleep logs.

Apparatus

ActiLife 6. This study will use ActiSleep+ devices and ActiLife 6 software, which are marketed by Actigraph. Each ActiSleep+ device, or actigraph, is a small, wristwatch-like device, which measures physical activity such as activity counts and vector magnitude. Using the

reported sleep window, the ActiLife software calculates each night's sleep latency, total sleep time, number and duration of awakenings, and sleep efficiency. The software calculates sleep latency as the number of minutes from the time the participant reports going to sleep on a particular night to sleep onset. Total sleep time is the total number of minutes that the participant was actually asleep. Each period of wake time between first sleep onset and final awakening were recorded as awakenings, and the duration of each was recorded in minutes. Wake after sleep onset (WASO) is the sum of the durations of all awakenings within a night. Sleep efficiency is calculated as a participant's total sleep time divided by the total amount of time they were in bed (obtained from participants' sleep logs).

RESULTS

Our analyses can be divided into two primary areas: the effect of anxiety sensitivity and dysfunctional beliefs about sleep on *objective* measures of sleep quality (e.g. latency, efficiency, wake after sleep onset [WASO], and number of awakenings after sleep onset as assessed by actigraphy) and their effect on *subjective* measures of sleep quality (e.g. self-reported latency, reported number of awakenings after sleep onset, perceived sleep quality, and feeling rested and/or refreshed). Due to minute differences in the amount of useable objective compared to subjective data, the number of data points used in these analyses differed slightly, however both sets of data come from the same sample of 137 subjects. Of 831 morning surveys, 54 were excluded since they were completed over 2 hours after the participant woke. This was a predetermined exclusion criterion as we determined that the reports may be invalid due to the long response time. Of 861 nights of useable actigraph data, 13 were excluded because they reported perfect efficiency. This was a predetermined exclusion criterion as it likely indicated that the participant did not actually wear the actigraph that night.

Descriptive Analyses

The final analysis of objective outcomes measures possessed 848 useable units/days of actigraph data from 137 subjects, whereas the final analysis of subjective outcome measures possessed 792 useable units/morning surveys from 137 subjects. Table 1 shows the means and standard deviations for number of useable observations of objective and subjective data (nights of actigraph data and morning surveys completed).

Table 1
Descriptive Statistics: Data Points

Data Type	N	Minimum	Maximum	Mean	SD
Actigraph Nights	**137**	**1**	**7**	**6.28**	**1.11**
Morning Surveys	**137**	**1**	**7**	**5.69**	**1.59**

Table 2
Descriptive Statistics: Level 2 Variables

Variable	N	Minimum	Maximum	Mean	SD
ASI	**137**	**0**	**50**	**17.04**	**11.01**
DBAS-16	**137**	**0.88**	**7.56**	**3.98**	**1.45**
NEO-N	**137**	**6**	**46**	**21.61**	**8.50**
MASQ-NSA	**137**	**11**	**43**	**21.12**	**6.63**
MASQ-AA	**137**	**17**	**55**	**24.45**	**6.71**
MASQ-NSD	**137**	**13**	**58**	**25.19**	**9.27**
MASQ-AD	**137**	**29**	**91**	**58.06**	**13.53**
TST	**137**	**254.0**	**492.8**	**381.59**	**47.23**

ASI: Anxiety Sensitivity Index, DBAS-16: Dysfunctional Beliefs about Sleep, NEO-N: Neuroticism, MASQ: Mood and Anxiety Symptom Questionnaire, NSA: Non-specific anxiety, AA: Anxious Arousal, NSD: Non-specific depression, AD: Anhedonic depression, TST: Total sleep time

Neither of these was correlated with any of the primary variables of interest. ANOVAs showed that neither statistic differed by sex, although gender difference on the number of morning surveys completed approached significance (F=.026, p=.873; F=2.777, p=.098). The mean number of morning surveys completed by women was 5.8675, compared to an average of 5.4074 morning surveys completed by men. Table 2 shows the means and standard deviations for the primary variables of interest. Table 3 shows the means and standard deviations for all daily survey and actigraph variables.

