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Sedative Hypnotic Medications: Effect on Negative Cognition and Insomnia

Austin L. Larsen

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Austin L. Larsen

Department of Clinical Psychology, Augsburg University

Jim Theisen, PhD, LP
Chair

Abby Hughes-Scalise, PhD, LP
Member

Marcia Bennett, Ph.D.
Member

Minneapolis, Minnesota

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Dedications and Acknowledgments

I would like to dedicate this work to Kelsey, who has been a constant strength, and light through grief. I would like to thank my family and friends for supporting me. Additionally, I am grateful for the support of Dr. Jim Theisen, Dr. Abby Hughes-Scalise, and Dr. Marcia Bennett. A quote that has stuck with me through this program is from The Lord of the Rings by John Ronald Reuel Tolkien 1991 and is as follows "His grief he will not forget; but it will not darken his heart, it will teach him wisdom."

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Abstract

The pursuit of healthy sleep in the United States often involves the use of prescription medications that were never intended for long-term treatment of insomnia. The use of medication to address sleep difficulty has multiple sources, including the medical system and a consumer driven economy within the United States. Behavioral interventions for insomnia have been increasing in availability and effectiveness over time, but medication use continues to be a first-line treatment for sleep problems. The focus of the current paper is the exploration of relationships between sedative use, sleep difficulties, and negative cognition in a sample of 104 adult participants who endorsed a history of sleep problems (mean age 55.6 years, 46.2% male) from the *Midlife in the United States* (MIDUS), a national study of health and well-being (History & overview of MIDUS, 2011; University of Wisconsin, 2017a; University of Wisconsin 2017b). Specifically, it was hypothesized that sedative use would be correlated with increased negative cognition. Negative cognition has been theorized to contribute to the development of insomnia, the disorder the medication was intended to alleviate. Those reporting sedative use reported more sleep difficulties than those not taking sedatives, though these groups did not differ on reported negative cognitions. No correlation was found between negative cognition and sleep difficulties for those reporting sedative use. For those not reporting sedative use, increased negative cognition was correlated with increased sleep difficulties. The lack of evidence for a relationship between negative cognition and self-reported insomnia for those who used medication could suggest that sedative use may minimize the relationship between negative cognitions and sleep difficulties. Clinical implications of the findings support non-pharmacological treatments in the reduction of negative cognition associated sleep difficulties.

CHAPTER ONE

Introduction

The quality of sleep is a source of suffering for many individuals in the United States; an estimated one third of the population reported at least one symptom of insomnia (Ohayon, 2002). A more recent estimation of the prevalence of insomnia is 3.9% - 22.1% depending on what diagnostic criteria were used to categorize the disorder (Roth et al., 2010). The presence of negative sleep in the general population within the United States may be increasing, as a previous estimate suggested that 30 to 50% of people reported symptoms of poor sleep initiation and maintenance (Taylor & Dietch, 2018).

Sleep is a necessary human behavior, although aspects of sleep can be highly variable between individuals (Jonasdottir et al., 2021). The variability in sleep onset, duration, frequency, and discontinuation is likely connected to an individual's thoughts, behaviors, environment, and development. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (5th ed. text rev.; *DSM–5 TR*; American Psychiatric Association [APA], 2022) defines insomnia as a clinically significant difficulty with falling asleep and staying asleep that is evident in poor job performance, decreased interpersonal functioning, or other areas of functioning. Cases of clinically significant insomnia within the population of individuals exhibiting symptoms are estimated to be 4 to 22 percent (APA, 2022).

As a guideline, the National Sleep Foundation recommends 7 to 9 hours of sleep ("Sleep and disease," n.d.). However, sleep architecture is just as important as the total number of hours slept and consists of various stages. Stage 1 is the state between being awake and falling asleep, when thinking and muscle activity start to slow. Stage 2 begins with the onset of light sleep; at

this stage, body temperature decreases, eye movement stops, and brain waves and heart rate gradually start to slow. The third stage of sleep is deep sleep. This is the stage of sleep that allows the body to recover from daily stressors. If we fail to initiate stage three and, ultimately, REM sleep, our bodies are not given the chance to recover from waking hours. Having the right mix of NREM (non-rapid eye movement) and REM (rapid eye movement) sleep is important in developing healthy sleep architecture because while we sleep, our bodies perform key tasks that play a role in learning, memory development, mood regulation, blood pressure, hormone production, cell repair, autoimmune system recovery, and appetite that are dependent on sleep architecture ("Sleep and disease," n.d.; Wolkove et al., 2007b).

It is common for individuals suffering from poor sleep to seek out over-the-counter or nonprescription medications (Dawson et al., 2023). Repeated use of nonprescription medications over longer periods of time than recommended has been shown to present serious risks including cognitive decline (Thomas et al., 2016), and possibly dementia (Basu et al., 2003). Three prevalent over-the-counter sleep medications available are diphenhydramine, doxylamine, and melatonin. When over the counter medications and alternative treatments are ineffective, individuals may seek medical assistance to address the symptoms associated with poor sleep. Despite findings that indicate best practices in alleviating symptoms of insomnia include both pharmacological and non-pharmacological interventions, many patients receive only pharmacological treatment (Bramness & Sexton, 2011; Holbrook et al., 2000; Lader 2011).

Benzodiazepines and z-drugs are classes of hypnotic medications with a wide range of side effects and are often prescribed to address sleep problems (Freedom, 2011; Neikrug & Ancoli-Israel, 2010; Neubauer, 2014; Wolkove et al., 2007a). Benzodiazepine medications were developed to address safety concerns in the use of an older class of medications called

barbiturates (Neubauer, 2014). Benzodiazepine medications are not processed quickly in the body and consequently result in long half-lives. This problem was addressed in the development of, yet another class of medications called z-drugs or non-benzodiazepines. Following the creation of benzodiazepines, second-generation non-benzodiazepines, or Z drugs, were created and include drugs like zolpidem, zaleplon, eszopiclone, and zopiclone (Brandt and Leong, 2017).

Arguably, the most common initial treatment for sleep problems is prescription medication (Neikrug & Ancoli-Israel, 2010). Medications commonly used for insomnia were found to include benzodiazepine receptor agonists and barbiturates. Most hypnotic sleep medications are prescribed to individuals in middle to late adulthood (Holbrook et, al., 2000). From a clinical research perspective, benzodiazepines and z-drugs are often prescribed due to evidence to support their efficacy in short term treatment of sleep disorders such as rapid eye movement sleep behavior disorders restless leg syndrome, insomnia, and periodic leg-movement disorder (Krystal et al., 2012; Wolkove et al., 2007a). Additionally, efficacy for pharmacological intervention has been found for the treatment of short-term effects resulting from sleep deprivation associated with shift work or other irregularities in sleep architecture (Roth et al., 2010). Clients may prefer pharmacological intervention to engaging in talk therapy, which may explain some of the prevalance of one intervention over the other (Garland et al., 2018). In treating insomnia, medications allow for variation in sleep frequency, onset and duration through medication type and dose (Bramness & Sexton, 2011).

A potential drawback of using both traditional benzodiazepines, as well as z-drugs, is that both can disrupt normal sleep cycles (Wolkove et al., 2007b). A common complaint from users of benzodiazepines is that they disrupt REM sleep in the third stage resulting in poor recovery. Z-drug medications have shown promising results in REM sleep, in that they do not circumvent

the REM phase of sleep like benzodiazepines do. The maintenance of the REM phase may suggest that the newer class of medication is less disruptive in its effect on sleep cycles; however, z-drugs do disrupt natural sleep progression and impact sleep architecture. Z-drugs vary in terms of time that they can be applied and elicit fewer side effects compared to benzodiazepine medications. However, they do have side effects and have time limitations for recommended use.

An assertion exists in the literature that suggests benzodiazepines should be prescribed for short term use and they should not be prescribed to address chronic conditions (Bixler et al., 1987; Committee O. T. R. O. M. 1980; Holbrook et al., 2000; Lader & File, 1987; Schutte-Rodin, et al., 2008). It is also clear that a substantial amount of the population who have been diagnosed with clinical insomnia have also been prescribed benzodiazepines (Heussler et al., 2013; Yang et al., 2011). Despite being banned for clinical application in many countries, sedative hypnotics, such as benzodiazepines, continue to be considered a viable option for the short-term treatment of insomnia (Lader, 2011). Some countries that continue to prescribe benzodiazepines to address sleep difficulty include the United States, Canada, various European countries, and several countries in southeast Asia.

Several of the negative side-effects of sedative medications used to treat sleep match the symptoms being treated (Lader, 2011). For example, some sleep medications have a negative side effect of increased anxiety, daytime drowsiness, and/or perceptual problems, all of which are also common sequelae with clinical sleep disorders. Similarity between the medication side-effects and symptom criterion of insomnia may contribute to doctors prescribing more of the drug for longer periods of time, despite a lack of evidence to support the efficacy of long-term repeated use of sedative hypnotic medication (Committee O.T.R.O.M, 1980). Research findings

also suggest the sole use of medication to address sleep difficulty could perpetuate and amplify a cycle involving medication prescription, as daytime drowsiness associated with sedative use may lead to physicians prescribing larger doses (Neubauer, 2014).

When treating insomnia, pharmacological treatments are used more frequently than alternative treatments such as Cognitive Behavioral Therapy for Insomnia (CBTI) (Heussler et al., 2013). Additionally, pharmacological interventions are used more than combined approaches that utilize both cognitive behavioral therapy in unison with pharmacological intervention. CBTI outperforms medication in long term treatment of insomnia (Morin et al., 1999), but the combined use of both CBTI and pharmacological therapy shows the best results (Yang et al., 2011). The cognitive behavioral theoretical framework provides a compelling explanation of the etiology of insomnia. This framework asserts that there are connections between our perception, cognition, sympathetic nervous system, and behavior that impact sleep quality and quantity. CBTI specifically focuses on interventions that work on cognitive restructuring and behavioral interventions meant to cultivate sleep health.

In their ground-breaking work on sleep and insomnia, Harvey (2002) suggested that the etiology of insomnia includes negatively toned cognitions. The theory suggests that negative cognition contributes to the development of insomnia. The connection between negative cognition and the development of insomnia was of interest prior to the model produced by Harvey and colleagues (2002), with one study looking at the relationship of sleep-related beliefs to disturbances in sleep (Edinger et al., 2000). The researchers found that individuals who endorsed insomnia symptoms also endorsed a higher level of negative cognitions compared to those who did not endorse symptoms of insomnia. One speculation made in this research study was that certain cognitions could raise the reported severity of insomnia symptoms in those

suffering from the disorder. Similar findings in a more recent study on individuals with insomnia showed a connection between the tendency to present with distortions in perception of total time spent sleeping and negative cognitions about sleep (Fernandez-Mendoza et al., 2011). The findings of this study indicated that perceptual distortions were common in a sample of insomniacs who slept a normal duration. Additionally, the control group displayed no such distortions, and did not report negative cognitions that were prevalent in the group of insomniacs. A review generated by Kaplan and colleagues (2009) assessing evidence supporting the connection between negative cognitions and the development of insomnia also outlined several key associations. Specifically, researchers found that experimental manipulation of negative cognition impacted subjective measures of insomnia across studies. They also found that heightened levels of negative cognition predicted shorter total sleep times.

The negative side-effects commonly associated with sleep medication like benzodiazepines and z-drugs include aspects of perception such as impaired coordination, drowsiness (Buscemi et al., 2007; Committee O. T. R. O. M. 1980; Neubauer, 2014), and poor psychomotor functioning (Freedom, 2011; Sweetman et al., 2020). Cognitive side effects commonly associated with z-drugs and benzodiazepine also include confusion, delayed motor skills, and poor balance (Wolkove et al., 2007a). The side effect of distorted perception is common among all sedative hypnotic medications to varying degrees (Lader, 2011). Several of the side effects of medications used to treat insomnia coincide with elements of Harvey's (2002) model of the etiology of insomnia, which include distortions in daytime perception and negative cognitions. Given the influence of perception on cognition outlined in Harvey's model, it is important to understand how the distortions in perception caused by sleep medications influence negative cognition and symptom severity of insomnia. However, literature exploring the effect of

sleep medication side-effects on negative cognitions is not established and represents a gap in our current knowledge. Side effects are outlined as dangerous for daytime activities due to distortions in perception during waking hours (Wolkove et al., 2007a). Because the symptoms seem to be less harmful than they are helpful in initiating sleep, many of the studies on efficacy support their use, but considerations of perceptual distortions on the development of negative cognitions have not been systematically considered. When conceptualizing the etiology of insomnia from a cognitive behavioral standpoint, the side effects of sedative hypnotic medications may go beyond daytime functioning and influence the development of disorder itself.

