BRIEF COGNITIVE BEHAVIORAL INTERVENTION FOR INSOMNIA IN OLDER ADULTS WITH AND WITHOUT A HISTORY OF CHRONIC PAIN

By

JACOB M. WILLIAMS

A THESIS PRESENTED TO THE GRADUATE SCHOOL
OF THE UNIVERSITY OF FLORIDA IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF SCIENCE

UNIVERSITY OF FLORIDA

2010

To all of those who have helped me learn the joy in a job well done.

ACKNOWLEDGMENTS

I would like to acknowledge my mentor, Dr. Christina McCrae, in helping me to refine my ideas and providing me the opportunity to train under her guidance. I would also like to thank the innumerable number of teachers and mentors I have had over the years, Dr. Daniel Taylor and Dr. Mary Carskadon who provided me the opportunity to prove myself and develop in this area of work.

I would like to give a particular thanks to my family: to my Mom, who has and continues to be an anchoring force in my life; my Dad, who helped me cultivate my passion and to seek out the “why” in life; and my sisters, who have always been a source of companionship and joy.

TABLE OF CONTENTS

page

LIST OF TABLES

LIST OF FIGURES

Abstract of Thesis Presented to the Graduate School
of the University of Florida in Partial Fulfillment of the
Requirements for the Degree of Master of Science

BRIEF COGNITIVE BEHAVIORAL INTERVENTION FOR INSOMNIA IN OLDER ADULTS WITH AND WITHOUT A HISTORY OF CHRONIC PAIN

By

Jacob M. Williams

May 2010

Chair: Christina S. McCrae
Major: Psychology

When treating sleep disorders in older adults, comorbid medical conditions can raise concerns about efficacy of treatment. Previous research has shown significant improvements are possible using Cognitive-Behavioral Therapy for insomnia (CBTi) with individuals with chronic pain but has never had a non-pain comparison. It was hypothesized that participants with insomnia and a history of chronic pain (HCP) who received CBTi would show significant improvements on the outcome measures: sleep onset latency (SOL), wake after sleep onset (WASO), sleep efficiency (SE), total sleep time (TST), sleep quality rating (SQR), and pain rating. However, across the sleep variables these improvements were predicted to be less than improvements among non-HCP participants. A sample of 53 older adults with insomnia (M_{age} = 69.1 years, SD_{age} = 7.0 years) completed daily sleep diaries and daily pain ratings throughout baseline, 4-week cognitive-behavioral treatment (stimulus control, sleep restriction, relaxation) or wait-list control, and post-treatment evaluation. Preliminary analyses revealed that participants receiving CBTi improved from baseline to post-treatment and at post-treatment the CBTi group reported significantly better sleep compared to the wait-list control. Using multiple analyses of variance (ANOVA) no significant differences

were found between the HCP and non-HCP participants receiving treatment. Equivalence was found between the HCP and non-HCP groups at post-treatment on the following sleep measures: SOL, WASO, SE, and SQR. HCP participants did not experience significant improvements in their pain rating but non-HCP participants did have improvements in pain. This study indicated that participants who received the treatment, both with HCP and without HCP groups, achieved equivalently high rates of average improvement across four of the sleep variables. It can be concluded that the current form of CBTi can be a brief but efficacious sleep intervention among individuals with HCP and the presence of a comorbid chronic pain conditions does not interfere with the treatment of chronic insomnia.

CHAPTER 1
INTRODUCTION

Cognitive behavioral therapy for chronic insomnia has been shown to be a highly efficacious treatment among older adults. Much of the past research with older adults has been focused on individuals suffering from insomnia alone, while less focus has been given to individuals with comorbid insomnia (insomnia that co-occurs with other physical or mental health conditions). However, in clinical samples a large majority of insomnia cases are comorbid in nature. Thus the exploration of treatment for these individuals is absolutely necessary. Previous research has indicated that there may be a link between sleep problems and chronic pain conditions through physiological, behavioral, and cognitive mechanisms. In addition, there is research to support the use of CBTi among individuals with different chronic pain conditions (Savard, et al., 2005). So far, no study has directly explored whether the presence of a chronic pain condition impacts CBTi when compared to individuals without chronic pain. According to the theory proposed by Spielman (discussed in greater detail below) the presence of predisposing, precipitating, and perpetuating factors drive the development of chronic insomnia. Research among individuals with primary insomnia, insomnia that is not comorbid with other mental or physical health problems, has demonstrated that over time predisposing and precipitating factors become less important in the maintenance of chronic insomnia. This same pattern has been suggested for individuals with insomnia comorbid to other chronic health conditions (Rybarczyk, Stepanski et al., 2005) but this theory has not been formally tested in this population. It is possible that chronic pain serves as a non-modifiable factor in the precipitance and perpetuation of chronic insomnia. This study attempted to show that individuals with a history of chronic pain

experience improvements with behavioral treatment, but their improvements would be less than individuals without a history of chronic pain. Two secondary analyses were conducted on data collected from a sample of older adults with chronic insomnia with the aim of better understanding how CBTi impacts the sleep and pain of individuals with a comorbid pain condition. The first analysis determined whether participants with a history of chronic pain had reduced response to insomnia treatment (compared to those without such a health history) as a result of underlying untreated pain mechanisms. The second analysis aimed to determine whether participants with chronic pain saw improvements in their pain experience as measured through daily pain ratings. Both of these aims hinged on the assumption that the underlying untreated mechanisms would preserve some pain symptoms and thus influence the amount of improvement in sleep. This study will not be able to directly measure the underlying physiological processes. Nevertheless, the subjective complaints of chronic insomnia and chronic pain should be considered the most important measures in the improvement of symptoms, since both chronic pain and chronic insomnia are identified through subjective complaint.

CHAPTER 2
REVIEW OF THE LITERATURE

Insomnia has a profound impact on the lives of millions of Americans and has been widely studied. However, to date the majority of psychological treatment studies have looked at insomnia in isolation (Lichstein, Wilson, & Johnson, 2000; Morin, Hauri et al., 1999). Less well understood is the role of sleep disturbance in the context of a host of comorbid chronic health conditions. While no causal relationship can be assumed, a clear overlap exists between sleep problems and chronic health conditions (D. J. Taylor et al., 2007). The wide overlap of insomnia with chronic health conditions necessitates a greater understanding of whether current treatments for insomnia are effective among these clinical populations (Ohayon, 2005; D. J. Taylor et al., 2007). In particular, the efficacy of insomnia treatment may be affected by co-morbid chronically painful, non-life threatening conditions (e.g. arthritis, irritable bowels syndrome, fibromyalgia, pain resulting from previous injury). Chronic non-life threatening pain has been reported to cause profound loss in quality of life and productivity (Gerstle, All, & Wallace, 2001). Insomnia may develop as a secondary condition to chronic pain, but over time takes on a separate course and becomes a self sustaining disorder (Currie, Wilson, Pontefract, & deLaplante, 2000). The effects of acute pain, emotional distress, functional changes, reduced activity, and medication reliance may drive the development of sleep problems among these individuals. Insomnia symptoms may then be perpetuated by excessive time in bed, irregular sleep schedule, napping, low activity, worrying in bed, and continued use of pain and sleep medication (Currie et al., 2000). As a result, an individual's insomnia can persist despite effectively managed chronic pain.

Pathogenesis of Chronic Pain

Chronic pain problems may arise from one or a combination of many different sources. Sources can include: a single event resulting in physical injury, a build up over time of minor injuries that compound to a more severe condition than any of the individual injuries, and/or the result of changes within the central and peripheral nervous system that result in hyper-sensitivity to pain stimuli or a sensitivity to stimuli that were not previously painful (anadonia). Change in the central nervous system to persistent enhancement of pain transmission, is called central sensitization. This results from long term potentiation (enhanced response) of nociceptors (pain signalers) in the dorsal horn neurons that results in increased pain sensation (Salter, 2002). High frequency stimulation of nociceptors has been experimentally shown to result in enhanced response to subsequent lower frequency stimuli, a phenomenon known as windup (Li, Simone, & Larson, 1999). While windup can serve as an experimental model, true central sensitization results in a longer term sensitization of nociceptors (Salter, 2002). This sensitization results in enhanced excitatory responses and depressed inhibition of nociceptors, causing amplified responses to innocuous as well as noxious input.

Inflammation may also play a significant role in chronic widespread pain conditions. The link between neural activity and the immune system, responsible for inflammation, is mediated by the presence of neurotransmitters, hormones, and cytokines. While these three classes of chemo-transmitters seem to have a clear division of function, the reality is that immune system cells can be a source of pituitary hormones and neurotransmitters and that cytokines from immune cells can function as hormones (Chapman, Tuckett, & Song, 2008). Consequently, the immune system can act as a sensory system to the brain via the hypothalamic-pituitary-adrenal (HPA) axis.

Cytokines most frequently act at a local level in response to injury as either proinflammatory or anti-inflammatory chemicals. The widespread release of proinflammatory cytokines is typical of the sickness response. Individuals with chronic widespread pain have been identified as having significantly lower levels of the anti-inflammatory cytokines interlukin-4 (IL-4) and interlukin-10 (IL-10) than controls (Uceyler et al., 2006). Anti-inflammatory cytokines have been identified as important mediators in immune system activity. Interlukin-4 has been linked to the production of endogenous opioids, and the active chemical has been found to produce hypoalgesia or reduced sensitivity to painful stimuli (Uceyler et al., 2006). In rodents reduced levels of the anti-inflammatory, IL-10, have been linked to signs of hyperalgesia, and administration of the chemical has been found to reduce hyperalgesia. In addition, differences in active night-time hormones' (cortisol, prolactin, and melatonin) circadian rhythmicity have been identified among individuals with severe rheumatoid arthritis. Prolactin and melatonin both have proinflammatory properties, while cortisol serves an anti-inflammatory role. As a result, individuals with rheumatoid arthritis continue to experience high levels of the pro-inflammatory chemicals tumor necrosis factor (TNF) and interlukin-6 (IL-6) until approximately 10 or 11 AM, which is approximately 2-3 hours later than healthy controls (Cutolo, Straub, & Buttgereit, 2008). This may account for some of the complaints of unrestorative sleep, stiffness, and fatigue often experienced by this population in the morning.

Development of Insomnia

The Three Ps Model of Insomnia

The course of chronic insomnia, according to the theory proposed by Spielman, includes predisposing conditions, precipitating circumstances, and perpetuating factors

(Spielman, 1986). Predisposing conditions alone are not sufficient to produce chronic insomnia but precede the onset of insomnia and increase the likelihood for its occurrence. In the context of chronic pain and insomnia, a trait-like tendency to interpret stimuli in a threatening way could be considered a predisposing factor for insomnia. Precipitating circumstances co-occur with the onset of insomnia and might include stressful personal events, substance use, poor sleep habits or rapid shifts in health. Insomnia is maintained and continued by perpetuating factors which might include changes in sleep/wake schedule, changes in compensatory daytime behavior, such as napping and caffeine use, and changes in cognitive processes.

Table 2-1. The Three Ps Model of insomnia across physiological, behavioral, and cognitive domains.