Table 3
Descriptive Statistics: Level 1 Variables

Variable	N	Minimum	Maximum	Mean	SD
Subjective Sleep Onset Latency	**798**	**0**	**320**	**17.65**	**23.63**
Subjective # of Awakenings	**779**	**0**	**10**	**1.64**	**1.60**
Subjective Sleep Quality	**787**	**0**	**4**	**2.50**	**.94**
Subjective Feeling Rested	**792**	**0**	**4**	**2.16**	**.99**
Actigraph Sleep Onset Latency	**848**	**0**	**239**	**11.96**	**17.84**
Actigraph Sleep Efficiency	**848**	**8.42**	**99.29**	**83.06**	**9.22**
Actigraph Wake after Sleep Onset	**848**	**1**	**346**	**67.15**	**44.42**
Actigraph # of Awakenings	**848**	**1**	**48**	**20.53**	**8.99**

Table 4
Correlations Between 7 Questionnaire Scores and Total Sleep Time

Measure	1	2	3	4	5	6	7	8
1. ASI	**-**							
2. DBAS-16	**.430****	**-**						
3. NEO-N	**.381****	**.521****	**-**					
4. MASQ-NSA	**.354***	**.519****	**.466****	**-**				
5. MASQ-AA	**.322****	**.364****	**.353****	**.601****	**-**			
6. MASQ-NSD	**.439****	**.674****	**.420****	**.553****	**.382****	**-**		
7. MASQ-AD	**.304****	**.441****	**.302****	**.177***	**.280****	**.519****	**-**	
8. TST	**-.132**	**.071**	**-.002**	**.058**	**.003**	**-.095**	**-.093**	**-**

ASI: Anxiety Sensitivity Index, DBAS-16: Dysfunctional Beliefs about Sleep, NEO-N: Neuroticism, MASQ: Mood and Anxiety Symptom Questionnaire, NSA: Non-specific anxiety, AA: Anxious Arousal, NSD: Non-specific depression, AD: Anhedonic depression, TST: Total sleep time

Many of the variables of interest were correlated (see Table 4). Scores on the non-specific depression subscale of the MASQ and the NEO-N were highly correlated (r=.674, p=.000) as were scores on the non-specific anxiety and the anxious arousal subscales of the MASQ (r=.601, p=.000). See Table 4 for a full list of correlations of the primary variables of interest.

Overview of Analyses

For each of the outcome variables of interest, the main effects of anxiety sensitivity and dysfunctional beliefs about sleep were calculated. Due to the nested structure of our data, multilevel modeling was used to calculate these effects. For example, if one is interested in finding the relationship between anxiety sensitivity and sleep latency, the level 1 regression equation is

$$Latency_{ij} = b_0 + r$$

And the level 2 equation is

$$b_0 = \gamma_{00} + \gamma_{01}(ASI) + u_0$$

The level 1 intercept is modeled as a function of an intercept component, a slope component indicating the effect for anxiety sensitivity on sleep onset latency, and a random error component. The variable $\gamma_{01}$is the change in average sleep onset latency for every point increase on the ASI. The same basic equation can be used to calculate the effect of possible confounding variables such as neuroticism and anxious symptoms (as assessed by the MASQ). Once identified, these variables could be controlled for in analyses by adding an additional term to the level 2 equation. For example, if one is interested in the same relationship as previously described while controlling for neuroticism, the level 2 equation is

$$b_0 = \gamma_{00} + \gamma_{01}(ASI) + \gamma_{02}(NEO{:}N) + u_0$$

This models the effect of neuroticism on sleep onset latency and therefore the ASI coefficient indicates the unique relationship between anxiety sensitivity and sleep onset latency.

In order to investigate the interactive effects of anxiety sensitivity and dysfunctional beliefs about sleep on measures of sleep quality, an additional term was required for the level 2 equation. This variable, ASIxDBAS, is the product of the differences of each individuals' score

on the ASI and the DBAS-16 from the average score on each measure (i.e.$(ASI_i - ASI_M) \times (DBAS_i - DBAS_M)$). To calculate the moderating relationship of these two variables on measures of sleep quality, the level 2 equation becomes

$$b_0 = \gamma_{00} + \gamma_{01}(ASI) + \gamma_{02}(DBAS) + \gamma_{03}(ASIxDBAS) + u_0$$

In this equation, γ_{03}represents the interactive effect of ASI and DBAS on sleep variables.

