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The Role of Dysfunctional Beliefs and Attitudes about Sleep (DBAS), Rumination, Psychological Inflexibility, and Insomnia Among College Students

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THE ROLE OF DYSFUNCTIONAL BELIEFS AND ATTITUDES ABOUT SLEEP (DBAS), RUMINATION, PSYCHOLOGICAL INFLEXIBILITY, AND INSOMNIA AMONG COLLEGE STUDENTS

by

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ABSTRACT

The Role of Dysfunctional Beliefs and Attitudes About Sleep (DBAS), Rumination, Psychological Inflexibility, and Insomnia among College Students

by

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Utah State University, 2022

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A handful of studies have provided evidence for implementation of Acceptance and Commitment Therapy for insomnia. Although the outcomes of this research are positive, a closer exploration into the specific studies highlights inconsistencies that necessitates a further investigation between psychological inflexibility and insomnia. Two potential variables that are found among various insomnia models and have research ties to each other that could help explain that relationship are (a) dysfunctional beliefs and attitudes about sleep and (b) rumination. That is, these variables may serially explain the relationship between psychological inflexibility and insomnia. The current study sought to understand this relationship among college students through serial mediation analysis with dysfunctional sleep-related cognitions and insomnia rumination as serial mediation variables. Data were collected at baseline and 1-month follow up. Specifically, questionnaires were distributed online to individuals who are enrolled in a 2- or 4-year

university, have access to the internet, and not diagnosed with another sleep disorder. Participants (n = 490) were recruited at Utah State University, Southern Illinois University, and Western Michigan University in the U.S. and began at the start of each semester (Fall 2021 and Spring 2022) by notifying students during class announcements and were assisted by two professors at their respective school. The participants that completed the baseline and 1-month follow up were composed of college students who are full-time (94.7%), undergraduate (99.8%), female (75.1%), or White (83.7%). Results showed correlation analyses between psychological inflexibility, dysfunctional sleep-related cognitions, insomnia rumination, and insomnia to all be significant. The serial mediation model showed that all three indirect effects (i.e., when the mediator was only dysfunctional sleep-related cognitions, only insomnia rumination, and both) were also significant—these results were predicted. These results suggest there is potential to assisting future interventions by understanding and targeting these negative repetitive thoughts (i.e., dysfunctional sleep-related cognitions and insomnia rumination). Future studies should aim to uncover treatment outcomes for these variables.

(53 pages)

PUBLIC ABSTRACT

The Role of Dysfunctional Beliefs and Attitudes About Sleep (DBAS), Rumination, Psychological Inflexibility, and Insomnia among College Students

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The purpose of the study was to understand additional variables that play a role between insomnia and the inability to stay present with internal feelings/emotions (i.e., psychological inflexibility) among college students. These variables that were predicted to help explain this relationship include dysfunctional sleep-related cognitions and insomnia rumination. To do this, college students were surveyed online at the start of the study and one month later. In addition, to be included in the study they must have been enrolled in a 2- or 4-year university, have access to the internet, and not diagnosed with another sleep disorder. Participants (n = 490) were from the mountain west and Midwest region of the U.S. A majority of participants were composed of college students who are full-time (94.7%), undergraduate (99.8%), female (75.1%), or White (83.7%). Results showed psychological inflexibility, dysfunctional sleep-related cognitions, insomnia rumination, and insomnia were significantly correlated with each other. The main analysis (i.e., serial mediation), showed that dysfunctional sleep-related cognitions, insomnia rumination helped explain the relationship between psychological inflexibility and insomnia. These results suggest there is potential to assisting future interventions by understanding and targeting these negative repetitive thoughts (i.e., dysfunctional sleep-related cognitions and insomnia rumination). Future studies should aim to uncover treatment outcomes for these variables.

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Guadalupe G. San Miguel

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CHAPTER I

INTRODUCTION

Many individuals have healthy sleep that leaves them feeling restful. To accomplish this, they will cycle in-and-out of various stages—at differing depths—of sleep during one sleep session. This normative sleep involves stages of sleep, and its process is called sleep architecture, which is composed of non-rapid eye-movement (NREM) and rapid eye-movement (REM; Altevogt & Colten, 2006). Within NREM there are four stages that each constitute a greater depth of sleep which then concludes with REM, and each stage has its own characteristics and importance. An individual will progress, complete and cycle through NREM and REM sleep with varying times at each stage during one sleep episode (Altevogt & Colten, 2006). But for many, this process does not occur.

Insomnia can be defined as a subjective dissatisfaction with sleep quality along with the inability to initiate sleep, stay asleep or fall back asleep after waking up in the middle of the night (American Psychiatric Association [APA], 2013). It also involves daytime impairments (e.g., fatigue). It needs to occur for at least three days per week for at least 3 months, even with the opportunity to sleep. Finally, it cannot be explained by another sleep disorder (e.g., circadian rhythm sleep—wake disorders) or substance (e.g., medication) resulting in impairment and/or distress (APA, 2013).

Insomnia tends to occur in between 3.9% and 22.1% of the population (Roth et al., 2011). The reason for the variability in rates is because of the different diagnostic criteria used (e.g., Diagnostic and Statistical Manual of Mental Disorders-fourth edition-

text revision vs International Classification of Diseases-Tenth Edition; Roth et al., 2011). Among college students, prevalence rates of insomnia worldwide range from 9.4% to 38.2% but has a weighted mean prevenance rate (18.5%); that is relatively high when compared to the general population (Jiang et al., 2015). There are a variety of variables that could lead to these high rates in college students.

As universities are efficient in the use of technology to disseminate education, and students have full autonomy as to when they use their devices, students' sleep can be hampered in relation to screen time throughout the day (Christensen et al., 2016) and before bed (Fossum et al., 2014). Additionally, it is common practice to consume caffeine for the purpose of completing school-related objectives (e.g., study for a test; Malinauskas et al., 2007). High alcohol use is seen in college students (Knight et al., 2002) and these beverages also have negative effects on sleep quality, specifically among college students (Lohsoonthorn et al., 2013; Singleton & Wolfson, 2009). College students are highly susceptible to developing an insomnia disorder due to a college culture that fosters screen use, stimulants designed to keep students awake, and alcohol use leading to sleep deficiencies, among other issues (Hershner & Chervin, 2014).

Consequences that stem from an inadequate amount of sleep include hampered academic performance, driving impairment, and depressive symptoms (Hershner & Chervin, 2014). These consequences go beyond other highly correlated impairments that are associated with sleep deficiencies among the general population (e.g., metabolic dysregulation; Knutson et al., 2007).

