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EFFECTIVENESS OF A CBT INTERVENTION FOR PERSISTENT INSOMNIA AND
HYPNOTIC DEPENDENCY IN AN OUTPATIENT PSYCHIATRY CLINIC

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
Philosophy at Virginia Commonwealth University

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Abstract

EFFECTIVENESS OF A CBT INTERVENTION FOR PERSISTENT INSOMNIA AND HYPNOTIC DEPENDENCY IN AN OUTPATIENT PSYCHIATRY CLINIC

By Hannah Lund Taylor, M.S.

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
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Previous research supports the efficacy of cognitive-behavioral therapy for insomnia (CBT-I) in patients with comorbid psychiatric diagnoses; however, questions remain about the effectiveness of CBT-I due to the fact that previous studies excluded patients with significant psychiatric symptoms and comorbid diagnoses. This study begins to address this gap in the insomnia literature by testing a five-session CBT-I intervention in a diverse sample of patients receiving mental health treatment in an outpatient psychiatry clinic (N=23) who continue to experience chronic insomnia despite receiving pharmacological treatment for sleep.

Participants were randomized to CBT-I (n=13) or a treatment as usual control group (n=10). Following one week of baseline sleep diary assessment, those in the CBT-I group completed five sessions of individual treatment; three in-person and two by phone. Those in the treatment as usual group continued with medication treatment as prescribed by their psychiatrist for a five-week period and were then given the opportunity to cross over to receive CBT-I.

Study results show that adding a brief CBT-I intervention to usual care led to significant improvements in sleep compared to treatment as usual. Effect sizes were generally large, illustrating the potency of CBT-I in a psychiatric sample. Sleep gains were largely maintained at two-month follow-up. No significant changes in depression or anxiety severity were seen in the CBT-I group, suggesting that sleep interventions alone may not have the same impact in a psychiatric population with more severe and chronic mental health symptoms. Quality of life in the area of social functioning was improved following CBT-I compared to treatment as usual; however, this gain was not maintained at two-month follow-up. Finally, CBT-I was not associated with a reduction in use of sleep medication. This may reflect this sample's high level of chronicity of insomnia or a propensity towards medication dependency.

In sum, the findings of this study suggest that selected patients with complex and chronic psychiatric conditions can obtain sleep improvements with CBT-I beyond those obtained with pharmacotherapy alone. Future research should focus on identifying factors that predict which “real-world” psychiatric patients are most likely to undergo and benefit from CBT-I.

Effectiveness of a CBT Intervention for Persistent Insomnia and Hypnotic Dependency in an Outpatient Psychiatry Clinic

Chronic insomnia is a common sleep disorder that affects approximately 10-15% of the adult population (Roth, 2001) and poses serious implications for daily functioning (Dement & Pelayo, 1997; Simon & VonKorff, 1997) and quality of life (Léger, Guilleminault, Bader, Lévy, & Paillard, 2002). Pharmacotherapy with hypnotic medications has been one of the most common treatments offered to patients with insomnia and continues to be the first line treatment in many medical practices (National Institutes of Health, 2005); however, there is increasing concern about the efficacy and safety of long-term use of these medications. The literature shows a lack of randomized, controlled trials to test the efficacy and safety of using these medications for more than three or four weeks (National Institutes of Health, 2005) and what little data exists to examine the durability of benefits suggests that the improvements gradually degrade over time (Morin, Colecchi, Stone, Sood, & Brink, 1999). Additional concerns about the use of hypnotic medications include the potential for abuse and dependence, the risk of tolerance and withdrawal, residual daytime effects, increased morbidity from falls or injuries, rebound insomnia, and increased mortality (Kripke et al., 1998; Kripke, 2000; Kupfer & Reynolds, 1997). As a result of these concerns and the paucity of data to support the use of hypnotic medications in treating chronic insomnia, cognitive behavioral therapy for insomnia (CBT-I) is becoming increasingly accepted as the preferred treatment for this sleep disorder. Existing research supports that the efficacy of CBT-I is comparable to that of hypnotic medications (e.g. Morin et al., 2006) and benefits have been shown to be more durable with behavioral treatment (Doghrramji, 2010); however, more research is needed to examine the effectiveness of CBT-I in real-world populations.

Insomnia is particularly pervasive in psychiatric populations (Smith, Huang, & Manber, 2005). Even when mental health conditions such as depression or anxiety are effectively managed, insomnia frequently persists (Smith et al., 2005). If left untreated, chronic insomnia can exacerbate mood or anxiety problems, prevent the full remission of depressive disorders (Watanabe et al., 2011), and otherwise reduce quality of life (Smith et al., 2005). Most research studies that have tested CBT-I in chronic insomnia have examined its efficacy in those with pure insomnia and have therefore excluded those with comorbid mental health diagnoses (e.g. Morin et al., 2006). The studies that have attempted to examine CBT-I for insomnia that is comorbid with psychiatric diagnoses have excluded those with significant symptoms (e.g. Espie, Inglis, Tessier, & Harvey, 2001). Due to the limitations of existing studies, more research is needed to understand the effectiveness of CBT-I for insomnia in psychiatric populations and to determine whether treating the insomnia may also improve the mental health conditions.

Psychiatrists and primary care physicians are frequently called upon to address insomnia in their depressed or anxious patients. Due to the common assumption that disturbed sleep is a symptom rather than a separate diagnosis, many physicians will focus only on targeting the underlying mental health condition and may fail to recognize insomnia as a potentially separate diagnosis (Neylan, 2011). Those physicians who do recognize that persistent symptoms of insomnia warrant individualized treatment may rely on hypnotic medications despite recognition of the concerns about their long term use. More than 65% of patients who are prescribed hypnotic medications will continue using them for more than one year (Ohayon & Caulet, 1996). When patients do not experience improvements in their sleep, physicians must manage their distress and may struggle to come up with treatment alternatives for persistent symptoms. Physicians who find hypnotic medications to be ineffective in treating their patients' symptoms

and understand the risks of prescribing them for an extended period of time may be open-minded to a behavioral treatment but may not have a feasible way to offer this to patients. For many providers, there is a lack of convenient access to behavioral sleep medicine specialists, who typically deliver CBT-I (Neylan, 2011) despite research suggesting that nurse practitioners or other medical staff without specific training in sleep medicine can effectively administer it (Buysse et al., 2011). Reducing hypnotic medications can be difficult after long term use, even if patients have been prescribed a low dose (Morin et al., 2004). A small number of studies have considered how CBT-I may help patients with chronic insomnia to reduce their use of hypnotic medication in addition to improving their sleep (e.g. Espie, Inglis, Tessier, et al., 2001; Morgan, Dixon, Mathers, Thompson, & Tomeny, 2003; Morin et al., 2004; Morin, Colecchi, Ling, & Sood, 1995). The results of these studies have been positive; however, more research is needed to understand if CBT-I is effective in reducing hypnotic use in patients with psychiatric comorbidities.

The present study built upon existing research by testing the effectiveness of a five-session CBT treatment for persistent insomnia in chronic users of sleep medication who were receiving mental health treatment in an outpatient psychiatry clinic. Participants were individuals with psychiatric diagnoses (e.g. major depression, generalized anxiety disorder) who continued to experience disturbed sleep despite the use of medications prescribed for sleep. Previous research has shown that CBT-I is an effective treatment for insomnia comorbid with psychiatric symptoms in sleep clinics (e.g. Dashevsky & Kramer, 2008; Perlis et al., 2000) and primary care clinics (e.g. Espie et al., 2007; Espie, Inglis, Tessier, et al., 2001); however, only one previous study has tested CBT-I in an outpatient psychiatry setting (Watanabe et al., 2011). Additionally, research has supported the use of CBT-I in reducing use of hypnotic medications

taken for sleep (e.g. Morgan et al., 2003); however, not specifically in a psychiatric population. In the following sections of this paper, a review of the relevant literature on insomnia and CBT-I will be presented. Thereafter, a statement of the problem and rationale for the study will be presented and specific hypotheses will be proposed based on the following research questions: 1) Is CBT-I effective at improving sleep and reducing hypnotic use in an outpatient psychiatric population? 2) Does CBT-I in this population lead to improvement in psychiatric symptoms (e.g. depression, anxiety)? and 3) How durable are the observed improvements in sleep disturbance? Following the study hypotheses, the method used to carry out the study will be described in depth, results will be reported, and a discussion of the findings will be presented along with study limitations and recommendations for future research.