Objective Sleep Quality

Objective sleep quality was assessed by examining four variables obtained from actigraphy: sleep onset latency, sleep efficiency, WASO, and number of awakenings after sleep onset. Sleep onset latency did not vary as a function of either anxiety sensitivity or dysfunctional beliefs about sleep independently (γ_{01}=.100, *p*=.272; γ_{01}=.285, *p*=.630), nor did it vary as a function of the interaction of the two variables (γ_{03}=-.071, *p*=.159). However, sleep onset latency was strongly affected by neuroticism (γ_{01}=.250, *p*=.009); participants scoring higher in neuroticism experienced longer times to sleep onset upon attempting to fall asleep than people with lower neuroticism scores. Similarly, the effect of anxiety sensitivity on sleep efficiency approached significance, such that higher sensitivity was associated with less efficiency, but efficiency did not vary as a function of dysfunctional beliefs about sleep (γ_{01}= -.118, *p* =.057; γ_{01}= -.267, *p*=.490). Furthermore, sleep efficiency varied as a function of the combined relationship of anxiety sensitivity and dysfunctional beliefs about sleep (γ_{03}= .081 *p*=.015). To determine exactly what this interaction effect was, the sample was split based on whether a participant scored higher or lower than the sample median on the ASI. The relation between dysfunctional beliefs about sleep and efficiency was tested in each half of the sample. For people who score higher in anxiety sensitivity, there is no significant relationship between dysfunctional beliefs about sleep and sleep efficiency objectively measured by actigraphy. However, for people

who score lower in anxiety sensitivity, increases in dysfunctional beliefs about sleep were associated with impaired sleep efficiency. This relationship remains significant when controlling for average total sleep time, which was independently associated with sleep efficiency (γ_{01}= .055, *p* =.001). See Figure 1 for an illustration of this interaction effect.

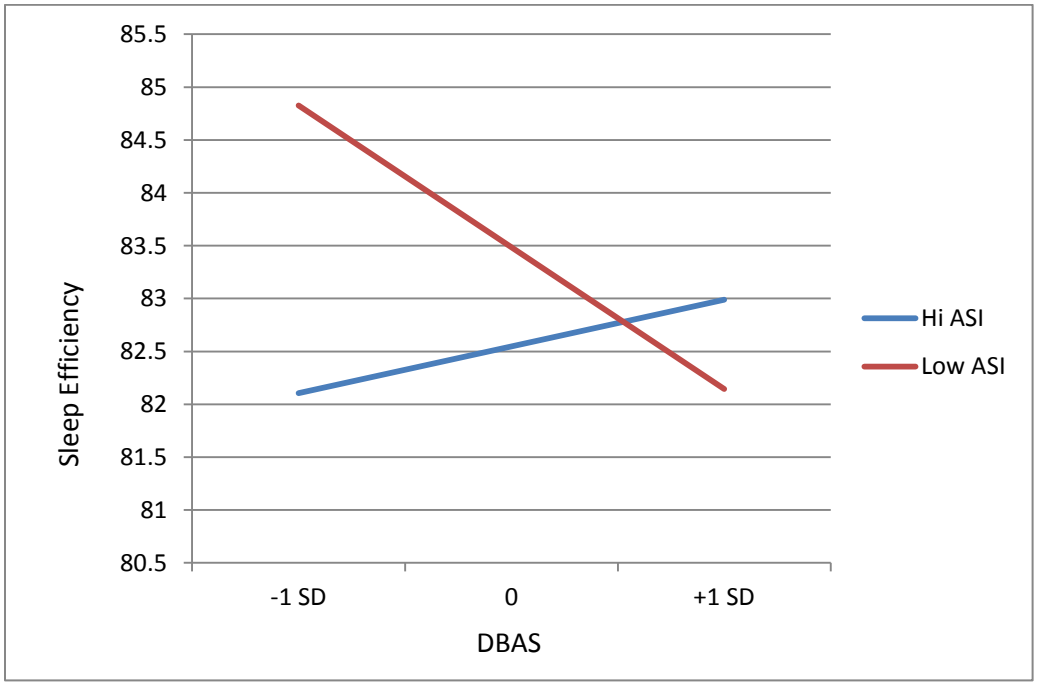

Figure 1. Sleep Efficiency as a function of Dysfunctional Beliefs about Sleep for subjects falling above and below the median score on the Anxiety Sensitivity Index