Another worthy item that necessitates highlighting, are the comorbidities that are

common with insomnia. Specifically, anxiety and depression have been shown to be highly comorbid with insomnia (e.g., Ohayon & Roth, 2003; Staner, 2010) and those with insomnia are more likely to have clinically significant anxiety and/or depression compared to individuals without insomnia (Taylor et al., 2005). This is not surprising considering there is some overlapping of symptomology among insomnia, anxiety, and depression. Researchers have attempted to elucidate the directionality of these comorbid disorders (i.e., insomnia predicting anxiety/depression vs anxiety/depression predicting insomnia) but empirical evidence has been inconclusive—or rather, suggest it is bidirectional (Alvaro et al., 2013).

These findings suggest, comorbid depression with insomnia requires specific interventions for each disorder due to the clinical course each disorder can take before, during, and/or after treatment (Staner, 2010). Anxiety also presents unique comorbid conflict as sleep deficiencies have an impact on anxiety symptom severity, indicating the greater sleep disturbance the greater symptom severity (Cox & Olatunji, 2016). As Cox and Olatunji suggest, a more efficacious anxiety intervention is one that assess for sleep deficiencies as well.

Models of Insomnia

A variety of models have attempted to explain the etiology of insomnia. One insomnia model that contributed and inspired sequential models is Spielman's 3-P model (Spielman, 1986; Spielman et al., 1987). Spielman presented predisposing vulnerability factors that contribute to the onset of sleep deficiencies, which alone is not enough to

develop insomnia. It is the combination of precipitating factors with predisposing ones that manifest into insomnia. Then, perpetuating factors help maintain the sleeping disorder. The 3-P model has clinical utility as a clinician's guide to case conceptualization and sleep history for insomnia (Spielman, 1986).

A decade earlier a behavioral approach was applied to insomnia. Rooted in an operant analysis of insomnia, Bootzin (1972) suggested sleep is a behavior that is learned and reinforced by specific stimuli. This is further extended into sleep difficulties where specific stimuli have failed to be established for sleep (Bootzin, 1972). Ensuing models of insomnia incorporate these behavioral aspects into their models.

One of the more popular models to embrace the behavioral aspect is Morin's (1993) integrative behavioral and cognitive model. Morin postulates insomnia is a multifaceted sleeping disorder that is the result of cognitions, perceived consequences, and behavioral factors with hyperarousal—coming in the form of emotional, cognitive, or physiological—being a key contributor to the onset of insomnia. Moreover, behavior and cognitions play a big role in maintaining insomnia leading to prime factors to target during interventions (Morin, 1993). Morin also emphasized dysfunctional sleep-related cognitions and their role in the emotional distress and sleep disturbance in insomnia (Morin, 1993; Morin et al., 1993). This led to the creation of the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) measure, which is typically used in studies assessing treatment efficacy during cognitive behavioral interventions (CBT) for insomnia.

Dysfunctional sleep-related cognitions and attitudes were later emphasized in Harvey's (2002) model of insomnia. Harvey, too, incorporated behavioral and cognitive

aspects into a comprehensive model. Specifically, negatively toned cognitions lead to arousal igniting selective monitoring of physiological and environmental reactions to sleep performance, and if the individual classified them as inefficient the individual would conclude there is a sleep deficit. Combined with the individual's dysfunctional beliefs and attitudes on sleep, maladaptive behaviors are then used to combat sleep deficiencies. Although these theoretical steps are sequential, insomnia can be initiated at any point during these processes leading to the ignition of the whole model. Beyond the empirical evidence Harvey presented in his original 2002 article, there has been a substantial amount of research supporting Harvey's model (Hiller et al., 2015).

A commonality between Harvey, and Lundh and Broman's suggested insomnia models are their focus on arousal, dysfunctional maladaptive behaviors, and interpretations. Specifically, Lundh and Broman (2000) presented sleep-interfering processes and dysfunctional sleep-interpreting factors that—mutually interact with each other—contribute to insomnia. Sleep-interfering processes include events or other internalizing emotions that cause arousal (i.e., emotional, cognitive) negatively affecting sleep. Sleep-Interpreting factors are dysfunctional perceptions and beliefs on sleep difficulties and consequences of sleep deficits. Additionally, when these two interact and independently reinforce one another they develop an insomnia variant that helps guide intervention choices based on which process, if not both, play a bigger role in the maintenance of insomnia (Lundh & Broman, 2000).

Espie took a slightly different approach to detailing characteristics involving insomnia maintenance and etiology. Espie's (2002) psychobiological model proposes

sleep is a natural occurrence that has automaticity and plasticity as the foundation and insomnia is simply a result of inhibitor factors. These subsystems that interact with the automaticity and plasticity of good sleep include physiological, cognitive, affective, and behavioral. Consequently, one inhibitory factor would negatively contribute to the disruption of good sleep considering all subsystems interact with one another. The other striking difference in Espie's model is the need for cognitive and affective factors to generate clinical insomnia. Meaning, one could be a poor sleeper and consider themselves a "good sleeper" because they have no concerns about any sleep deficits, thus have no reason to be bothered about the possibility of insomnia.

Building on different cognitive models of insomnia, Ong et al. (2012) suggested the importance of metacognitions during the development and maintenance of insomnia. Their model proposes two levels of cognitive arousal in the form of primary arousal and secondary arousal, which maintain and cause insomnia. At the primary level, cognitions are content-specific in relation to sleep deficits and the secondary level is concerned with judgmental thoughts based on the primary arousal. Ong et al. theorize mindfulness and acceptance-based interventions for insomnia target these metacognitions which leads to acceptance of the primary cognitions thus resulting in the reduction of insomnia-like symptoms.

Among the models presented are two common reoccurrences: negatively toned cognitions and dysfunctional cognitions about sleep. Of course, these commonalities are not representative in all the models, but they do present a pattern that is worth investigating. Specifically, psychological inflexibility could be the process by which

negative or dysfunctional cognitions result in insomnia. While psychological inflexibility and insomnia has shown to be correlated, there is a lack of understanding as to why this is. Understanding processes involved will aid in the development of a more efficacious model for insomnia.

Dysfunctional Beliefs and Attitudes about Sleep

Measuring these destructive sleep-related cognitions can be accomplished with the 30-item, self-reported DBAS scale (Morin, 1993). This questionnaire contains five subscales: misconceptions about the causes of insomnia, misattribution/amplification of its consequences, unrealistic sleep expectations, diminished perception of control and faulty beliefs about sleep-promoting practices. Statements for each item were manufactured from clinicians with insomnia expertise in their clinical practice (Morin, 1993). Some of the statements on the questionnaire do not have a right or wrong answer (e.g., I need 8 hours of sleep to feel refreshed and function well during the day), but it is the degree to which the individual holds that belief that can become problematic. Similar to other questionnaires, there are various forms such as the 16-item version (Morin et al., 2007).