If the medications used to treat insomnia are producing measurable changes to perception and negative cognitions that have been linked to the etiology of insomnia, then both cognition and perception should be considered in exploring the efficacy of pharmacological interventions. The current dilemma is that the literature outlining the side effects of sleep medications do not consistently account for negative cognitions as an unwanted side effect and consequently the impact of sleep medications on negative cognitions, a known component of the etiology of insomnia, is not well understood. To assess the efficacy of a medication, measures of negative cognition and perception should be used to capture any impact a medication may have on these constructs. A review article that assessed subjective and objective screening tools available for assessment of insomnia speculated that health professionals are typically unaware of tools available, and consequently neglect to use available measures (Luyster et al., 2015). The researchers outlined subjective screening tools that are available, but some of the prominent assessments did not screen for negative cognitions (e.g., Pittsburgh Sleep Quality Index) while others did (e.g., PROMIS Sleep; Luyster et al., 2015). The prevalence of these measures was not

explicitly outlined, but a review assessing clinical care of insomnia indicated that under one third of patents with insomnia are diagnosed, and that clients seldom discuss disturbances in sleep when visiting a physician. Given the lack of assessment of insomnia, it may be speculated that when clinically assessing the efficacy of hypnotic medications in the treatment of insomnia, cognitions are seldom evaluated by clinicians, if evaluated at all. There is a clear connection between negative cognitions and the increase of problematic symptoms of insomnia (Edinger et al., 2000, Fernandez-Mendoza et al., 2011; Harvey, 2002; Harvey et al., 2017; Hiller et al., 2015). If side effects of the medications are influencing the expression of elements within Harvey's model (e.g., distorting daytime perception and/or increasing negative cognitions), the efficacy of benzodiazepine and z-drug medications may be called into question. This discrepancy could have significant implications in the clinical application of medications in the treatment of insomnia because it may be the case that medications' effects on perception, cognition, and behavior may be exacerbating the symptoms they intend to cure.

This study aimed to assess the effect that sedative use had on negative cognitions, and consequently self-reported insomnia to evaluate the potential for medications' involvement in the etiology of insomnia. To examine this issue, this research project assessed the relationship between sedative use and self-reports of negative cognition and self-reported insomnia. This study hoped to refine our understanding of sedative-hypnotics' relationship to cognition and the effect of sedative use on sleep.

CHAPTER TWO

Literature Review

Etiology of Insomnia

Disruptions in sleep can occur from a seemingly infinite range of origins, but many psychological researchers have theorized that sleep problems develop due to cognition, environment, and co-morbid illness (Buysse et al., 2011; Ebben & Spielman, 2009; Harvey, 2002). Cognitive explanations of sleep problems rely on an understanding of physiological responses to real and perceived threat. For example, a cognitive explanation for sleep problems could be ruminating on or worrying about the quality of sleep and its perceived or actual effect on daytime performance. Behavioral conceptualizations of sleep difficulty suggest that there are patterns of behavior that result in poor sleep health. Examples of behavioral explanations of sleep difficulty include irregular waking schedules, excessive time spent in bed awake, and caffeine consumption. Cognitive and behavioral models are not mutually exclusive and often reference one another conceptually and in practice.

Harvey's Model of Insomnia

In a well-known theoretical article, Harvey (2002) presented a cognitive model explaining the development of sleep related symptomology, specifically insomnia. Harvey's model attempted to define the mechanisms that create and maintain insomnia. She proposed that an individual's difficulty attaining healthy sleep could be due to an inability to self-correct feelings, thoughts, and behaviors that produce poor sleep. Their model contains six parts: negative cognitive activity, arousal/distress, selective attention and monitoring, distorted perception, safety behaviors, and beliefs (see Figure 1 below).

Figure 1

Cognitive Model for the Etiology of Insomnia
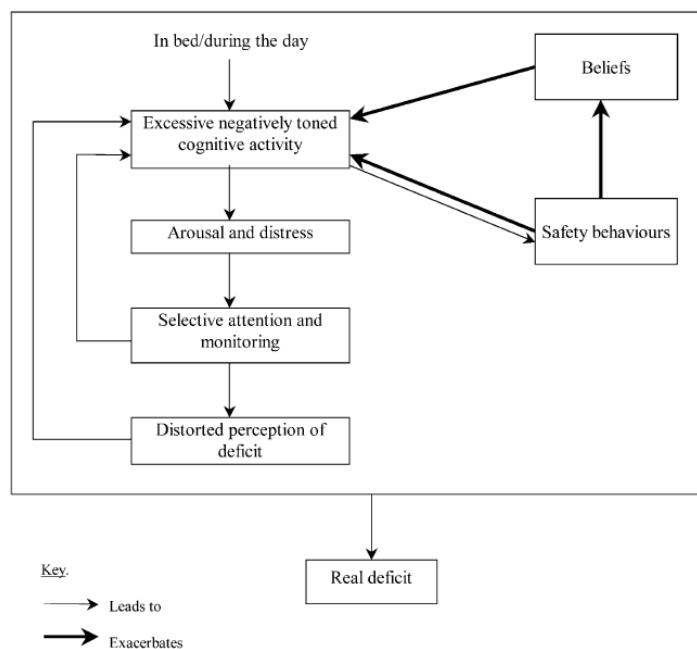
Note: This model was produced by Harvey in 2002, summarizing the Cognitive Model for the Etiology of Insomnia. From "A cognitive model of insomnia," by Allison G Harvey, 2002, *Behavior Research and Therapy*, *40*8, 69-893. p. 872.

Harvey believed that excessive negative thoughts about sleep could heighten levels of physiological arousal and affect an individual's perceptual sensitivity during waking hours, before sleep onset, and during sleep (Harvey, 2002). This suggests that high levels of negative thoughts about sleep would result in an individual being more sensitive to sound, light, and other sensory experiences. Heightened emotions and nervous system acuity could then result in an individual becoming hyper aware of internal and external stimuli, making sleep difficult. An increase in awareness is thought to result in fixation on sleep related problems, and an increase in monitoring external stimulation. Harvey proposed that this heightened level of sensitivity results

in less accurate assessment of senses resulting in distorted perception. One example of distorted perception is misjudgment of total sleep time. Distorted perception and misjudgment of qualities of sleep then serve to reinforce negative cognitions about sleep. This cycle of negative thoughts, distress/arousal, monitoring, and distorted perception perpetuate until actual symptoms manifest. The inability to correct this cycle results in insomnia.

Two additional elements that are defined in Harvey's model are beliefs and safety behaviors (Harvey, 2002). Examples of maladaptive safety behaviors that result because of negative cognition include "thought control, imagery control, emotional inhibition, and difficulty problem solving" (Harvey, 2002, p. 1). Safety behaviors exacerbate beliefs that reinforce further negative cognition. Negative cognition can lead to safety behaviors that affect beliefs, and these beliefs can further influence negative cognitions, creating a cycle of escalation. These additional elements focus on behavior, but they do not comprise the bulk of Harvey's theory. Others have proposed additional behavioral causes of insomnia (Buysse et al., 2011; Ebben & Spielman 2009; & Perlis et al, 2014).

Alternative Models of Insomnia Etiology

A behavioral conceptualization of insomnia developed by Ebben and Spielman (2009), proposes that negative cognitions and anxiety are precursors to insomnia and that these cognitions and anxiety are merely side effects of learned behaviors that cause sleep problems. These researchers suggested that targeting the symptoms of insomnia does not target the actual cause, which is theorized to be a behavioral pattern that inflates the symptoms. The primary difference between Ebben and Spielman's (2009) model and Harvey's (2002) model, is the emphasis on behavior rather than cognition. The interventions proposed by Ebben and Spielman (2009) focus heavily on sleep hygiene relation behaviors such as sleep routine, time spent in bed,

light exposure, diet, and exercise. This behavioral model emphasizes an approach to treatment utilizing behavioral manipulation that includes sleep restriction therapy, stimulus control, progressive muscle relaxation, biofeedback, and paradoxical intention.

The study of genetic predisposition is also helpful in conceptualizing etiology for insomnia. A meta-analysis conducted to explore the heritability of insomnia was conducted by two reviewers who gathered 5644 studies and selected 12 that fit the inclusion criterion of studies focusing on samples of twins (Barclay et al., 2021). The studies that were selected explored the heritability of insomnia symptoms, and intra-class correlation resulted in a heritability estimate of 40%. The researchers also examined moderation effects on heritably by age, sex, and reported symptoms. Stronger heritability was found in females compared to males, and in parent-reported insomnia symptoms compared to self-reports. The lack of significant moderator effects may have been due to the small number of studies utilized in the final analysis.

An additional study conducted by Partinen et al. (1983) used a sample of 2238 adult monozygotic twins and 4545 dizygotic twins to assess genetic predisposition for sleep length and sleep quality. Monozygotic twins are genetically identical whereas dizygotic twins are not, which allowed the researchers to control for environmental factors on the etiology of insomnia. The researchers were able to exclude participants that had a pre-existing illness, were unemployed, or doing shift work. The participants provided self-report data indicating average sleep attained per night, sleep quality, and if they shared living space with their twins. Intraclass correlations for measures of participant sleep quality and length were used to calculate Falconer's heritability estimate. The findings of this study also suggested a genetic component for the etiology of insomnia.

Sleep Health and Development in Adulthood

Qualities of sleep such as duration, onset, and frequency change as people age. The National Sleep Foundation indicates that aging adults, especially women, are at high risk for suffering from insomnia ("Sleep and disease," n.d.). Middle and late adulthood are full of shifts in circadian rhythm as well as physical and mental deterioration. Though sleep changes substantially over the lifespan, additional changes related to context also need to be considered. Unique consideration for aging adults includes changes in social involvement, comorbid disorders, environmental changes, and medical conditions that may be factors that interrupt normal sleep (Li et al., 2018).

The aging process has many age-related changes such as a decreased sleep duration, wake after sleep onset (WASO), nighttime awakenings, and daytime napping. In a 2018 review of normal age-related sleep changes, Li et al. reported that decreases in sleep duration and increase in WASO of ten minutes per decade started in young adulthood and plateaued around age 60 (Li et al., 2018). The number of nighttime awakenings does increase over the lifespan, with frequency of awakenings increasing as we age. Napping during the day appeared to change because of age, with younger adults napping less frequently compared to older adults. The increase in frequency of daytime napping appeared to be connected to biological changes. Some additional influences on sleep disruptions in aging adults were related to changes in lifestyle such as physical activity level, social engagement, and work involvement.

Another area in which sleep related change occurred is in sleep stages, and circadian rhythms. In the 2018 review of normal age-related sleep Li et al., outlined a general finding that indicated a decrease in slow wave sleep during adulthood. The researchers found that stage one and stage two sleep increased slightly while slow wave and REM sleep decreased slightly as

individuals aged. Some conflicting evidence was also reviewed that indicated an increase in REM between 75 to 85 years of age which suggested that this alteration in sleep structure may not be a consequence of normal aging.

The circadian rhythm is a natural pattern of the body that regulates various aspects of physiology that include core temperature, various hormone production, blood pressure, heart rate, sleep wake patterns (Li et al., 2018). The review conducted by Li and colleagues (2018) indicated that aging decreases flexibility of the circadian rhythm, in that changes in sleep wake cycles are not adapted to as readily compared to younger adults. A general finding outlined by the researchers was a deterioration of structures thought to be responsible for the circadian rhythm, such as the suprachiasmatic nucleus. This deterioration is thought to change the circadian rhythm, as evidenced by a decrease in stability of circadian rhythm as we age. Specific changes that occurred because of decreased circadian rhythm were: advanced sleep timing, reduced ability to adjust to changes in rhythm, and decrease in circadian amplitude. Advanced sleep timing was defined as the tendency of sleep onset to become earlier with age. A reduced ability to adjust to changes in rhythm was defined as inflexibility when adjusting to sleep disruptions. Changes in circadian amplitude included irregular levels of neurotransmitters associated circadian rhythm, and irregularities in core body temperature. Age related instability of the circadian rhythm was associated with undesired waking, being tiered earlier than wanted, change in core temperature patterns, and irregular production of melatonin and cortisol. In the 2018 review, Li and colleagues found these changes to be normative and not associated to medication or medical condition. This review suggested that when assessing changes in sleep, there are normal age-related changes that increase susceptibility to sleep related challenges.

In a review of age-related changes to sleep, common assumptions about sleep were reviewed, including the assumption that sleep initiation, sleep efficacy, and self-reported sleep quality drastically change during the aging process (Li et al., 2018). While the ability to initiate sleep prior to sleep and after nighttime waking decreases significantly with age, the effect size of studies related to this phenomenon are modest. Additionally, significant differences in sleep initiation, efficacy, and quality were found in studies that compared young children to older adults, which suggests a gradual shift opposed to a drastic difference. The amount of time spent in bed awake remained consistent in early adulthood and slowly decreased after age 60. Poor satisfaction with quality of sleep is assumed to be associated with age. However, when comorbidities and health factors are controlled for, older adults are not more likely to report poor sleep compared to younger adults. However, when clients do not have comorbid health conditions, they are less likely to be impacted by these developmental changes to sleep and consequently less likely to suffer from sleep related symptomatology (Li et al., 2018; Miner & Kryger, 2020).

A developmental period often associated with poor sleep is menopause. In a study intended to distinguish qualities of sleep secondary to menopause, a sample of 6179 women between the ages of 45 and 60 were separated into a pre and post menopause groups and completed sleep related measures. The measures included were self-reports on various qualities of sleep as well as medical and psychiatric conditions. Findings indicated an association between postmenopausal status and higher rates of sleep onset insomnia. This finding suggested that attaining satisfactory sleep may be more difficult in postmenopausal state. Additionally, the researchers found a tendency for symptoms of insomnia to increase both before and after menopause, which suggested a link between insomnia and menopause (Zolfaghari et al., 2020).