	Predisposing	Precipitating	Perpetuating
Physiological	• Underlying genetic propensity for pain condition	• Trauma from injury or illness	• Sedatives disrupting the normal sleep cycle
Behavioral	• Tendency maintain an irregular sleep/wake schedule	• Spending excessive time in bed as a result of injury	• Napping and low activity
Cognitive	• Personality trait to interpret stimuli as threatening	• Emotional distress due to disability or pain	• Worry about the next night's sleep

A distinction must be drawn between individuals suffering from acute versus chronic insomnia. Acute insomnia is marked by distress due to non restorative sleep, difficulty initiating sleep or maintaining sleep which lasts less than four weeks and can be linked to a specific cause (American Academy of Sleep Medicine, 2001). Chronic insomnia exhibits the same pattern of subjective complaints but lasts for at least 6

months or more and a specific cause may not be identifiable (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). Acute insomnia is more likely to be driven by the effects of precipitating circumstances and the impact of predisposing conditions. The development of chronic insomnia is often related to a combination of predisposing, precipitating, and perpetuating factors that manifest themselves as physiological, behavioral, and cognitive mechanisms that serve to disrupt normal sleep (see Table 1.).

Physiological Mechanisms

Among individuals suffering from chronic pain conditions there are a number of physiological factors which may act at the predisposing, precipitating, and perpetuating factor in the development of chronic insomnia. The initial traumatic injury can result in dramatic increases in inflammatory chemicals (i.e. IL-4, IL-10), along with reductions in anti-inflammatory chemicals (i.e. TNF, IL-6). These chemicals can disrupt the natural circadian rhythm for both sleep and wakefulness. Over time chronic injuries may result in changes in the sensitivity of the central and peripheral nervous system. Thus, it may become more difficult for the individual to relax and become comfortable while in bed. The effects of central sensitization (to be discussed in more detail below) may also impact patients' sleep by causing greater responsivity to all noxious stimuli including night time sensations and noises that were previously not problematic. The use of prescription analgesics (e.g. opioids) and sedatives (e.g. benzodiazepines) among many chronic pain patients may be an important factor in the development and preservation of sleep difficulties. Many of these medications have well known deleterious effects on sleep including reduced slow wave (delta) and rapid eye movement (REM) sleep. It is widely believed that sleep plays essential role in the restorative processes of the brain. Recent research has established that changes in

cellular, neuronal, hormonal, and immunological functions take place during various sleep stages (Dang-Vu, Desseilles, Peigneux, & Maquet, 2006; Irwin, Clark, Kennedy, Christian Gillin, & Ziegler, 2003; Irwin, Wang, Campomayor, Collado-Hidalgo, & Cole, 2006; Van Cauter et al., 2007). The long term disruption of the normal sleep process has been found to result in significant hormonal alterations, detrimental impact on learning and memory (particularly following REM sleep deprivation), and impaired immune functioning, particularly following slow wave sleep deprivation (Dang-Vu et al., 2006; Irwin et al., 2003; Irwin et al., 2006; Van Cauter et al., 2007). Non-opioid analgesics have been found to increase sleep latency and the number of awakenings and to decrease slow wave sleep among healthy adults. Opioids used as analgesiacs are known to decrease slow wave and REM sleep among drug users and healthy individuals (Kay, 1975; Kay et al., 1979). Sedatives and opioids, in particular, may also serve to disrupt the circadian rhythm of patients by causing greater sleepiness during the daytime, disrupted digestion, less activity, and less exposure to sunlight during normal waking hours.

Behavioral Mechanisms

Changes in behavior, such as daytime naps, excessive time in bed, consuming caffeinated drinks, and smoking too late in the day may be particularly relevant in the development of chronic insomnia among individuals with chronic pain conditions. Specifically, daytime naps may disrupt the sleep homeostat (drive for sleep that increases the longer one is awake) by meeting some of the sleep drive that typically builds during the day. As a result sleep drive, also known as homeostatic pressure for sleep, near bed time decreases and as a consequence it takes longer for the individual to fall asleep. Spending an excessive amount of wakeful time in bed during the day is a

common coping response for individuals who have difficulty sleeping. This response makes intuitive sense; unfortunately it frequently backfires as the additional wake time in bed strengthens the association between the individual's bed or bedroom and wakefulness. Stimulants such as caffeine and nicotine late in the day can result in reduced sleep drive due to the arousing effects these chemicals have on the central nervous system. A reduction of daytime activity often accompanies chronic pain conditions and chronic insomnia due to increased fatigue and pain. This reduction in activity may reduce patients' daytime light exposure thus modifying their natural circadian rhythm. In addition, reduced activity can lead to spending more time in bed and result in increased opportunity for napping. A moderate increase in daytime activity is a common clinical suggestion for individuals with sleep problems. The basis of this recommendation is largely rooted in anecdotal evidence and assumed connections between exercise and sleep. While it is true that exercise has been linked with improvements in cardiovascular and mental health, objective support for the direct effects of exercise on sleep problems remains inconclusive (Irwin et al., 2003; Youngstedt, O'Connor, & Dishman, 1997).

Cognitive Mechanisms

Cognitive distortions related to sleep have also been found to be important factors related to elevated emotional arousal and worsening sleep problems. The two main cognitive models developed by Morin and Harvey (Belanger, Savard, & Morin, 2006; Harvey, 2005; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993) assert that negative changes in beliefs, attitudes, and interpretations related to sleep problems serve to exacerbate and maintain insomnia symptoms by stimulating the sympathetic nervous system and increasing cognitive arousal. Morin emphasizes that change in

dysfunctional beliefs is best achieved through education and verbal techniques to challenge and dispel dysfunctional beliefs and attitudes. Harvey, on the other hand, believes that change is best achieved through formulating cognitive and behavioral experiments for each patient in order to challenge these beliefs, attitudes, and interpretations.

Worry is a common reaction to sleep disturbances that can further disrupt sleep by increasing anxiety via the sympathetic nervous system. Individuals' worries about their sleep or other stressors in their life may increase both cognitive and physiological arousal. Individuals with sleep problems also report increased sensitivity and attention to sleep related threats (Tang & Harvey, 2004; L. M. Taylor, Espie, & White, 2003). This process often operates in a highly cyclical and self perpetuating process. Increased arousal and worry about sleep can lead to heightened attention to sleep difficulties, which may in turn lighten or fragment sleep and thus increase worry the next day

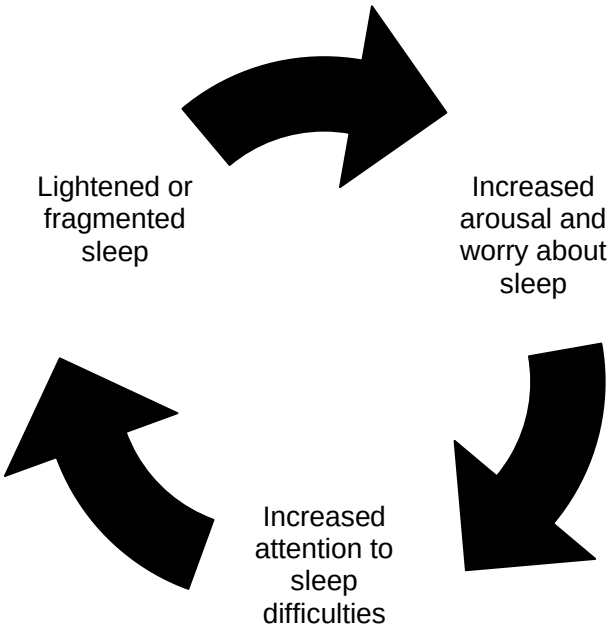

Figure 2-1. The cyclical effects of worry on sleep

(see Figure 2-1). It is also common for misperceptions to form about how long it takes for the individual to fall asleep and how long they are awake in the middle of the night.

Unhelpful or distorted beliefs related to sleep and daytime impairments can also serve to further exacerbate sleep problems. The cognitive distortions related to sleep generally fit into the Beck (1976) model that can include all-or-nothing thinking, catastrophizing, discounting positives, emotional reasoning, should and must statements, and mind reading. In addition, individuals with insomnia may hold one or more of the following maladaptive beliefs about sleep: unless a certain amount of sleep in attained they will be unable to function, insomnia is dangerous, insomnia is out of their control, they can force themselves to sleep, and they should be able to sleep like everyone else (Belanger et al., 2006).

As described above, physiological, behavioral, and/or cognitive mechanisms can drive the maintenance of chronic insomnia, although the order in which these factors act can vary from person–to–person. In addition, the temporal relationship of the predisposing, precipitating, and perpetuating factors can be difficult to determine within an individual. Over time the impact of the predisposing and precipitating factors declines as the perpetuating behavioral and cognitive factors take control. This process can be seen in Figure 2-2. The presence of the initial predisposing factors is not sufficient to cause insomnia. Precipitating events then occur to move the individual to a level where insomnia symptoms begin to appear in the acute form. At the same time the importance of the predisposing factors begin to decline. The occurrence of the perpetuating cognitive and behavioral factors drives the insomnia to a higher level and maintains the insomnia over time so that it receives a classification of chronic insomnia.

Simultaneously, the importance of the precipitating factors begins to decline and the importance of predisposing factors continues to decline. As a result, the factors of primary importance in the maintenance of chronic insomnia are the cognitive and behavioral perpetuating factors. These factors should then be the primary focus in the treatment of insomnia. The literature regarding the treatment of cognitive and behavioral factors in chronic insomnia will be discussed later in this paper. This model is based on the assumption that predisposing and precipitating factors will decline in importance over time. However, situations may arise where the precipitating factors do not decline but instead continue to play an important role in the insomnia. This non-modifiable precipitating factor, as it might pertain to individuals with chronic pain, will be discussed later in this paper.

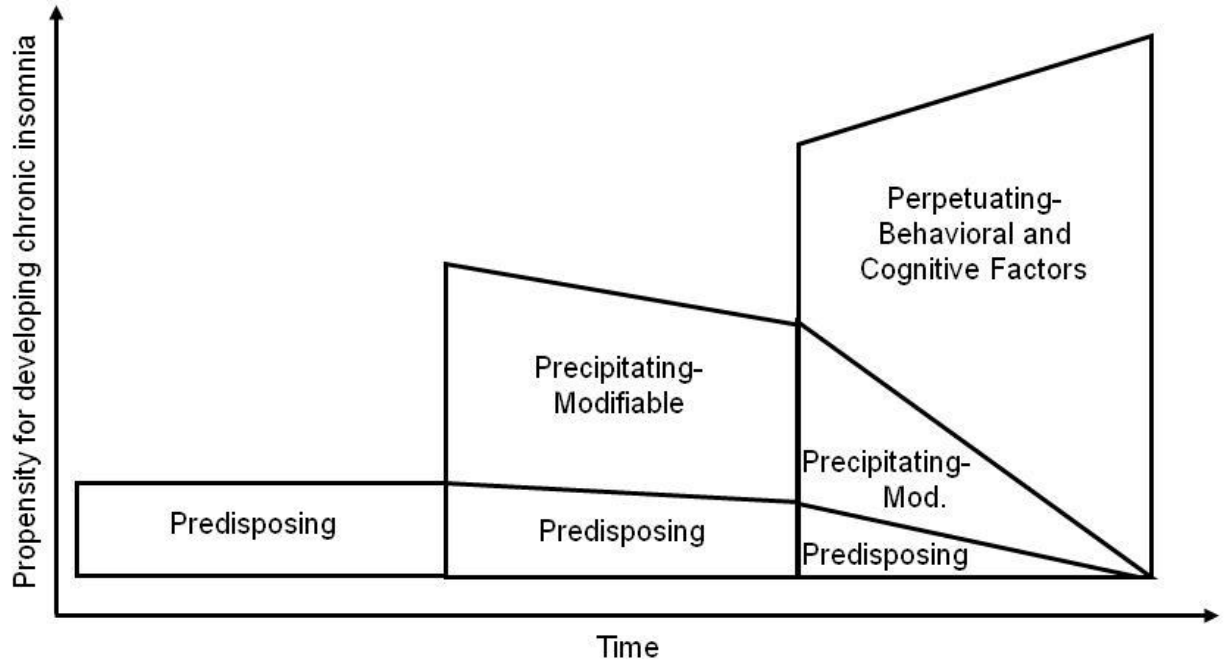

Figure 2-2. Three Ps Model of the development of insomnia with biological component of pain having a modifiable impact on sleep

For example, an individual may experience financial stress that causes worry and acute insomnia. At same time the deleterious effects of a medication on the individual's sleep may be identified by a health professional. The individual has a lifelong pattern of cognitive processing that resulted in worry that often results in catastrophic thinking. By the time they meet with a sleep specialist, the individual's worry about that night's sleep, along with excessive daytime napping, caffeine use, and associations between their bed and wakefulness keeps them awake for up to two hours a night, almost every night of the week. This shows how the predisposing, precipitating, and perpetuating factors described in Spielman's model can be accounted for by a combination of these physiological, behavioral, and cognitive mechanisms.