Review of the Literature

Insomnia. Insomnia is defined as a difficulty initiating or maintaining sleep, or a complaint of non-restorative sleep, for a duration of at least one month (American Psychiatric Association, 2000). The sleep disturbance or associated daytime fatigue must cause significant distress or impairment. Insomnia that occurs independent of other conditions, referred to as *Primary Insomnia*, can be challenging to treat; however, treatment can be complicated further when the sleep disturbance is accompanied by medical or psychiatric comorbidities, such as arthritis or depression. Formerly termed *Secondary Insomnia*, insomnia that co-occurs with other medical or psychiatric conditions is now referred to as *Comorbid Insomnia*, after a 2005 National Institutes of Health (NIH) State of the Science conference statement refuted the common assumption that insomnia is a direct consequence of a more primary medical condition. The statement suggested that insomnia should be conceptualized as a separate diagnosis that warrants individualized treatment (National Institutes of Health, 2005).

Chronic insomnia affects approximately 10-15% of the adult population (Roth, 2001); with the majority of affected individuals reporting symptoms comorbid with other medical or psychiatric conditions (Stepanski & Rybarczyk, 2006). In one study, clinical interviews conducted with patients presenting for insomnia assessment at a sleep disorders center revealed that 20% of patients met criteria for a diagnosis of primary insomnia, while 44% of patients met criteria for insomnia associated with a comorbid mental health disorder (Buysse et al., 1994). With psychiatric diagnoses such as depression and anxiety being the most common disorders associated with comorbid insomnia (Stepanski & Rybarczyk, 2006), this sleep disorder is particularly prevalent in psychiatric populations. In one study, Katz and McHorney (1998) reported that odds ratios for patients with major depression experiencing mild to severe insomnia ranged from 2.3 to 8.2, respectively. Another study of 1200 adults between the ages of 21 and 30 who were randomly selected from a 400,000-member health maintenance organization found a lifetime prevalence of insomnia of 24.6%, with increased prevalence of major depression (31.1% compared to 2.7%) and any anxiety disorder (35.9% compared to 19.1%) in those with insomnia compared to those without (Breslau, Roth, Rosenthal, & Andreski, 1996).

Etiological Theory of Comorbid Insomnia. The increased prevalence of insomnia in psychiatric populations is understood through the standard theoretical framework of insomnia, which relies upon a diathesis-stress model to conceptualize how insomnia develops (Morin & Espie, 2004; Spielman, Caruso, & Glovinsky, 1987). This model posits that the onset of a disorder is the result of an existing vulnerability that emerges when triggered by environmental stress. For insomnia, Spielman's 3-Ps model expands on this idea, stating that a predisposition to sleep disturbance (e.g. genetic traits, underlying personality characteristics) coupled with precipitating factors (e.g. life stressors, schedule changes) may lead to an episode of insomnia

(Spielman et al., 1987). When maladaptive behavior and thinking patterns, referred to as perpetuating factors, develop as a consequence of insomnia (e.g. spending excess time in bed, consuming alcohol to induce sleep, negative thoughts about the consequences of poor sleep), the sleep disturbance may be maintained and insomnia may become a chronic issue (Spielman et al., 1987). Work-related stress or the birth of a new baby are common examples of precipitating factors; however, a psychiatric diagnosis can also trigger an episode of insomnia (Rybarczyk, Lund, Mack, & Stepanski, 2009). Psychiatric disorders have been thought to predispose an individual to developing insomnia but they may also function as precipitating factors (Erman, 2007). Symptoms of depression (e.g. fatigue, negative affect) or anxiety (e.g. worry, hyperarousal) and the distress associated with these symptoms may interfere with sleep and sleep scheduling, thereby precipitating a period of disturbed sleep (Erman, 2007). Both as a direct result of psychiatric symptoms and due to attempts to ameliorate disturbed sleep, behavioral changes may take place that perpetuate the sleep problem and maintain it over time. For example, a depressed individual experiencing fatigue and a loss of interest in normal activities may engage in frequent napping. Alternatively, an individual with an anxiety disorder may experience excessive pre-sleep arousal (Smith et al., 2005).

Importance of Treating Comorbid Insomnia. The literature supports that mental health disorders do not only precipitate insomnia but insomnia may precipitate mental health disorders. Through this reciprocal relationship, distress associated with disturbed sleep has been conceptualized as a risk factor for the development of anxiety or depression (Ohayon & Roth, 2003). In those who have existing mental health diagnoses, the stress of an episode of insomnia may exacerbate the condition and promote a recurrence of symptoms in those who have experienced partial or full remission (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). In

otherwise healthy people, insomnia places a significant burden on the individual by reducing quality of life (Léger et al., 2002), impairing cognitive function, and increasing accident risk and work absenteeism (Walsh, 2004). In individuals who also suffer from psychiatric diagnoses, however, the burden of insomnia can be even greater. For example, multiple studies have suggested that comorbid insomnia occurring in depressed individuals not only provides a major source of distress but may also interact with other depressive symptoms to convey greater illness severity (McCall, Reboussin, & Cohen, 2000; Perlis, Smith, & Orff, 2002; Turek, 2005).

Because insomnia is often viewed as a secondary symptom of psychiatric disorders, it frequently goes untreated in psychiatric populations (Stepanski & Rybarczyk, 2006). The standard approach to treating comorbid insomnia has historically involved a focus on the primary disorder with the expectation that insomnia would improve as the symptoms of the disorder abate (Stepanski & Rybarczyk, 2006). Contrary to this approach, however, insomnia rarely improves following specific treatment for the psychiatric condition (Stepanski & Rybarczyk, 2006). Following the reconceptualization of comorbid insomnia at the start of the millennium, insomnia is no longer viewed as a secondary process but is now recognized to be a separate disorder that warrants individualized treatment (Rybarczyk et al., 2009). A solid base of literature highlights the importance of treating insomnia that co-occurs with mental health disorders, with the majority of the research focusing on insomnia comorbid with major depression.

Comorbid Insomnia and Depression. Several studies have shown that persistent sleep disturbance is associated with slower recovery from depression and lower rates of remission (Buysse et al., 1997; Dew et al., 1997; Winokur & Reynolds, 1994). The presence of comorbid insomnia may therefore hinder response to antidepressant treatment (Smith et al., 2005). Insomnia has also been shown to threaten the stability of response to treatment (Dew et al.,

1007). Nierenberg et al., (1999) found insomnia to be the most common persistent residual symptom in individuals who complete depression treatment, with 44% of full responders in their study reporting continued symptoms of insomnia at post-treatment. This finding is of particular concern due to the fact that insomnia may present a significant risk for relapse (Reynolds et al., 1997). In a study by Reynolds et al. (1997), two thirds of patients with persistent insomnia at the end of treatment for depression with an antidepressant medication and psychotherapy experienced relapse of depressive symptoms within one year. In contrast, 90% of patients in the study without sleep disturbance at the end of treatment remained well within the year after discontinuing the treatment (Reynolds et al., 1997). As an additional concern, depressed patients with comorbid insomnia may exhibit increased suicidality (Agargun, Kara, & Solmaz, 1997). In one study, Agargun et al. (1997) found that depressed patients who reported disturbed sleep also exhibited higher rates of suicidal behavior in comparison to those without sleep problems.

Comorbid Insomnia and Anxiety Disorders. In the context of anxiety disorders, sleep disturbance is a prominent feature of two anxiety disorders in the DSM-IV; post-traumatic stress disorder (PTSD) and generalized anxiety disorder (GAD; American Psychiatric Association, 2000). It is also commonly associated with several other diagnoses; panic disorder, obsessive-compulsive disorder, and social phobia (Becker, 2006; Stein & Mellman, 2005). Despite its common presentation as a symptom, sleep disturbance in anxious patients may also indicate the presence of a separate diagnosis of comorbid insomnia that may not improve with response to anxiety-specific treatment (Becker, 2006). Because of the considerable overlap between symptom and separate diagnosis, it is especially important to assess and manage disturbed sleep in anxious patients as a part of effective psychiatric treatment (Stein & Mellman, 2005). Less research has highlighted the importance of treating comorbid insomnia in the specific context of

anxiety disorders; however, studies in PTSD patients have found that sleep disturbance may maintain or exacerbate PTSD symptoms (Spoormaker & Montgomery, 2008). Sleep-focused interventions have been found to improve both sleep disturbances and PTSD symptoms (DeViva, Zayfert, Pigeon, & Mellman, 2005; Germain, Shear, Hall, & Buysse, 2007; Ulmer, Edinger, & Calhoun, 2011).

Given the potential consequences of leaving comorbid insomnia untreated in psychiatric patients, including prolonged distress and higher risk of suicidality, the potential to exacerbate and hinder treatment of the psychiatric condition, and increased risk of relapse, there is good reason for physicians treating patients for depression or anxiety to separately target the presence of insomnia. Research has shown that not only does treating comorbid insomnia improve sleep and reduce distress associated with chronic sleep disturbance but these sleep-specific benefits may translate into improvements in the psychiatric diagnosis (Rybarczyk et al., 2009; Watanabe et al., 2011).