Age related and gender differences related to sleep were examined in a study conducted by Jonasdottir et al. (2021), in which aspects of sleep including timing, duration, onset, and variability were assessed using data generated by consumer wearable devises. The study utilized a data set containing sleep observations of 11.14 million nights of sleep from 69,650 adults. Participants in the sample were gathered from Japan, Germany, Russia, Taiwan, and the United Kingdom. The study resulted in a general finding that sleep duration and timing is the same between genders in similar age ranges. Men sleep less compared to women across the lifespan, and frequency of nighttime awakenings are more prevalent for women. The largest gender differences in nighttime awakenings were found in middle adulthood and may be associated with child-rearing. Age related differences indicated that younger adults differ in the length of time sleeping compared to older adults. Younger adults tend to go to sleep later, wake later in the day, and have a greater discrepancy between sleep wake time for the weekday and weekend compared to older adults. Consistent with Li et al (2018), the findings in Jonasdottir et al., (2021) suggest that older adults wake more frequently, have a shorter sleep duration compared to younger adults, and little to no changes in sleep onset variability over the lifespan. A marginal difference between genders was found for the misalignment of circadian rhythm in middle adulthood. However, large differences in circadian rhyme alignment over the lifespan appeared to be more associated with geographic region, suggesting cultural differences as opposed to biological differences. The findings in this study suggested that mean differences exist between genders, but the overlap in sleep times between genders over the lifespan may imply that overgeneralization about gender differences may be inaccurate.

Contextual aspects of aging like social engagement, sedative use, lifestyle changes, role changes, culture, and presence of medical or psychiatric comorbidity appear to play a role in

sleep health (Jonasdottir et al., 2021). Normal development may account for variation in aspects of sleep, but normal aging is not a direct cause of clinically significant sleep disturbance. Sedative use is one contextual aspect associated with sleep health and ageing. Sleep sedative use among adults is increasing, (Bertisch et al., 2014), with approximately 50% of those who report sleep problems endorsing use of sleep medication (Neikrug & Ancoli-Israel, 2010). It has been suggested that use of sedative medication increases the likelihood of older adults developing disordered breathing and REM sleep behavior disorder. Sleep medication is frequently prescribed to the elderly despite little evidence supporting chronic use of benzodiazepines is effective in treating insomnia, its potential for high-risk side effects (Bramness & Sexton, 2011; Heussler et al., 2013; Holbrook et al., 2000) and the potentially superior performance of non-pharmacological interventions such as CBTI (Dolan et al., 2010; Järnefelt et al., 2012; Sivertsen et al., 2006).

Treating Insomnia

Cognitive Behavioral Therapy for Insomnia

Cognitive behavioral therapy as a way of alleviating symptoms for insomnia is a relatively new treatment compared to pharmacological approaches but is considered a front-line treatment for the disorder (Garland et al., 2018). Cognitive Behavioral Therapy for Insomnia (CBTI) is a non-pharmacological approach to addressing sleep related symptoms that focuses on cognitions and behaviors that are connected to or directly associated with sleeping. The aim of CBTI is to change behavioral patterns and decrease the occurrence of negative cognitions. The typical length of treatment when using CBTI is between 4 to 8 weeks. This treatment for insomnia utilizes cognitive restructuring, stimulus control, and sleep restriction. Ebben and Spielman (2009) outlined additional behavioral interventions for treating insomnia which are

utilized in CBTI that included: progressive muscle relaxation, biofeedback, and paradoxical intention. A review conducted by Sateia and colleagues (2017) was developed to provide guidelines for clinical practice when treating insomnia. The researchers found that CBTI was the preferred due to its comparable outcome with other interventions, such as pharmacologic intervention, due to its more advantageous benefit to risk ratio.

Cognitive restructuring is an intervention in CBTI that is intended to manage negative cognitions related to sleep. The intention of cognitive restructuring is to evaluate beliefs that may be influencing sleep and to modify them (Garland, et al., 2018). Modification of beliefs consists of identifying cognitive distortions to raise subjective awareness of specific thoughts. Patients are asked to begin to first identify thoughts, and then to question the thoughts (e.g., asking if the thought is accurate, helpful, or if it is time to accept new information). The identified cognitions are viewed within the context of emotion and then the patient is coached on ways to restructure the original cognition to produce a different emotional reaction. Various thought restructuring techniques used to restructure thoughts could be testing hypotheses, de-catastrophizing, attention shifting, reappraisal, and reattribution (Morin et al., 2016). The techniques utilized in CBTI target cognition and help to lower negative cognitions that are thought to harm sleep.

Stimulus control is a behavioral intervention utilized in CBTI that is intended to target daytime functioning and aspects of the sleeping environment that can affect sleep (Garland, et al., 2018; Morin, 1987; Perlis et al., 2014). This is often delivered in the form of psychoeducation and common suggestions include: avoiding your bed unless sleepy, restricting activities in the bed to sleeping and intimacy, limiting the amount of time spent in bed awake to 15 to 20 minutes, waking at a consistent time, and eliminating daytime naps.

Sleep restriction is a behavioral intervention utilized in CBTI that limits patients allotted time to sleep (Miller et al., 2014). Implementing this intervention involves an assessment of the patient's sleep efficacy (SE). Sleep efficacy is a ratio of time spent in bed over time spent sleeping multiplied by 100. Measuring this score is intended to lower the time spent in bed awake by restricting the allowed time in bed. A consistent wake time is established, and records of sleep diaries are produced to generate current sleep ability (Garland et al., 2018; Miller et al., 2014).

Progressive muscle relaxation is another behavioral intervention that calls for intentionally flexing or tensing a muscle area and then relaxing while focusing on the subjective experience (Ebben & Spielman, 2009). This technique is intended to reduce physical tension in the body and reduce physiological arousal through the connection between our body and mind. An additional behavioral intervention that utilizes this connection is biofeedback. Biofeedback involves clients using real-time measurements of their physiology including body temperature, muscle tension, and brain activity. The client is then coached on ways to increase or decrease these measurements using guided imagery, diaphragmatic breathing, or other emotion regulation techniques.

An additional intervention suggested by Ebben and Spielman (2009) is paradoxical intention which is when a client goes through a process of amplifying disturbing symptoms to gain insight into the issues. An example of this would be instructing a client to try and stay awake in bed. This intervention is intended to alleviate stress the individual may feel about getting to sleep. Ebben and Spielman (2009) theorized that behavioral interventions will lower cognition before sleeping. One example discussed by these researchers speculated that stimulus control before sleeping, along with minimizing activity, would lower cognition before sleep. In

theory, this would help an individual associate the bed with only two activities and would inevitably lower cognitive load by means of behavioral modification. While behavioral and cognitive conceptualizations are helpful in developing treatments for insomnia, a physiological approach through pharmacological intervention is also a part of treating insomnia.

Locating providers that offer CBTI can be problematic for many people in the United States, as clinics that specialize in the treatment of sleep disorders are under established. One study evaluating the availability of Behavioral Sleep Medicine or BSM, specifically CBTI, found that 88% of all clinicians who specialize in BSM are located within the United States (Thomas et al., 2016). Additionally, the researchers found that only twelve of the fifty states had specialists, with most providers being located in cities with a population greater than 150,000. The findings suggested that in 2015 only 752 clinicians were trained in behavioral sleep medicine and 206 were licensed to administer CBTI (Thomas et al., 2016). A narrative review conducted by Koffel and colleagues (2018) found three primary barriers for the implementation and utilization of CBTI. These include systemic barriers limiting access to the therapy, underutilization of CBTI by clinicians, and a lack of engagement by clients (Koffel et al., 2018). The reason for the underutilization of CBTI likely has its origins in several aspects of the medical system. For example, CBTI occurs over multiple sessions and must be actively implemented by the patient and may be outperformed in the short term by utilizing medications to treat symptoms of insomnia. Another factor may be the medical systems' close relationship with pharmaceutical companies and the income associated with the industry. Many of the diagnoses in the DSM-5 have criteria that include sleep problems, yet insomnia is often considered a consequence rather than a primary target for intervention (Garland et al., 2018).

Support for cognition impact on sleep and efficacy of CBTI. A study conducted by Cronlein et al. (2014) found that the extent to which negative cognition perpetuates difficulties with sleep, may be relevant to multiple sleep disorders. These researchers administered the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS; Morin et al., 2007) to a clinical population at a sleep center. The disorders represented in this sample were primary insomnia, sleep apnea syndrome (SAS), restless legs syndrome (RLS), comorbid SAS with RLS, hypersomnia, and narcolepsy. They also utilized a control group that carried no sleep disorder diagnoses. The study utilized 229 participants, 84 of which were part of the control group. The mean age of the sample was 47.9 years. The severity of the sleep disorders was assessed using polysomnographic variables, subjective sleep parameters, the Beck Depression Inventory, and the Regensburg Insomnia Scale. Findings supported the notion that cognitive interventions play an important role in the management of sleep disorder symptom severity, as evidenced in cognitive interventions correlating with measures of sleep structure that include N2, TST, WASO, and SE%.

Robabeh (2015) found that the use of CBTI was effective in treating sleep difficulties in those struggling with methamphetamine addiction. In the study, 22 male patients with insomnia due to methadone maintenance therapy were recruited and grouped into either CBTI or a control group that used behavioral placebo therapy (BPT) for eight weeks. The BPT group treatment focused on reducing conditioned arousal. The researchers helped clients in this group develop a hierarchical list of arousal-producing stimuli, such as worrying about sleep and watching the clock. A natural list was also created and then paired with items on the list of arousing items. Interventions used in the CBTI group were sleep restriction, stimulus control, a cognitive component, and psychoeducation about sleep hygiene. The Pittsburgh Sleep Quality Index

(PSQI; Buysse et al., 1989) was used to assess sleep in each group. Both CBTI and BPT groups demonstrated significant improvement after five and eight weeks of treatment. The results of this study appeared to indicate a small, but significant, improvement of symptoms when using both cognitive and behavioral intervention compared to only the behavioral intervention of stimulus control. These results support the hypothesis that negative cognitions impact symptoms of insomnia and that they may be more efficacious than behavioral intervention alone.

The findings in the reviewed literature supports the presumption that cognitions are a key component in managing insomnia. Given that cognition appeared to have an impact on sleep architecture for multiple disorders, and improved outcome compared to behavioral interventions in several sleep disorders the effect of treatment on cognition should be considered. In testing for efficacy of treatment the impact sleep medications have on negative cognition must be considered.

Pharmacological Approaches to Insomnia

Substance use to help with sleep quality is not a new concept and is potentially as old as the use of alcohol and opium, which were two early substances used to manipulate sleep prior to the development of barbiturates and hypnotic medications (Neubauer, 2014). Sedative-medications used for sleep include benzodiazepines, barbiturates, and second-generation anxiolytics. The term sedative was originally given to any class of medication that suppressed the central nervous system. As medications for sedation broadened and became more diverse, so did their clinical utility. Sedatives could address anxiety and sleep difficulty in addition to sedation for medical procedures. Terminology for sedatives that were used to address anxiety was developed to differentiate medications by function and these medications were called anxiolytics. Sedative medications used to aid sleep were called hypnotics (Lader, 2011).

The first sedative medications to be developed were barbiturates, which had a narrow therapeutic range compared to benzodiazepines and second-generation anxiolytics (Lader, 2011). Compared to the use of barbiturates, the use of benzodiazepines is a relatively new practice. The use of barbiturates to assist with sleep was approved for the public about 90 years ago in the early 20th century. Barbiturates would be used as a sleep aid until benzodiazepine was approved for use in 1960. Benzodiazepine became the top prescribed drug in America, with diazepam tablet sales breaking 2 billion from 1969 to 1982. While these new drugs were substantially safer than the barbiturates being used in 1960, they still had many side effects. Second generation non-benzodiazepines, or Z drugs, were developed following the production of benzodiazepines and include medications such as zolpidem, zaleplon, eszopiclone, and zopiclone (Brandt & Leong, 2017; Lader, 2011). A study conducted in the early 2000s suggests z-drugs were frequently prescribed in the United States and Europe (Morlock et al., 2006).

Mechanisms of Action. There are various biological mechanisms of action when considering the effects of sedative-hypnotics on the CNS (Meyer & Quenzer 2005). The effects of sedative-hypnotic medications on the body have been connected to the neurotransmitter (NT) GABA. Sedative-hypnotic medications, such as benzodiazepines and barbiturates, act on receptors that are activated by the NT GABA at the GABA$_A$ receptor. GABA binds to the GABA$_A$ receptors and is responsible for changes inside the cell that cause inhibitory effects that range from mild sedation to general anesthesia. In this class of medication, the dose can vary in its effects on the subject, with higher doses producing sleep and general anesthesia, and lower doses producing relief from anxiety and sedation. At high non-therapeutic doses, these medications can be fatal. Both benzodiazepines and barbiturates appear to act on the same type of receptors with slight differences that make benzodiazepines marginally more therapeutic due

to their inability to simulate GABA (Meyer & Quenzer, 2005). The difference between barbiturate medications and benzodiazepine can be found in their effect on the CNS or the pharmacodynamics of the medication (Meyer & Quenzer 2019). Both barbiturate and benzodiazepine medications act on the $GABA_A$ receptor. Barbiturates are the less therapeutic of the two when treating sleep problems, due to barbiturates' tendency to produce a more powerful and less controlled level of sedation (Meyer & Quenzer, 2005). However, these medications act in similar ways physiologically. The action of a medication on the body plays a critical role in the symptoms and the therapeutic efficacy of the medication. Similarly, z-drugs also interact with GABA and have a propensity to selectively effect $GABA_A$ a1 receptors. This propensity increases its ability to target sedation and avoid other types of effects that may be undesirable (De Haas et al., 2010). The most prevalent receptors in our CNS are for GABA, therefore, medications that regulate the effects of this NT are widespread in their effect.