Connection between Pain and Insomnia

There is considerable epidemiological evidence that supports the link between chronic pain conditions and insomnia. In a study (*N* = 1769) by Taylor and colleagues (2008), 50.4% of people with chronic insomnia reported chronic pain (e.g. arthritis, back pain, migraines), while chronic pain was only reported by 18.2% of individuals without insomnia. Among individuals with chronic pain, 48.6% reported having chronic insomnia and of those with chronic pain, only 17.2% did not have insomnia. A study of almost 19,000 European community dwelling adults (18 and older) assessed the prevalence of chronically painful physical conditions along with insomnia complaints. Around 23% of individuals with chronic pain conditions reported at least one insomnia complaint and 40% of individuals with insomnia symptoms reported at least one chronic pain condition. Rheumatoid arthritis has been associated with sleep disturbance at rates of 54% to 70% (Devins et al., 1993; Hart, Taylor, & Huskisson, 1970; Wright, 1985). Arthritis is also associated with an increased number of night time awakenings, longer wake time after

sleep onset, and lower sleep efficiency than matched controls but only minor differences in sleep stages (Devins et al., 1993; Hart et al., 1970).

As with the conditions in isolation, the links that have been made between chronic pain and sleep difficulties center around three major areas: changes in biological mechanisms, changes in behavioral processes, and changes in cognitive processes.

Physiological Mechanisms

Changes at the chemical, cellular and bodily systems may account for some of the overlap of chronic pain and chronic insomnia. One possible explanation is related to the theory of central sensitization which emphasizes changes in nociceptive transmitters (pain information transmitting cells) that result in hyperactivation of these cells in the spinal cord and brain (Salter, 2002). Reduced pain threshold, spatial spreading of pain sensation, and increased temporal length of pain sensation are characteristic of central sensitization (Latremoliere & Woolf, 2009). A chronic wide spread pain condition, known as fibromyalgia, illustrates the shared relationship between sleep and pain conditions through central sensitization. Fibromyalgia is often characterized by a wide range of symptoms including widespread muscular and non –muscular pain, irritable bowl syndrome, headaches, mood disorders, fatigue, and sleep disturbance –which are best explained by the effects of central sensitization (Cassisi et al., 2008). Sleep and pain may also be linked through the effects of peripheral sensitization, which cause peripheral nociceptors (cells that detect potentially painful stimuli) to have a reduced threshold for and increased response to noxious stimuli and to the inflammatory processes (Latremoliere & Woolf, 2009). Among healthy adults, total and partial sleep deprivation has been found to decrease mechanical pain thresholds, a characteristic of

peripheral sensitization (Onen, Alloui, Gross, Eschallier, & Dubray, 2001; Onen, Onen, Courpron, & Dubray, 2005).

In addition to the neurological changes, changes in the role of nervous-endocrine-immune super-system, known as the HPA axis, may lead to hyperarousal and the additional release of inflammatory chemicals. The widespread release of proinflammatory cytokines, particularly IL-6, is known to be related to fragmented and lightened sleep (Irwin et al., 2006; Mills et al., 2007). Both of these symptoms are frequent characterizations of insomnia.

The central sensitization and HPA axis explanations are not contradictory but instead simply emphasize different communication systems within the body. Sleep difficulties may feed both systems information by signaling threat and thus, heightening the sympathetic nervous system response which stimulates both nervous and endocrine system responses. Despite these possible biological connections between sleep and pain, the convergence of biological and cognitive/behavioral mechanisms remains unclear. A lack of definitive evidence for this shared biological/cognitive/behavior process necessitates careful consideration in evaluating the links between these processes.

Cognitive/Behavioral Processes

Chronic insomnia and chronic pain likely share considerable overlap resulting from changes in behavior and cognitive processes. Individuals' attempts to cope with these two conditions seem to share similar patterns and characteristics. A recent exploratory factor analysis of sleep problem and pain symptomatology revealed overlap in three different dimensions: safety behaviors of behavioral orientation, safety behaviors of cognitive orientation, and catastrophizing (MacDonald, Linton, & Jansson-

Frojmark, 2008). Safety behaviors of behavioral orientation are defined as directly observable changes in daily activities (such as inactivity) to avoid worsening of symptoms. Safety behaviors of cognitive orientation are defined as covert changes with the goal of reducing stimulation, social interaction, and leisure pursuits. Catastrophizing is generally defined as a negative prediction of the future without considering other more likely outcomes (Beck, 1976). These dimensions were assessed using a composite measure, called the Safety Behaviors and Catastrophizing Scale with items taken from the Fear Avoidance Beliefs Questionnaire (Waddell, Newton, Henderson, Somerville, & Main, 1993), the Cogni-phobia Scale (Martelli, Zasler, Grayson, & Liljedahl, 1999), and the Pain Catastrophizing Scale (Sullivan, Bishop, & Pivik, 1995). Test-retest reliability for the three domains of the measure were .63, .64 and .68. Further multi-nomial analysis revealed that the both pain and insomnia conditions share catastrophic profiles and that the safety behaviors of the two conditions exacerbate one another.

This body of research lends strong support to the idea that insomnia and chronic pain do not act independently. Both conditions revolve around heightened sensitivity to environmental stimuli, and this heightened activation creates a continuous loop of distress. Pharmaceutical treatment of pain among osteoarthritis and fibromyalgia patients has been shown to improve sleep quality and sleep quantity despite the fact that some medications can contribute to sleep deficits (Arnold, 2009; Rosenthal et al., 2007). The effective treatment of sleep has been shown to significantly improve daytime consequences such as depression, anxiety, and fatigue among individuals with co-morbid chronic pain (this literature will be reviewed below). Due to their similar

underlying mechanisms, improvements in sleep may translate into improvements in pain. Effective treatment of chronic insomnia may result in reduced activation of the central nervous system, peripheral nervous system, and reduced inflammatory response. In addition, treatment of insomnia may reduce worry and stress among chronic pain patients and bring one aspect of their lives under their control. Targeting these underlying biological, behavioral, and cognitive mechanisms via cognitive/behavior sleep interventions may be a particularly efficacious route since sleep cognitions and behaviors are generally highly modifiable. However, as was previously stated this link remains uncertain. It cannot be assumed that causal associations and relationships exist based on correlational information.

Cognitive-Behavioral Therapy for Insomnia

Non-pharmacological therapies for insomnia have been identified as highly efficacious when treating primary insomnia among young (Morin, Hauri et al., 1999; Morin, Mimeault, & Gagne, 1999; Wang, Wang, & Tsai, 2005) and older adults (65 years and older (Morin, Mimeault et al., 1999). Previous research has shown that 70-80% of individuals treated with non-pharmacological interventions had improved sleep and between 40-64% of individuals with primary insomnia had clinically significant changes in sleep onset latency and wake after sleep onset. Treatment procedures that have been evaluated include sleep hygiene, stimulus control therapy (Bootzin, 1972), cognitive therapy (Morin, Savard, & Blais, 2000), paradoxical intention, sleep hygiene education, sleep restriction (Spielman, Saskin, & Thorpy), and relaxation therapies. These treatments attempt to identify and modify factors that perpetuate insomnia, including maladaptive sleep habits, autonomic and cognitive arousal, dysfunctional beliefs and attitudes about sleep, and non-healthy sleep habits (Morin, Hauri et al.,

1999; Morin et al., 1993). A meta-analysis of non-pharmacological insomnia treatment studies (*N* = 2,102) has revealed modest to large effect sizes on specific sleep disorder variables: sleep onset latency (*d* = .87 - .88), wake after sleep onset (*d* = .65), number of awakenings (d = .53 -.63), total sleep time (*d* = .42-.49), and sleep quality (*d* = .94; Morin, Hauri et al., 1999). When the different treatment methods have been compared, stimulus control and sleep restriction have shown to be the most efficacious treatments in isolation among younger and older adults (Morin, Hauri et al., 1999; Morin, Mimeault et al., 1999). Specific factors have been identified to be predictive of a better response to CBTi and include later age of onset (25 and older), longer duration of insomnia, and less psychopathology (Lacks and Powlishta, 1989).

The majority of insomnia research has focused on primary insomnia. Prior to a 2005 NIH State of the Science Conference on Manifestation and Management of Chronic Insomnia in Adults, insomnia was defined as either primary or secondary. Under this model, if insomnia symptoms were related to another physical, psychological or drug induced condition, that condition was considered the primary diagnosis while insomnia was considered to be “secondary” to that primary diagnosis. Thus, the focus of treatment was on the primary diagnosis, and it was assumed that if the primary diagnosis was successfully treated than the “secondary insomnia” would resolve. However, there is evidence to show that the treatment of primary conditions does not necessarily result in remitting insomnia (Hauri, Chernik, Hawkins, & Mendels, 1974). Prevalence studies have estimated that “secondary insomnia” accounts for 90% of insomnia cases in the general population (Ford & Kamerow, 1989; Kay et al., 1979); Klink et al., 1992; Mellinger et al., 1985). As previously mentioned, insomnia and

chronic pain are frequently comorbid conditions. The conceptualization of insomnia as "secondary" often denies these patients access to treatment that may alleviate their insomnia symptoms (Harvey, 2001). In 2005, a panel of insomnia experts participated in an NIH State of the Science Conference on the Manifestation and Management of Chronic Insomnia in Adults, and they recommended that the term, "secondary" be dropped in favor of the term "comorbid." This recommendation was made for the following reasons: 1) framing the conditions with a causal relationship is problematic since the underlying mechanisms between insomnia and other health problems are not well understood, 2) insomnia thought of as secondary insomnia is less likely to receive treatment, and 3) when perpetuating factors take over, factors that predispose and precipitate insomnia may become less relevant. As a consequence, chronic insomnia takes on a separate and independent course from its original manifestation. A simplified but still pertinent example might entail the following situation: an individual who experienced a specific injury would initially develop sleep problems due directly to the injury preventing them from becoming comfortable and relaxed when in bed. Over time patients begin to associate their bed with wakefulness, arousal, and discomfort. Even after the initial impact of the injury has passed these individuals continue to experience difficulty sleeping due to the learned association with the bed. Thus, the initial injury becomes less relevant as time goes on and the learned associations become the perpetuating factors in the maintenance of the insomnia.

The criteria for the diagnosis of insomnia in the DSM-IV, ICSD, and ICD-10 do not provide sufficient specificity to make them meaningful in research. Due to this limitation a study was conducted by Lichstein and colleagues (2003) which reviewed 61 insomnia

trials and used sensitivity-specificity analyses to determine the cutoffs that provided the highest levels of sensitivity and specificity of insomnia diagnosis. Based on this research a minimum diagnostic criteria was suggested for sleep onset latency and wake after sleep onset of (a) at least 31 minutes; (b) occurring at least 3 nights per week; (c) occurring for at least 6 months (Lichstein et al., 2003). Based on clinical consensus and empirical research, a patient is considered to be successfully treated of sleep problems if their sleep onset latency and wake after sleep onset onset are less than 31 minutes and their sleep efficiency is greater than 85% among older adults (Lacks and Morin, 1992).