Treatment for Insomnia. Pharmacotherapy remains the most common first-line treatment for insomnia (Edinger, Wohlgemuth, Radtke, Coffman, & Carney, 2007); however concerns about long term use of hypnotic medications and drug therapies have led to increased consideration of behavioral treatments. Despite support for the short-term use of hypnotic medication, there is a lack of data to support the long-term efficacy of these medications (National Institutes of Health, 2005). Additionally, adverse events have been associated with the use of these drugs, including tolerance, dependence, residual daytime sedation, cognitive impairment, rebound insomnia, increased risk for falls and car accidents, and increased mortality (Kripke, 2000; Kripke et al., 1998; Kupfer & Reynolds, 1997). The high prevalence of hypnotic use among insomnia patients may in part be due to the ubiquitous marketing of hypnotic

medications. Due to pervasive and convincing ads on television or online, many patients adopt beliefs that their sleep can only be improved with the help of a medication or that there must be a "quick fix" to address their difficulties with sleep (Kripke, 2000). Primary care physicians or psychiatrists who field patient requests for specific hypnotic medications may choose to prescribe these medications due to the research supporting their effectiveness for acute insomnia but may continue to prescribe them long-term at a patient's request in order to preserve their relationship with that patient (Kripke, 2000). Despite continued symptoms of insomnia, many patients will push to remain on hypnotics as a result of their addictive properties or due to the unpleasant withdrawal symptoms experienced during past attempts to come off the medications (Kripke, 2000). Long-term use of sleep medications may therefore reinforce patient beliefs about the permanence of their sleep problems or their inability to sleep without the help of medication.

Cognitive-Behavioral Therapy for Insomnia (CBT-I). An alternative treatment for insomnia works to address these beliefs and involves making changes to maladaptive behavior patterns in place of pharmacotherapy (Morin, 2004). CBT-I is a multiple-component behavioral treatment that combines stimulus control, sleep restriction, sleep hygiene, and cognitive restructuring, with an added relaxation component used less routinely (Smith et al., 2005). Because chronic insomnia is conceptualized to be a product of maladaptive behaviors that maintain sleep disturbance, three core components of CBT-I (stimulus control, sleep restriction, and sleep hygiene) work to address these behaviors by modifying poor sleep habits and regulating the sleep-wake schedule (Morin, 2004).

Individuals with insomnia commonly find that they no longer respond to sleep-related stimuli (e.g. the bedroom or bedtime) with drowsiness but instead associate these stimuli with

wakefulness. This concept has been termed "conditioned arousal." *Stimulus control*, which was first introduced by Bootzin (1972), works to repair the association between sleep stimuli and falling asleep by limiting the time patients spend awake in bed. It also aims to reestablish synchronized circadian rhythms (Morin, 2004). Five rules guide this portion of treatment: 1) Go to bed only when sleepy, 2) Use the bedroom only for sleep or sex, 3) Get out of bed when unable to sleep after 15 minutes and only return when sleepy again, 4) Arise at the same time every morning, and 5) Do not nap during the day (Morin, 2004). *Sleep restriction* is intended to overpower conditioned arousal by creating a temporary state of sleep deprivation (Morin, 2004). By limiting the time spent in bed, sleep restriction helps to shorten sleep onset latency and produce greater sleep continuity and improved sleep quality, ultimately improving the efficiency of one's sleep. Sleep restriction is conducted by first assessing how much time a patient spends actually sleeping during the night and then establishing a "sleep window" based on this estimation. This sleep window will determine the extent to which a patient's sleep time will be restricted during the course of treatment with CBT-I (Morin, 2004). For example, if a patient reports sleeping an average of 6 hours during the night, he or she will be asked to restrict their time in bed to 6 hours and will set prescribed bed and rise times to align with this. *Sleep hygiene* involves educating the patient about the ways in which external factors (e.g. light/noise in the bedroom, caffeine use too close to bedtime, consuming alcohol late in the evening) can contribute to disturbed sleep (Morin, 2004). Patients are taught to recognize factors that could compromise the quality of their sleep and take steps to minimize them.

An additional CBT-I component, *cognitive restructuring*, addresses the faulty beliefs and unrealistic expectations insomnia patients often have about their sleep (Morin, 2004).

Maladaptive behavior patterns that perpetuate insomnia are often bolstered by misconceptions

about the causes of insomnia (e.g. "My sleep is poor because I'm getting old"), misattributions and amplifications of the consequences of insomnia (e.g. "If I don't get a full night's sleep, I'll get sick"), and faulty beliefs about sleep-promoting practices (e.g. "If I catch up on sleep in the morning, it will help my insomnia"; Morin, 2004). In addition, many patients endorse unrealistic expectations about sleep (e.g. "I have to get 8 hours of sleep every night") or experience performance anxiety or learned helplessness in relation to sleep, which serve to make sleep more difficult to achieve (Morin, 2004). Cognitive therapy helps to change patients' underlying ideas that perpetuate their insomnia and gives them tools to combat maladaptive thinking patterns, such as: 1) Never try to sleep, 2) Do not catastrophize after a poor night's sleep, and 3) Keep realistic expectations (Morin, 2004). As an adjunct component, *relaxation* is sometimes included to reduce cognitive and autonomic arousal through progressive muscle relaxation, imagery training, or meditation (Morin, 2004).

Efficacy of CBT-I. Research supports the clinical efficacy of CBT-I, with numerous studies reporting an average symptom reduction of 50% to 60% on primary outcomes of sleep onset latency (SOL), which is the time between laying down to sleep and sleep onset, and wake time after sleep onset (WASO), which is the time spent awake after sleep onset (e.g. Espie, Inglis, Tessier, et al., 2001; Edinger, Wohlgemuth, Radke, Marsh, & Quillian, 2001; Morin, Colecchi, et al., 1999; Morin, Culbert, & Schwartz, 1994; Morin, Hauri, et al., 1999; Smith et al., 2002). Following treating with CBT-I, SOL and WASO are typically reduced to below the 30 minute criterion used in identifying sleep onset insomnia or sleep maintenance insomnia, respectively (Morin, 2004). Total sleep time (TST) has also been shown to increase by an average of 30 to 45 minutes after treatment with CBT-I. Sleep efficiency (SE), which is calculated by dividing the time spent asleep by the time spent in bed, has been shown to improve

to approximately 85% (Edinger, et al., 2001; Espie, Inglis, Tessier, et al., 2001; Morin, Colecchi, et al., 1999; Morin, Culbert, et al., 1994; Morin, Hauri, et al., 1999; Smith et al., 2002).

Treatment Format. CBT-I has been shown to be efficacious when delivered as a brief treatment, with the typical treatment format consisting of four to six weekly sessions lasting 50 to 75 minutes each (Morin, 2004). Edinger et al., 2007 sought to identify the optimal "dose" of CBT-I in a study comparing one, two, four, or eight sessions of CBT-I over an eight week treatment period with an eight week no treatment waiting period. Results of this study showed that optimal outcomes were seen in those patients who received four biweekly sessions of CBT-I (Edinger et al., 2007). CBT-I has typically been carried out through individual, in-person sessions with clinicians who specialize in sleep medicine. Recent studies; however, support that CBT-I is equally efficacious when delivered in an individual format, group format, or when therapy is conducted over the phone (Bastien, Morin, Ouellet, Blais, & Bouchard, 2004). Additionally, randomized, controlled trials, such as a study by Buysse et al. (2011), have shown that CBT-I is efficacious when delivered by nurses or other allied health professionals. In the study by Buysse et al. (2011), all treatment was delivered by a masters-level nurse practitioner.

CBT-I versus Pharmacotherapy. Multiple controlled studies have examined the efficacy of CBT-I in comparison with pharmacotherapy (e.g. Jacobs, Pace-Schott, Stickgold, & Otto, 2004; Morin, Colecchi, et al., 1999; Sivertsen et al., 2006). One study in young adult and middle aged patients with sleep onset insomnia compared CBT-I, pharmacotherapy with the hypnotic medication zolpidem (Ambien), or combination therapy with placebo (Jacobs et al., 2004). Results indicated that CBT-I, alone or in combination with pharmacotherapy, was more efficacious than pharmacotherapy or placebo. On most outcome measures (diary reports of SE and SOL), CBT-I alone proved to be equal or superior to combination therapy with CBT-I and

pharmacotherapy (Jacobs et al., 2004). Of all treatments tested, CBT-I yielded the greatest number of normal sleepers after treatment (measured by a SOL of 30 minutes or less and a SE of 85% or more). A second study comparing CBT-I, pharmacotherapy with temazepam (Restoril), combination therapy, and placebo in older adults extended these results by examining long term outcomes (Morin, Colecchi, et al., 1999). CBT-I was found to yield the most durable improvements in insomnia symptoms compared to the other treatment conditions or placebo, with no significant changes in treatment gains between post-treatment and 24-month follow-up. An additional finding to note from this study was that patients receiving CBT-I were more satisfied with this behavioral approach to treatment than those receiving only pharmacotherapy. Patients, significant others, and clinicians all rated CBT-I to be the more effective treatment (Morin, Colecchi, et al., 1999).