The effects of the body on the medication are important in determining the efficacy of the medication and explaining how different medications work so they can be administered with optimal efficacy. When considering the body's effect on medications (i.e., pharmacokinetics), the focus for defining the process is on evaluating the ways in which the body processes the medication (Meyer & Quenzer, 2005). Differences between benzodiazepine and z-drugs are found in the rate that they are processed by the body, with z-drugs being processed by the body faster compared to benzodiazepines. Aside from differing processing rates, both medications act on identical physiological mechanisms to produce sedative effects.

Side Effects. Benzodiazepine and z-drugs fall under a broad class of medication known as anxiolytics and the common side effects of these medications vary in severity and quality depending on the specific medication being used. Daytime side effects after habitual use as well

as prescribed brief use of benzodiazepines include: impaired coordination, drowsiness, compromised judgment, ataxia, and cognitive decline (Committee O. T. R. O. M. 1980; Sys et al., 2020).

Common side effect of both benzodiazepines and z-drugs impact aspects of cognition, medical health, and psychomotor functioning (Freedom, 2011; Sweetman et al., 2020). Additional effects associated with benzodiazepines and z-drugs are increased tolerance, withdrawal, and alteration in sleep behavior. The addiction potential for benzodiazepines is low, but withdrawal symptoms are significant and include anxiety, apprehension, tremor, insomnia, nausea, and vomiting (Brandt & Leong, 2017; Committee O. T. R. O. M. 1980). Some medical health side effects include nausea, somnolence, headaches, dizziness, and drowsiness (Brandt & Leong, 2017; Buscemi et al., 2007; Neubauer, 2014). Adverse psychomotor and cognitive side effects including confusion, delayed motor skills, and poor balance (Brandt & Leong, 2017; Wolkove et al., 2007a).

The use of sleep medications among adults, especially aging adults, has increased in the last ten years according to a nationally representative sample (Bertisch et al., 2014). In a sample of 32,328 participants that were given an in-person survey inquiring about sleep related topics, 55% reported using multiple sedatives to help with poor sleep. Multiple prescriptions make it increasingly difficult to predict side effects. Examples presented in this mini-review (i.e., Neikrug & Ancoli-Israel, 2010) suggest that sedative medications used for sleep increase the likelihood of older adults developing sleep problems, such as sleep disordered breathing and REM sleep behavior disorder.

Clinical Application of Sleep Medications. The clinical utility for sedative-hypnotic medications increases as residual daytime side effects are minimized and the propensity for tolerance is lowered (Lader, 2011; Meyer & Quenzer, 2005). Medications that can be quickly eliminated from the body are of interest when considering the treatment of insomnia because they presumably lower the chances of additional daytime side effects. There are some countries that do not allow prescriptions of benzodiazepines and some that do, suggesting a lack of clarity around their efficacy and safety. The efficacy of the drug, even for short term use, is not fully understood because getting to sleep and staying asleep are not necessarily indicative of healthy sleep. Lader (2011) took a more conservative and skeptical approach to determining the safety and benefits of benzodiazepines. Evidence presented in Lader's review suggests that long term exposure to benzodiazepines results in loss of attention, perception, cognitive ability with an emphasis on verbal memory, and a resulting host of potentially hazardous behaviors. An additional concern with the use of this medication is the development of tolerance.

A more favorable take on the use of medication is presented by Neubauer (2014), where characteristics of hypnotic medications are reviewed by their qualities including half-lives, side effects, route of application, and dose, to clarify how each medication can address specific forms of sleep disorder symptomology. The argument is made in favor of benzodiazepines and z-drugs used to promote sleep. Neubauer argues that medications vary in their characteristics, and thus, may have increased utility depending on the client's individual needs. The perspective in Neubauer's review casts benzodiazepine medications and z-drugs in a favorable light, concluding that they are relatively safe, which challenges the observations made in Lader's (2011) and Okajima and colleagues (2013) research.

Ultimately, the negative effects of long-term use suggest that the most appropriate use of sleep medication is in short term treatment. One reason that short term use is suggested is that the medication does well with decreasing sleep latency and lowering the number of times that people wake during the night (Wolkove et al., 2007a). While short term use will help individuals get to sleep and stay asleep, extended use may result in negative side effects (Freedom, 2011), the most common of which are outlined above.

Both pharmacodynamic and pharmacokinetic factors, as well as individual demographics, determine the effectiveness of a medication (Neubauer, 2014). Pharmacologists have been grappling with developing substances that have fewer side effects and have made some advancements with the creation of z-drugs. Z-drugs have a different molecular structure than benzodiazepines, but the biological mechanism of action in the body is the same. One strength of this new medication is that its molecular shape allows it to have a much shorter half-life, which lasts about 1 to 6 hours as compared to that of benzodiazepines, which last for several days. However, this benefit does not exempt users from several of the negative side effects commonly associated with benzodiazepines, such as headaches, nausea, and disorientation. The shorter half-life means that individuals whose sleep is interrupted by nocturnal awakening may have the option of taking a sleep aid when they wake up as long as it is within the time limitations prescribed. Neubauer (2014) argued that the reason patients experience negative side effects is that the dose, timing, route, or medication type are incorrect. He makes the case that pharmacological treatment of insomnia can vary significantly in terms of the dose, route, time, and medication used, which is considered one strength of the approach.

Pharmacological intervention is often a primary treatment for insomnia and providers may be underestimating the significance of side effects due to a lack of needed knowledge

regarding side effects. One study assessing clinician self-reported knowledge of common side-effect of hypnotic medication indicated that over half the sample of 181 clinicians endorsed a lack of knowledge around side effects (Heussler et al., 2013). In addition to a minimization of potentially key side effects, the length of prescription for benzodiazepine type medications is often longer than what is recommended, which is concerning as therapeutic value of these medications lowers over time (Bramness & Sexton, 2011). Aging adults constitute a large proportion of users of sleep medication use, as well as those with clinically significant insomnia symptoms. Aging adults account for roughly 50% of those that report sleep problems. It may be that the reported sleep problems in aging adults may not be a consequence of aging, but rather, a result of peripheral factors such as medication use, cultural expectations, and environment (Neikrug & Ancoli-Israel, 2010).

Best Practices for Treating Insomnia

Treating Insomnia Using CBTI

The two primary intervention types for insomnia have historically been behavioral/ psychological treatments and pharmacological interventions. A review conducted by The American Academy of Sleep Medicine developed a guideline for clinicians in the application of behavioral and psychological treatment for chronic insomnia (Edinger et al., 2021). The task force created to develop the review assessed current literature to determine the efficacy of cognitive and behavioral interventions, and to develop a gradient for effectiveness of different interventions within the scope of cognitive behavioral treatments. Efficacy was assessed using The Grading of Recommendations Assessment, Development and Evaluation. This assessment looked at four major factors that included ratio of harm to benefit, patient preference, resource consideration and quality of empirical evidence. Using this method, the research team developed

two recommendation types of strong and conditional. Strong recommendations were given to interventions that should be utilized for most adult clients suffering from chronic insomnia. Conditional recommendations were given to interventions that the research team was less certain of its efficacy given one of the four considerations mentioned above. Consequently, interventions that received a conditional recommendation would rely more heavily on clinical judgment and consideration of the client's preference, value, and context.

The use of CBTI received a strong recommendation from the review (Edinger et al., 2021). A strong recommendation was also given to the use of emotion regulation strategies such as relaxation training, and the course of this treatment was typically four to eight sessions. Fewer sessions ranging from one to four received a conditional recommendation for use. The use of stimulus control, sleep restriction, and relaxation by themselves for single session treatments all received conditional recommendations for use. The implementation of single session of general sleep hygiene received a conditional recommendation against use. Additionally, there is some evidence to support the idea that CBTI is superior to some sleep medication in its ability to alleviate symptoms (Sivertsen et al., 2006), reduce dependence on benzodiazepines (Dolan et al., 2010), and treat medication resistant clients (Järnefelt, et al., 2012; Okajima et al. 2013).

Treating Insomnia Using Pharmacological Intervention

In general, medications for insomnia are recommended for short-term use not exceeding five weeks (Bixler et al., 1987; Holbrook et al., 2000; Lader & File, 1987; Sateia et al., 2017; Schutte-Rodin, et al., 2008;). The general recommendation of short-term use has some exceptions for clients who continue to improve the symptoms of insomnia, and experience minimal negative side effects with long term use under regular assessment by clinicians (Sateia et al., 2017). Best practice treating insomnia with pharmacological intervention is informed by

efficacy studies. The efficacy of sedative hypnotic medication is somewhat contested in psychological literature for a variety of reasons and therefore, best practice when administering said medications can be controversial.

A meta-analysis intended to provide guidance on when and how to administer common sleep medicines to adults indicated that for many benzodiazepines and z-drugs the literature does not provide strong enough evidence to say how to best use medications and emphasizes shortfalls of our current knowledge (Sateia et al., 2017). The considerations made in this analysis to assess the quality of empirical support for medication use in treating insomnia included assessment for inconsistent results between studies, assessment of publication bias evident in research funding being provided by drug representatives, and analysis of results that pass clinical significance thresholds given a 95% confidence interval. Using these considerations, a grading system was created that indicated the quality of research to support the various sleep medications. In addition to quality of evidence, the researchers considered benefit to harm ratios, as well as treatment effect in relation to burden on clients. Of note, the data available for benefit to harm ratios using sleep aids and knowledge of adverse events related to use are limited, thus most decisions made in the review relied on the clinical judgment of the research team. All benzodiazepine and z-drugs involved in the meta-analysis resulted in a weak recommendation because the evidence of benefit to the client was unclear, and the quality of empirical support was low. This method differed from methodology used by the FDA in approving substances, in that the researchers did not exclusively rely on statistical significance of objective and subjective measures of sleep health in relation to a placebo group.

Considering the evidence reviewed, the researchers provided recommendations on medication dose, sleep maintenance, sleep onset, use with comorbid disorder, length of

treatment, and developmental considerations (Sateia et al., 2017). Dose recommendations were dependent on the dose used in the studies conducted utilizing the medication. The recommended does for z-drugs ranged from 2 milligrams (mg) to 10 mg. The recommended dose for benzodiazepines ranged from .25 mg to 15 mg. All benzodiazepine and z-drugs included in the review were useful in treating sleep maintenance and sleep onset symptoms aside from Zaleplon and Triazolam, which were only recommended for use in sleep onset insomnia. Very few studies were found concerning the application of pharmacological intervention in cases of insomnia with comorbidities. Consequently, little guidance as it relates to best practice in instances of comorbid disorders is available. The review did indicate a limited number of studies that suggest some level of efficacy for both benzodiazepines and z-drugs. However, it was highly suggested that clinicians utilize clinical judgment in those cases given the low quality of empirical support. Treatment time for sleep medications is generally short term (no more than five weeks) except for some short acting z-drugs that may be useful for longer term treatment given appropriate conditions. If a client is not able to receive CBTI or is not receptive to the therapeutic intervention, chronic use may be necessary. When using sleep medications long-term, it is recommended that patients' sleep quality is monitored, individual considerations are made for the appropriateness of the medication, and regular medical consultation is given to assure no adverse effects develop. In general, long-term use of sleep aids is discouraged when non-pharmacological therapies are available such as CBTI.

Developmental considerations for best practice include the differing pharmacokinetics and pharmacodynamics of benzodiazepines and z-drugs for aging clients (Sateia et al., 2017). In geriatric populations, lower doses of benzodiazepines and z-drugs were recommended. It was also recommended that benzodiazepines be avoided due to an increased risk of negative side

effects compared to younger adults, and z-drugs be restricted to short term treatment not exceeding 90 days. The researchers indicated that medications with shorter half-lives and lower effective doses reduce negative symptoms common in medications that have long half-lives with high effective doses. Consideration of individual circumstances was strongly advised for filling prescriptions. Essentially, it was suggested that providers use clinical judgment and patient preference when considering pharmacological intervention for adults with insomnia, as literature on efficacy of medication use is weak. Before considering the use of medication, clinicians should use behavioral treatment and exhaustively review the patients' profile and history for symptoms that may have developed after previous medication use (i.e., Neikrug & Ancoli-Israel, 2010).

Discrepancies Between Empirical Support and Clinical Standard

In an archival study using records from The Norwegian Prescription Database (NorPD), Bramness & Sexton (2011) obtained the data of patients who had filled a minimum of one prescription for benzodiazepine medications between January of 2004 to October of 2009. The sample size for this study was 513,558 participants. The researchers estimated the number of individuals using benzodiazepines at around 61 per 1000 persons. They also found that 65% of all users of benzodiazepines were women. The data indicated that patients seldom used benzodiazepines and only sporadically filled their prescriptions. The average amount of medication prescribed to participants that filled at least two prescriptions was .3 milligrams per day. The researchers estimated the typical length of use to be between 9 and 15 years, with longer durations of use in patients who used larger amounts of the medication. In their estimate of the timeline, the researchers assumed that some participants may have been engaging in long-term use prior to the sample time, which is a limitation of the study. The findings in this study

were compelling for several reasons. First, the significant periods of time that individuals have continued to take medications for insomnia suggest a lack of adherence to recommended use. Second, the low volume of medication taken was typical for individuals receiving long term prescriptions, suggesting that chronic use occurs at low doses. Finally, the stability of use over time indicted that individuals were not filling prescriptions periodically (Bramness & Sexton, 2011). Though this study may not generalize the population in the United States due to its sample being gathered in Norway, it demonstrated a contradiction between empirically supported advice advocating for short term use, and the reality of prescriptions being long term.