CBTi and Chronic Pain

A number of studies have been conducted evaluating the efficacy of cognitive and behavioral therapies for chronic insomnia comorbid with various chronic pain conditions. Morin and colleagues (1990) conducted the first study investigating the effects of CBTi secondary to chronic pain. There were three patients with no significant psychiatric history in their 30s-40s who had pain caused by work related injuries (each lasting 5 years, 18 months, and 2 years). Treatment consisted of 6 weeks of one hour sessions covering stimulus control and sleep restriction. The researchers reported approximately 20 minute improvements in sleep onset latency and wake after sleep onset at 2 and 6 months follow-ups. They reported very little change in the number of awakenings and inconsistent change in total sleep time. However, there was no true statistical testing of these improvements. Consequently, limited conclusions can be drawn from this study. The study did not report any meaningful change in night-time pain ratings.

Currie and colleagues (2000) conducted an efficacy study of insomnia secondary to chronic pain in general. This study worked under the assumption of the Spielman 3-Ps Model, that insomnia may begin as a result of chronic pain but overtime develops into a separate problem that requires separate treatment. This situation is the result of the development of habits that are counteractive to a good sleep environment and produce an irregular sleep routine. Sixty participants diagnosed with insomnia secondary to a general medical condition of chronic pain where included in the study. Participants in the treatment group received 7 two hour sessions of group CBTi. The first hour was focused on discussing sleep diaries and progress in the treatment. Active treatment included education on insomnia and sleep, sleep hygiene, sleep restriction, stimulus control, sleep restriction, relaxation training, and restructuring of maladaptive sleep related cognitions as proposed by Morin's (1993) cognitive therapy approach. At post-treatment, the CBTi group had significant improvements in sleep onset latency, both compared to themselves at baseline (25 minutes less) and compared to the waitlist-control group at post-treatment (30 minutes less). Sleep efficiency was 15% higher in the treatment group than for waitlist-group and wake after sleep onset was 50 minutes less for the treatment group than the waitlist-control group. All of these improvements were maintained at the 3 month follow-up. There was not a significant improvement in pain severity ratings, number of awakening, or in the total sleep time. The lack of improvement in total sleep time is not surprising given that previous literature has shown CBTi to primarily improve sleep by reducing the impact of unwanted awake time in the middle of the night.

A more recent randomized clinical trial was conducted comparing CBTi, sleep hygiene alone, and usual care among 47 individuals with fibromyalgia and chronic insomnia (Edinger, Wohlgemuth, Krystal, & Rice, 2005). The CBTi treatment consisted of psycho-education about sleep, stimulus control, and sleep restriction. Participants receiving the CBTi reported greater improvements on sleep diaries at post-treatment on sleep efficiency, total wake time, and sleep onset latency than did individuals receiving care as usual for fibromyalgia. Of the participants receiving CBTi, 57% showed clinically significant improvements in sleep, which was defined as a total sleep time of 6.5 hours or longer, a mean total wake time of less than 60 minutes, and a mean sleep efficiency of 85% or greater. The CBTi group did not show a significant improvement in pain assessment using the McGill Pain Questionnaire (MPQ) and the Brief Pain Inventory (BPI) when compared to usual care. However, those receiving sleep hygiene showed improvements in pain assessment compared to usual care on the MPQ (approximately 10 points lower) and on the BPI (approximately one and half point lower). The authors noted that this effect was possibly seen because some of the individuals self implemented sleep restriction but this does not explain the lack of benefit for the CBTi participants.

Most recently Vitiello, Rybarczyk, Von Korff & Stephanski (2009) published an article investigating the effects of CBTi on pain and sleep among 51 older adults with osteoarthritis. Treatment consisted of eight sessions of group intervention including stimulus control, sleep restriction, cognitive restructuring, relaxation training, and sleep hygiene. This intervention was compared to an attention control condition which did not include any active treatment components. Sleep variables were measured using self

report sleep logs. At post-treatment participants receiving the CBTi treatment reported significant improvements (all < .05) compared to their own baseline on sleep efficiency (~13%), sleep onset latency (~15 minutes), and wake after sleep onset (~35 minutes). In addition, these participants reported significant improvements in their pain (~10 points) at post-treatment as measured using the SF-36. Attention controls did not report any significant improvement in their sleep or pain.

These previous studies have shown that significant improvements in insomnia symptoms can be made using non-pharmacological (behavioral/cognitive) therapies among individuals with chronic pain conditions. However, all of these studies centered on individuals suffering from chronic pain and as a result did not attempt to compare the change made in this population to changes made among individuals without chronic pain. The current study intends to fill this gap in the literature. The effects of the treatment will be examined among older adults with a history of chronic pain (HCP) and a direct comparison will be made with older adults without a history of chronic pain (non-HCP).

CHAPTER 3
STATEMENT OF THE PROBLEM

Specific Aim 1

The first specific aim will determine whether HCP participants have a decreased treatment response as compared to non-HCP participants on the five sleep outcome variables at post-treatment including sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST), sleep efficiency (SE), and sleep quality rating (SQR). No previous research has directly compared HCP participants to those without a HCP in their response to psychological treatment for insomnia.

Hypothesis for Specific Aim 1

It is hypothesized that HCP participants will exhibit significantly less improvement on each of the five sleep outcome variables compared to non-HCP participants. This reduced treatment response would be expected if the impact of chronic pain serves as a fixed or non-modifiable precipitating factor in the development and perpetuation of chronic insomnia (see Figure 3-1). The previous model discussed (Figure 2-2), would suggested that there will be a gradual decline in the importance of precipitating and predisposing factors in the maintenance of insomnia. In the current model (Figure 3-1), pain remains a stable contribution to insomnia and fails to decline over time. The two conditions are thus predicted to have an additive effect, making HCP participants more resistant to change. As was previously discussed, chronic pain and chronic insomnia share similar maladaptive cognitive and behavior profiles. Cognitive-behavioral therapy for insomnia specifically targets behaviors and cognitions that interfere with a good night's sleep and these changes in cognitions and behaviors are expected to be effective for all individuals' insomnia regardless of comorbid condition.

However, changes leading to central sensitization, peripheral sensitization, alteration of the HPA axis or other changes to the individuals' fundamental physiology are predicted to remain despite improvements in the behavioral and cognitive components responsible for the development and perpetuation of chronic insomnia. As can be seen in Figure 3-1, the precipitating factor of non-modifiable pain does not decline over time but instead remains stable. Thus, at the time of intervention this precipitating factor continues to play an important role in the maintenance of insomnia and it predicted to interfere with improvements in sleep.

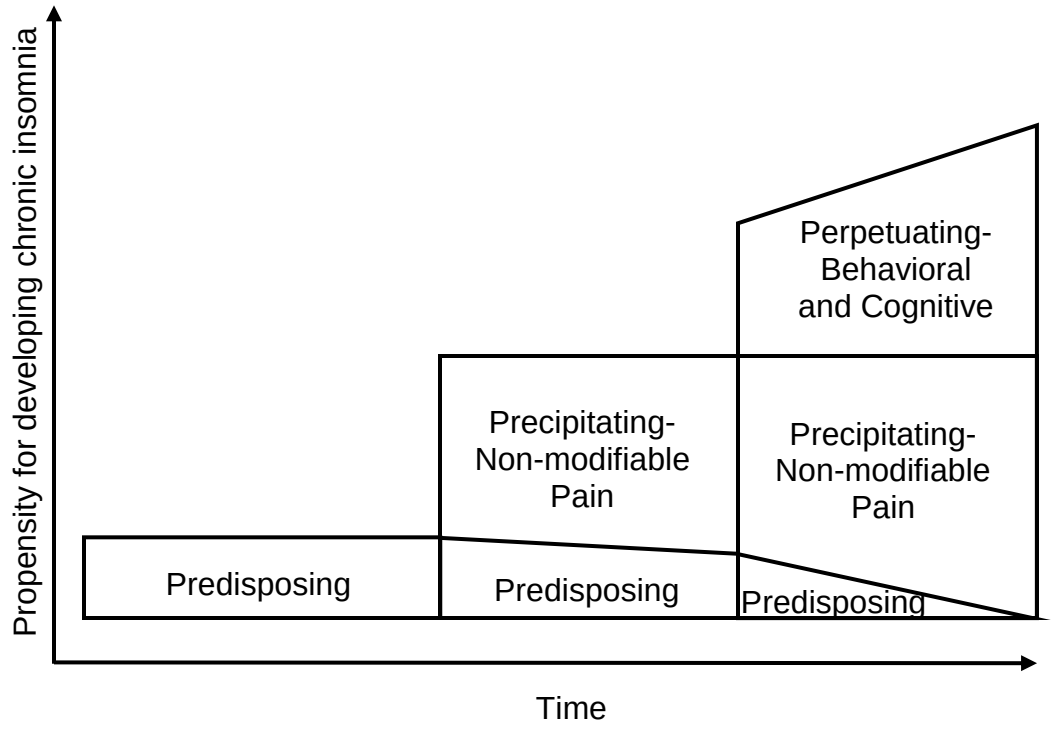

Figure 3-1. Three P Model of the development of insomnia with biological component of pain having a non-modifiable impact on sleep

Specific Aim 2

The second aim of the study is to determine whether the CBTi for chronic insomnia improves average daily pain ratings pre-treatment to post-treatment in HCP participants.

Hypothesis for Specific Aim 2

It is hypothesized that HCP participants will see a significant improvement, although modest, in pain from baseline to post-treatment. Mixed results in pain improvement have been reported in the previously reviewed literature (Currie, Wilson, & Curran, 2002; Currie et al., 2000; Edinger et al., 2005; Morin, Kowatch, & O'Shanick, 1990; Vitiello, Rybarczyk, Von Korff, & Stepanski, 2009). It is predicted that there is a non-modifiable physiological link between chronic pain and chronic insomnia. Although CBTi may have no impact on the underlying mechanisms (e.g. central sensitization, peripheral sensitization, HPA axis response), there should be improvements in the portion of pain that is related to behavioral and cognitive processes. A great deal of uncertainty remains regarding the shared biological/cognitive/behavior process between chronic pain and chronic insomnia and as a result the impact of CBTi on pain symptoms remains difficult to predict.

CHAPTER 4
METHODS

Participants

A total of 77 older adults (65 years or older) were recruited from the North Central Florida community and surrounding areas through newspaper and other community advertisements for treatment of late life insomnia. This project was possible due to a grant from the National Institute of Health/National Institute of Aging (AG024459-01, Christina S. McCrae, PhD, PI NIH/NIA). Participants completed a screening phone interview to address the basic inclusion and exclusion criteria. Based on this interview, potential participants underwent an in-person interview to assess if they qualified for the study. Inclusion criteria included a) report of sleep onset or awake time during night greater than 30 minutes that was present at least 3 nights per week for more than 6 months, b) expressing daytime dysfunction due to insomnia (mood, cognitive, social or occupational impairment), and c) must not have used prescribed or over-the-counter sleep medication for at least 1 month, or were stabilized on medication for more than six months. Exclusion criteria included a) presence of significant medical (e.g., cancer) or neurological disorder (e.g., dementia), b) major psychopathology (e.g., bipolar disorder, substance abuse), c) endorsement of any other sleep disorders (e.g., sleep apnea, periodic limb movements), d) a score of 24 or higher on the Beck Depression Inventory-Second Edition (Beck, Steer, Garbin, 1996) or e) a score of 13 or higher on the Geriatric Depression Scale (Yesavage, 1983), f.) a Mini-Mental State Exam (MMSE) score lower than 23 if 9^{th} grade education or higher or if less than 9^{th} grade education a score lower than 18(Folstein, Folstein, & McHugh, 1975; Murden, McRae, Kaner, & Bucknam, 1991), g.) presence of sleep apnea (an apnea-hypopnea index, AHI, above 15.1, or a

SA O^2 > 93%), measured using a single night of ambulatory monitoring of blood oxygen concentration and respiration with a Compass F10 apnea screening device, and f.) use of any psychotropic or other medications (e.g., beta-blockers) known to alter sleep. Participants were not excluded if they were deemed to have clinically significant levels of depression or anxiety. These two mental health conditions are widely found among individuals with chronic insomnia (Buysse et al., 1994; Coleman et al., 1982; Ford & Kamerow, 1989) and are generally included in studies of chronic insomnia. However, participants were excluded if they were found to have depression with suicidal ideation and were referred to community mental health providers.