In a more recent study, Morin et al. (2009) sought to determine whether adding hypnotic medication to CBT-I would improve treatment outcomes in comparison to CBT-I, alone. Adults with persistent primary insomnia underwent either six weekly sessions of CBT-I or underwent the same CBT-I treatment with an added 10 mg dose of zolpidem (Ambien) taken at bedtime. Patients were also given six months of extended therapy to assess long term outcomes (Morin et al., 2009). Results showed that CBT-I, alone or in combination with zolpidem, produced significant improvements in SOL, WASO, and SE during the initial six week treatment phase. There were no significant differences between those receiving CBT-I and those receiving the combined treatment, suggesting that treatment outcomes were not enhanced by the addition of hypnotic medications (Morin et al., 2009). The best long term outcome was reportedly achieved by patients who were initially treated with combination therapy for the first weeks but then received maintenance CBT-I, alone, during the following six months (Morin et al., 2009).

Efficacy of CBT-I in Comorbid Insomnia. In addition to the base of studies that have documented the efficacy of CBT-I for primary insomnia, numerous studies have been conducted with comorbid insomnia. These studies provide evidence to support that CBT-I is efficacious not only for patients with a single diagnosis of insomnia but also for those who suffer from medical or psychiatric comorbidities. CBT-I has been shown to be an efficacious treatment for comorbid insomnia in patients with diverse medical conditions, including cancer (Epstein & Dirksen, 2007; Savard, Simard, Ivers, & Morin, 2005), coronary artery disease (Rybarczyk et al., 2005), fibromyalgia (Edinger, Wohlgenuth, Krystal, & Rice, 2005), pulmonary disease (Rybarczyk et al., 2005), and osteoarthritis (Rybarczyk et al., 2005; Vitiello, Rybarczyk, Von Korff, and Stepanski, 2009). Studies focused on comorbid insomnia in the context of psychiatric comorbidities have provided support for the efficacy of multicomponent CBT-I; however, fewer randomized controlled studies have been carried out. Several nonrandomized studies have found support for CBT-I comorbid with post-traumatic stress disorder (PTSD; DeViva et al., 2005; Krakow et al., 2001), mild depression (Taylor, Lichstein, Weinstock, Sanford, & Temple, 2007), and serious mental illness (Dopke, Lehner, & Wells, 2004). The handful of randomized studies with psychiatric comorbidities support that multicomponent CBT-I is efficacious in treating comorbid insomnia in alcoholism (Currie, Clark, Hodgins, & el-Guebaly, 2004), mixed medical and psychiatric comorbidities (Lichstein, Wilson, & Johnson, 2000), depression (Manber et al., 2008; Watanabe et al., 2011; Karlin, Trockel, Taylor, Gimeno, & Manber, 2013), and PTSD (Ulmer et al., 2011).

CBT-I Effectiveness Studies. A broad base of research exists to support the efficacy of CBT-I; however, these studies do not answer the question of whether this treatment is as effective as it is efficacious. Without examining a treatment's effectiveness, it is difficult to be

certain that study results are generalizable to heterogeneous populations, which is an important consideration due to the fact that insomnia is so frequently associated with comorbid medical and psychological disorders (Morin, Stone, McDonald, & Jones, 1994). A treatment may produce desired outcomes when study participants have pure primary insomnia without comorbidities or when they self-refer to a treatment study due to a high level of motivation or as a result of compensation in return for their time, but are the same outcomes observed when participants are not self-selected, reimbursed, or limited by strict exclusion criteria? A handful of studies have sought to answer this question, beginning with several case-series studies that tested CBT-I in patients who sought treatment at sleep clinics (Dashevsky & Kramer, 1998; Morin, Stone, et al., 1994; Perlis et al., 2000; Perlis, Sharpe, Smith, Greenblatt, & Giles, 2001).

Case-Series Studies. One early study by Morin, Stone, et al. (1994) tested CBT-I in 100 adults who presented for insomnia treatment at a university hospital sleep disorders clinic. In contrast to many of the efficacy studies mentioned above, participants were not solicited through advertisements but were informed of the study opportunity upon presenting for sleep treatment in a clinical setting (Morin, Stone, et al., 1994). Patients who were admitted into the study were not excluded due to psychiatric or medical comorbidities (aside from other sleep disorders or serious impairments due to psychosis or head injuries); however, those who were determined to have psychiatric comorbidities severe enough to be considered their primary diagnosis were not included in the data analysis (e.g. Morin, Stone, et al., 1994). Patients diagnosed with drug-dependent insomnia who were taking prescribed or over-the-counter medications for sleep were admitted into the study and offered a withdrawal plan to decrease or eliminate medication usage (Morin, Stone, et al., 1994). Results of this study showed that participants reported improvements in SOL, WASO, and early morning awakening, and that the magnitude of

improvement was comparable to what has been shown by efficacy studies that involve stricter exclusion criteria (Morin, Stone, et al., 1994). A significant reduction in the use of sleep aids was also noted, with hypnotic use decreasing by 54% by the end of the study. A primary limitation of this study is its lack of a control group, which precludes definite conclusions as to whether the outcomes observed were due to the specific treatment components of CBT-I (Morin, Stone, et al., 1994). This limitation is inherent in case-series research; however, it is important to note. An additional limitation is that only those with a primary diagnosis of insomnia were included in the analyses. The study results, therefore, do not represent individuals being treated for a primary diagnosis of depression or anxiety who also have comorbid insomnia.

A second case-series study (Perlis et al., 2000) did not explicitly state that patients with primary psychiatric diagnoses were excluded; however, no participants were reported to have comorbid insomnia and 73% were reported to have primary insomnia, which is not consistent with prevalence estimates reported in other studies (e.g. Buysse et al., 1994). In a replication study by some of the same authors (Perlis et al., 2001); however, the investigators chose to assess whether medical and/or psychiatric comorbidity might influence outcome. Patients were 89 adults who were either self- or physician-referred to a sleep disorders center for insomnia treatment (Perlis et al., 2001). Of these patients, 30% were diagnosed with primary insomnia, 30% with insomnia comorbid with depression, 35% with insomnia comorbid with circadian rhythm disturbances or other sleep disorders, and 5% with insomnia comorbid with psychiatric disorders other than depression. Of the 28 subjects who completed treatment, 50% reported primary psychiatric disorders, with depression and anxiety being the most commonly reported, and 53% reported primary medical disorders (Perlis et al., 2001). Results showed that after an average of seven sessions of CBT-I, significant improvements were observed in SOL, WASO,

and TST. Compared to pharmacotherapy treatment norms from the insomnia literature, CBT-I produced comparable results for three out of the four sleep parameters assessed (Perlis et al., 2001). Although this study also lacked a control group, CBT-I was found to be an effective treatment and medical and/or psychiatric comorbidity was not found to significantly influence treatment outcome (Perlis et al., 2001).

In another attempt to focus more explicitly on insomnia patients who have psychiatric comorbidities, Dashevsky and Kramer (1998) tested a CBT-I intervention in 48 psychiatrically ill adults who had previously failed to respond to pharmacotherapy for insomnia. Patients had been either self- or physician-referred to a sleep disorders center for behavioral therapy for chronic insomnia. A reported 79.2% of patients were taking hypnotic medications at the start of the study and over half of these discontinued or significantly decreased their dosage by 12 months after the pre-treatment assessment (Dashevsky & Kramer, 1998). Results indicated a statistically significant change in all self-reported sleep parameters after eight weeks of CBT-I and benefits were maintained at 12-month follow-up. One interesting additional finding from this study was that there was a reported tendency for patients who were not initially taking hypnotics to drop out of the study. This suggests that there may be some benefit to beginning behavioral therapy while patients are still taking hypnotics rather than titrating off the medications before initiating treatment, as has been the procedure in other studies (e.g. Perlis et al., 2000).

A recent study by Karlin et al. (2013) assessed the effectiveness of a six-session CBT-I intervention delivered by therapists in training in 102 veterans presenting for treatment in various mental health or primary care clinics within the Veteran's Health Administration (VHA). Veterans with current depression were admitted into the study, with mean scores on the Beck

Depression Inventory-II (BDI-II) at baseline indicating a moderate level of baseline depression; however, participants were excluded due to diagnosis of bipolar disorder, severe daytime sleepiness, or "uncontrolled medical or mental health conditions," a term that was not further explained. Results showed significant improvement in Insomnia Severity Index (ISI) Score, depression, and quality of life (Karlin et al., 2013).