When compared to good sleepers, those with insomnia showed a stronger belief in the consequences of sleep, diminished perception of control, sleep-promoting practices, and unrealistic sleep expectations (Morin et al., 1993). Therefore, changing these beliefs, should result in improvements in sleep. In a comparison to relaxation and a control group, cognitive-behavioral treatments produced improvements in sleep and scores on the

DBAS (Edinger et al., 2001). Additional studies found similar results. Specifically, one study found lower scores on the DBAS (less dysfunctional cognitions about sleep) were linked to improvements in sleep efficiency (time asleep divided by time in bed multiplied by 100), recorded by sleep dairies, at post treatment and consecutive follow-ups (i.e., 3-month, 12-month, and 24-month; Morin et al., 2002). A second paper used three different studies to strengthen the argument for the clinical utility of DBAS by discriminating between those with insomnia and good sleepers, DBAS response to CBT, and identifying specific improved items that were correlated to clinical outcomes for insomnia at post treatment (Carney & Edinger, 2006). Sleep-related beliefs and attitudes play a small role in predicting overall clinical improvements for insomnia but has a larger role in predicting sleep quality (26% of the variance; Jansson-Fröjmark & Linton, 2008). Finally, change in dysfunctional sleep-related cognitions over the course of a behavior therapy, cognitive therapy or CBT were related to insomnia symptom improvement at post-treatment and follow-up visits (Eidelman et al., 2016). The information provided shows the prominent role dysfunctional sleep-cognitions have in perpetuating individuals with insomnia. This also offers the clinical implications of targeting this specific maintaining factor through any intervention.

Rumination

One process that could potentially be a byproduct of dysfunctional sleep-related cognitions, and is a common theme among many insomnia models, is negative cognitive arousal. That is, active cognitive arousal—or lack of cognitive de-arousal—has been

proposed as a perpetuating factor to the development, or maintenance, of insomnia (e.g., Espie, 2002; Harvey, 2002; Lundh & Broman, 2000; Morin, 1993). Repetitive thoughts play a large role in pre-sleep cognitive arousal (Harvey, 2002; Lundh & Broman, 2000; Morin, 1993) and are also unique to individuals diagnosed with insomnia compared to healthy subjects (Harvey, 2000).

While worry and rumination do fall under the same umbrella of repetitive negative valanced cognitions (i.e., repetitive thought) that manifest in internalizing disordered symptoms (Segerstrom et al., 2000), and have both been framed as interchangeable, they each have unique content making them distinct from one another. Worry is a predominantly verbalized, negatively valanced cognition concerned with future events that is used as a cognitive avoidance strategy (Borkovec et al. 1998). Whereas rumination is used to gain insight on an individual's past conflict, believing they can prevent, or problem solve the event (Lyubomirsky & Nolen-Hoeksema, 1993; Watkins & Baracaia, 2001). Recently, empirical evidence has provided support for worry and rumination being separate in the context of insomnia. A clinical sample of individuals with insomnia that were allocated into high or low ruminators had significant differences in sleep quality, sleep efficiency and wakefulness after sleep onset but no main effects were found in the high, low worriers (Carney et al., 2010). These findings were conducted while controlling for depression, further supporting rumination playing an independent role in insomnia absent of depression (Carney et al., 2010). Above all, factor analysis revealed that rumination and worry are in fact separate constructs (Carney et al., 2010).

While the bulk of worry has consumed the research landscape in relation to insomnia, rumination has recently shown to have ties to sleep and insomnia. First, among college students, higher rumination scores were found to be associated—all measured by the Pittsburg Sleep Quality Index (PSQI)—with poorer subjective sleep quality, longer sleep latency, and higher total PSQI scores (Thomsen et al., 2003). Most importantly, when controlling for depressive, anxious, and angry mood, total PSQI scores were still correlated with rumination (Thomsen et al., 2003). Similarly, in a cross-sectional study, poor sleepers (i.e., PSQI less than or equal to 6) had relatively higher ruminations scores compared to good sleepers (Carney et al., 2006).

Another study appointed college students as low or high trait ruminators depending on their responses to the Ruminative Response Scale of the Response Styles Questionnaire (RSS), then randomized into a rumination condition or distraction condition (Guastella & Moulds, 2007). In the rumination condition, participants were asked to reflect on their performance and results of the exam they took that day right before they went to bed. Participants in the distraction condition were asked to think of non-performance related items (e.g., ”think about clouds forming in the sky”) before bed. High-trait participants allocated to the rumination condition reported poorer sleep quality compared to the high-trait participants in the distraction condition (Guastella & Moulds, 2007). Similarly, college students underwent an induced psycho stressor to further expand the directionality of rumination and sleep using objective and subjective measures (Zoccola et al., 2009). State and trait rumination were found to be a predictor of objective sleep-onset latency (SOL-O; Zoccola et al., 2009). Results also showed an interaction

between trait and stressor-specific rumination to predict SOL-O (Zoccola et al., 2009). That is, participants who reported greater trait rumination and engaged in more stressor specific rumination tend to have an objectively longer time to enter sleep from full wakefulness. Finally, albeit among college student endorsing sever depressive symptomology (Beck Depression Inventory-II, mean = 37.03), pre-sleep rumination predicted longer SOL-O (Pillai et al., 2014). Specifically, after taking account for baseline sleep disturbance and depressive symptoms, a one standard deviation increase on the RRS was associated with a 7-minute increase in time to enter sleep from a wakeful state measured with an actigraphy. Furthermore, presleep rumination was also predictive of objective and subjective SOL (Pillai et al., 2014). Together, these studies show that rumination not only has ties to sleep but offers support of rumination possibly preceding sleep quality. Although, these data do not account for the effect insomnia symptomology could possibly have on rumination.

Insomnia-specific rumination is related to insomnia. Specifically, repetitive thoughts about what has caused fatigue and cognitive deficiencies has shown to predict insomnia severity (Carney, Harris, Falco, & Edinger, 2013). Furthermore, one comparative study among individuals with obstructive sleep apnea syndrome, insomnia, and healthy subjects found higher rumination among those only with insomnia (Palagini et al., 2015). Not only is rumination unique to sleep related disorders, but it is also unique to insomnia, albeit this study only compared it to one other sleep related disorder (Palagini et al., 2015). Another study established that those diagnosed with insomnia and poor inhibitory control—an executive function involved with controlling responses and

reasoning with them—in combination with high depressive symptoms and high cognitive reprisal predicted higher rumination (Ballesio et al., 2019). Together, not only does rumination contribute to sleep deficits but evidence also supports the maintaining role rumination plays in insomnia.