Procedure

A preliminary telephone screening involved a 20 minute, structured interview to address study inclusion/exclusion criteria, rule-out other sleep disorders (i.e., sleep apnea), and to establish a probable diagnosis of insomnia. Following the prescreening an in-person appointment was made with each participant. During the initial 1- 1½ hour interview, participants read and signed an informed consent form approved by the University of Florida Institutional Review Board, IRB.2. Participants also completed the Beck Depression Inventory-Second Edition (Beck, Steer, Garbin, 1996), Geriatric Depression Scale (Yesavage, 1983) and the MMSE (Folstein et al., 1975; Murden et al., 1991) during this appointment. Demographic and health survey data were also obtained at this time and participants were sent home wearing the apnea screening device for night to determine the presence of breathing related sleep disorders. The participants were advised to complete daily self report sleep diaries for 14 days. At the end of the first and second week the sleep diaries were collected from the participants. The two

weeks of baseline sleep diaries were used in order to confirm insomnia. Participants were then randomized to four weeks of CBTi or to four weeks of waitlist-control. Following completion of treatment or control period participants completed an additional 14 days of sleep diaries. At the end of the study, the waitlist-control participants were offered the treatment at no cost.

Multicomponent Cognitive Behavioral Treatment for insomnia

The CBTi consisted of psycho-education about sleep, sleep hygiene, stimulus control, relaxation training, and sleep restriction. The psycho-education component included information about sleep stages, circadian rhythms, aging, insomnia, medical conditions, and other sleep disorders. Sleep hygiene recommendations included: restricting caffeine intake to before 12 PM, no nicotine consumption within two hours of bedtime, no large meals within two hours of bedtime, and no exercise within two hours of bedtime (Riedel, 2000). Stimulus control included the recommendation that if one was unable to fall asleep within 15-20 minutes, to get out of bed and engage in mildly stimulating activity in another room until they became sleepy and then to get back into bed (Bootzin, 1972, 2000). This same process was to be repeated if participants awoke during the middle of the night. Relaxation training included a brief (10-minute) technique to target cognitive and physiological arousal (Lichstein, 1988). Sleep restriction consisted of limits/restricts on the time allowed for sleep each night with the goal of regulating the sleep-wake cycle by tailoring the time spent in bed to each patient's true sleep need (Spielman, Saskin, & Thorpy, 1987). This therapy has been supported as an efficacious treatment of insomnia in numerous studies (Lichstein & Morin, 2001).

Measures

Demographics and Health Survey

This survey consists of 13 items collecting information on demographics, sleep disorder symptoms, physical health, and mental health information regarding medical condition/ diagnoses that was administered at intake into the study. Participants were asked to report medical conditions including heart attack, other heart problems, cancer, AIDS, hypertension, neurological disorder (seizures, Parkinson's), breathing disorders (asthma, emphysema, allergies), urinary problems (kidney disease, prostate problems), diabetes, pains (arthritis, back pain, migraines), gastrointestinal disorders (stomach, irritable bowels, ulcers, gastric reflux), and other medical conditions. From these endorsements, a total number of conditions reported was calculated.

History of chronic pain condition: This was determined from the reported medical conditions with the following question: "Do you have chronic pain such as arthritis, back pain, or migraines? If yes, please explain."

Number of health conditions: The number of health conditions that participants complained of was summed. Scores could range from 0-10 complaints.

Education: This was defined as the number of years of formal education.

Insomnia duration: This was defined as the number of months an individual reported having difficulties due to insomnia. This was later converted to number of years.

Number of medications: This was defined as the total number of prescription medications that were self reported.

Subjective Daily Variables

Sleep diary

Participants completed a sleep diary (Lichstein, Riedel, & Means, 1999) each morning for 14 days, providing subjective estimates of five commonly reported sleep–wake variables: 1) sleep onset latencies (time from initial lights out until sleep onset); 2) wake time after sleep onset (time spent awake after initial sleep onset until last awakening); 3) total sleep times (computed by subtracting total wake time from time in bed); 4) sleep efficiency percentages (TST divided by the total time in bed and multiplied by 100); 5) sleep quality rating (given on a 1 to 5 scale, where 1 represented very poor and 5 represented excellent sleep). Variables calculated based on self report daily sleep diaries were averaged for the two weeks of baseline and post-treatment periods, respectively.

Pain rating scale

Pain was subjectively evaluated daily through participants' response to the question, "What is your current pain level?" Participants rated their pain on a 0 (No Pain) to 10 (Worst Possible Pain). This item was to be completed in the morning. This item corresponds to criteria recommended in a consensus statement by chronic pain researchers (Dworkin, Turk, Wyrwich, Beaton, Cleeland et al., 2008).

Analysis

SPSS 17.0 was used for data analysis. All significance tests were conducted with an alpha level of .05. Multiple repeated measures analysis of covariance (ANCOVA) was used to determine whether individuals who have a HCP differed in their response to treatment from those without a HCP. The main effect analysis included the repeated factor of time measurement (baseline or post-treatment), treatment condition (treatment or control), and presence or absence of a history of chronic pain. The outcome variables included SOL, WASO, TST, SE, SQR, and pain rating. The primary focus of the

analyses was not on the main effects but rather the specific interactions within time by condition model and the time by condition by history of chronic pain model. Since the focus was on changes within each variable and not on an aggregate change or an underlying construct, a multiple ANOVA approach is more appropriate than the multivariate analysis of variance (MANOVA; Huberty & Morris, 1989). The standard assumption is that the MANOVA protects against Type I error when conducting multiple ANOVAs. However, it has been argued that the use of this analysis should be more based on the research question than attempts at protection (Huberty & Morris, 1989). To determine if the treatment was effective for HCP participants, their mean values at baseline were be compared to their values at post-treatment. In addition, the post-treatment values for treatment HCP group were compared to post-treatment values of the waitlist-control HCP group for statistical significance of difference of means.

CHAPTER 5
RESULTS

The main objective of this study was to determine the treatment response to CBTi among older adults with chronic insomnia and HCP as compared to older adults with insomnia but who do not have HCP. This objective was achieved by examining mean reports of SOL, WASO, SE, TST, SQR, and pain rating in relation to time (baseline vs. post-treatment), condition (wait-list control v treatment), and history of chronic pain (absence vs. presence of HCP). The main objective was achieved through these two aforementioned specific aims. Results will be discussed separately for each aim.

Sample Characteristics

A total of 484 individuals initially responded to advertisements for participants. Three hundred and twenty eight individuals declined participation after receiving further information over the phone. Of the 156 persons who attended the screening appointment, 55 dropped out of the study for personal reasons, 12 were ruled out because they did not meet other criteria, 11 were ruled out for possible apnea/hypopnea, and one did not have insomnia. Thus, 77 individuals participated in baseline assessment. However, an additional 24 individuals had substantial missing sleep and pain data and were thus excluded from the present analyses. Participants who had completed both baseline and post-treatment sleep and pain data were retained in the analyses.

The sample analyzed included 53 older adults with insomnia (M_{age} = 69.1 years, SD_{age} = 7.0 years). Specific sample descriptive characteristics (including demographics, sleep, and pain information) can be found in Table 5-1. Self report of pain conditions

revealed the following: 19 with arthritis, 4 with lower back pain, 2 with osteoporosis, 1 with fibromyalgia, and 29 without a specific pain condition.

A significant difference between the treatment and control conditions was found for the number of medical conditions, and consequently, this variable was used as a covariate throughout the model. Means and standard deviations for sleep (SOL, WASO, SE, TST, and SQR), and pain (PRS) outcomes by time, treatment, condition, and HCP are provided in Table 5-2. They are broken down by time, treatment condition, and presence of chronic pain.

Preliminary Analysis of Parent Project

There is currently a paper in progress is to assess whether the all participants included in this study who received CBTi, saw significant improvements in sleep outcome variables compared to their own baseline and compared to the control condition. This main effect is not a specific aim of the current study; however, it was deemed a necessary step in assessing the effects of the treatment. For the time effects it was hypothesized that participants receiving CBTi would see a significant improvement at post-treatment across all 5 sleep outcome variables compared to baseline. In addition, the control group was not expected to have a significant improvement in the sleep outcome variables from baseline to post-treatment. On the condition interaction effect, it was hypothesized that participants receiving the treatment would see significant improvements to sleep at post-treatment from participants not receiving the treatment on SOL, WASO, TST, SE, SQR and mean pain rating.

Efficacy of Treatment – Parent Project

Multiple ANCOVAs were conducted comparing the outcome variables SOL, WASO, TST, SE, and SQR for main effect of time (pretreatment and post-treatment)

and condition (treatment vs. control). The condition main effect was compared the above mentioned variables and controlled for the number of medical conditions. This was done to determine if the number of medical conditions had a specific effect on any of the variables. Number of medical conditions was not a significant condition interaction predictor on any of the outcome variables (for all p > .05).

The time (baseline vs. post-treatment) × condition (treatment vs. control) interaction was significant for SOL, $F(1, 48) = 4.59$, $p < .05$, $\eta^2 = .087$, WASO, $F(1, 48) = 11.45$, $p < .01$, $\eta^2 = .19$, SE, $F(1, 48) = 15.40$, $p < .001$, $\eta^2 = .24$, and SQR, $F(1, 48) = 7.75$, $p < .01$, $\eta^2 = .14$. Time interaction effects were not significant for TST. Post analyses revealed a significant improvement at post-treatment for the treatment condition on SOL by 26 minutes, $F(1, 48) = 22.18$, $p < .001$, on WASO by 36 minutes, $F(1, 48) = 39.56$, $p < .001$, on SE by 13%, $F(1, 48) = 55.06$, $p < .001$, and on SQR by .4 points $F(1, 48) = 18.94$, $p < .01$. In the control condition, there was no significant difference between baseline and post-treatment on any of the outcome variables.

Condition interaction post-hoc analyses were conducted to determine differences between the treatment and waitlist-control conditions. There were no significant differences at baseline. Further analyses revealed a significant difference between the treatment group and the waitlist-control group at post-treatment on SOL, $F(1,48) = 7.99$, $p < .05$, $\eta^2 = .14$, SE, $F(1,48) = 6.17$, $p < .05$, $\eta^2 = .114$, and SQR, $F(1,48) = 3.93$, $p < .05$, $\eta^2 = .08$. Condition interaction differences were not significant for WASO or TST. At post-treatment the treatment group took an average 26 fewer minutes to fall asleep than waitlist-control, the treatment group was approximately 8.5% higher in SE, and rated their sleep about .4 points better.

Condition interaction post-hoc analyses were also conducted to determine differences in mean pain rating between the treatment and waitlist-control groups. Among those receiving the treatment there was a .55 point decrease in the mean pain rating at post-treatment, $F(1, 50) = 8.31$, $p < .01$, $\eta^2 = .059$. There was not a significant improvement among those in the control group.