Effectiveness Studies in the Primary Care Setting. Additional effectiveness studies have been conducted outside of specialty sleep clinics and have tested CBT-I in the primary care setting delivered by those who do not have specialized training in sleep medicine (Espie et al., 2007; Espie, Inglis, & Harvey, 2001; Espie, Inglis, Tessier, et al., 2001). Unlike the case-series studies, the inclusion of a control group in these studies produces more compelling evidence in support of CBT-I due to improved methodological rigor. In one example (Espie, Inglis, Tessier et al., 2001), 139 adults with insomnia were randomized to either six group sessions of multi-component CBT-I or a six week self-monitoring control condition. CBT-I was conducted by primary care nurses who had no specialization in sleep medicine but underwent specific training before delivering the treatment (Espie, Inglis, Tessier, et al., 2001). Results showed that CBT-I significantly reduced SOL and WASO compared to the control condition, with improvements observed to persist at one-year follow-up. An additional finding was that 84% of those patients who began the study while taking hypnotic medications were drug-free at one-year follow-up (Espie, Inglis, Tessier, et al., 2001). A limitation of this study was that individuals with major depression and a subsample of participants with "other diagnoses" were excluded from participating. This limits the generalizability of the results to primary care populations, considering that significant depressive symptoms are endorsed by an estimated 20% of patients seeing primary care physicians (Stafford, Ausiello, Misra, & Saglam, 2000).

A similar study also excluded those with primary psychiatric diagnoses (Espie, Inglis, & Harvey, 2001); however, a more recent study (Espie et al., 2007) attempted to make fewer exclusions. In this study, patients with physical or psychiatric problems were reportedly included in the study; however, the fact that those exhibiting "new," "untreated," or "serious" disorders were excluded from the study is, again, limiting. In a description of those participants who were excluded from the study, which appears later in their paper, Espie et al., (2007) indicate that multiple patients were excluded due to "pain," "depression," and "anxiety." Without any additional information provided about these excluded participants, it is unclear why they were not included in this effectiveness study when these three complaints are some of the most widely prevalent among primary care populations (Means-Christensen, Roy-Byrne, Sherbourne, Craske, & Stein, 2008).

The lack of true CBT-I effectiveness studies in the existing literature highlights a need for further research that does not exclude participants who have more severe psychiatric comorbidities or who carry primary diagnoses such as depression or anxiety. More research is also needed to understand the effectiveness of CBT-I among chronic hypnotic users, who make up a significant portion of individuals living with chronic insomnia (Ohayon & Caulet, 1996). Several CBT-I effectiveness studies, mentioned above, provide preliminary evidence for the use of CBT-I in reducing chronic hypnotic use (e.g. Espie, Inglis, Tessier, et al., 2001); however, additional studies that tested CBT-I specifically for this purpose provide additional support.

CBT-I and Hypnotic Medication Reduction. In one early study, Morin et al. (1995) tested CBT-I along with a supervised medication tapering schedule in five hypnotic-dependent insomnia patients and found that four out of the five patients had discontinued their medication within six to eight weeks. The fifth patient had decreased drug intake by 90% since the start of

treatment. Whereas SE initially deteriorated as the patients withdrew from their hypnotics, at three-month follow-up, this trend had reversed (Morin et al., 1995). A later study randomized patients to either a 10-week supervised medication withdrawal program, CBT-I, or supervised withdrawal plus CBT-I (Morin et al., 2004). Results showed that more patients who received both the medication taper program and CBT-I were medication-free at post-treatment compared to those who had one or the other. No significant withdrawal symptoms or adverse events were reported with the medication tapering and those patients who received CBT-I, either alone or with the taper program, reported greater subjective sleep improvements than those who received medication taper, alone (Morin, 2004). Reductions in medication use were sustained at 12-month follow-up and sleep improvements became more noticeable during this period (Morin et al., 2004). A third study showed that CBT-I was effective in reducing hypnotic use when delivered in a routine primary care setting by non-sleep specialists (Morgan et al., 2003). Two hundred and nine patients were randomized to either CBT-I or treatment as usual and results showed that those receiving CBT-I reported significantly improved sleep efficiency and significantly reduced SOL and hypnotic use compared to those who did not receive behavioral treatment (Morgan et al., 2003).

CBT-I and Improvements in Quality of Life. An additional finding from the study by Morgan et al., (2003) was that CBT-I led to improvements in vitality on the SF-36, a measure of health-related quality of life. A second study (Dixon et al., 2006) compared baseline SF-36 profiles of adult hypnotic users with insomnia with the profiles of age-matched individuals from a primary care reference group in order to assess the effect of CBT-I on scores of health-related quality of life. Results showed that despite poorer health-related quality of life in the CBT-I group prior to treatment, SF-36 scores were significantly higher in this group at six-month

follow-up for the domains of physical functioning, emotional role limitation, and mental health (Dixon et al., 2006). A third study looked at quality of life as measured by the World Health Organization Quality of Life - BREF (WHOQL-BREF) and found that scores in physical, psychological, social, and environmental domains were all improved following six sessions of CBT-I (Karlin et al., 2013). These findings indicate that treatment with CBT-I may not only improve sleep or reduce hypnotic use but may also have a positive effect on daily functioning for individuals with chronic insomnia.

Statement of the Problem

Previous research supports the efficacy of CBT-I for comorbid insomnia in patients with psychiatric diagnoses (Lichstein et al., 2000; Manber et al., 2008; Ulmer et al., 2011; Watanabe et al., 2011) and a handful of studies have begun to examine the effectiveness of this treatment (Dashevsky & Kramer, 2008; Perlis et al., 2000; Espie, Inglis, Tessier, et al., 2001; Espie et al., 2007). Despite preliminary evidence provided by these previous research studies, questions still remain about the effectiveness of CBT-I due to the fact that most studies have excluded patients with significant psychiatric symptoms. Studies have tested CBT-I in sleep clinics (e.g. Dashevsky & Kramer, 2008; Perlis et al., 2000) and primary care clinics (e.g. Espie, Inglis, Tessier, et al., 2001; Espie et al., 2007) but only one study to date has been carried out in an outpatient psychiatry setting (Watanabe et al., 2011). This study, by Watanabe et al., (2011), tested a brief CBT-I intervention in individuals with depression and comorbid insomnia recruited from outpatient psychiatric clinics in Japan and found that adding a brief CBT-I intervention to usual care led to significant improvements in sleep and depressive symptoms compared to a treatment as usual group. This study made a significant contribution to the comorbid insomnia literature; however, the study findings cannot be generalized to heterogeneous psychiatric

populations due to the fact that patients with psychiatric comorbidities other than depression were excluded and the investigators specifically targeted patients with mild, moderate, or partially remitted depression. Also, Watanabe et al. (2011) allowed use of hypnotics or other medications during the study but did not specifically target hypnotic reduction as a treatment aim. A small base of research supports the use of CBT-I in reducing hypnotic medication use for chronic insomnia (e.g. Morgan et al., 2003); however, the use of CBT-I for this purpose has not yet been specifically tested in a psychiatric population.

The purpose of this study was to build upon existing research by testing the effectiveness of a five session CBT treatment for persistent insomnia in chronic hypnotic users receiving treatment for mental health conditions in a psychiatric clinic located in an urban academic medical center. Participants with significant symptoms of depression or anxiety were not excluded from participation and all patients were chronic hypnotic users who continued to experience significant symptoms of insomnia despite receiving pharmacotherapy. The effectiveness of CBT-I is important to examine in a psychiatric population considering the pervasiveness of this sleep disorder among patients with mental health diagnoses and the consequences of leaving it untreated. By testing the effectiveness of CBT-I in this particular setting, we can determine whether brief behavioral treatment is a potential solution to the problems of long term hypnotic use, which is associated with numerous concerns in regard to safety (Kripke, 2000) and residual insomnia symptoms, which exacerbate psychiatric problems (Watanabe et al., 2011) and reduce quality of life (Smith et al., 2005). In this study, individuals were randomized to receive either CBT-I or treatment as usual (TAU) and were assessed at baseline, post-treatment, and at two-month follow-up.

Statement of Hypotheses

Based on the literature and the aims of the proposed study, the following hypotheses were tested:

1. Participants in the CBT-I group will report significantly improved sleep compared to the TAU group at post-treatment. Specifically, the CBT-I group will show improvement in a global measure of insomnia severity, significantly higher sleep efficiency (SE), shorter sleep onset latency (SOL), longer total sleep time (TST), and shorter wake time after sleep onset (WASO).

2. Participants in the CBT-I group will report significantly reduced hypnotic use compared to the TAU group at post-treatment.

3. Improvements in sleep resulting from the intervention will lead to secondary improvements in psychiatric symptoms and quality of life compared to TAU controls at post-treatment; specifically, reduced symptoms of depression and anxiety and improved health-related quality of life.

4. Improvements in sleep and psychiatric symptoms and reductions in hypnotic use will be maintained at two-month follow-up for participants in the CBT-I group.