WASO also varied as a function of the moderating relationship of anxiety sensitivity and dysfunctional beliefs about sleep (γ_{03}=-.362 *p*=.016), but did not vary as a function of either variable independently (γ_{01}= .411, *p* =.141; γ_{01}= 1.608, *p*=.407). For people who score high in anxiety sensitivity, more dysfunctional beliefs about sleep was not significantly associated with wake time after sleep onset. People scoring lower on the ASI experienced more wake after sleep onset with increases in dysfunctional beliefs about sleep. See Figure 2 for an illustration of this interaction effect.

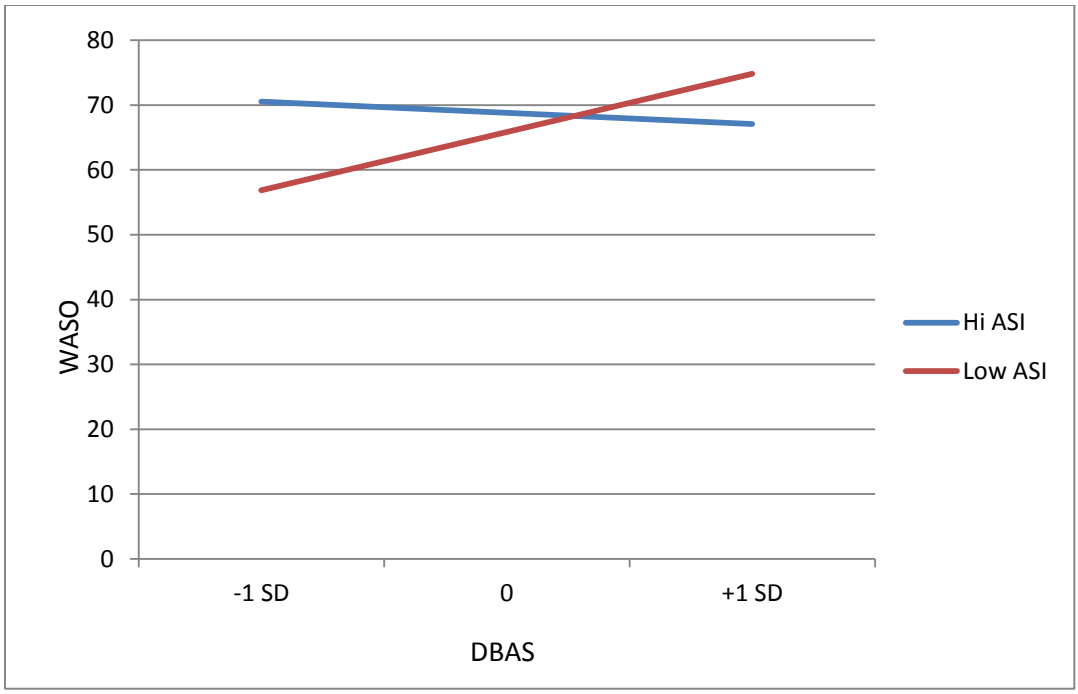

Figure 2. WASO as a function of Dysfunctional Beliefs about Sleep for subjects falling above and below the median score on the Anxiety Sensitivity Index

Similar results were found with the number of awakenings experienced after sleep onset; the number of awakenings experienced after sleep onset varied as a function of the moderating relationship of anxiety sensitivity and dysfunctional beliefs about sleep (γ_{03}=-.068 *p*=.017), but did not vary as a function of either variable independently (γ_{01}= .007, *p* =.141; γ_{01}= .266, *p*=.473). Splitting the sample into high scorers and low scorers on the ASI revealed that for people high in anxiety sensitivity, there is no significant relationship between dysfunctional beliefs about sleep and number of awakenings. However, for people scoring low in anxiety sensitivity, higher scores in dysfunctional beliefs about sleep was significantly associated with more awakenings per night. This relationship remained significant when controlling for average total sleep time, which was also independently related to number of nightly awakenings. See Figure 3 for an illustration of this interaction effect.