The overutilization of prescription sleep medication over non-pharmacological approaches appears to contradict data supporting the claim that CBTI outperforms hypnotic medications in short-term treatment of insomnia as well as long-term treatment (Sivertsen et al., 2006). Sivertsen and colleagues conducted a randomized double-blind placebo-controlled study that compared CBTI to a non-benzodiazepine called Zopiclone. The researchers studied the efficacy in the application of these interventions separately in a population of older adults. The number of participants used in this study was 46 and the mean age was 60.8 years old. The primary outcome measures in this study were ambulant clinical polysomnographic data and sleep diaries. These measures were used to develop a sleep efficacy score and assess slow wave sleep. The measures were gathered at baseline, 6 weeks follow up, and 6 month follow up. Improvements in sleep efficacy and polysomnographic data were observed after treatment and after follow-up for the group receiving CBTI. Zopiclone did not differ from placebo and the researchers proposed that CBTI was superior to Zopiclone as an intervention for insomnia in older adults. Though this study did not account for other types of non-benzodiazepine medications, it does have implications in the application of sleep related intervention. The

findings suggest that when treating older adults, CBTI may be superior to Zopiclone and similar non-benzodiazepine type medications.

CBTI and the Reduction of Hypnotic Medication

A study conducted by Dolan et al. (2010) found that when CBTI was administered to a clinical population taking medication for insomnia, their use of hypnotic medication decreased without prompting from the clinicians. The researchers of this study recruited 32 participants who were actively taking a hypnotic medication for sleep difficulty and were able to complete eight sessions of CBTI. The outcome measures used in the study were sleep diaries, an insomnia severity self-report, a self-report measure of negative beliefs and attitudes about sleep, and self-report. Using a chi-square goodness-of-fit test, the researchers found that medication usage at baseline (87%) compared to medication usage after treating patients with CBTI (59.3%) lowered significantly. While this study did not disprove the effectiveness of hypnotic medication for the short-term treatment of insomnia, it did promote the idea that CBTI was a viable option to promoting less drug dependency in populations using hypnotic medications. When sleep disorders were treated with medication alone, a potential risk could be an increased dose and the length of time prescribed. Medication effectiveness decreases with longer use in most circumstances due to the development of tolerance (Freedom, 2011; Sweetman et al., 2020). The introduction of CBTI resulted in a decrease in medication use which could be considered a modification of behavior in addition to the treatment of negative cognitions.

Developing a tolerance when using benzodiazepines is a common problem for individuals who have been prescribed sleeping medication (Freedom, 2011; Sweetman et al., 2020). CBTI provides a promising alternative for individuals who develop tolerance to medication, and as such CBTI should be considered in treatment development. In a study

conducted by Okajima et al. (2013), non-pharmacological intervention consisting of CBTI, in combination with behavioral interventions, was compared to pharmacological intervention using only medication. A sample of 68 participants were recruited from a clinical population that suffered from chronic insomnia and who had been unresponsive to prior pharmacological treatments. Participants were allowed to choose between two groups: a treatment as usual (TAU) group that consisted of medication prescription, and a CBTI group that also included the elements of the TAU group. The treatment group that used sleep medication and CBTI produced lager changes in the scales used to measure symptoms of insomnia compared to the group that was given only medication and clinical advice. As mentioned previously, all participants were unresponsive to prior pharmacological treatments. The findings would suggest that CBTI is effective in alleviating symptoms of insomnia when clients are resistant to pharmacological intervention. The study demonstrated that pharmacological intervention did not produce symptom relief for chronic insomnia and that CBTI did. Negative cognitions are targeted in the CBTI group and not in the treatment as usual group. The lack of symptom relief in the treatment as usual group suggested that negative cognition played a role in symptom relief.

Another study conducted through an occupational resources department at the Finnish Broadcasting Company in Helsinki (Järnefelt, et al., 2012) assessed the use of CBTI with 33 employees that reported symptoms of insomnia and were motivated to participate in treatment. The participants were predominantly women, with 27 out of the 33 participants identifying as women. All participants had experienced symptoms of insomnia for an average of 7 years and 67% of them had taken sleep promoting medications (SPM). The most common SPMs used were benzodiazepine-like hypnotics and 86% of participants used this type of medication. The results suggested that for individuals not experiencing symptom relief from medication use, CBTI may

be an alternative to hypnotic medication and may be an attractive treatment modality for individuals looking to improve productivity considering many of the negative side effects of hypnotic medication can lead to a decrease in productivity. Discrepancies in productivity may be a consequence of distortions in perception which is a side effect of benzodiazepine-like hypnotics. The findings supported a relationship between increased productivity when CBTI was used as a primary treatment rather than the use of benzodiazepine-like hypnotic medications. The clinical application of CBTI in response to decreased effectiveness or addiction to medications is promising. Several studies reviewed suggest that CBTI is generally applicable in its efficacy for most clients, but the use of medication is less generalizable (Bixler et al., 1987; Committee O. T. R. O. M. 1980; Holbrook et al., 2000; Lader & File, 1987; Schutte-Rodin, et al., 2008). Medication and CBTI are the most effective interventions when treating insomnia. These treatments are considered best practices and are a first line of treatment when addressing insomnia in a clinical setting.

Taken together, the use of CBTI in the treatment of insomnia in adult populations is strongly recommended due to its superior risk benefit ratio compared to medication, stability of symptom remission over time, ability to treat medication resistant clients, and ability to lower dependence on sleep medication. Benzodiazepine and z-drugs were both recommended for short term treatment. The duration of z-drug prescription was slightly longer due to z-drugs shorter half-life compared to benzodiazepine. Current evidence to inform the use of benzodiazepines and z-drugs was lacking and consequently recommendations for CBTI were preferable. Clinical practice in the treatment of insomnia in adults does not appear to coincide with empirical recommendation. Specifically, the length of treatment of pharmacological intervention, and utilization of pharmacological intervention compared to CBTI are inconsistent with current

literature on best practice. It is important to note that the treatment of insomnia using CBTI specifically incorporates the assessment and alteration of negative cognitions. Targeting negative cognitions seems to be one explanation as to why CBTI is more efficacious in the treatment of insomnia compared to medication only approaches.

Rationale

Hypnotic medication may interrupt the elements of healthy sleep that are necessary for a restful night's sleep (Manconi et al., 2017), but this may not be evident to the individual taking the medication because hypnotic medication is effective at activating sleep initiation and maintenance (Neubauer, 2014). Further, sleep progression is complex, so taking a sleeping medication for a short duration of insomnia may be effective in convincing patients that they have received successful treatment, as client perception of sleep quality is often inaccurate compared to objective measures such as polysomnography (Trimmel et al., 2021). Given the complicated relationships between negative cognitions, sleep medication, and changes in sleep quality secondary to these two factors, the use of anxiolytic medications and the impact on negative cognitions should be considered when assessing the efficacy of sleep medication.

Support for the role of negative cognitions in sleep issues can be seen in literature that supports targeting cognitions to improve sleep quality. Specifically, the implementation of cognitive and behavioral approaches to treat insomnia has produced promising results. However, from a clinical practice perspective, the implementation of CBTI is overshadowed by the use of prescription medication. Additionally, clinicians conducting intake sessions with patients often do not ask about insomnia and, consequently, it often goes undiagnosed with comorbid disorders (Garland et al., 2018). The literature reviewed in this proposal supported the use of CBTI for long term treatment of insomnia when compared to treatment with hypnotic medication

(Colecchi, 1999; Dolan et al., 2010; Järnefelt, et al., 2012; Lader, 2011; Morin & Sivertsen et al., 2006; Sateia et al., 2017; Schwartz & Carney, 2012; Sivertsen et al., 2006).

Measures used to assess the efficacy of medications in studies reviewed have previously included measures of cognitive performance, polysomnography, and variations of self-report measures that assess sleep onset (Krystal et al., 2012; Kyle et al., 2017; Rosenberg et al., 2005; Roth et al., 2010). None of the studies reviewed assessing medication efficacy utilized measures that evaluate negative cognitive functioning. The relationship between negative cognitions when using hypnotic medications compared to negative cognitions when not using hypnotic medications may not be assessed given the absence of measures related to negative cognition in such studies. Assessing negative cognition is necessary as the etiology of insomnia relies on the development and proliferation of such negative cognitions (Buysse et al., 1989; Cronlein et al., 2014; Harvey, 2002).

Using data from the *Midlife in the United States* (MIDUS), a national study of health and well-being, an evaluation of medication use and its effect on self-reports of cognition and self-reported insomnia will be conducted (History & overview of MIDUS, 2011; University of Wisconsin, 2017a; University of Wisconsin 2017b). The aim of the research was to assess the relationship between medication use and negative cognitions, which was identified as a mechanism of action when understanding sleep difficulties from a cognitive behavioral framework (Harvey 2002). To examine this research aim, this study included three hypotheses:

Hypothesis 1: For those with a history of sleep problems, those who use sedatives will show greater sleep difficulties than those who do not use sedatives. Medications become ineffective after repeated use, suggesting they do not address the underlying mechanisms involved in healthy sleep (Ebben & Spielman, 2009). Common side effects of benzodiazepine

type medications are alterations in sleep behavior (Freedom, 2011; Sweetman et al., 2020), somnolence, and drowsiness (Buscemi et al., 2007; Neubauer, 2014). These common side effects appear to suggest that sedative use could lead to increased sleep difficulties, despite their intended use being to decrease sleep issues.

Hypothesis 2: For those with a history of sleep problems, those who report use of sedatives will report greater negative cognitions than those who do not use sedatives. CBTI utilized a theoretical connection between excessive negative cognition, physiological arousal, attention, and perception (Harvey 2002) to suggest a possible connection between negative cognition to the development of insomnia through distortions in perception. A well-documented side effect of hypnotic medication is altered daytime performance, psychomotor functioning, dizziness, and changes in cognitive functioning (Buscemi et al., 2007; Freedom, 2011; Neubauer, 2014; Sweetman et al., 2020) that result in distorted perception (Bramness & Sexton, 2011; Committee O. T. R. O. M. 1980; Neubauer, 2014; Heussler et al., 2013; Holbrook et al., 2000). Perception may inform our cognition, and if increased negative cognition is a consequence of altered perception brought on using sedative medications, it follows that those who use said medications would report more negative cognitions relative to those who do not.

Hypothesis 3: For participants with a history of sleep problems who endorse current sedative use, there will be a positive relationship between self-reported insomnia and negative cognition, such that, as negative cognition increases, self-reported insomnia severity also increases. For participants with a history of sleep problems who do not endorse current sedative use, negative cognition will not be correlated with self-reported insomnia. A theoretical connection outlined by Harvey and colleagues (2002) proposed an explanation for the development of insomnia: as dissatisfaction with self-reported insomnia increases, so do severity

of negative cognitions about sleep. The impact of behaviors on beliefs then contributes to the

development of negative cognitions (Ebben & Spielman, 2009; Harvey, 2002). The relationship

between cognition and self-reported insomnia may be influenced by talking hypnotic medication,

as sedative use can result in perceptual distortions (Brandt & Leong, 2017; Buscemi et al., 2007;

Freedom, 2011; Neubauer, 2014; Sweetman et al., 2020; Sys et al., 2020; Wolkove et al., 2007a)

which in turn may increase negative cognitions (Harvey, 2002). The increase in negative

cognitions may then disrupt sleep and create sleep problems (Edinger et al., 2000, Fernandez-

Mendoza et al., 2011; Harvey et al., 2017; Hiller et al., 2015). If negative cognition is an

underlying mechanism responsible for the development of insomnia, such that negative cognition

increases when self-reported insomnia decreases, then introducing medications that increase

negative cognition through perceptual distortion should further the deterioration of self-reported

insomnia. If hypnotic medications are indirectly exacerbating the development of negative

cognitions, and negative cognition exacerbates symptoms of insomnia, then medication use may

worsen self-reported insomnia. If this is the case, it will be evident in self-reports of sleep

initiation, maintenance, daytime performance, and measures of negatively toned cognitions.

CHAPTER THREE

Methods

Procedures

The data used for this study was from *The Midlife in the United States* (MIDUS), a survey carried out by the John and Catherine MacArthur Foundation, which sought to gather data on development during the midlife between the ages of 25 to 74. The aim of the MIDUS survey was to examine the role of social factors, behavior, psychological factors, and variation in health among adults. The survey has been conducted at various times following its founding in 1995; some of the participants from previous years returned to participate in following waves of the survey, which created longitudinal data. The sample size varied by year; during the original sample, the number of participants was 3487. Oversampling of urban areas was conducted to increase the generalization of the sample. Psychological factors assessed by the survey included but were not limited to personality traits, well-being, positive and negative affect, goal commitments, and sense of control. A more detailed outline of the data set may be found at MIDUS website, http:// midmac.med.harvard.edu/research.html (History & overview of MIDUS, 2011).

Data for the (MIDUS) study was gathered during separate time points and sorted into groups based upon endorsement of sedative use for sleep and the respective time point (History & overview of MIDUS, 2011). Each section, or time point, is called a wave. This study utilized two waves of data collection. Wave one was carried out between 1995 and 1996 (University of Wisconsin, 2017a) and wave two was carried out between 2002 and 2009 (University of Wisconsin 2017b). Participants were provided a monetary incentive of $20 for completing a

phone interview and written survey at wave one and $60 for completing the same phone interview and survey at wave two. Subjects were contacted by phone and asked to participate. Participants were randomly selected using existing telephone banks and provided verbal consent to the operator. After consenting to participate in the study, a 30-minute phone interview was conducted. Following the phone interview, two self-administered questionnaires of approximately 55 pages in length were mailed to participants for completion. The self-administered questionnaires were returned by mail.