Specific Aim 1: Impact of HCP on sleep outcomes

The effect of a HCP was tested in the main effect model including time × condition × pain condition (presence vs. absence of HCP). This main effect revealed no significant difference between HCP and non-HCP participants receiving treatment, when compared at baseline or post-treatment on SOL, WASO, TST, SE, and SQR (see Table 5-2). Since there was no significant HCP interaction at this level the HCP and non-HCP participants were compared for equality of means.

Comparison of Means for Equality

Based on the finding of no significant difference across outcome measures in the time × condition × pain condition main effect the treatment HCP and non-HCP groups were separately compared for equality. The treatment HCP group was also compared to the waitlist-control HCP group. These groups were compared across the 4 measures that showed the most consistent treatment effects: SE, WASO, SOL, and SQR. Equality of means was determined using equivalency z-tests with equivalency windows set based on clinically relevant criteria. A 10% significance range of equivalence was used on SE. On WASO and SOL, a 20 minute significance range was used. On SQR a .5 point range was used for significance testing. This approach is used to determine if two groups are sufficiently near to one another to be considered equivalent based on

clinically or empirically driven criteria (Rogers, Howard, & Vessey, 1993). The null hypothesis is that the difference between the groups is as large as the difference specified by the researcher and the alternative hypothesis is that difference between the groups is smaller than the difference specified by the researcher. The means of two groups are considered equivalent if they differ by less than some difference (delta) in the positive and negative direction. Thus, two simultaneous one-sided z-tests are performed to determine if the mean of sample two is too far to the left or too far to the right of sample one to be considered equivalent. In order to establish equivalency the null hypothesis must be rejected in both directions. The positive or negative difference with the greatest absolute value will be the statistic that is formally tested. If the null hypothesis is rejected for the greatest absolute difference, the other value will necessarily have a smaller p-value and thus testing is unnecessary as it would also be rejected. Since only one test is performed there is no risk of alpha inflation and consequently corrections are not needed for individual variables assessed.

The test for the equivalency is performed by generating a z statistic using the following formula:

$$Z_1 = [(M_1 - M_2) - \delta_1] / S_{M1-M2}$$

$$Z_2 = [(M_1 - M_2) - \delta_2] / S_{M1-M2}$$

Where M_1 represents the mean of group 1, M_2 represents the mean of group 2, δ_1 represents the positive difference, δ_2 represents the negative difference, and S_{M1-M2} represents the standard error for the two group means. A p value is then computed from the z statistic.

The results of these analyses are summarized in Table 5-3. On all 4 measures the treatment HCP and treatment non-HCP groups were equivalent; SE: $z(29) = 7.71$, $p < .001$, SOL: $z(29) = 3.44$, $p < .01$; WASO: $z(29) = 5.59$, $p < .05$; SQR: $z(29) = 7.38$, $p < .001$]. On all 4 measures the treatment CHP and waitlist-control HCP groups were not equivalent.

Specific Aim 2: Impact of HCP on Pain outcome

The time × condition × pain condition (presence vs. absence of HCP) main effect was significant for Pain rating, $F(1, 50) = 4.16$ $p < .05$, $\eta^2 = .077$. Interaction analyses revealed that among those receiving treatment there were significant improvements in pain rating among non-HCP participants, $F(1, 50) = 9.97$, $p < .01$, but there was not a significant improvement among those with a HCP.

Table 5-1. Total sample group differences for descriptive variables

	Treatment Group (n = 27)		Control Group (n = 26)		History of Pain (n = 26)		No History of Pain (n = 27)	
Characteristic	M	SD	M	SD	M	SD	M	SD
Age	68.2	5.53	71.1	8.67	70.2	7.1	70	8.3
Insomnia duration (in years)	11.4	13.7	16.1	16.5	14.0	16.3	14.6	15.0
Education (in years)	15.8	2.8	16.7	2.5	16.6	2.8	15.9	2.5
Number of medical conditions	2.4 [a]	1.5	2.9 [a]	1.7	3.2	1.4	1.6	1.3
Gender	n	% [b]	n	% [b]	n	% [b]	n	% [b]
Male	10	18.8%	12	22.6%	11	20.8%	11	20.8%
Female	17	32%	14	26.2%	15	28.3%	16	30.1%

[a] Indicates significant difference between groups in the row ($p < .05$) [b] Percent of total sample

Table 5-2. Means and standard deviations for sleep and pain outcomes by treatment condition, time, and history of chronic pain

		Pain				No Pain			
		Baseline		Post-treatment		Baseline		Post-treatment	
Measure	Condition	M	SD	M	SD	M	SD	M	SD
Sleep onset Latency (minutes)	Waitlist	45.88	14.68	41.92[b]	9.51	47.89	12.96	35.59[b]	8.39
	Treatment	40.43	12.95	16.82[a,b,c]	8.39	36.41	13.89	8.36[a,b,c]	8.99
Wake after sleep onset (minutes)	Waitlist	53.85	10.95	45.66	9.49	47.60	9.67	42.43	8.38
	Treatment	52.41	9.66	30.06[a,c]	8.37	77.79	10.37	28.8[a,c]	8.98
Sleep efficiency (%)	Waitlist	74.31	4.1	76.44[b]	3.51	75.89	3.62	79.11[b]	3.10
	Treatment	75.64	3.62	86.04[a,b,c]	3.10	70.58	3.88	86.49[a,b,c]	3.33
Total Sleep Time (minutes)	Waitlist	363.28	22.96	380.31	21.51	352.51	20.27	374.01	18.99
	Treatment	375.72	20.26	373.76	18.99	359.98	21.73	377.05	20.36
Sleep quality rating	Waitlist	2.89	.16	2.88[b]	.19	2.97	.14	3.01[b]	.17
	Treatment	3.04	.14	3.32[a,b,c]	.17	2.79	.15	3.31[a,b,c]	.18
Pain Rating	Waitlist	1.79	.40	1.56	.38	.92	.35	1.07	.33
	Treatment	1.81	.36	1.60	.35	.93	.37	.13[a]	.35

[a] There was a significant within group difference from baseline to post-treatment *(p* < .05). [b] There was a significant difference between groups in this column *(p* < .05). [c] There was statistical equivalence between the groups compared in this row (*p* < .01). Sleep quality rating was conducted using a 1-5 scale; Pain Rating was conducted using a 0-10 scale

Table 5-3. Results of equivalency testing for SE, SOL, WASO, and SQR for the treatment and chronic pain subgroups

		SE (10%)		WASO (20 min)		SOL (20 min)		SQR (.5 points)	
	N	Z	SD	Z	SD	Z	SD	Z	SD
CBTi and History of Chronic Pain	13								
and		7.71[a]	1.24	5.59[a]	3.35	3.44[a]	3.36	7.84[a]	.06
CBTi and No History of Chronic Pain	14								
CBTi and History of Chronic Pain	13								
and		.30	1.23	1.26	3.51	1.45	3.52	.75	.07
Waitlist-Control and History of Chronic Pain	13								

[a] There was statistical equivalence between the groups compared ($p < .01$)

CHAPTER 6
DISCUSSION

Review of Findings

Aim 1

The first hypothesis, that the two treatment groups (HCP and non-HCP) would differ, was not confirmed. Both treatment groups achieved high rates of average improvement in SOL, WASO, and SE. Equivalency testing showed that regardless of a HCP, patients made significant improvements in their sleep. At post-treatment HCP and non-HCP participants receiving treatment had an average SOL below 30 minutes, WASO at or below 30 minutes, SE above 85% and improved sleep quality rating. These results suggest that CBTi for insomnia can be a brief but efficacious sleep intervention among individuals with HCP. This gives further support to the argument that comorbid insomnia can be treated using a cognitive behavioral approach, despite the presence of another physical condition. These results suggest that the Three P Model presented in Figure 2-2 is an equally accurate representation of the pattern of chronic insomnia among HCP and non-HCP participants. Precipitating effects of chronic pain do appear to have declined over time for the HCP participants and chronic pain did not significantly interfere with improvements from the treatment. More focus should be placed on expanding this efficacious treatment to the wider comorbid insomnia population. In addition, there is no statistical reason to separate older adult chronic pain and non-pain populations (that have similar characteristics to the current sample).

Aim 2

The second hypothesis, that those with a HCP receiving treatment would see improvements in their pain ratings, was not confirmed. Participants reported little or no

improvement in their pain ratings when comparing post-treatment to baseline. This suggests that changes in sleep related behaviors and cognitions that resulted from CBTi did not result in improvements in the behavioral and cognitive components in chronic pain. The fact that the treatment effects did not carry over into the pain domain further indicates that, while sleep and pain share similar behaviors and cognitions, they are not interchangeable. In addition, the interaction between chronic insomnia and chronic pain appears to be highly nuanced.

Interestingly, non-HCP participants saw significant improvements in pain rating, rather than the HCP group. However, the clinical importance of this decrease may be limited as these participants started with relatively low average pain ratings (below 1 out of 10) and decreased to a near zero level. In one prior study, chronic pain participants improved regardless of whether they were in the treatment or control group (Edinger et al., 2005). Thus, it may be that any changes observed in the current study are an artifact of the measurement of pain over time, such that initial pain ratings are higher than subsequent pain ratings for non-HCP. The relatively low baseline levels of pain among the HCP participants also limited the possibility for improvement following the treatment In addition, when previous sleep treatment studies that have found significant changes in pain they have used longer treatment protocols. It may be that a four week treatment is not sufficient time to reduce the effect of sleep problems on daily pain sensation. It would be interesting for subsequent research to explore the relationship of length of CBTi with change in pain rating.

The use of cognitive-behavioral interventions for insomnia is well supported, as previously documented, and provides significant improvements in 4 weeks (in the

current study) and has been shown to be efficacious when used in shorter two session treatment models (Edinger & Sampson, 2003; McCrae, McGovern, Lukefahr, & Stripling, 2007). There has been and continues to be a severe lack of cognitive-behavioral intervention providers relative to the need for these services. The frequency of insomnia among individuals with chronic pain and many other chronic health conditions clearly illustrates the need for this intervention in primary care. However, the time and expertise necessary for more extensive previously designed treatments have rendered their use in primary care impractical. The implementation of these short versions of behavioral interventions in primary care settings could provide more extensive treatment and could be used across disciplines and in a variety of settings. However, more research is needed to explore the effectiveness and practicality of providing these services within primary care settings.

Of the total sample of HCP participants in this study, 21 endorsed a diagnosis of arthritis or osteoarthritis. Arthritis is an inflammatory process in the joints of the body that results in swelling in the joint, joint pain, and reduced ability to move the joint. A large body of research has documented the detrimental impact of arthritis on sleep quality, nocturnal body movements, sleep fragmentation, daytime sleepiness fatigue, and depressed mood (Devins et al., 1993; Hirsch et al., 1994; Mahowald, Mahowald, Bundlie, & Ytterberg, 1989; Moldofsky, Lue, & Smythe, 1983; Power, Perruccio, & Badley, 2005). There is some research to suggest that sleep architecture among arthritic patients is lighter (less time in EEG measured delta wave patterns characteristic of deep sleep) and more likely to show alpha wave patterns (Moldofsky et al., 1983). However, others have failed to show these EEG abnormalities (Hirsch et al., 1994).

Regardless of changes in sleep architecture, all of the articles reviewed agree that subjective complaints of sleep disruption indicate that insomnia is a widespread and major concern among arthritic patients. Thus, it is important for this treatment to be further studied among arthritic patients as an efficacious and effective treatment among this population.