Method

Study Design

This study explored the effectiveness of a CBT for insomnia treatment in individuals from an outpatient psychiatry clinic who were experiencing continued symptoms of insomnia despite use of hypnotic medication. Participants were randomized to either a CBT-I treatment or TAU group. Those in the CBT-I condition participated in five sessions of CBT-I over a period of approximately five weeks. These sessions included individual meetings as well as brief

check-in sessions via phone. As a pre-treatment assessment, all participants completed a one-week sleep diary and several self-report questionnaires (see Measures section for further detail) within 1-2 weeks prior to the start of treatment. Post-treatment assessment using the same sleep diary and questionnaires took place following the treatment period. Participants who completed the CBT-I treatment were also asked to participate in a two-month follow-up assessment. Participants in the TAU group were contacted biweekly by phone until the post-treatment assessment was completed. This phone check-in was included for the purpose of maintaining participant involvement in the study. During these calls, the doctoral student reassured participants of their inclusion in the study and reminded them to fill out their sleep diary. No advice regarding sleep was given. After one week of baseline data collection using sleep diaries, five weeks of receiving treatment as usual, and one more week of post-treatment sleep diary data collection, participants in the TAU group were given the opportunity to participate in an equivalent CBT-I treatment. By including participants who elected to undergo CBT-I after initial randomization to the TAU group as "crossovers" in the study, their data during the CBT-I treatment phase has been included in some analyses, thereby increasing sample size for the CBT-I condition. Due to time and staff limitations, crossovers were not asked to complete two-month follow-up data.

Participants

Study participants were 23 individuals receiving medication management at the Virginia Commonwealth University Outpatient Psychiatry clinic. This clinic is located in an urban academic medical center and serves in large part as a safety net clinic, treating a significant number of underserved groups. In most cases, treatment is provided by psychiatry residents under the supervision of an attending psychiatrist. Of the 23 individuals enrolled in the study, 12

were randomized to CBT-I and 10 were randomized to TAU. An additional participant who was initially found ineligible for the study due to no evidence of insomnia after being randomized to TAU and completing baseline assessments was later re-enrolled in the study and re-randomized to the CBT-I group, bringing the total number of participants randomized to CBT-I to 13. All participants were recruited between May 2012 and March 2013 and were identified as appropriate candidates either by chart review prior to their regularly scheduled psychiatry appointment or by their treating psychiatrist. Appropriate study referrals were patients who reported continued symptoms of insomnia despite use of sleep medication and who were receiving treatment at VCU Outpatient Psychiatry for comorbid psychiatric diagnoses (e.g. depression, anxiety). To confirm each patient's eligibility for the study, the doctoral student conducted a brief interview through which she reviewed each inclusion criterion with the potential participant. Inclusion criteria consisted of: a) current symptoms of chronic insomnia, defined as (1) at least three episodes of insomnia per week for at least six months (with an episode consisting of a 30-minute SOL, WASO of at least 60 minutes, or TST of less than 6.5 hours per night) and (2) daytime consequences of insomnia, such as fatigue, irritability, or difficulty concentrating. This definition of insomnia is based on research criteria used commonly in other studies (Edinger et al., 2004; Rybarczyk et al., 2005); b) current use of hypnotic medication prescribed for sleep by the patient's psychiatrist; c) continued symptoms of insomnia despite treatment with hypnotic medication for a period of at least 2 weeks, and d) comorbid psychiatric diagnosis/diagnoses other than insomnia for which the patient is currently receiving medication management at VCU Outpatient Psychiatry. To facilitate enrollment, "hypnotic medication" under inclusion criterion b was interpreted as any medication prescribed as least in part for the purpose of improving sleep (for example, the antidepressant medication

trazodone that is commonly used to treat sleep disturbance in addition to depressed mood). This definition was approved by the study's medically responsible investigator and no participants were approached about the study unless their treating psychiatrist identified the medication they were taking for sleep as safe to be tapered.

Before accepting a referral to the study, the doctoral student requested that each patient's treating psychiatrist confirm that he or she was on an appropriate medication to meet inclusion criterion b and noted which medication would be targeted for taper should the patient be interested. Exclusion criteria consisted of: a) meeting criteria for current alcohol or substance abuse or dependence, b) unstable or untreated bipolar disorder, c) current mania or psychosis, d) diagnosis of a seizure disorder, e) diagnosis of severe depression without stable medication treatment for a minimum of two months; f) diagnoses of untreated restless leg syndrome, sleep apnea, or other sleep disorders as indicated by patient report and medical record review; g) medical conditions that are highly likely to cause sleep disturbances such as Parkinson's disease (Rybarczyk et al., 2005); and h) prescription for primary sleep medication from a provider outside of VCU Outpatient Psychiatry.

See Table 1 for the demographic and other characteristics of participants in the study. T-tests and chi-square analyses were used to test the success of randomization by detecting any baseline differences between groups. Because these analyses are sensitive to violations of normality, the normality of the data was assessed prior to data analysis. Due to high skewness and kurtosis (values greater than or less than 1 and -1, respectively) outlier values were adjusted according to the method described in the results section (see page 52). No differences were found between the CBT-I group and the TAU group with regard to age, gender, education, race, duration of insomnia, number of medical conditions, number of psychiatric conditions, number

of all medications, or number of psychiatric medications (all $ps > .05$). When the CBT-I group was compared with "crossovers" (those participants who were initially randomized to TAU but chose to cross over to the CBT-I group after completion of the treatment as usual period; $n = 4$), again no differences between these variables were found between groups.

Table 1.
Demographics and Other Baseline Characteristics for Participants by Treatment Group, (N=23)

Characteristics	CBT-I (n=11)	TAU (n = 8)	Crossovers (n = 4)
Gender	9 Females (82%) 2 Males (18%)	8 Females (100%) 2 Males (0%)	4 Females (100%) 0 Males (0%)
Age (Mean, SD)	50.1 (13.1) Range = 26 - 67	50.9 (8.5) Range = 37 - 61	49.75 (8.9) Range = 37 - 56
Race	6 Caucasian (54%) 4 African-American (36%) 1 Biracial (1%)	5 Caucasian (62%) 2 African-American (25%) 1 Unknown/Other (13%)	2 Caucasian (50%) 1 African-American (25%) 1 Unknown/Other (25%)
Education	1 Some Middle School 1 Middle School 3 Some High School 1 High School 3 Some College 2 Graduate School	1 Some High School 4 Some College 3 College	2 Some High School 2 College
Mean Number of Medical Conditions (SD)	5.2 (2.3)	3.4 (1.7)	4.25 (1.7)
Mean Number of Psychiatric Conditions (SD)	2.1 (.7)	2.1 (.8)	2.2 (1.3)
Mean Number of Current Medications (SD)	10.8 (4.3)	7.9 (3.6)	10 (3.5)
Mean Number of Current Psychiatric Medications (SD)	2.9 (1.1)	3.5 (1.2)	3.5 (.6)
Current Sleep Medications*	5 Trazodone 2 Ambien 2 Temazepam 2 Clonazepam	2 Trazodone 2 Ambien 2 Seroquel 2 Perphenazine	2 Seroquel 2 Perphenazine
Mean Frequency of Sleep Medication Use (Nights per week)	5.6 (2.4)	6.7 (1.1)	5.7 (2.5)
Mean Duration of Insomnia (Months; SD)	192.3 (159.7)	222.8 (211.0)	280.2 (255.7)

*All medications were prescribed for their effects on sleep; however, some (e.g. perphenazine) were prescribed for more than one purpose. For all participants enrolled in the study, the treating psychiatrist targeted the prescribed medication for tapering.

Baseline sleep and health/mental variables were also compared between groups. Results of t-tests showed no significant differences between those randomized to the CBT-I group and those randomized to TAU (all $ps > .05$) in regard to total score on the Insomnia Severity Index (ISI), sleep onset latency, total sleep time, wake time after sleep onset, sleep efficiency, depression score on the PHQ-9, anxiety score on the GAD-7, or physical and mental health components and eight domain scales on the SF-36, a measure of health-related quality of life. Additional t-tests were conducted between the CBT-I group and the group of crossovers ($n=4$). Again, no significant differences were found between these baseline variables (all $ps > .05$). See Table 2.

Table 2.
Sleep Variable and Mental Health Variable Comparisons between Groups at Baseline (N=23; Outliers Adjusted)

	CBT (n = 11)	TAU (n = 8)	Combined (n = 19)	Crossover (n = 4)
Insomnia Severity Index (ISI) Score	21.7 (3.8)	22.9 (3.2)	22.2 (3.5)	21.2 (3.4)
Sleep Onset Latency in hours (SOL)	1.1 (1.1)	0.9 (1.0)	1.0 (1.0)	1.2 (1.1)
Total Sleep Time in hours (TST)	5.7 (2.2)	6.3 (1.4)	6.0 (1.9)	5.1 (2.1)
Wake Time After Sleep Onset in hours (WASO)	0.9 (0.5)	1.0 (0.4)	1.0 (0.5)	0.6 (0.3)
Sleep Efficiency % (SE)	58.0 (22.8)	73.4 (10.3)	64.5 (19.8)	70.3 (19.8)
Mean Days per Week of Sleep Medication Use	5.6 (2.4)	6.6 (1.1)	6.1 (2.0)	5.8 (2.5)
PHQ-9 Score	16.2 (7.9)	18.5 (4.8)	17.2 (6.7)	15.0 (4.5)
GAD-7 Score	13.4 (6.4)	16.1 (4.8)	14.5 (5.8)	13.5 (5.1)
SF-36 Physical Component Score	37.7 (10.1)	42.7 (13.2)	39.8 (11.4)	41.5 (5.4)
SF-36 Mental Component Score	29.3 (10.6)	26.9 (6.7)	28.3 (9.0)	23.5 (5.2)

Study participants had a range of psychiatric diagnoses, with 84% having two or more diagnoses and 21% having three or more diagnoses, as documented by their treating psychiatrist. See Table 3 for the list of psychiatric diagnoses across groups.