Surprisingly, understanding the relationship between rumination and dysfunctional sleep-related cognitions has not been at the forefront of insomnia research. One study used a multiple regression model to help reveal dysfunctional sleep-related cognitions - as measured by the DBAS - plays a significant part in elevated sleep related rumination among individuals with insomnia (Palagini et al., 2015). Furthermore, greater insomnia severity, poorer sleep quality, and greater dysfunctional sleep-related cognitions was unique to individuals with insomnia (Palagini et al., 2015). The paucity of information about how rumination and dysfunctional beliefs and attitudes about sleep play a role in insomnia leaves room to examine their roles together.

Psychological Inflexibility

Psychological flexibility is living in the present moment, regardless of distressing states while dictating behavior based on one's values (S. C. Hayes et al. 2006). It is commonly visualized with a hexagon—coined hexaflex—with each vertices detailing a dimension that together makes up psychological flexibility: acceptance (willingness to experience any unwanted experiences), cognitive defusion (detachment from thoughts), values (personal desired qualities), self-as-context (having perspective of oneself), mindfulness (contact with the present moment), and committed action (guided behavior

based on values; S. C. Hayes et al., 2006).

On the contrary, psychological inflexibility reflects rigidity to be adaptive to their environment. This inability to be flexible often leads to maladaptive behaviors (e.g., avoidance) as a coping mechanism which often manifests into internalizing disorders resulting in a poor quality of life and causing an individual to live a life not devoted to their personal values. Similarly to the psychological flexibility hexaflex, is the psychological inflexibility hexaflex—its six dimensions include: experiential avoidance (unwilling to experience distress), cognitive fusion (judgmental of one's thoughts), attachment to conceptualized self (judgmental of previous experiences culminating in a biased perspective of self), lack of values clarity (having no clear and concise values), avoidant persistence (lack of valued-based driven behavior), and inflexible attention (inability to have contact with the present moment; S. C. Hayes et al., 2011).

Together, these 12 dimensions are often written as opposite ends of a psychological flexibility spectrum. It is rather easy to fall for this assumption, considering the explanation of psychological flexibility is written as if the absence of this trait results in psychological inflexibility and are both on one continuum (e.g., S. C. Hayes et al., 2011). However, this is not the case. Rolffs et al. (2018) explored this paradigm and found psychological flexibility and inflexibility are separate constructs that consists of six dimensions each. Additionally, each process offered distinct and unique information to global (in)flexibility (Rolffs et al., 2018). Meaning, an individual could show higher levels of cognitive defusion yet not exhibit an increase to higher levels of mindfulness. These results were later validated and added flexibility was tied to overall well-being,

while inflexibility showed ties to depressive symptoms (Rogge, Daks, Dubler, & Saint, 2019). From this we can deduce that—although correlated—these are two distinct constructs that aid in the understanding of wellness and psychological distress

Psychological Inflexibility, Rumination, and DBAS

Dysfunctional beliefs and attitudes about sleep that aid in the onset and maintenance of insomnia are signs of high rigidity among an individual. Specifically, psychological inflexibility could be a prominent underlaying variable to those with strong beliefs in their sleep-related cognitions. For example, an individual with high psychological inflexibility would be less likely to accept outcomes of having an inadequate amount of sleep or be unwilling to defuse from any of their strongly held sleep-related beliefs. Ong et al. (2012) did hint at the notion of an individual that has flexibility to their sleep deficient consequences could possibly promote healthier reactions rather than generating faulty beliefs that usually aid into the development of insomnia. To date, there are no studies that have explored the relationship these two constructs have together. This leaves open the door to further understand how these three constructs (i.e., rumination, psychological inflexibility, and DBAS) react with one another.

Furthermore, ties between rumination and psychological inflexibility are limited but there is research suggesting the inability to be cognitively flexible and rumination have a relationship. Rumination is similar to other emotion dysregulation and coping strategies, including rigid cognitive flexibility (Nolen-Hoeksema et al., 2008). For

instance, not only has rumination been tied to the inability to switch from unhelpful strategies to helpful ones in order to complete tasks, but the negative repetitive thought reinforces justification for behavioral withdraw (e.g., not experiencing distressing environmental stimuli) thus leading to a firm behavioral pattern (Nolen-Hoeksema et al., 2008). Second, affective flexibility (emotional processing)—a specific branch of cognitive flexibility—has predictive power in rumination (Genet et al., 2013). That is, inflexibility to move away from the emotional meaning of positive material predicted lower use of ruminative thoughts, while inflexibility to move away from the emotional meaning of negative material was associated with higher use of ruminative thoughts (Genet et al., 2013). When investigating rumination and any form of cognitive flexibility among those with insomnia, poor executive functions—specifically, cognitive inhibition—has predictive power of rumination (Ballesio et al., 2019). Emotional dysregulation—the inability to adequately regulate and attend to inner experiences—and hyperarousal was explained by insomnia specific rumination (Palagini, Moretto, Dell'Osso, & Carney, 2017). Additionally, the same study found those with insomnia, reported higher levels of rumination, arousal and emotional dysregulation compared to good sleepers (Palagini et al., 2017). Together, rumination and cognitive flexibility variants show promise as interacting variables but exploring rumination and psychological inflexibility through the lens of insomnia is nonexistent.

Psychological Inflexibility, and Insomnia

Albeit a novel intersection of two constructs, there is evidence to indicate there is

a link between psychological inflexibility and insomnia. In one study among 159 individuals with chronic pain, dimensions of psychological flexibility (i.e., acceptance, mindfulness, and values-based action) helped explain 19% of the variance when predicting insomnia severity (McCracken et al., 2011). Although these dimensions were contributing factors to insomnia, when demographic variables and pain severity were controlled for variance dropped to 9.9% (McCracken et al., 2011). Another study among Japanese college students, found greater psychological inflexibility to be correlated to higher levels of sleep difficulty, even after controlling for depressive symptoms (Kato, 2016).

Similarly, among U.S. college students, psychological inflexibility indirectly corresponded to changes in sleep quality and depressive symptoms, and psychological inflexibility indirectly linked the association between sleep quality and suicidal ideation (Peltz et al., 2020). Interestingly, in the same study, psychological flexibility did not have any statistically significant indirect paths with changes in sleep disturbance and depressive symptoms, or sleep disturbance and suicidal ideation (Peltz et al., 2020). This further illustrates the separate constructs between psychological flexibility and inflexibility, and a need to explore differences in healthy sleepers and poor sleepers on both constructs.