Limitations

This study was limited by the use of secondary data. The sample was drawn as a sample of older adults with insomnia but did not specifically target individuals with pain conditions. Since the pain sample that was drawn was one of convenience there was less control over the participant selection. In addition, the use of a no treatment/control group is a less powerful test of the treatment efficacy than using a placebo group. Once participants were separated into the four comparison groups, the size of the groups may have reduced the ability to detect small but significant improvements. Changes that were not significant in the current study might have reached significance in a larger sample. Specifically trends toward improvement in pain rating among the HCP treatment group may have been significant if a larger sample was collected.

There might be some concern regarding the use of subjective measurement of sleep. However, since insomnia is inherently a subjective complaint self report data is the most appropriate means of collecting data for both diagnoses and treatment. This is the approach used throughout the CBTi literature so use of this data type allows for easier comparison of the current results to that of past research. The reporting of pain conditions was self-report, and was not followed by a review of the individual's medical files or any other confirmatory procedure. However, since pain conditions are intrinsically subjective, the use of a subjective medical history does not greatly spoil this

study. The collection of daily pain ratings was limited by the fact that this information was only collected once during each day and is consequently subject to bias by extreme pain levels at one time of the day. In addition, the rating of pain was only completed at one time in the day (morning) and consequently changes during other times of the day went unrecorded. The sample of older adults was from a group of younger older adults and as a result may not be representative of older adults, older than 69.1 (mean age) + 7.0 (standard deviation). The sample was also more highly educated than the average older United States citizen, which is about 12 years education (U.S. Census Bureau, 2000).

The use of a single two week post-treatment time as the outcome period may have impacted the findings. It has been recommended by a consensus meeting of pain researchers, that multiple time point measurement serve as the most appropriate means of evaluating the efficacy of a treatment (Turk et al., 2008). The two week outcome period used in the current study could be considered a multiple time point measurement as pain ratings over multiple days reduces the potential for Type I errors (false detection of significant differences). However, this approach does not allow for the same degree of certainty that temporally separated time points would allow for. In interpreting results the current results is also important to consider that changes in pain symptoms may require a longer period of time than the two weeks that were measured. Perhaps a longer period of good sleep is necessary before changes take place in the peripheral and central nervous system and the HPA axis. Examination of follow-up data for study participants would provide this information with a minimal investment on the part of participants and researchers.

Future Directions

Additional research on the role of the HPA axis and inflammation in comorbid chronic pain and insomnia could lend greater understanding to the etiology of both conditions. This could also aid in the development of more specifically targeted treatments. The circadian rhythm is known to regulate the patterns of endorphins and encephalins, as well as reports of pain intensity (Junker & Wirz, 2009). By reestablishing a regular sleep schedule, circadian modified systems may be brought into a more stable pattern. Although individuals with HCP did not see significant improvements in their pain rating, the potential may exist for improvement of pain symptoms through sleep treatment. The lack of finding in this area may be a product of the timing of sleep and pain measurement. The pain item was completed in the morning, shortly after awakening. It is possible that pain and stiffness that comes with awakening was not greatly affected by improved sleep. Indeed, the highest ratings of pain intensity among those with fibromyalgia (Bellamy, Sothern, & Campbell, 2004) and rheumatoid arthritis (Cutolo & Straub, 2008) are reported in the morning. Among arthritis patients this heightened pain level has been linked to the effects of proinflammatory hormones and cytokines that reach a peak just prior to awakening (Cutolo & Straub, 2008). In the current study it is possible that there was a differential impact on pain intensity during the day and prior to bedtime compared to the higher intensity morning pain. Future research should include more time points of pain measurement over the course of the day to account for the circadian nature of pain and to better assess the impact of sleep treatment on these patterns.

While the current study did not find improvement in pain ratings among HCP participants, previous research has shown that treating sleep can improve pain. Erikson

and Ursin (2002) posit that many subjective health complaints not corroborated with medical procedures (fibromyalgia, irritable bowel, food insensitivity, chemical insensitivity, etc.) maybe the result of chronic stress, negative affect, poor sleep, and hypervigilance to bodily sensations. This theory, known as Cognitive Activation Theory, helps to explain many of the findings of the central sensitization model (Eriksen & Ursin, 2002) among chronic pain patients. In addition, this theory helps to explain why previous sleep treatment studies have found that improvements in sleep have resulted in improvements in pain perception. This theory is likely less applicable to the current study since a large majority of the HCP group reported arthritis, an inflammatory disease, rather than a sensitivity disease. Further research on the role of central sensitization in chronic insomnia should focus more on sensitivity diseases (e.g. fibromyalgia, irritable bowels syndrome) and should include direct measurement of the typical patterns seen in central sensitization, such as reduced pain threshold, spatial spreading of pain sensation and increased temporal length of pain sensation.

It seems particularly interesting that non-HCP participants reported significant improvements in their pain rating. This finding may suggest that the treatment is helpful in reducing the common aches and pains and acute pain among older adults with insomnia. Pain sensitivity among this group could be linked to sleep problems through the previously mentioned effects of sleep deprivation on pain thresholds and peripheral sensitization (Onen et al., 2001; Onen et al., 2005). However, as was previously noted is important to consider that non-HCP participants started with low average levels of pain, and these individuals did not initially complain of pain as a clinical concern. With this in mind, research is needed on the use of CBTi to provide improvements in other

domains of life (such as pain), among the general older adult population. Indeed improvements in mood, energy, vitality, social functioning, and pain have been observed in a numerous studies among various populations (Morgan, Dixon, Mathers, Thompson, & Tomeny, 2003; Quesnel, Savard, Simard, Ivers, & Morin, 2003; Rybarczyk, Lopez, Schelble, & Stepanski, 2005).

Affect is one domain of life in which improvements have been widely found using CBTi. Negative affect and sleep difficulties co-occur to such as degree that the DSM-IV lists insomnia as a major symptom of all depressive syndromes. Mood disorders have been found to co-occur with insomnia at a rate of 35% – 46% (Buysse et al., 1994; Coleman et al., 1982). However, improvements in affect are often seen as a result of improved sleep (Nowell & Buysse, 2001). In fact, CBTi has resulted in significant improvements in mental well-being and mood among individuals with various comorbid conditions including major depression, breast cancer, and fibromyalgia (Dirksen & Epstein, 2008; Edinger et al., 2005; Manber et al., 2008). Improvements in this essential quality of life domain are an area that needs more thorough investigation. It would be interesting to determine whether changes in sleep and mood predict changes in pain.

Summary of the Study

The current study found that older adults with comorbid chronic insomnia and a history of chronic pain can see significant improvements in their sleep from CBTi. Improvements across multiple measures of sleep in the history of chronic pain group were shown to be comparable to the improvements seen among older adults without a history of chronic pain. However, the study did not find improvements in pain ratings among the history of chronic pain participants. This study makes another important step in advancing efficacious treatment for patients with insomnia and comorbid pain. It also

lends support for the treatment of the many forms of comorbid chronic insomnia. Further research is needed in order to explore potential benefits beyond improved sleep that comorbid insomnia populations may experience from the successful treatment of their insomnia. As was previously mentioned, changes in affect and diagnosis specific symptoms (e.g. pain sensitivity, bowels problems) may result from the successful treatment of chronic insomnia. It is now essential that sleep clinicians and researchers translate this treatment to real world settings and carry it to their patients through clinical practice and effectiveness trials.

REFERENCES

American Academy of Sleep Medicine (2001). International classification of sleep disorders, revised: Diagnostic and coding manual. Chicago, Illinois: American Academy of Sleep Medicine.

Arnold, L. M. (2009). Strategies for managing fibromyalgia. *Am J Med, 122*(12 Suppl), S31-43.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* New York: International Universities Press.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1996). Beck Depression Inventory-Second Edition. The Psychological Corporation: San Antonio, TX.

Belanger, L., Savard, J., & Morin, C. M. (2006). Clinical management of insomnia using cognitive therapy. *Behav Sleep Med, 4*(3), 179-198.

Bellamy, N., Sothern, R. B., & Campbell, J. (2004). Aspects of diurnal rhythmicity in pain, stiffness, and fatigue in patients with fibromyalgia. *J Rheumatol, 31*(2), 379-389.

Buysse, D. J., Reynolds, C. F., 3rd, Hauri, P. J., Roth, T., Stepanski, E. J., Thorpy, M. J., et al. (1994). Diagnostic concordance for DSM-IV sleep disorders: a report from the APA/NIMH DSM-IV field trial. *Am J Psychiatry, 151*(9), 1351-1360.

Cassisi, G., Sarzi-Puttini, P., Alciati, A., Casale, R., Bazzichi, L., Carignola, R., et al. (2008). Symptoms and signs in fibromyalgia syndrome. *Reumatismo, 60 Suppl 1*, 15-24.

Chapman, C. R., Tuckett, R. P., & Song, C. W. (2008). Pain and stress in a systems perspective: reciprocal neural, endocrine, and immune interactions. *J Pain, 9*(2), 122-145.

Coleman, R. M., Roffwarg, H. P., Kennedy, S. J., Guilleminault, C., Cinque, J., Cohn, M. A., et al. (1982). Sleep-wake disorders based on a polysomnographic diagnosis. A national cooperative study. *JAMA, 247*(7), 997-1003.

Currie, S. R., Wilson, K. G., & Curran, D. (2002). Clinical Significance and Predictors of Treatment Response of Cognitive-Behavior Therapy for Insomnia Secondary to Chronic Pain. *Journal of Behavioral Medicine, 25*(2), 135-153.

Currie, S. R., Wilson, K. G., Pontefract, A. J., & deLaplante, L. (2000). Cognitive-Behavioral Treatment of Insomnia Secondary to Chronic Pain. *Journal of Consulting and Clinical Psychology, 68*(3), 407-416.

Cutolo, M., & Straub, R. H. (2008). Circadian rhythms in arthritis: hormonal effects on the immune/inflammatory reaction. *Autoimmun Rev, 7*(3), 223-228.

Cutolo, M., Straub, R. H., & Buttgereit, F. (2008). Circadian rhythms of nocturnal hormones in rheumatoid arthritis: translation from bench to bedside. *Ann Rheum Dis, 67*(7), 905-908.

Dang-Vu, T. T., Desseilles, M., Peigneux, P., & Maquet, P. (2006). A role for sleep in brain plasticity. *Pediatr Rehabil, 9*(2), 98-118.

Devins, G. M., Edworthy, S. M., Paul, L. C., Mandin, H., Seland, T. P., Klein, G., et al. (1993). Restless sleep, illness intrusiveness, and depressive symptoms in three chronic illness conditions: rheumatoid arthritis, end-stage renal disease, and multiple sclerosis. *J Psychosom Res, 37*(2), 163-170.

Dirksen, S. R., & Epstein, D. R. (2008). Efficacy of an insomnia intervention on fatigue, mood and quality of life in breast cancer survivors. *J Adv Nurs, 61*(6), 664-675.

Edinger, J. D., & Sampson, W. S. (2003). A primary care "friendly" cognitive behavioral insomnia therapy. *Sleep, 26*(2), 177-182.

Edinger, J. D., Wohlgemuth, W. K., Krystal, A. D., & Rice, J. R. (2005). Behavioral insomnia therapy for fibromyalgia patients: a randomized clinical trial. *Arch Intern Med, 165*(21), 2527-2535.

Eriksen, H. R., & Ursin, H. (2002). Sensitization and subjective health complaints. *Scand J Psychol, 43*(2), 189-196.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res, 12*(3), 189-198.

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *JAMA, 262*(11), 1479-1484.

Gerstle, D. S., All, A. C., & Wallace, D. C. (2001). Quality of Life and Chronic Nonmalignant Pain. *Pain Management Nursing, 2*(3), 98-109.