Table 3.

Participant Psychiatric Diagnoses By Group (Number With Each Diagnosis and Percentage; N=23)

CBT (n = 11)	TAU (n = 8)	Crossover (n = 4)
5 Major Depressive Disorder (45%)	3 Major Depressive Disorder (37%)	2 Major Depressive Disorder (50%)
4 Anxiety Disorder NOS (36%)	3 Panic Disorder (37%)	2 Mood Disorder NOS (50%)
4 History of Drug/Alcohol Dependence (36%)	2 Mood Disorder NOS (25%)	2 PTSD (50%)
2 Mood Disorder NOS (18%)	2 Anxiety Disorder NOS (25%)	1 Panic Disorder (25%)
2 Panic Disorder (18%)	2 PTSD (25%)	
1 Depressive Disorder NOS (9%)	1 Depressive Disorder NOS (12%)	
1 Bipolar II Disorder (9%)	1 Bipolar I Disorder (12%)	
1 PTSD (9%)	1 Social Anxiety Disorder (12%)	
1 Generalized Anxiety Disorder (9%)	1 Dysthymia (12%)	
1 Dysthymia (9%)	1 Attention Deficit Disorder (12%)	
1 Borderline Personality Disorder (9%)		

Procedure

Recruitment. Prior to the first psychiatry appointment of each clinic day, the doctoral student was given access to the medical records of all patients scheduled to come in to the clinic. The doctoral student identified and flagged any patients who were taking hypnotic medication for sleep and discussed these cases with the patient's treating psychiatrist during the morning rounds. This meeting was held immediately prior to each clinic block and typically included an attending psychiatrist, two psychiatry residents, two or three third year medical students, and occasionally a nurse specialist. By participating as a team member in this meeting, the doctoral student was able to learn which patients coming in for the clinic block were eligible for the study and likely to benefit from behavioral treatment of insomnia. To allow for collaboration between the doctoral student and other clinicians working to treat a patient's insomnia symptoms, those being treated with sleep medication prescribed by an outside provider were excluded from the study.

In some cases, patients were not identified during this meeting but were identified by psychiatry attendings or residents after they learned of the study through a one-time meeting with the doctoral student about the study, through a flyer put in their mailbox, or through word of mouth from other providers in the clinic. Any patient identified as an eligible candidate for the study was informed of the study by their treating psychiatrist. This provider introduced the CBT-I treatment as an alternative or adjunct treatment to chronic use of hypnotic medication. Typically, a paper handout was given to patients as a reminder of the referral opportunity and to outline inclusion/exclusion criteria. If a patient expressed interest in the study or was willing to hear more about the opportunity, the doctoral student subsequently met with him or her to formally assess eligibility and to provide a standardized verbal introduction to the treatment. On a few occasions, this conversation occurred by phone. In this brief meeting, the student also listened to patient questions or objections to behavioral treatment and provided standardized responses to patient concerns. If a patient was eligible and expressed interest in the study, the doctoral student asked him or her to sign a consent form and administered baseline assessments. If this discussion did not take place in person, a time was scheduled for the patient to meet with the doctoral student to complete enrollment and baseline assessments.

The study protocol was approved by the Virginia Commonwealth University Institutional Review Board (IRB); HM #14329, and all participants were asked to give written informed consent at the time of enrollment in the study. The doctoral student reviewed the consent forms with eligible individuals to ensure that they understood the study procedure and their rights as participants. Once enrolled, each patient was given a one-week sleep diary to complete at home along with instructions for how to complete it.

Randomization. During the enrollment process, the doctoral student randomized each participant to either begin CBT-I treatment in approximately one week as a member of the treatment group or to begin CBT-I treatment in approximately seven weeks as a member of the TAU group (after one week of baseline assessment, five weeks of treatment as usual, and one week of post-treatment assessment). This randomization process included the use of a random number generator. Prior to the beginning of the study, the doctoral student listed the treatment and control assignments in a systematic order (e.g. T, C, T, C...) so that there were 30 total assignments. Then, a random permutation of integers from 1 to 30 was created using the randomization tool on www.randomization.com. This list was used to attach a random number to assignment, in order. For example, the first three numbers of the random permutation were 7, 19, and 23. These numbers were recorded by the 1st, 2nd, and 3rd assignment in the list so that the 7th patient to be randomized was in the treatment condition (T), the 19th was in the control condition (C), and the 23rd was in the treatment condition (T). Once the list was complete, the doctoral student referred to this for each interested participant, and, beginning with number 1, determined the group to which the patient was randomized by what assignment (T or C) was beside the number for that patient. The first patient to be randomized corresponded to the number 1, the second was 2, and so on.

All participants in both the CBT-I and TAU groups completed a one-week sleep diary in addition to self-report questionnaires prior to the start of treatment or the TAU period. A check-in phone call three to five days into baseline sleep diary data collection was made by the doctoral student to confirm that patients were correctly completing their sleep diaries and to answer any questions about this task. All participants also completed post-treatment measures and a one-week sleep diary at the completion of the treatment/TAU period. For TAU group participants,

post-treatment assessment took place following baseline assessments and randomization, one week of sleep diary assessment, and five weeks of treatment as usual.

Attrition. Forty-eight (48) patients were approached by their psychiatrist or psychologist about the study and of these individuals forty-two (42) expressed interest in learning more about the study and subsequently met briefly with the doctoral student or spoke with her via phone. See Figure 1 for the study flow chart, which diagrams the details of enrollment and drop-out. Twenty-six patients approached about the study declined to participate. Reasons for this are outlined in Table 4.

Table 4.
Reasons for Decision not to Participate in Study (n=26)

Reason	Number of People (n=26)
Lost to follow-up, no response to calls	9
Too difficult to make study visits	5
Too many other life stressors to commit to study	4
Little reported stress about sleep	4
Concerned treatment may not work	1
Not sure about participating, "not ready yet"	1
Not enough compensation for study participation	1
Not interested at this time, no other specific reason given	1

Ten participants were randomized to the TAU group and nine completed baseline assessments including a one-week sleep diary. One participant failed to return the baseline sleep diary and did not respond to calls about the study. Another participant whose sleep diary showed no symptoms of insomnia was excluded from the study but re-enrolled five months later and was

re-randomized to the CBT-I group after her insomnia symptoms returned. The eight remaining participants in the TAU group were called bi-weekly in an effort to keep the participants involved in the study through the point of post-treatment assessment. Seven participants completed the five-week treatment as usual period, while one failed to return post-treatment assessments or respond to calls about the study. This participant's data was included in analyses as an intent-to-treat dropout, with her baseline values included as both pre- and post-treatment data. Participants who completed the TAU period and assessments were given the option to cross over to the CBT-I group once they completed the TAU period and five chose to do so.

The individuals in the CBT-I condition participated in five CBT-I sessions that were typically held over a five-week period. Because some patients needed to reschedule study sessions or failed to show up for scheduled appointments, patients were permitted to complete the study sessions across a longer period of time, if needed. Thirteen participants were randomized to the CBT-I group, two of whom dropped out before attending the first treatment session or returning the pre-treatment sleep diary. One additional participant dropped out mid-treatment without submitting any completed sleep diaries beyond the baseline diary. This participant's data was included in analyses as an intent-to-treat dropout, with her pre-treatment values included as both baseline and post-treatment data. A total of 10 participants completed the treatment in 6-10 weeks and seven of these participants returned all sleep diaries and post-treatment assessments. Three participants completed the treatment but failed to return the final post-treatment diary. For these participants, their most recent sleep diary (e.g. diary completed during week 4 of treatment) was used in the analyses in place of post-treatment data. All participants who completed the CBT-I treatment, including crossovers, were asked to participate in a two-month follow-up assessment that involved completing study measures and a one-week

sleep diary. These were mailed to the participants along with a self-addressed envelope in which to return the completed questionnaires. Patients received a reminder phone call following the mailing of the two-month follow-up assessments to encourage return of this data.