A handful of studies have attempted to improve psychological inflexibility among people with insomnia via ACT. Unfortunately, many of these outcomes were dependent on the measure used to capture sleep quality or sleep disturbance (Salari et al., 2020). In addition, few studies have investigated ACT with the aim of reducing insomnia severity

among people with primary insomnia.

Of the four studies to date, one study improved sleep quality (via sleep diary) and sleep related quality of life following 6 weekly group sessions in a sample of 10 participants that were partial or non-responders to CBT for insomnia (Hertenstein et al., 2014). Nonetheless, Hertenstein and colleagues (2014) failed to have any statistically significant effects for total sleep time, sleep onset latency, wake time after sleep onset, insomnia severity (via Insomnia Severity Index [ISI]) and the PSQI. The author did mention Acceptance and Commitment Therapy's (ACT) trait to be non-disorder specific and being more aligned with improving quality of life, leaving the door open for conflict when attempting to improve disorder specific symptomology among people with insomnia. Beyond the concerns brought upon by the author, is the discrepancy in improving sleep quality. Two different measures (i.e., PSQI and sleep diary) were used in the study to assess sleep quality, yet only one (sleep diary) showed any statistical significance (Hertenstein et al., 2014). There are unknown variables that may be contributing to this disparity.

The second study improved sleep quality, sleep duration, sleep distress, and depressive symptomology via a six week online delivered ACT intervention among 43 participants with at least subclinical insomnia (Lappalainen, Langrial, Oinas-Kukkonen, Muotka, & Lappalainen, 2019). Surprisingly, Lappalaienen et al. did not find any positive effects post-intervention on the psychological inflexibility and mindfulness measures. The authors noted this may be due to a ceiling effect and necessitates further investigation as to why this could happen.

Third, Baik (2015) provided feasibility, acceptability, and perceived helpfulness of an ACT intervention for insomnia; yet insomnia related symptom reduction was not shown. The author notes that a more methodological study (i.e., larger sample size and longer treatment length) could have provided statistical effects because there were slight insomnia symptom improvements from pre- to post-treatment (Baik, 2015). Finally, a small study with four participants with insomnia found improvements in the DBAS, experiential avoidance, sleep difficulty acceptance, and sleep quality during an eight session ACT intervention (Zakiei & Khazaie, 2019). This study increased sleep duration and decreased onset sleep latency among three out of the four participants (Zakiei & Khazaie, 2019).

These irregularities are also observed when researchers implement a modified, hybrid version of ACT. For example, quality of life and ACT processes have a greater improvement than insomnia related symptoms when treated with an ACT-like intervention for insomnia (Dalrymple et al., 2010; Daly-Eichenhardt et al., 2016). As previously stated, results like this are perhaps due to ACT not being predominantly disorder centered. Results in ACT-related studies for insomnia are further complicated by the lack of psychological (in)flexibility measures and insomnia specific cognitive measures. Although having positive effects towards insomnia is a great outcome—and the primary aim of most of the studies discussed—the lack of understanding of clear processes of change is a limitation.

Together, results suggest ties between psychological inflexibility and insomnia, yet the inconsistencies and drawbacks create further questions. Specifically,

nonsignificant findings in insomnia-related symptoms and ACT processes post-ACT leads to believe aiming to improve overall psychological inflexibility among those with insomnia may not be so straight forward and there may be other variables contributing to the relationship between these two. Researching the processes that play a role in this association is pivotal for manufacturing a highly efficient and acceptable ACT intervention for patients with insomnia.

Current Study

The current study seeks to understand the processes that explain the relationship between psychological inflexibility and insomnia among college students through dysfunctional beliefs, and attitudes about sleep and rumination. In theory, the inability to deviate from an individual's ridged truths about sleep would likely give rise to concerns about their past performance. And because of their ridged sleep-related beliefs, the individual would exhibit clinical distress regarding their sleep deficiency when attempting to problem solve their past sleep performance at a cognitive level. To clarify the relationship between psychological inflexibility and insomnia, depression, age, fear of COVID, and gender will be controlled for in all analysis.

First, we predict there will be significant correlations between all variables: psychological inflexibility, insomnia, rumination, and DBAS. Secondly, we predict DBAS and rumination will serially mediate the association between psychological inflexibility and insomnia among college students. That is, we expect increases in psychological inflexibility will be a predictive variable for insomnia severity among

college students with dysfunctional sleep-related cognitions (measured via the DBAS) and rumination assisting in the explanation between these two constructs (i.e., psychological inflexibility and insomnia) by presenting statistically significant indirect paths.

CHAPTER II

METHODS

Participants

Participants were college students enrolled in at least one class at a 2- or 4-year university, older than 18 years of age attending school, and had access to the internet. Exclusion criteria included of presence of another self-reported sleep-related disorder that was not insomnia (e.g., sleep-related breathing disorders).

To help determine the adequate sample size needed to calculate a serial mediation model with power ≥.80 and assumptions of correlations ≥.40 between the independent, dependent, and mediating variables, a Monte Carlo Simulation was used (Schoemann et al., 2017). The study aimed to recruit between 400-410 participants.

Procedure

Participants were recruited at Utah State University, Southern Illinois University, and Western Michigan University in the U.S. Recruitment began at the start of each semester (Fall 2021 and Spring 2022) by notifying students during class announcements and were assisted by two professors at their respective school. Longitudinal data were collected from participants via a unique link hosted by REDCap that guided them to the consent and appropriate questionnaires. First, agreeing participants completed a screener and if deemed eligible, they were directed to the questionnaires. Participants who completed the questionnaires at baseline were asked to complete the same questionnaires

at the 1-month follow-up. At the conclusion of the follow-up, participants were given the option to participate in the electronic gift card raffle as part of their compensation. Twenty electronic Amazon gift cards, priced at $50 each, were raffled to participating individuals.

Measures

Demographics

Participants were asked a sequence of demographic questions including age, gender, ethnicity, race, marital status, employment status, living situation (e.g., on-campus with/without roommate, off-campus with/without roommate, with family), transfer student (yes/no), first-generation student (yes/no), year in school, current number of credit hours enrolled and household income.