Hart, F. D., Taylor, R. T., & Huskisson, E. C. (1970). Pain at night. *Lancet, 1*(7652), 881-884.

Harvey, A. (2005). A cognitive theory and therapy for chronic insomnia. *Journal of Cognitive Psychotherapy: An International Quarterly, 19*(1), 41-59.

Hauri, P., Chernik, D., Hawkins, D., & Mendels, J. (1974). Sleep of depressed patients in remission. *Arch Gen Psychiatry, 31*(3), 386-391.

Hirsch, M., Carlander, B., Verge, M., Tafti, M., Anaya, J. M., Billiard, M., et al. (1994). Objective and subjective sleep disturbances in patients with rheumatoid arthritis. A reappraisal. *Arthritis Rheum, 37*(1), 41-49.

Huberty, C. J. & Morris, J.D. (1989). Multivariate analysis versus multiple univariate analyses. *Psychological Bulletin. Vol 105*(2), 302-308.

Irwin, M. R., Clark, C., Kennedy, B., Christian Gillin, J., & Ziegler, M. (2003). Nocturnal catecholamines and immune function in insomniacs, depressed patients, and control subjects. *Brain Behav Immun, 17*(5), 365-372.

Irwin, M. R., Wang, M., Campomayor, C. O., Collado-Hidalgo, A., & Cole, S. (2006). Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. *Arch Intern Med, 166*(16), 1756-1762.

Junker, U., & Wirz, S. (2009). Chronobiology: influence of circadian rhythms on the therapy of severe pain. *J Oncol Pharm Pract*.

Kay, D. C. (1975). Human sleep during chronic morphine intoxication. *Psychopharmacologia, 44*(2), 117-124.

Kay, D. C., Pickworth, W. B., Neidert, G. L., Falcone, D., Fishman, P. M., & Othmer, E. (1979). Opioid effects on computer-derived sleep and EEG parameters in nondependent human addicts. *Sleep, 2*(2), 175-191.

Latremoliere, A., & Woolf, C. J. (2009). Central sensitization: a generator of pain hypersensitivity by central neural plasticity. *J Pain, 10*(9), 895-926.

Li, J., Simone, D. A., & Larson, A. A. (1999). Windup leads to characteristics of central sensitization. *Pain, 79*(1), 75-82.

Lichstein, K. L., Durrence, H. H., Taylor, D. J., Bush, A. J., & Riedel, B. W. (2003). Quantitative criteria for insomnia. *Behav Res Ther, 41*(4), 427-445.

Lichstein, K. L., Wilson, N. M., & Johnson, C. T. (2000). Psychological treatment of secondary insomnia. *Psychol Aging, 15*(2), 232-240.

MacDonald, S., Linton, S. J., & Jansson-Frojmark, M. (2008). Avoidant safety behaviors and catastrophizing: shared cognitive-behavioral processes and consequences in co-morbid pain and sleep disorders. *Int J Behav Med, 15*(3), 201-210.

Mahowald, M. W., Mahowald, M. L., Bundlie, S. R., & Ytterberg, S. R. (1989). Sleep fragmentation in rheumatoid arthritis. *Arthritis Rheum, 32*(8), 974-983.

Manber, R., Edinger, J. D., Gress, J. L., San Pedro-Salcedo, M. G., Kuo, T. F., & Kalista, T. (2008). Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep, 31*(4), 489-495.

Martelli, M. F., Zasler, N. D., Grayson, R. L., & Liljedahl, E. L. (1999). *Kinesiophobia and cogniphobia: Assessment of avoidance conditioned pain related disability (ACPRD).* Retrieved April 18, 2009, from www.angelfire.com/va/MFMartelliPhD/nanposters.html.

McCrae, C. S., McGovern, R., Lukefahr, R., & Stripling, A. M. (2007). Research Evaluating Brief Behavioral Sleep Treatments for Rural Elderly (RESTORE): a preliminary examination of effectiveness. *Am J Geriatr Psychiatry, 15*(11), 979-982.

Mills, P. J., von Kanel, R., Norman, D., Natarajan, L., Ziegler, M. G., & Dimsdale, J. E. (2007). Inflammation and sleep in healthy individuals. *Sleep, 30*(6), 729-735.

Moldofsky, H., Lue, F. A., & Smythe, H. A. (1983). Alpha EEG sleep and morning symptoms in rheumatoid arthritis. *J Rheumatol, 10*(3), 373-379.

Morgan, K., Dixon, S., Mathers, N., Thompson, J., & Tomeny, M. (2003). Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. *Br J Gen Pract, 53*(497), 923-928.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep, 22*(8), 1134-1156.

Morin, C. M., Kowatch, R. A., & O'Shanick, G. (1990). Sleep Restriction for the Inpatient Treatment of Insomnia. *Sleep, 13*(2), 183-186.

Morin, C. M., Mimeault, V., & Gagne, A. (1999). Nonpharmacological treatment of late-life insomnia. *J Psychosom Res, 46*(2), 103-116.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol Aging, 8*(3), 463-467.

Murden, R. A., McRae, T. D., Kaner, S., & Bucknam, M. E. (1991). Mini-Mental State exam scores vary with education in blacks and whites. *J Am Geriatr Soc, 39*(2), 149-155.

Nowell, P. D., & Buysse, D. J. (2001). Treatment of insomnia in patients with mood disorders. *Depress Anxiety, 14*(1), 7-18.

Ohayon, M. M. (2005). Relationship between chronic painful physical condition and insomnia. *Journal of Psychiatric Research, 39*, 151-159.

Onen, S. H., Alloui, A., Gross, A., Eschallier, A., & Dubray, C. (2001). The effects of total sleep deprivation, selective interruption and sleep recovery of pain tolerance thresholds in healthy subjects. *Journal of Sleep Research, 10*, 35-42.

Onen, S. H., Onen, F., Courpron, P., & Dubray, C. (2005). How pain and analgesics disturb sleep. *Clin J Pain, 21*(5), 422-431.

Power, J. D., Perruccio, A. V., & Badley, E. M. (2005). Pain as a mediator of sleep problems in arthritis and other chronic conditions. *Arthritis Rheum, 53*(6), 911-919.

Quesnel, C., Savard, J., Simard, S., Ivers, H., & Morin, C. M. (2003). Efficacy of cognitive-behavioral therapy for insomnia in women treated for nonmetastatic breast cancer. *J Consult Clin Psychol, 71*(1), 189-200.

Rogers, J. L., Howard, K. I., & Vessey, J. T. (1993). Using significance tests to evaluate equivalence between two experimental groups. *Psychol Bull, 113*(3), 553-565.

Rosenthal, M., Moore, P., Groves, E., Iwan, T., Schlosser, L. G., Dziewanowska, Z., et al. (2007). Sleep improves when patients with chronic OA pain are managed with morning dosing of once a day extended-release morphine sulfate (AVINZA): findings from a pilot study. *J Opioid Manag, 3*(3), 145-154.

Rybarczyk, B., Lopez, M., Schelble, K., & Stepanski, E. (2005). Home-based video CBT for comorbid geriatric insomnia: a pilot study using secondary data analyses. *Behav Sleep Med, 3*(3), 158-175.

Rybarczyk, B., Stepanski, E., Fogg, L., Lopez, M., Barry, P., & Davis, A. (2005). A placebo-controlled test of cognitive-behavioral therapy for comorbid insomnia in older adults. *J Consult Clin Psychol, 73*(6), 1164-1174.

Salter, M. W. (2002). The neurobiology of central sensitization. *Journal of Muscoloskeletal Pain, 10*(1), 22-33.

Spielman, A. (1986). Assessment of insomnia. *Clinical Psychology Review, 6*, 11-25.

Sullivan, M. J., Bishop, S. R., & Pivik, J. (1995). The pain catastrophizing scale: Development and validation. *Psychological Assessment, 7*, 524-532.

Tang, N. K., & Harvey, A. G. (2004). Correcting distorted perception of sleep in insomnia: a novel behavioural experiment? *Behav Res Ther, 42*(1), 27-39.

Taylor, D. J., Mallory, L. J., Lichstein, K. L., Durrence, H. H., Riedel, B. W., & Bush, A. J. (2007). Comorbidity of chronic insomnia with medical problems. *Sleep, 30*(2), 213-218.

Taylor, L. M., Espie, C. A., & White, C. A. (2003). Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behav Sleep Med, 1*(4), 200-212.

Turk, D. C., Dworkin, R. H., McDermott, M. P., Bellamy, N., Burke, L. B., Chandler, J. M., et al. (2008). Analyzing multiple endpoints in clinical trials of pain treatments: IMMPACT recommendations. Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials. *Pain, 139*(3), 485-493.

Uceyler, N., Valenza, R., Stock, M., Schedel, R., Sprotte, G., & Sommer, C. (2006). Reduced levels of antiinflammatory cytokines in patients with chronic widespread pain. *Arthritis Rheum, 54*(8), 2656-2664.

U.S. Census Bureau. (2000). *Sex by age by educational attainment for the population 18 years and over.* Retrieved February 11, 2010 from http://factfinder.census.gov/ servlet/DTTable?_bm=y&-geo_id=01000US&-ds_name=DEC_2000_SF3_U&-redoLog=false& mt_name=DEC_2000_SF3_U_PCT025

Van Cauter, E., Holmback, U., Knutson, K., Leproult, R., Miller, A., Nedeltcheva, A., et al. (2007). Impact of sleep and sleep loss on neuroendocrine and metabolic function. *Horm Res, 67 Suppl 1*, 2-9.

Vitiello, M. V., Rybarczyk, B., Von Korff, M., & Stepanski, E. J. (2009). Cognitive Behavioral Therapy for Insomnia Improves Sleep and Decreases Pain in Older Adults with Co-Morbid Insomnia and Osteoarthritis *Journal of Clinical Sleep Medicine, 5*(4), 355-362.

Waddell, G., Newton, M., Henderson, I., Somerville, D., & Main, C. J. (1993). A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain, 52*(2), 157-168.

Wang, M. Y., Wang, S. Y., & Tsai, P. S. (2005). Cognitive behavioural therapy for primary insomnia: a systematic review. *J Adv Nurs, 50*(5), 553-564.

Wright, V. (1985). Measurement of outcome in rheumatic diseases. *J R Soc Med, 78*(12), 985-994.

Youngstedt, S. D., O'Connor, P. J., & Dishman, R. K. (1997). The effects of acute exercise on sleep: a quantitative synthesis. *Sleep, 20*(3), 203-214.

BIOGRAPHICAL SKETCH

Mr. Williams received his Master of Science in clinical psychology in the spring of 2010 and is continuing his studies toward a doctorate. He is a graduate student in the Department of Clinical and Health Psychology at the University of Florida and is studying to be a clinical health psychologist. Mr. Williams has been involved in sleep research for 4 year. His training began in the sleep research lab of Dr. Daniel Taylor at University of North Texas, where he received a Bachelor of Science in psychology. He continued his research experience under the guidance of Dr. Mary Carskadon at Brown University as part of the Williams C. Dement Sleep Research Apprenticeship. He is currently studying chronic health conditions and sleep in older adults in the research lab of Dr. Christina McCrae. He received the 2009 TSS Merit Based Award at the meeting of Associated Professional Sleep Societies. Research interests include coping behaviors used by individuals suffering from chronic health conditions and psychological/behavioral etiological and mediating factors of chronic illness. Of particular interest is how poor sleep can lead to, be caused by, and exacerbate chronic health conditions. Mr. Williams is committed to helping make cognitive-behavioral treatments for sleep problems more integrative, accessible, and effective in clinical practice.