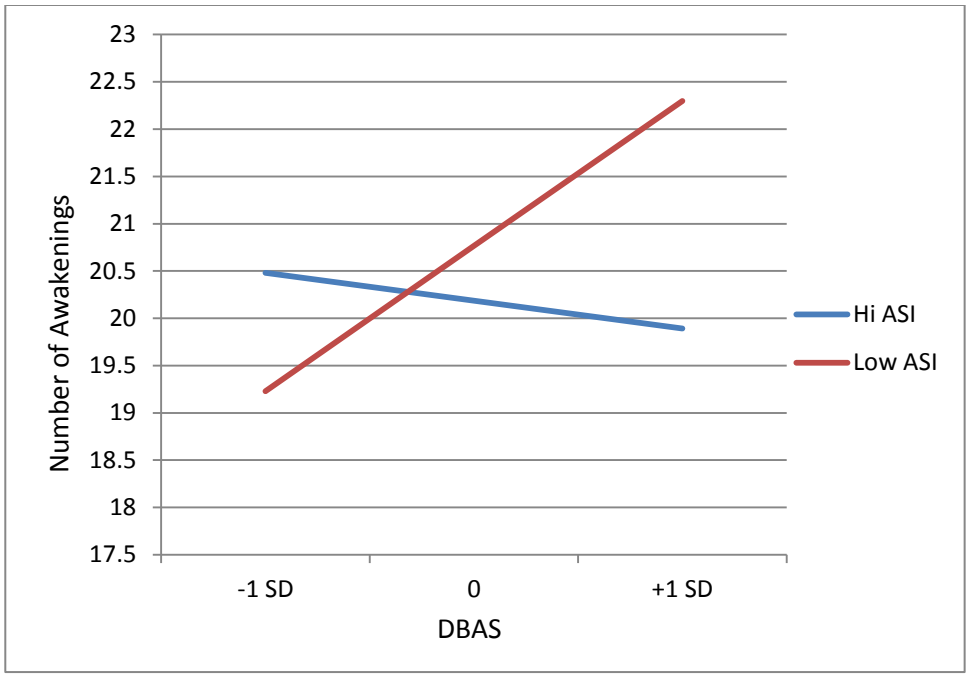

Figure 3. Number of Awakenings as a function of Dysfunctional Beliefs about Sleep for subjects falling above and below the median score on the Anxiety Sensitivity Index

Subjective Sleep Quality

Subjective sleep quality was assessed using four variables obtained from morning surveys: self-reported sleep onset latency, self-reported number of awakenings after sleep onset, self-reported feelings of sleep quality, and self-reported feelings of being rested and/or refreshed upon waking. Self-reported sleep onset latency did not vary as a function of either anxiety sensitivity or dysfunctional beliefs about sleep, independently (γ_{01}= .127, *p* =.310; γ_{01}= .624, *p*=.458) or in a moderating relationship (γ_{03}=-.089, *p*=.135). Nor did self-reported number of awakenings vary as a function of either anxiety sensitivity or dysfunctional beliefs about sleep, independently (γ_{01}= .001, *p* =.832; γ_{01}= .014, *p*=.829) or in a moderating relationship (γ_{03}=-.002, *p*=.733). Subjective sleep quality varied independently as a function of anxiety sensitivity (γ_{01}=-.010, *p*=.018) and neuroticism (γ_{01}=-.011, *p*=.025). Subjects who scored higher in either anxiety sensitivity or neuroticism also subjectively reported poorer sleep quality. It did not vary

as a function of dysfunctional beliefs about sleep (γ_{01}=-.046, *p*=.255) or the moderating influence of anxiety sensitivity and dysfunctional beliefs about sleep (γ_{03}=-.001, *p*=.860). Similarly, self-reported feelings of being rested and/or refreshed varied independently as a function of scores the NEO-N (γ_{01}=-.016, *p*=.003), anxiety sensitivity (γ_{01}=-.012, *p*=.008) and scores on the non-specific depression symptoms and anhedonic depression symptoms portions of the MASQ (γ_{01}=-.013, *p*=.021; γ_{01}=-.009, *p*=.042 respectively). It did not vary as a function of dysfunctional beliefs about sleep (γ_{01}=-.020, *p*=.661) or the moderating relationship of anxiety sensitivity and dysfunctional beliefs about sleep (γ_{03}=-.002, *p*=.590).