Insomnia Severity Index

The Insomnia Severity Index (ISI; Morin, 1993) is a self-reported 7-item measure is designed to assess insomnia symptomology. Each item is scored on a 5-point scale ranging from 0-4. Total scores range from 028, with cutoffs including: no clinically significant insomnia (0-7), subthreshold insomnia (8-14), moderate severity (15-21), severe (22-28). Higher scores indicate a higher insomnia symptomology. The ISI is a recommended assessment to measure insomnia severity (Buysse et al., 2006), and has shown sufficient internal consistency and good discriminant validity (Bastien et al., 2001). Previous studies have successfully implemented the ISI among college students participating in some form of sleep intervention (Gellis, Arigo, & Elliott, 2013; Taylor et

al., 2014). Internal reliability for this measure was good (α = 0.84).

Depression Anxiety Stress Scale-21

The Depression Anxiety Stress Scale-21 (DASS-21; Lovibond & Lovibond, 1995) is an assessment composed of 21 self-reported items. The measure is used to assess anxiety and depression symptomology and overall distress, which includes 3 subscales (anxiety, depression distress) rated on a 4-point scale ranging from 0 (never) to 4 (almost always). Each subscale is scored individually with each having its own cut-off score categorized from normal to extremely severe. The DASS short form (21 items) has shown to be reliable and valid compared to the original 42-item version (Antony et al., 1998). The DASS-21 has been used in previous samples of college students with some form of sleep deficiencies (e.g., Becker et al., 2018). Internal reliability for the depression scale was excellent (α = 0.91).

Dysfunctional Beliefs and Attitudes About Sleep

The Dysfunctional Beliefs and Attitudes About Sleep (DBAS; Morin et al., 2007) is a shortened version of the original 30-item self-reported measure (Morin, 1993) designed to assess insomnia related cognitions. This 16-item version contains four subscales (perceived consequences of insomnia, worry/helplessness about insomnia, sleep expectations, and medication) with individuals responding to each item on a visual analog scale that ranges from 0 (strongly disagree) to 10 (strongly agree). The DBAS has shown to have adequate and acceptable convergent and construct validity (Morin et al., 2007). Internal reliability for this measure was good (α = 0.84).

Daytime Insomnia Symptom Response Scale

The Daytime Insomnia Symptom Response Scale (DISRS; Carney et al., 2013) is a 20-item self-reported measure designed to assess daytime rumination related to sleep. Seven of the items stems from the Symptom-Focused Rumination Subscale (Bagby, Rector, Bacchiochi, & McBride, 2004) of the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991) with an additional 12-items added with the assistance of insomnia experts. Individuals are asked about their behavior when they were tired using a 4-point scale ranging from 1 (almost never) to 4 (almost always) accumulating in a total score ranging from 20 to 80. Higher scores indicate higher levels of rumination. The DISRS has shown to have great internal consistency ($\alpha > 0.9$) across two different samples (Carney et al., 2013). Internal reliability for this measure was excellent ($\alpha = 0.95$).

Fear of COVID-19 Scale

The Fear of COVID-19 Scale (FCV-19S; Ahorsu et al., 2020) is a 7-item self-reported scale developed to capture fears regarding COVID-19. Each item is rated on a 5-point scale, ranging from 1 (Strongly Disagree) to 5 (Strongly Agree). Total scores range from 7 to 35 with higher scores indicating a greater fear of COVID-19. The FCV-19S has shown strong psychometric properties among a general Iranian sample (Ahorsu et al., 2020) and later validated among a U.S. college sample (Perz et al., 2020). Items 1 and 7 were altered to reflect common American English expressions highlighted by Perz et al. Internal reliability for this measure was good ($\alpha = 0.86$).

Multidimensional Psychological Flexibility Inventory-Inflexibility

The Multidimensional Psychological Flexibility Inventory-Inflexibility (MPFI-Inflexbility; Rolffs et al., 2018) shortened version—from the original 60-item measure—is a self-reported questionnaire designed to assess psychological flexibility (12-items) and inflexibility (12-items). There are a total of 12 subscales between the flexibility (acceptance, present moment awareness, self as context, defusion, values and committed action) and inflexibility (experiential avoidance, lack of contact with the present moment, self as content, fusion, lack of contact with values and inaction) measures. Each item is rated on a 6-point scale ranging from 1 (never true) to 6 (always true). Higher scores correspond to higher global composite score (i.e., flexibility or inflexibility). The MPFI has shown good validity and reliability (Rogge et al., 2019; Rolffs et al., 2018; Seidler et al., 2020). Internal reliability for the inflexibility subscale was excellent ($\alpha = 0.90$). The current study used the inflexibility subscale to capture psychological inflexibility.

Data Analytic Plan

All analysis (i.e., correlations and serial mediation) were conducted using the R statistical environment (R Core Team, 2020). Serial mediation was conducted using the 'semTools' package (Jorgensen et al., 2022) to calculate three indirect effects of DBAS (M_1) on psychological inflexibility (X) and insomnia (Y), rumination (M_2) on psychological inflexibility (X) and insomnia (Y), and DBAS (M_1) and rumination (M_2) on psychological inflexibility (X) and insomnia (Y). A total effect (c)—which is produced by the X variable predicting the Y variable withholding the proposed mediating variables (M_1

or M_2)—is composed of three indirect effects and one direct effect (c'). Of the three indirect effect one consists of *X* predicting *Y* through M_1, another is *X* predicting *Y* through M_2, and the last one is *X* predicting *Y* through both M_1 and M_2 serially. Finally, the direct effect is *X* predicting *Y* while controlling for both mediating variables. An indirect effect is deemed significant from the absence of a zero within the 95% confidence interval (A. F. Hayes, 2017). Gender, age, fear of COVID, and depression were used in all regressions as covariates. To account for change over time, 1-month follow up data was regressed onto baseline data for the ISI, DASS-21, DABS, DISRS, and MPFI-Inflexibility, then the residuals from these regressions were used in the serial mediation model. Missing data was accounted for using multiple imputation because missing data was completely at random, at an item level, and the highest percentage of missing data for any one item—that did not include demographics—was no higher than 1.8%.

CHAPTER III

RESULTS

Participants

There were 660 participants that completed only the baseline and 490 participants that completed the baseline and 1-month follow up. Those that completed the baseline and 1-month follow up were used for analysis in this paper. Much of the sample was composed of college students who are full-time (94.7%), undergraduate (99.8%), female (75.1%), and White (83.7%). On average students who participated in the study completed 1.3 years (*SD* = 1.2) of college. Among 294 participants who reported having a paid or non-paid job, the average number of hours worked per week was 19.6 (*SD* = 10.2). Students who are the first in their family to attend college only made up 18.8% of the sample. There was mixed reporting when it came to the participants living situation. Specifically, 286 participants (58.4%) reported living off-campus (on-campus 41.2%) and only 54 participants (11%) reported living alone (living with roommate or housemate 88.8%). Table 1 provides greater details of participant demographics.