Participants

The current study consisted of 104 participants from the MIDUS dataset. Clients who reported sedative hypnotic use during wave 1 and who continued participating in the study at wave 2 were included in the final analysis. Only participants who endorsed the use of sedative hypnotic medication in wave 1 were used, to ensure that all participants had a history of sleep problems.

The ages of participants ranged from 33 and 83 years old with an average age of 55.6 years old. Of the participants who completed the study, 48 (46.2%) were male and 56 (53.8%) were female. Of the 104 participants that were included in the final analysis 3 (2.9%) identified as multiracial, 90 (86.5%) as White, 4 (3.8%) as Black and/or African American, 2 (1.9%) as Native American or Aleutian Islander or Eskimo, 1 (1%) as Asian or Pacific Islander, and 4 (3.8%) as other.

Measures

Self-Reported Insomnia—The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed. text rev.; *DSM–5 TR*; American Psychiatric Association [APA], 2022) defined insomnia as a clinically significant difficulty with falling asleep and staying asleep. To assess symptoms

related to self-reported insomnia in the current study, insomnia symptoms were assessed on the

MIDUS wave 2 with the question: "During the past 30 days, how often have you experienced

trouble getting to sleep or staying asleep?" Ideally, it would have been better to gather more

information about sleep structure and daytime performance to assess symptoms of insomnia.

However, a single item question has been previously used to assess the relationship between

insomnia and well-being (e.g., Karlson et al., 2013). Additionally, single question responses have

been used in previous research to assess the relationship between mortality rates and the duration

of sleep (Kripke et al., 2002). The data in the MIDUS was scored so that high scores reflected

low insomnia symptoms.

Excessive Negative Cognition— The MIDUS dataset does not contain a standardized

measure to capture the construct of negative cognition. Consequently, a measure was developed

by the current researcher using available survey items. Previous measures for the construct of

excessive negative cognition were used to guide the development of the current measure for

negative cognition. A systematic review identifying assessment instruments that were utilized to

measure key aspects of the cognitive model of insomnia model developed by Harvey and

colleagues (2002), provided a list of measures of negative cognitions related to sleep (Hiller et

al., 2015). The review further identified two cognitive styles present in negative cognitions:

rumination and worry. Ruminations were defined as negative cognitions in which a prediction is

made about the cause of subjective mood. The construct of worry was defined as future oriented

prediction. These researchers specifically identified a scale called the Glasgow Sleep Efficiency

Scale as a measure that had previously been used to measure negative cognitions in those with

sleep difficulties (GSES; Broomfield & Espie, 2005). In particular, the GSES included items that

assess worry, as defined by Hiller and colleagues (2015). The authors noted that the GSES and

other measures associated with the construct of negative cognitions do not have cut offs for clinical significance, which make the utility of the measure in clinical settings more difficult. Additionally, the GSES lacks replication that would help bolster its reliability and validity (Broomfield & Espie, 2005; Hiller et al., 2015). The GSES was identified as a potential example measure for generation of an excessive negative cognitions measure for the current study. One consideration for the utilization of this measure was its psychometric properties.

Broomfield & Espie (2005) assessed the psychometric properties of the GSES utilizing a sample of individuals suffering from insomnia (n=89) and a group of individuals who reported having healthy sleep (n=102). Total item correlation on average was 0.64 with a range of 0.49 to 0.73. These results suggested that internal consistency was not acceptable within the measure because item correlation did not exceed 0.80 (Broomfield & Espie, 2005). The GSES did positively correlate with the Dysfunctional Beliefs and Attitudes about Sleep (Morin et al., 1993), and did not correlate with a measure of anxiety, which bolstered its concurrent validity (Broomfield & Espie, 2005). The findings indicated that the GSES distinguished between those with insomnia and those without, which supported the discriminant validity of the measure. It should be noted that data was available to distinguish sedative use within the sample of insomnia patients. The GSES, however, was not able to differentiate between those taking medication and those not taking medication.

The MIDUS data set did not contain a specific measure of negative cognitions related to sleep, and consequently, a new measure needed to be created using existing self-report survey questions available in the MIDUS data set to measure negative cognitions. An available scale utilized in the MIDUS assessed anxiety, and some of the individual items on this scale

specifically asked about sleep and negative cognitions. Items within this anxiety scale asked

participants if, in the past 12 months, they experienced the following reactions:

- Were you restless because of your worry?

- Were you keyed up on edge or had a lot of nervous energy?

- Were you irritable because of your worry?

- Did you have trouble falling asleep?

- Did you have trouble staying asleep because of your worry?

- Did you have trouble keeping your mind on what you were doing?

- Did you have trouble remembering things because of your worry?

- Were you low on energy?

- Did you tire easily because of your worry?

- Did you have sore or aching muscles because of tension?

Items with the MIDUS anxiety scales included questions such as (a) "did you have

trouble remembering things because of your worry?", and (b)"were you irritable because of your

worry?" did not specifically ask about nighttime cognitions, and as such were problematic

because available measures used to assess negative cognitions all assess nighttime negative

cognitions specifically (Hiller et al., 2015). Several of the items within these scales assessed

cognitions, and some items within these scales aligned with items of measures utilized to

measure negative cognitions. An item analysis of these scales resulted in an acceptable number

of consistencies between the MIDUS anxiety items and the GSES items.

The GSES contains a series of seven items that assess negative cognitions (Broomfield &
Espie, 2005). Similarities between the items on the MIDUS anxiety scale and items on the GSES
are as follows:

- The GSES item "*I put too much effort into sleeping when it should come
  naturally*" is similar to the anxiety scale in the MIDUS data set item *"had trouble
  staying asleep because of your worry."*

- The GSES item *"I put off going to bed at night for fear of not being able to sleep"*
  is similar to the anxiety scale in the MIDUS data set item *"were restless because
  of your worry."*

- The GSES item *"I worry about not sleeping if I cannot sleep"* is similar to the
  anxiety scale in the MIDUS data set item *"had trouble falling asleep."*

- The GSES item *"I am no good at sleeping"* is similar to the anxiety scale in the
  MIDUS data set item *"Were low on energy."*

- The GSES item *"I get anxious about sleeping before I go to bed"* had no
  comparable items to items from the scales in the original MIDUS data set that
  were not already used to represent other GSES items.

- The GSES item *"I worry about the consequences of not sleeping"* is similar to the
  anxiety scale in the MIDUS data set item *"tired easily because of your worry."*

The final scale developed from the original scale items in the MIDUS study contained a
total of five items that resemble items in the GSES scale (see Table 1).

Table 1

MIDUS to GSES

MIDUS Question	GSES Question
NO COMPARABLE QUESTION	I put too much effort into sleeping when it should come naturally
Did you have trouble staying asleep because of your worry	I feel I should be able to control my sleep
Were you restless because of your worry	I put off going to bed at night for fear of not being able to sleep
Did you have trouble falling asleep	I worry about not sleeping if I cannot sleep
Were you low on energy	I am no good at sleeping
NO COMPARABLE QUESTION	I get anxious about sleeping before I go to bed
Did you tire easily because of your worry	I worry about the consequences of not sleeping

Note: This table depicts what MIDUS questions were used to represent items of the GSES.

High scores on the *MIDUS to GSES* measure would be indicative of decreased levels of negative cognitions. The range of scores possible using this measure could be 5 to 20. Each survey item can receive a subscore between 1 and 4 (except for the trouble falling asleep item --- that needed to be re-scaled to be in line (original: 1 = yes, 2=no; adapted: 1 = yes, 4 = no). High scores reflected high frequency of negative cognitions and were coded as follows: most days = 4, about half the days = 3, less than half the days = 2, never = 1, DK= 0 (see table 1). Survey item B1SA10G falling asleep needed to be modified so that responses for *no endorsement* (scoring -1) were accounted for. Responses for no endorsement on this survey item were coded as 0 so they would not influence participant scores.

To assess reliability of this *MIDUS to GSES* measure for the current study, a Cronbach's alpha was utilized. A Cronbach's alpha ranges from 0 to 1, with higher values indicative of stronger internal validity. The current study measured the construct of negative cognition and achieved a Cronbach's alpha of 0.75. This result suggests adequate reliability.

Sedative Use— To assess the presence or absence of sedative use, a question in the MIDUS study that was related to barbiturate and sleep pill use was utilized during wave 2. The first question asked was "Did you ever use any of the following substances on your own in the past 12 month: sedatives, including either barbiturates or sleeping pills on your own (e.g., Seconal, Halcion, Methaqualone)?". The possible responses to this question were coded as *yes* = 1 and *no* = 0 with scores of 1 indicating the presence of sedative use and scores of 0 indicating an absence of sedative use. To control for a history of sleep problems, participants used in the final analysis needed to endorse the sedative use variable in wave 1. Responses on this item from wave 1 of the MIDUS study were used as inclusion criteria and wave 2 responses on this item were used in the analyses.

Data Analysis

Research Design

Hypothesis 1: For participants with a history of sleep problems, those who use sedatives will show greater sleep difficulties than those who do not use sedatives. The independent variable used in this hypothesis was sedative use. This dichotomous variable created one group that endorsed sedative use and one group that did not. The dependent variable was insomnia symptoms, which was measured using a continuous Likert scale of self-reported insomnia intended to measure severity of sleep disturbance. This hypothesis will be tested using an independent samples t-test with sedative use as the grouping variable.

Hypothesis 2: For participants with a history of sleep problems, those who report use of sedatives will report greater negative cognitions than those who do not use sedatives. The independent variable used in this hypothesis was sedative use, a dichotomous categorical variable. The dependent variable was the excessive negative cognitions scale developed to resemble the GSES. As the dependent variable is continuous, this hypothesis will be tested using an independent samples t-test with sedative use as the grouping variable. The focus of interest in this analysis is participants differing based upon endorsement of sedative use, and this analysis will allow us to compare the means of participants' excessive negative cognitions related to sleep across these two groups (those who endorse sedative use and those who do not).

Hypothesis 3: For participants with a history of sleep problems who endorse current sedative use, there will be a positive relationship between self-reported insomnia and negative cognition, such that, as negative cognition increases, self-reported insomnia severity also increases. For participants with a history of sleep problems who do not endorse current sedative use, negative cognition will not be correlated with self-reported insomnia.

In the first correlation, only those who endorse sedative use at wave 2 will be included in the bivariate Pearson correlation to investigate the relationship between negative cognition and self-reported insomnia. To ensure a robust confidence interval, bootstrapping will be utilized in the analysis. In the second correlation, only those who do not endorse sedative use at wave 2 will be included in the bivariate Pearson correlation to investigate the relationship between negative cognition and self-reported insomnia. To ensure a robust confidence interval, bootstrapping will also be utilized in the analysis. The continuous variables in both correlations are excessive negative cognitions, which was assessed using a scale developed to resemble the GSES, and insomnia symptoms.

Power and Effect Size

The error rates for this study consisted of an alpha of .05 and a beta of .08. Error rates of .05 and a beta of .08 have been used in similar studies (Jonasdottir et al., 2021; Krystal et al., 2012) and are standard in current psychological literature. The anticipated effect size in this study was moderate, considering previous research using a similar data set produced a medium effect size (Karlson et al., 2013). However, the sample size used in Karlson et al (2013) was much larger (n=4,014) than the sample being used in this analysis (n=104). A sensitivity power analysis using G*power (Faul et al., 2007) was used to estimate the effect size of the hypothesis in the present study. The power analysis produced an r = 0.27, which indicates that the study would be unable to reliably detect correlations smaller than r = 0.27.

Software

The statistics program IBM SPSS Statistics Grad Pack 29.0 PREMIUM was used to conduct all data analysis.

Ethical Issues

Consent

When the original data was collected (History & overview of MIDUS, 2011), participants in the study were informed that the survey was designed to study health and well-being during the middle years of life. They were told that participation would entail completing a telephone interview and two mail questionnaires. A study fact brochure was mailed to respondents who asked for more information before deciding and a re-contact telephone appointment was made after they received the brochure. Participants who agreed to participate gave verbal consent to

participate and the phone interview was conducted. As an archival, publicly available dataset was used in the current study, informed consent for the current study is not required.

Risks

Risks of physical harm, mental harm, and social discomfort to participants in the original study were minimal. The risk of physical injury because of completing a self-survey is minimal as it does not require physical exertion aside from writing a response or verbally responding to the question. Responding to the survey was optional, so the risk of causing mental harm was unlikely as participants could end participation at any point. Participation in the original study included taking four surveys over the course of approximately nine years. The phone-survey and self-survey took approximately an hour to complete and monetary compensation was provided for the hour of time it took to complete the survey. Participants risked disclosure of sensitive and personal information, but the data was not connected to participants' identifying information. The survey and all data gathered from the survey were deidentified, which preserved participant anonymity and eliminated the possibility of social discomfort for the current archival study.

Deception

No deception was used when gathering the survey data for the National Study of Midlife in the United States, and no deception was used in the current archival study.

Confidentiality

All data for the MIDUS study was de-identified prior to its release to communities conducting research. As all available data was already de-identified, no further measures to protect confidentiality were taken for the current study.

Information and Debriefing

Given the nature of the original study (i.e., participants agreed to participate in a study that contributed to a publicly held/available database), debriefing for the original study was not necessary. Providing an opportunity for participants to see the results of the current study is not possible because identifying information for participants was not available. However, the results of this study will be provided in a summary to the University of Wisconsin-Madison, Institute on Aging, as this group is responsible for the MIDUS.

Retention of Data

The data that I utilized for this research will be deleted once it is sent to IDUN.