DISCUSSION

Anxiety Sensitivity

As predicted, anxiety sensitivity was not significantly associated with objective measures of sleep quality, but was significantly associated with some subjective measures of sleep quality. These findings are consistent with the research of Walker and Vincent (2001) which reported that subjects higher in anxiety sensitivity are more likely to subjectively report more daytime impairment due to perceived sleep disturbance, even when experiencing no differences in objectively assessed sleep. These subjects likely focus more attention on their anxious symptoms and therefore say they feel less rested in the mornings and believe they experience poorer sleep quality. We did analyze the individual subscales of the ASI as well, but were not able to replicate the findings of Hoge et al. (2011) or of Babson et al. (2008). In fact, the subscales of the ASI were not independently correlated with any variable of interest, rather it was the total ASI score that was found to be significant.

Dysfunctional Beliefs about Sleep

Counter to our predictions, dysfunctional beliefs about sleep were not significantly associated with any measure of sleep quality, either objective or subjective. This finding is inconsistent with the research of Morin, Blais, and Savard (2002) which showed that reductions in DBAS scores in insomniacs who received CBT-I were highly correlated with improvements in sleep efficiency measured both subjectively and objectively. However, this difference could be a consequence of looking at two different samples: a non-clinical sample primarily composed of college students, compared to a clinical sample of patients diagnosed with insomnia with an average age of 64.7. It could also be the case that CBT-I produces change in sleep efficiency

through some additional mechanism not related to identifying and countering dysfunctional beliefs about sleep.

Anxiety Sensitivity as a Moderator of Dysfunctional Beliefs about Sleep and Sleep Quality

Our prediction regarding the moderating influence of anxiety sensitivity on the relationship between dysfunctional beliefs about sleep and sleep quality was not supported by our results. Indeed, moderation effects were present, but it appeared that dysfunctional beliefs about sleep had a stronger effect on objective sleep indices in the low ASI group, not the high group as predicted. These findings with regard to objective but not subjective measures of sleep quality counter to the findings of Babson et al. (2008) which showed that anxiety sensitivity acted as a moderator of the relation between sleep anticipatory anxiety and *subjective* sleep onset latency. Additionally, the relationship appears more complex than our hypothesis, which stated that high levels of the two variables would interact to produce poorer sleep outcomes. Instead, different levels of anxiety sensitivity and dysfunctional beliefs about sleep have different impacts on sleep objective sleep parameters. For people who score above the median in anxiety sensitivity, dysfunctional beliefs about sleep appear to have no significant impact on any objective sleep variables. However, for people who score below the median in anxiety sensitivity, more dysfunctional beliefs about sleep is detrimental, causing poorer sleep efficiency and more awakenings and more time awake after sleep onset. This finding suggests that dysfunctional beliefs about sleep act as a vulnerability factor which can lead to objective sleep deficits in people with lower levels of anxiety sensitivity, but has little impact in those with higher levels of anxiety sensitivity. We hypothesized, in agreement with Harvey's cognitive model of chronic insomnia, that higher levels of anxiety sensitivity would amplify worry and selective attention and monitoring of anxious symptoms. It was thought that this would lead