Correlations

Table 2 shows correlations that were conducted for variables used in the serial mediation model. The independent variable (psychological inflexibility), mediating variables (DBAS and insomnia rumination), and the dependent variable (ISI) were all significantly positively correlated.

Table 1

Demographics (*N* = 489)

Variable	*n*	%
Age *M (SD)*	20.60	4.1
Student status		
Full-time	463	94.68
Part-time	26	5.31
NA	1	0.20
Undergraduate	488	99.80
Graduate	1	0.20
NA	1	0.20
Transfer student		
Yes	106	21.63
No	381	77.76
NA	3	0.61
First generation student		
Yes	92	18.78
No	396	80.82
NA	2	0.41
Living situation		
On-campus	202	41.22
Off-campus	286	58.37
NA	2	0.41
Living alone	54	11.02
Living w/roommate or housemate	435	88.78
NA	1	0.20
Housing insecurity		
Never	214	43.67
Rarely	108	22.04
Sometimes	109	22.24
Usually	40	8.16
Always	17	3.47
NA	2	0.41
Food insecurity		
Never	145	29.59
Rarely	113	23.06
Sometimes	142	28.98
Usually	66	13.47
Always	20	4.08
NA	4	0.82

(*table continues*)

Variable	*n*	%
Gender		
Male	109	22.24
Female	368	75.10
Transgender	1	0.20
Nonbinary	6	1.22
Other	4	0.82
NA	2	0.41
Sexual orientation		
Heterosexual	392	80.00
Bisexual	68	13.88
Gay or Lesbian	10	2.04
Other	16	3.27
NA	4	0.82
Race		
Native American/American Indian/Alaska Native/Indigenous	4	0.82
Middle Eastern/North African (Non-White)	3	0.61
Pacific Islander/Native Hawaiian	-	-
Asian	16	3.27
Black	16	3.27
White	410	83.67
Latinx/Hispanic (Non-White)	22	4.49
Multiracial	16	3.27
Other	1	0.20
NA	2	0.41
Family income		
$0 - $20,000	48	9.80
$20,001 - $40,000	60	12.24
$40,001 - $80,000	123	25.10
$80,001 - $120,000	135	27.55
$120,001 - $200,000	82	16.73
More than $200,000	31	6.33
NA	11	2.24

Serial Mediation

Table 3 show results from all regressions of the serial mediation model. Analysis revealed increases in psychological inflexibility led to increases in dysfunctional beliefs and attitudes about sleep (Model 1; $B = 0.28$, confidence interval [CI]: 0.16 - 0.40) over a 1-month period. Meaning for every 1-point increase in psychological inflexibility (via

Table 2

Correlations for Variables at Baseline

Measures	1	2	3	4	5	6	7	8
1. Insomnia	-	0.48***	0.56***	0.12**	0.57***	0.43***	0.07	0.10*
2. DASS-D		-	0.44***	0.19***	0.67***	0.63***	0.02	0.12*
3. DBAS			-	0.27***	0.58***	0.42***	0.13**	0.12**
4. Fear of COVID				-	0.30***	0.25***	0.05	0.17***
5. Insomnia rumination					-	0.67***	0.05	0.18***
6. Psychological Inflexibility						-	-0.02	0.18***
7. Age							-	-0.01
8. Gender								-

Note. DASS-D = Depression Anxiety Stress Scale-Depression, DBAS = Dysfunctional Beliefs and Attitudes About Sleep.

* $p < .0\ 5$.
** $p < .01$.
*** $p < .001$.

MPFI-Inflexibility), there would be a corresponding 0.28-point increase in college students' beliefs and attitudes about sleep (via DBAS) over that 1-month. Age (B = -0.01), gender (B = 0.03), fear of COVID (B = 0.02), and depression (B = 0.05) were used as controlling variables for Model 1.

Second, dysfunctional beliefs and attitudes about sleep led to increases in daytime insomnia rumination (Model 2; B = 1.39, CI: 0.61 - 2.18) over a 1-month period. Meaning for every 1-point increase in dysfunctional beliefs and attitudes about sleep, there would be a corresponding 1.39-point increase in college students' daytime insomnia rumination (via DISRS) over that 1-month. Age (B = -0.13), gender (B = 0.55), fear of COVID (B = 0.03), and depression (B = 0.87) were used as controlling variables for Model 2.

Third, daytime insomnia rumination led to increases in insomnia symptomology

Table 3

Regressions for Serial Mediation Model

	Model 1 DBAS				Model 2 Insomnia rumination				Model 3 Insomnia			
Outcomes	*B*	SE	t	CI	*B*	SE	t	CI	*B*	SE	t	CI
MPFI-Inflexibility	0.28	0.06	4.54	0.16-0.40	2.48	0.55	4.53	1.41-3.55	-0.41	0.23	-1.74	-0.86-0.05
DBAS					1.39	0.40	3.50	0.61-2.18	0.59	0.17	3.51	0.26-0.92
Insomnia rumination									0.06	0.02	3.31	0.03-0.10
Age	-0.01	0.01	0.17	-0.03-0.01	-0.13	0.08	-1.55	-0.30-0.04	-0.01	0.04	-0.36	-0.08-0.06
Gender	0.03	0.07	0.47	-0.11-0.17	0.55	0.63	0.88	-0.67-1.78	0.11	0.26	0.40	-0.41-0.62
Fear of COVID	0.02	0.01	1.80	-0.002-0.05	0.03	0.11	0.28	-0.19-0.25	-0.02	0.05	-0.36	-0.17-0.07
DASS-D	0.05	0.01	3.93	0.03-0.08	0.87	0.12	7.42	0.64-1.10	0.22	0.05	4.33	0.12-0.33

Note. MPFI = Multidimensional Psychological Flexibility Inventory, DBAS = Dysfunctional Beliefs and Attitudes about Sleep, DASS-D = Depression Anxiety Stress Scale-Depression.

(Model 3; *B* = 0.06, CI: 0.03 - 0.10) over a 1-month period. Meaning for every 1-point increase in daytime insomnia rumination, there was a corresponding 0.06-point increase among college students' insomnia symptomology (via ISI) over that 1-month. Age (*B* = -0.01), gender (*B* = 0.11), fear of COVID (*B* = -0.02), and depression (*B* = 0.22) were used as controlling variables for Model 3.