Permissions

The data used was over 5 years old and available for public use at https://www.icpsr.umich.edu/web/ICPSR/series/203/studies. All materials used in conducting this research are in the public domain and require no purchase or permission.

CHAPTER FOUR

Results

Sedative use in wave one was used as a screening variable to demonstrate a history of sleep difficulties. Those participants who endorsed use in wave one and completed the required survey questions in wave two were included in the final analysis. Consequently, 104 participants were viable for use in the final analysis. The ages of participants ranged from 33 to 83 years old with an average age of 55.6 years. Of the participants who completed the study, 48 (46.2%) were male and 56 (53.8%) were female. Regarding race and ethnicity, 3 (2.9%) identified as multiracial, 90 (86.5%) as White, 4 (3.8%) as Black and/or African American, 2 (1.9%) as Native American or Aleutian Islander or Eskimo, 1 (1%) as Asian or Pacific Islander, and 4 (3.8%) as other.

The three variables utilized in this statistical analysis included: a Likert scale survey item assessing self-reported sleep difficulties, a categorical variable assessing sedative use, and an ordinal variable assessing negative cognition. The variable assessing self-reported sleep difficulties had 104 participants respond. Of those participants, the mean response was 3.3 with a standard deviation of 1.74, suggesting that, on average, participants struggled with sleep once a week to several times a month.

The second variable assessed current sedative use. Frequency analysis of this survey item indicated that 18 participants endorsed using sedative hypnotic medication use, and 69 did not endorse sedative use, leaving 17 participants who did not respond to the survey item.

The third variable assessed negative cognition. Participants who were unwilling to provide a response or who indicated that they did not understand the question were excluded from the final analysis. The average score for participants within this variable was approximately

12.19, with a standard deviation of 3.5. Only 47 participants provided a response for each item

making up the negative cognitions scale, leaving 57 participants with this data.

All planned analyses for the current study were parametric tests. A primary assumption of

parametric tests is that the sampling distribution is normally distributed. To check for abnormal

distribution, *Figure 1* shows a bar graph that depicts the number of participants per scale for the

Likert scale variable assessing self-reported insomnia. The graph depicted in *Figure 1* suggests

that a greater number of participants reported high frequencies of sleep problems compared to

participants who reported lower frequencies. However, the sample size used was greater than 30,

so normality of the data can be assumed.

Figure 1
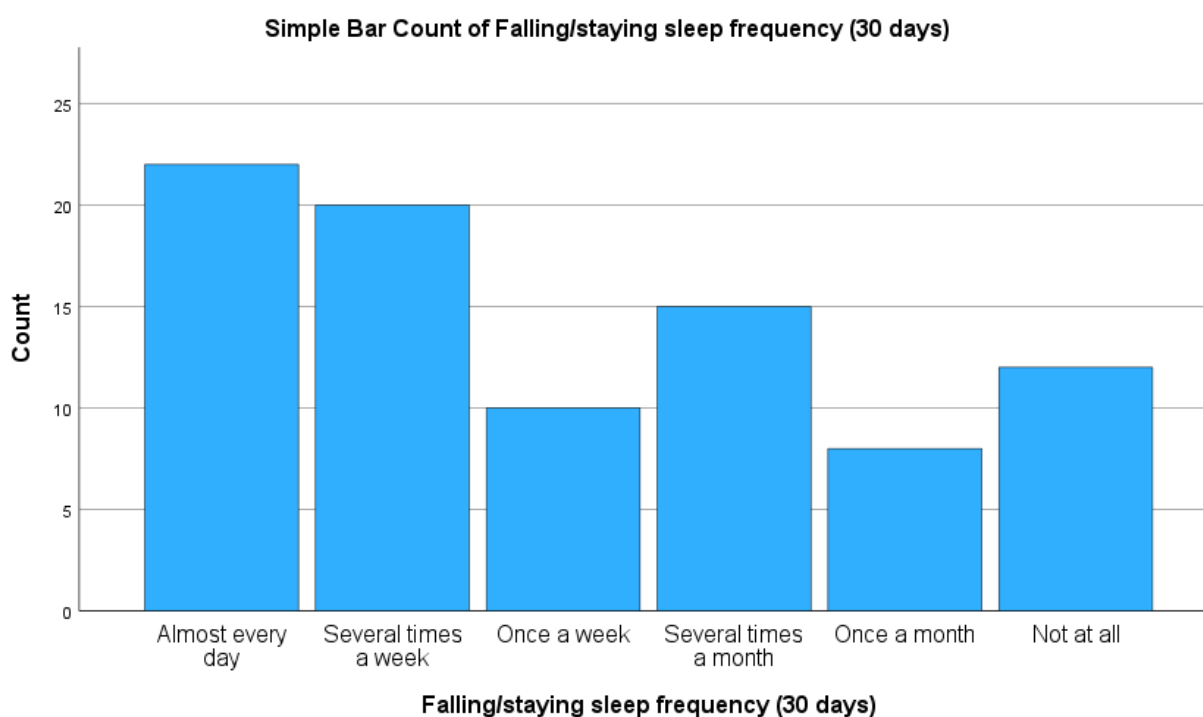
To check for abnormal distribution, *Figure 2* shows a bar graph that depicts the number

of participants in each scoring category from 5 to 20 in the scale created for negative cognition.

The chart suggests that scores on the fringes of the measure are less prevalent compared to scores

in the middle of the measure. The data appears to be normally distributed within this variable and

the sample size used was greater than 30, so normality of the data can be assumed.

Figure 2

Simple Bar Count of NegCogSleep
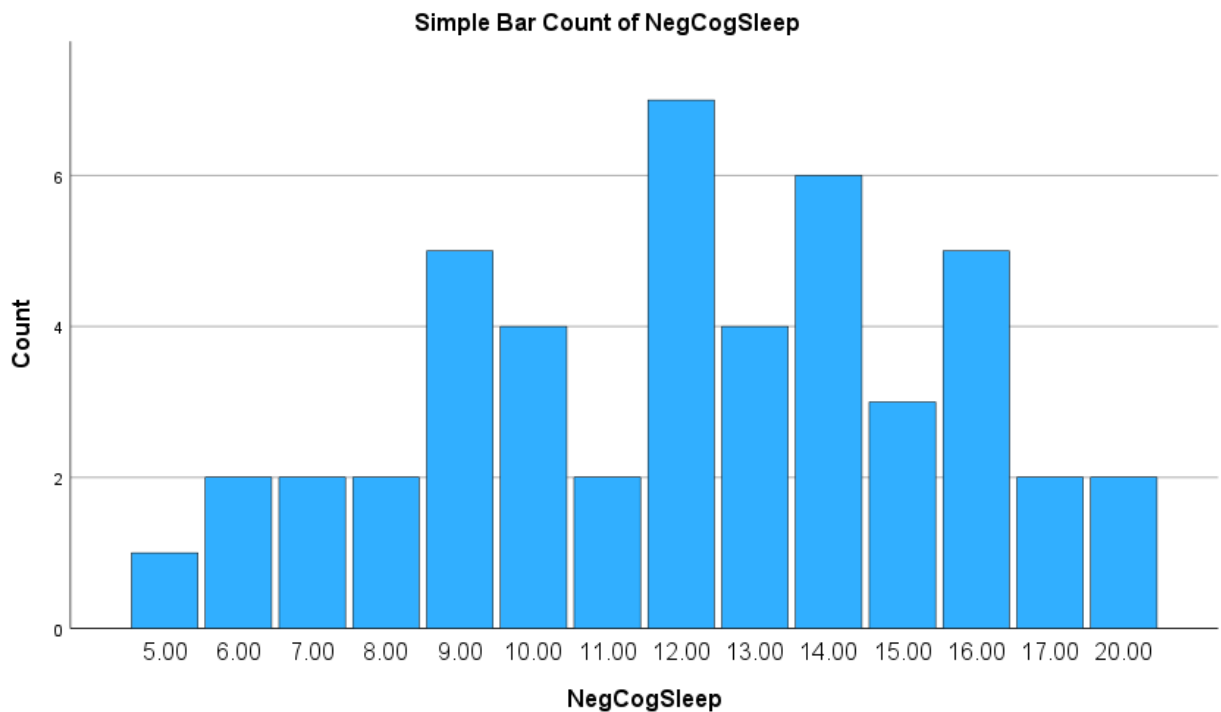
To further assess the normality of the data, a probability-probability plot was developed

which compared the probability of participant self-reported insomnia against the normal

distribution. The S-shape of the data in Figure 3 suggests skewness in the sample of participant

self-reported insomnia. However, the sample size was larger than 30, so normality can be

assumed.

Figure 3

Normal P-P Plot of Falling/staying sleep frequency (30 days)
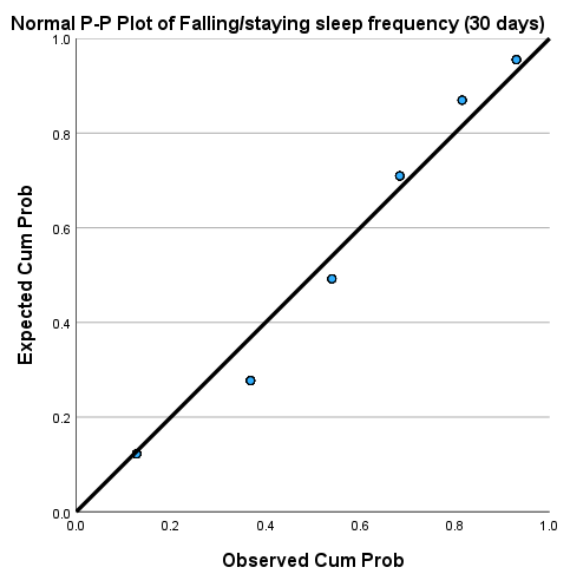
An additional measure taken to further assess the normalcy of the data was the development of a probability-probability plot which compared the probability of participant negative cognition against the normal distribution. The slight S-shape of the data in figure 4 suggests skewness in the sample of participant negative cognition. However, the sample size was larger than 30, so normality can be assumed.

Figure 4

Normal P-P Plot of NegCogSleep
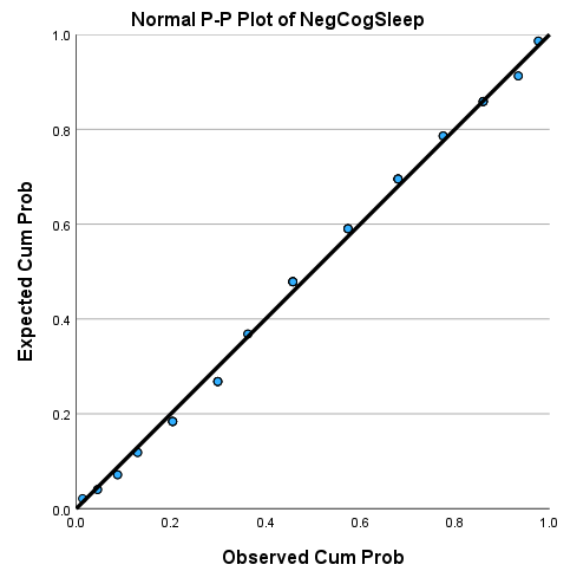
The figures presented above indicate that the data may have slight abnormalities in its distribution. However, the number of participants included is large enough to meet assumptions of a parametric model. To reduce bias, bootstrapping was utilized for all analyses. Bootstrapping is a robust method of correcting for bias.

Hypothesis One

An independent t-test was used to investigate hypothesis one: For participants with a history of sleep problems at wave one, those who used sedatives (n=18) at wave two were hypothesized to show greater sleep difficulties and report more symptoms of insomnia than those who did not use sedatives (n=69) at wave two. Sedative use at wave two was used as the grouping variable. On average, participants who used medication reported worse sleep (M = 2.17, SD = 0.34), than those who did not use medication (M = 3.26, SD = 0.089). It was found that self-reported symptoms of insomnia where significantly higher in groups using sedatives compared to those who did not use sedatives $t(85) = -2.431$, $p = 0.017$ BCa 95% CI [-1.989, -0.199].

Hypothesis Two

An independent samples t-test was used to investigate the second hypothesis: For participants with a history of sleep problems at wave one, it was hypothesized that those who reported use of sedatives at wave two (n=18) would report greater negative cognitions than those who did not use sedatives at wave two (n=69). Sedative use at wave two was used as the grouping variable. The dependent variable was the excessive negative cognitions scale developed to resemble the GSES. Despite a slight difference in the level of negative cognitions reported by those who used medication (M = 12.63, SD = 4.07) compared to those who did not use medication (M

= 12.03, SD = 3.64), the reported level of negative cognition was not significantly different across groups t(38) = 0.403, p = 0.689 BCa 95% CI [-2.39, 3.57].

Hypothesis Three

Two separate bivariate Pearson correlations were used to investigate the third hypothesis: For participants with a history of sleep problems who endorsed current sedative use, it was hypothesized that there would be a positive relationship between self-reported insomnia and negative cognition, such that as negative cognition increased, self-reported insomnia severity also increased. For participants with a history of sleep problems who did not endorse current sedative use, negative cognition will not be correlated with self-reported insomnia. One correlational analysis investigated sedative users (n=18) and a second correlational analysis investigated non-sedative users (n=69). Findings of these analyses indicated that for individuals using sedative medications, negative cognition was not correlated with self-reported insomnia, r(8) = .425 p=0.294. For individuals not taking sedative medications, negative cognition was significantly correlated with self-reported insomnia, r(32)= .430 p=.014.