directly to more arousal, exacerbating the cycle which keeps people awake at night. This appears not to be the case. Although higher scores on the ASI tend to be associated with greater latency to sleep onset, poorer sleep efficiency, more wake time after sleep onset, and more nighttime awakenings; none of these relationships is significant. Dysfunctional beliefs about sleep have a significant impact only in people scoring lower in anxiety sensitivity. This impact is significantly negative for both sleep efficiency and number of awakenings, and approaches significance for wake time after sleep onset. It is possible that people high in anxiety sensitivity who have more dysfunctional beliefs about sleep engage in more safety behaviors to ensure they get adequate amounts of undisturbed sleep, which could explain why dysfunctional beliefs about sleep are detrimental to those lower in anxiety sensitivity, who may not direct as much attention to their sleep behaviors. We considered that these results may be particular to our sample, which was largely comprised of university undergraduate students and who may experience erratic sleep schedules or restricted total sleep time. Yet these relationships remain even when controlling for average total sleep time, suggesting that they are present regardless of whether a person obtains adequate amounts of sleep. It should be noted that this relationship exists only for objective sleep measures. Thus, subjective reports are driven by some other variables. Our results suggest that anxiety sensitivity may be independently responsible for self-reports of poor sleep quality and greater impairment in daytime functioning.

Limitations and Directions for future research

This research was conducted with a non-clinical sample consisting primarily of college students. While we desired to observe the effects of anxiety sensitivity and dysfunctional beliefs about sleep in a non-clinical sample, this is a unique population which limits the generalizability

of the results. Circadian rhythms change over the lifespan, with a major change occurring at the onset of puberty and continuing into early adulthood. During this time, an individual's circadian rhythm shifts to a later bedtime and a later wake time, with an elongated sleep requirement (Carskadon et al., (1980). Additionally, the primary reason for insufficient sleep in a college undergraduate sample is schoolwork or recreation rather than difficulty falling asleep. Therefore, our results may be specific to the college undergraduate population. Conversely, our sample could have been too heterogeneous with regards to their sleep habits and behaviors to demonstrate the effect of anxiety sensitivity on sleep quality found by Vincent and Walker (2001) in chronic insomniacs, or the effect of dysfunctional beliefs found by Morin et al. (2002) on sleep efficiency. While we did exclude patients who were diagnosed with a sleep disorder, we only assessed this in a single question in the demographics questionnaire portion of our baseline measures. Many sleep disorders can go undiagnosed for many years, and a more thorough screening process may have excluded additional participants who exhibited clinical levels of a sleep disorder. The presence of daily stressors is an additional potential confound that was not addressed. It is possible that impaired sleep quality is better predicted by day-to-day stressors, or perceived stress, than by level-2 variables such as anxiety sensitivity and dysfunctional beliefs about sleep. The use of actigraphy for our objective data, while allowing us to study subjects in their natural sleeping environments, did not provide the depth of information that could have been obtained by other objective tests, such as a polysomnogram. Additionally, the accuracy of the data was limited by self-report. Even our "objective data" determined by actigraphy was subject to self-report, as the actigraph did not indicate when participants went to bed or woke up in the morning, and so we relied on participants to document sleep periods in their morning survey. Thus, the time interval analyzed by actigraphy depended on participants' sleep logs. It is

possible that more significant relationships could be found with more accurate or objective reporting of time in bed and time of final awakening. Another possible confound to both our objective and subjective data is the possibility that participants altered their normal sleep patterns and habits in response to monitoring by actigraphy and daily surveys. Ideally, individuals should be given time to adapt to the use of the actigraph and survey instruments before data is collected. Consequently, an extended observation time of two weeks to a month would have been preferable to our limited one-week observation window.

Conclusion

It is clear that anxiety sensitivity and dysfunctional beliefs about sleep interact to produce a significant impact on an individual's sleep quality, although our understanding of this relationship is not complete, and the relationship may change depending on the population in question. In our non-clinical sample, neither scale is independently related to objective measures of sleep quality, but increased dysfunctional beliefs about sleep negatively affect most sleep outcomes in individuals scoring low in anxiety sensitivity. Subjective reports of sleep quality appear to be driven by anxiety sensitivity and/or neuroticism. This could have implications for cognitive behavior therapy for insomnia, which has traditionally focused on addressing the specific cognitions and beliefs maintaining insomnia and countering them with new cognitions as well as education on good sleep hygiene. While dysfunctional beliefs tend to diminish as a consequence of CBT-I, our results suggest that anxiety sensitivity is a significant contributor to both subjective and objective sleep deficits. Targeting anxiety sensitivity specifically within therapy could lead to increased treatment efficacy.

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