Finally, all three indirect paths are shown in Table 4. The first path (MPFI-Inflexibility -> DBAS -> Insomnia) had significant findings (*B* = 0.39, *p* = 0.01, CI: 0.11 - 0.66). The second path (MPFI-Inflexibility -> Insomnia Rumination -> Insomnia) also had significant findings (*B* = 0.09, *p* = 0.02, CI: 0.02 - 0.16). Significant findings (*B* = 0.02, p = 0.03, CI: 0.002 - 0.05) were also present in the final path (MPFI-Inflexibility -> DBAS -> Insomnia Rumination -> Insomnia). In addition, after controlling for age, gender, fear of COVID, and depression for both the total and direct effect, the total path was not significant (*B* = 0.09, CI: -0.46 - 0.65) and the direct path was not significant (*B* = -0.41, CI: -0.86 - 0.05). These results indicate that dysfunctional sleep-related cognitions and insomnia rumination serially and independently help explain the relationship between psychological inflexibility and insomnia among college students.

Table 4

Indirect Effects

Indirect effects	*B*	*SE*	*t*	*p*	CI
MPFI-Inflexibility -> DBAS -> Insomnia	0.39	0.14	2.77	0.01	0.11-0.66
MPFI-Inflexibility -> Insomnia Rumination -> Insomnia	0.09	0.04	2.43	0.02	0.02-0.16
MPFI-Inflexibility -> DBAS -> Insomnia Rumination -> Insomnia	0.02	0.01	2.15	0.03	0.002-0.05
Total	0.09	0.28	0.33	0.75	-0.46-0.65

Note. MPFI = Multidimensional Psychological Flexibility Inventory, DBAS = Dysfunctional Beliefs and Attitudes about Sleep.

CHAPTER IV

DISCUSSION

The current study investigated additional variables (i.e., dysfunctional sleep-related cognitions and insomnia rumination) that could potentially help explain the relationship between psychological inflexibility and insomnia among college students. Correlation analysis between variables involved with the serial mediation model were conducted. As predicted, these correlation analyses showed psychological inflexibility, dysfunctional sleep-related cognitions, insomnia rumination, and insomnia were all significantly correlated. While the total effect (i.e., psychological inflexibility predicting insomnia) was not significant, the serial mediation model showed that all three indirect effects (i.e., when the mediator was only dysfunctional sleep-related cognitions, only insomnia rumination, and both) were also significant—these indirect effects results were also predicted.

Over the course of 1 month, changes in psychological inflexibility, dysfunctional sleep-related cognitions, and insomnia rumination led to increases in dysfunctional sleep-related cognitions, insomnia rumination, and insomnia, respectively. One item to highlight is dysfunctional sleep-related cognitions predicting insomnia rumination over the course of a month. As dysfunctional sleep-related cognitions are a more established perpetuating cognitive feature of insomnia, insomnia rumination has only recently been researched. This outcome provides evidence for an association between dysfunctional sleep-related cognitions and insomnia rumination. In addition, Palagini et al. (2015) that found dysfunctional sleep-related cognitions to have a significant impact on insomnia

rumination among people who were diagnosed with insomnia.

All three indirect effects that make up the serial mediation model were shown to be significant. Once both mediators (i.e., dysfunctional sleep-related cognitions and insomnia rumination) were tested, the amount of influence these mediators had the outcome reduced in size. This could have occurred due to unknown additional variables (e.g., insomnia worry) that could assist in the explanation between psychological inflexibility and insomnia. Dysfunctional sleep-related cognitions had the largest influence on the outcome when it was the only variable to help explain the connection between psychological inflexibility and insomnia. This result strengthens previous cognitive models that have suggested dysfunctional beliefs about sleep perpetuate insomnia (e.g., Harvey, 2002). While this study did not implement an intervention, these results are similar to Parsons et al. (2021) who found changes in dysfunctional sleep-related cognitions to help explain the relationship between treatment groups and changes in insomnia among seven randomized controlled trails testing CBT-I efficacy. Fewer studies have investigated the role changes in dysfunctional sleep-related cognitions has among those with sleep difficulties or insomnia (e.g., Kloss et al., 2016).

Although psychological inflexibility having predictive power for insomnia was not present in this study, dysfunctional sleep-related cognitions and insomnia rumination contributing to this relationship is still relevant. Hayes (2017) and other reserachers (e.g., Rucker et al., 2011) have cited in the importance of looking beyond the casual step approach (X and Y having a significant association prior to testing for a mediation effect of M), introduced by Baron and Kenny (1986), when testing for indirect effects. While

the confidence interval for each indirect effect does not include a zero—indicating significance—it is important to highlight the possibility of the covariates having an impact on psychological inflexibility not predicting insomnia. One previous study (McCracken et al., 2011) showed a decrease in predictive power of psychological flexibility dimensions (e.g., mindfulness, acceptance) for insomnia after demographic information was included in the analysis. Additionally, considering the relationship between psychological inflexibility and insomnia has only been investigated in the past few years, future studies should examine the impact of demographics on this association.

As insomnia rumination is a novel construct to insomnia research, the findings of this variable assisting in the explanation between psychological inflexibility and insomnia offer new evidence that insomnia specific rumination plays a role in insomnia, specifically among college students. Furthermore, psychological inflexibility is often researched within the context of improving insomnia symptomology (e.g., Hertenstein et al., 2014) and not in understanding the variables that are at play within insomnia. This dichotomy assists in providing ample evidence for the former but not the latter. Meaning, studies that aim to test interventions that improve psychological inflexibility and insomnia do not extend themselves to investigating other insomnia related variables (e.g., insomnia rumination) leaving gaps between contextual behavioral sciences and insomnia research.

It is well known how avoidance precipitates and/or perpetuates maladaptive behavior sometimes leading to a psychological disorder (Chawla & Ostafin, 2007), and this is seen within the current study and previous attempts to implement ACT among

those that have insomnia. These results, in addition, extend insomnia research that has investigated negative repetitive thinking (e.g., insomnia rumination) as a precipitating and/or perpetuating variable in insomnia, but through the lens of contextual behavioral sciences.

Outcomes from this study should be contextualized prior to generalizing. First, the serial mediation analysis was conducted with an attempt to view change in each variable over time and because of this, it is difficult to determine directionality. Second, there is reason to suggest another negative repetitive valanced cognition could be at play (i.e., insomnia worry) because of the small coefficients produced by the indirect effects that included insomnia rumination. Finally, the sample consists of only college students. This makes it difficult to generalize to the public due to the other factors (e.g., high alcohol use, screen time) that could influence sleeping difficulties that is specific to this population. Still, college students are worthy of individualized studies because their environment plays a role in their sleep habits differently compared the general population.

Together, findings show DBAS and insomnia rumination having a significant association with psychological inflexibility and insomnia among college students. There is potential to assisting future interventions by understanding and targeting these negative repetitive thoughts. Future studies should aim to uncover treatment outcomes for these variables.

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