CHAPTER FIVE

Discussion

The goal of the current study was to explore the relationship between negative cognition and sleep. Specifically, the primary area of focus for the current study was to better understand the relationship between negative cognition and sleep quality when taking sleeping medication. Exploration of the influence sleeping medications may have on negative cognition will help clinicians inform the treatment of insomnia, and will also help researchers direct future exploration of potential effects of sleeping medication on negative cognition.

Major Findings

First Hypothesis

The first hypothesis in this study predicted that individuals using sleep medication would endorse worse sleep compared to people who do not use sleep medications. Findings of the current study indicated that individuals who utilized sedative medications reported significantly worse sleep when compared to individuals who did not utilize sedative medications. These findings support the first hypothesis.

This finding aligns with previous literature suggesting that increased acuity of the symptoms of insomnia are treated using medication (Heussler et al., 2013; Yang et al., 2011). In many respects, it is intuitive that a significant difference in sleep difficulty was found between groups of individuals who use sedative medications and groups of individuals who do not. If individuals are experiencing higher acuity in symptoms of insomnia, it logically follows that individuals experiencing such sleep difficulties will pursue medical advice to treat their poor sleep. Greater symptom endorsement may result in the medical system utilizing pharmacological

intervention. The tendency to seek medication in the presence of higher acuity is likely due in part to the promotion of sedative use within the population.

Second Hypothesis

The findings from the t-test utilized in the second hypothesis indicated that those who used medication and those who did not use medication did not differ significantly in self-reported negative cognition. This finding did not support the second hypothesis, which suggested that individuals who utilized medication for sleep may have greater occurrences of negative cognition compared to a sample of people who do not take medication for sleep.

Literature on the side effects of medications used to treat insomnia have yet to explore medication's role in negative cognition, instead focusing primarily on medical health and psychomotor functioning (Freedom, 2011; Sweetman et al., 2020). Documented side effects of sleep medication include increased tolerance, alteration in sleep behavior, and withdrawal symptoms, including anxiety, apprehension, tremor, insomnia, nausea, vomiting (Brandt & Leong, 2017; Committee O. T. R. O. M. 1980), somnolence, headaches, dizziness, and drowsiness (Brandt & Leong, 2017; Buscemi et al., 2007; Neubauer, 2014). Several of the adverse side effects of sedative medication involve alterations in perception. Well-documented side effects of hypnotic medication include altered daytime performance, psychomotor functioning, dizziness, and changes in cognitive functioning (Buscemi et al., 2007; Freedom, 2011; Neubauer, 2014; Sweetman et al., 2020) that result in distorted perception (Bramness & Sexton, 2011; Committee O. T. R. O. M. 1980; Heussler et al., 2013; Holbrook et al., 2000; Neubauer, 2014). Although a significant result was not found in the current study, further exploration of the relationship of distorted perception caused by sedative medication and its influence on negative cognition is warranted.

Third Hypothesis

The third hypothesis utilized two separate correlations to investigate differences in the relationship between negative cognition and self-reported insomnia based on the presence or absence of sleep medication. It was expected that taking medication for sleep would result in a positive relationship between self-reported insomnia and negative cognition, such that as negative cognition increases, self-reported insomnia severity also increases. In contrast, the findings from the first correlational analysis indicated that there was no relationship between poor sleep and negative cognition for individuals who reported using sedative medication. These findings contradict cognitive explanations for the etiology of insomnia, as theories for the etiology of insomnia identify negative cognition as a driving component of the development of insomnia (Edinger et al., 2000, Fernandez-Mendoza et al., 2011; Harvey et al., 2017; Hiller et al., 2015). Consequently, a relationship between poor sleep and negative cognition was expected. The absence of significant findings in this correlation may suggest that medications are not addressing the underlying etiology of the disorder.

In the second correlation, a significant relationship was observed between self-reported insomnia and negative cognition for individuals who did not endorse the use of sedative medication. As sleep became worse, negative cognition increased in the current sample of individuals who did not endorse sedative use. While this finding was contrary to the predicted relationship in the current study, it can still be interpreted through the broader theoretical framework for the development of insomnia (Harvey, 2002). One potential explanation for this result is that people who were not taking medications were experiencing poor sleep and receiving no treatment. Consequently, the relationship between negative cognition and sleep quality may have been more pronounced compared to the group that was receiving treatment. This

interpretation is supported by the broader literature in its connection between insomnia and negative cognition, as outlined in the theory of the etiology of insomnia proposed by Harvey (2002). Harvey proposed that insomnia is caused by negative cognition. Before Harvey's model, a study by Edinger et al. (2000) examined the relationship between sleep-related beliefs and sleep disturbances. They found that individuals with symptoms of insomnia had more negative cognitions compared to those without symptoms which overlaps with the findings in hypothesis three where a relationship was observed between negative cognition and self-reported insomnia in the second correlation.

Clinical Implications

A meaningful finding from the current study was that people who reported higher sleep difficulties were more likely to take medication for aid in sleep. This finding can inform clinicians about sedative use for individuals who struggle with sleep. For example, if we know that self-reported insomnia is worse in populations who report sleep sedative use, further assessment for the presence of disordered sleep may be more important for a client taking medication for sleep compared to a client who is not reporting sleep medication use. Additionally, this finding would suggest that those who use sleep medication are at higher risk for experiencing higher acuity of insomnia symptoms compared to those who do not take sleep medication, and consequently would benefit more from targeted treatment for disordered sleep.

Medical advice when treating insomnia is typically to use pharmacological intervention (Heussler et al., 2013; Yang et al., 2011), despite several of the commonly prescribed substances used for sleep aid being banned for clinical application in many countries (Lader, 2011). Previous research has shown that individuals who take medications for insomnia over long

periods of time suggest a lack of adherence to recommended use, and that these individuals do not fill prescriptions periodically (Bramness & Sexton, 2011).

A meaningful relationship was not observed between negative cognition and self-reported insomnia for participants who were taking medication to help with sleep quality. The lack of an observable relationship could mean that taking medication for sleep problems does not address negative cognition, a known cause of insomnia. Non-pharmacological treatments, such as CBTI, focus specifically on negative cognitions to reduce symptom severity of insomnia (Garland et al., 2018). The absence of a relationship between negative cognition and self-reported insomnia for participants taking medication in the current study provides support for the use of non-pharmacological treatments to address chronic sleep difficulties.

The significant relationship between negative cognition and self-reported insomnia for participants who did not take medication suggests that a measure of negative cognition would likely be needed to assess the progress of treatment for insomnia. A meaningful relationship between negative cognitions and greater endorsement of symptoms of insomnia in participants not taking sleep medications suggests that measuring negative cognition when assessing sleep health is important. Additionally, this finding suggests that prudent assessment of the clinical utility of pharmacological and nonpharmacological treatments for insomnia should include screening for negative cognition. Assessments that screen for negative cognition such as the one developed in the current study are easily administered. However, the lack of significant results on the first correlation in the third hypothesis of this study suggests that observable effects from pharmacological intervention may need more sophisticated or more sensitive measures of sleep. More sophisticated measures of sleep such as a polysomnogram may be needed to observe changes of pharmacological intervention on sleep architecture. The measure of negative

cognition developed for the present study may not be sufficient in its sensitivity to measure changes in cognition from pharmacological intervention, but might be suitable for use as a screening instrument for vulnerability to the development of insomnia.

Limitations

The impact of the current study on the broader literature may benefit from the discussion of the study's limitations. First, the inclusion criterion used in the current study reduced the sample of participants significantly. This restriction was needed to ensure that all participants had a history of medication use. While the sample size in the current study was adequate for statistical analysis, the small sample size made the results less generalizable to the population within the United States, particular in relation to participant race and ethnicity. Of the 104 participants who were included in the final analysis 3 (2.9%) identified as multiracial, 90 (86.5%) as White, 4 (3.8%) as Black and/or African American, 2 (1.9%) as Native American or Aleutian Islander or Eskimo, 1 (1%) as Asian or Pacific Islander, and 4 (3.8%) as other. The population estimate for race within the United States as of July 1, 2022 provided by the United States Census Bureau is 75.5% White, 13.6% African American, 1.3% American Indian and Alaska Native, 6.3% Asian, 3.0 % two or more races (United States Census Bureau, 2022). Based on these statistics, the current sample contained more individuals who identified as White and Native American/Aleutian Islander or Eskimo compared to the national average. Additionally, individuals who identified as multiracial, African American, and Asian were underrepresented compared to the national average. The discrepancies in the sample would suggest that results from the current study are less likely to generalize to individuals who identify with these underrepresented groups.

When considering the limitations of the methods utilized in the current study, a predominant concern was each measure's ability to assess the constructs of interest. Creating measures that assess constructs like insomnia and healthy sleep is challenging for a variety of reasons. The various factors that determine whether sleep is healthy or disordered include the time it takes to initially fall asleep, the total amount of sleep, the length of time spent awake during the night, and the frequency of night-time wakings (APA, 2022). Moreover, sleep is a complex, ever-changing process that is mostly not able to be directly observed by patients unless they take part in a sleep study. Additionally, some of our sleep stages are not well understood. In addition to the complexities of the construct of sleep, the way that measures are developed also generate methodological concerns.

The measures of self-reported sleep difficulties in the current study have limitations. The survey item used to measure insomnia is considered a proxy variable because it did not directly measure the construct of insomnia. Insomnia, along with many other psychological disorders, cannot be quantified without using a proxy variable that is quantifiable and presumed to measure the construct. In this study, the single variable utilized to capture self-reported insomnia asked, "During the past 30 days, how often have you experienced trouble getting to sleep or staying asleep?" utilized a Likert scale to capture severity. This survey item may not have been sensitive enough to capture variation in self-reported insomnia. The variable was gathered from an archival dataset with little to no additional resources, so the sensitivity of the item was limited. The methods used to assess self-reported insomnia did not include data regarding fluctuations of self-reported insomnia over time for participants. For example, no data involving polysomnogram or sleep schedule were included in the measure developed for self-reported insomnia in the current study. It should be noted that many of the methodological limitations

noted above are primarily related to limitations in the archival dataset used in the analysis. Further, the significant findings in the present study demonstrate that the variable utilized to capture insomnia is sufficient to produce an observation of the relationship between negative cognition and sleep health.

An additional methodological concern was the single item self-report question used for assessing the use of sleep medication. The question utilized did not ask about specific sleep medications being taken and asked only broadly about barbiturates or sleeping pills. As discussed previously, barbiturates are an outdated class of sedative medication that is not commonly used for sleep problems. The vague language of the question in this study (i.e., sleeping pills) may have impacted the results because the pharmacodynamic and pharmacokinetic implications of different medications on negative cognition could vary across medications though this is speculation because negative cognition is not accounted for as a side effect (Brandt & Leong, 2017; Buscemi et al., 2007; Committee O. T. R. O. M. 1980; Freedom, 2011; Neubauer, 2014; Sweetman et al., 2020; Wolkove et al., 2007a).

Similarly, the variable developed to assess negative cognition in the current study had limitations in its ability to measure the construct of interest. The items utilized in this study has multiple datapoints, but is a newly developed measure which limits its reliability. The measure used is considered a proxy variable because it indirectly measures the construct of negative cognition. Internal dialogue, or cognition, can be directed towards anything that can be thought of. The cognition utilized for the measure developed in the current study would ideally have isolated cognition that contains negative content related to sleep. One of the items used did not include sleep asking *Were you restless because of your worry*. Two of the items only mentioned energy level asking *Were you low on energy,* and *Did you tire easily because of your worry*. An

additional complication was that one of the items may not have assessed cognition asking *Did you have trouble falling asleep.* However, all items used were directed towards sleep or negative cognition which satisfied the basic needs of the study.

Despite limitations, the results of the present study represent a unique contribution to our understanding of the relationships between sleep medication, negative cognition, and sleep. It is important for these relationships to be explored, given the already extensive and pronounced list of negative side effects associated with taking sleep medication.

Directions for Future Research

The focus of this study was to obtain information about the relationship that sleep medication has with negative cognition and self-reported sleep difficulties. The relationship between negative cognition and the development of insomnia is well established in the literature (Cronlein et al. 2014; Garland et al., 2018; Morin et al., 2016; Robabeh, 2015; Sateia et al., 2017). Future research on the relationship between sedatives and negative cognition is important for the treatment of insomnia, as few studies concerning the efficacy of pharmacological intervention look specifically at negative cognition. Some examples of specific areas of exploration regarding the impact of pharmacological interventions on negative cognition may include more detailed measures of frequency of sedative use, amount of sedative used, specific sedative used, duration of use, and tracking of additional medications used in conjunction with sleep medication. Specifically, future research could explore the impact of sleep medications on negative cognition longitudinally, which would likely provide further clinical implications.

Further research that refines the measure of self-reported insomnia could be useful in exploring the relationship between self-reported insomnia and sedative use. Additional research could also utilize more experimental and prospective studies involving pre-established measures

of self-reported insomnia and sedative use, which would allow for higher construct validity for these measures.

Finally, given the extensive empirical support for the efficacy of cognitive and behavioral treatments for insomnia, the standards and procedures associated with the regulation and recommendation of medication protocols would benefit from the incorporation of a cognitive component when investigating unwanted side effects of sleep medications. Cognitive treatments for insomnia, like CBTI, explain the etiology of insomnia as the development and maintenance of negative cognitions about sleep. Despite ample literature to suggest evidence for the cognitive theory for the etiology of insomnia, many trials assessing the efficacy of sleep medication do not incorporate screening for, or assessment of, negative cognition. A cognitive component of medication regulation should be used to assess the utility of sleep medication in treating insomnia.

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