Compensation. At the time of enrollment, participants were informed verbally and through the study consent form that there would be some small compensation provided for their participation in the study. Any participant in the CBT-I group who completed all five treatment sessions along with pre-treatment and post-treatment assessments and sleep diaries received a \$5.00 gift card to Target. If they completed follow-up assessments, including a one-week sleep diary, they were mailed a second \$5.00 Target gift card. Participants in the TAU group were given a \$5.00 gift card to Target after completing pre-treatment assessments and sleep diary, the five-week treatment as usual period, and post-treatment assessments and diary. Those who chose to cross over to complete the CBT-I treatment were given the opportunity to receive two additional gift cards, as explained above, one for completion of the treatment and post-treatment assessments and one for completion of follow-up assessments.

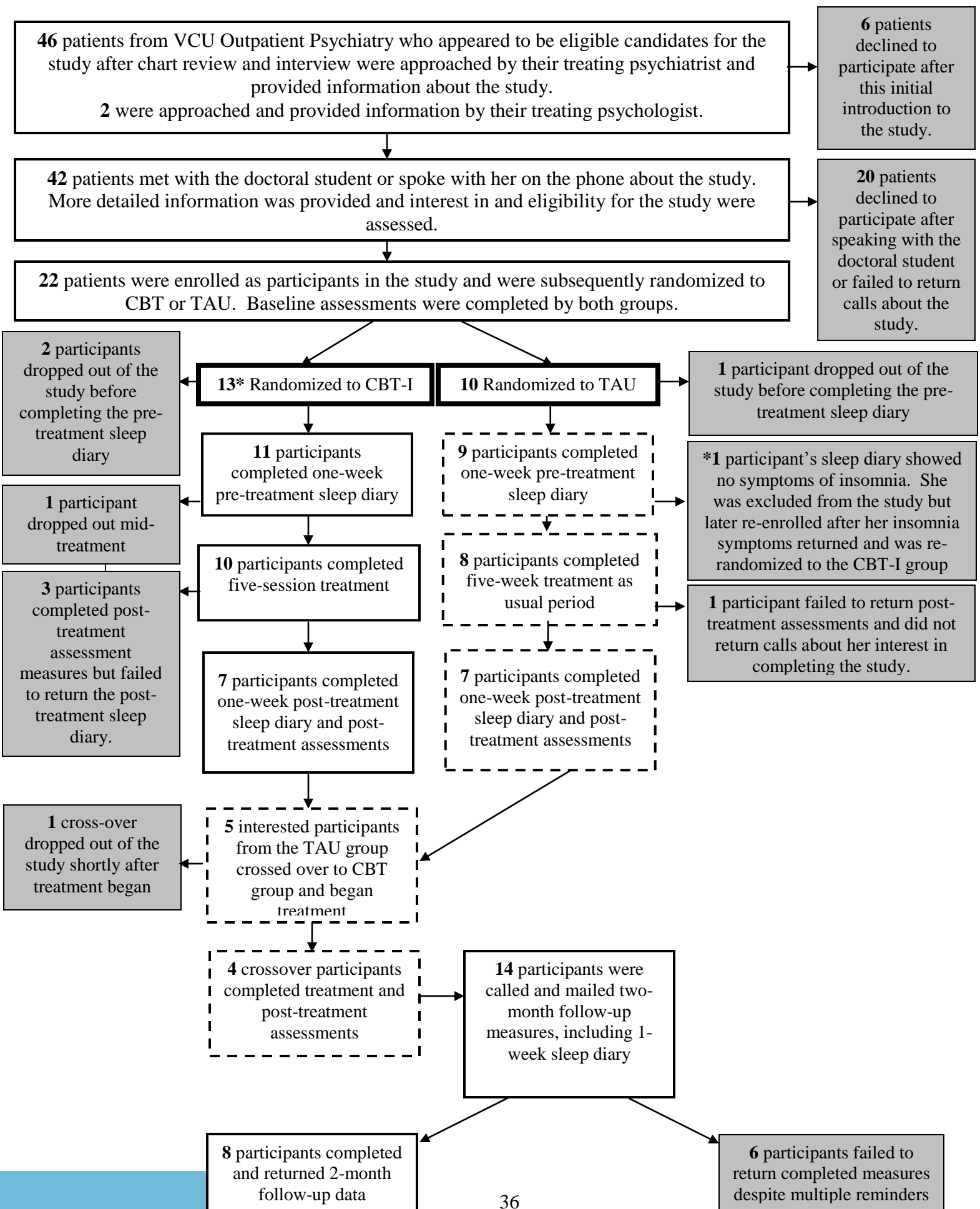


Figure 1. Study Flow Chart for CBT and TAU Groups

Reasons for dropout after beginning CBT-I were collected from each of the four participants who failed to complete the treatment. See Table 5, below.

Table 5.
Reasons for Dropout (n=4)

Reason for Dropping out of Study after Beginning CBT-I	Number of People (n=4)
Too difficult to follow treatment guidelines	1
Sleep has gotten better on its own	1
Too many other stressors & medical issues	1
Not interested in completing study; no further reason specified	1

Intervention

The intervention program consisted of five treatment sessions intended to occur one week apart. The content of these sessions was largely based on the insomnia treatment programs by Perlis, Jungquist, Smith, & Posner (2005) and Morin (1993); however, the brief model of delivery that included both in-person and telephone sessions was most similar to the brief behavioral treatment for insomnia (BBTI) described by Troxel, Germain, and Buysse (2012). All sessions involved a standardized powerpoint guide that provided visuals and bullet points as a supplement to the information provided verbally and through paper handouts by the doctoral student. Session 1 was a 60-minute individual meeting between the participant and the doctoral student, Hannah Lund Taylor, MS. This first session involved an introduction to behavioral treatment as an alternative or adjunctive treatment to hypnotic medications and provided basic information about sleep and insomnia, including prevalence rates and the relationship between insomnia and mental health diagnoses. The two-process model of sleep was also introduced,

including a discussion of how homeostatic and circadian systems work together to determine patterns of sleep and wake. Basic education on Spielman's 3-Ps Model was then provided, which outlines the factors that contribute to the development and maintenance of disturbed sleep. In the latter half of this first session, behavioral treatment components were introduced and a rationale for pursuing behavioral treatment versus medication treatment was verbally provided.

The doctoral student offered to participants an optional hypnotic reduction module in this first session that involved initiating a medication taper schedule with the approval of each patient's treating psychiatrist. Prior to this session, each participant's treating psychiatrist indicated his or her approval for the patient to gradually reduce hypnotic medication in the case that the patient elected to complete this optional module. The completion of this module was not a requirement but was presented as an option for a second time at the beginning of session 3 if they did not complete it in session 1. Beginning a taper schedule prior to behavioral treatment has been shown to produce better sleep outcomes than initiating the taper after behavioral treatment (Morin, 1993). Additionally, initiating the taper at the same time that CBT-I treatment components are introduced has been suggested to increase patient willingness to reduce hypnotic use (Morin, 1993). Despite the evidence to support these points, it was anticipated that many patients would still be unwilling to taper off their sleep medications. Rather than excluding these individuals from participating in the study, they were welcomed to participate. Before making their decision to receive or decline the medication reduction module, all participants were provided with brief education in session 1 about the potential adverse effects of long term hypnotic use and the potential benefits of treating insomnia with alternative methods (i.e. behavioral techniques).

If patients had elected to complete the hypnotic reduction module, the plan was to have created a specific tapering schedule for each individual using guidelines appearing in Morin's (1993) insomnia treatment manual, along with individualized recommendations provided by the patient's treating psychiatrist. It was anticipated that the first step of the tapering schedule would be to stabilize the patient on the lowest available dose of the hypnotic medication currently in use. Once the patient was stabilized on the low dose, a second step would involve introducing drug-free nights so that medication was only allowed on pre-determined nights (Morin, 1993). In order to reduce the association between sleeplessness and drug-taking behavior, this scheduling was designed to be coupled with a rule of taking any hypnotic medication a specific amount of time before bed rather than when the patient has difficulty sleeping (Morin, 1993). By increasing the number of drug-free nights each week, each patient would gradually taper off of his or her hypnotic medications.

Of all participants randomized to CBT-I, all of whom were offered the medication reduction module along with information about the consequences of long term use of sleep medications and the evidence for behavioral treatment, none were interested in coming off their medication in session 1. Several reported that they may be interested in considering this at a later time but were not willing to do so at the time as part of the study.

The first session of the intervention also included a review of the participant's completed sleep diary and an introduction to the concepts of sleep restriction and stimulus control. The doctoral student calculated the patient's average total sleep time using the data from the sleep diary and asked the patient to limit his or her time in bed, or "sleep window," to this amount of time (with a lower limit of 4.5 hours of sleep per night). Participants in the CBT-I treatment arm of the study continued to track their sleep using the sleep diary for the duration of the study.

Following this first session were four individual sessions that were held either in-person with the doctoral student or via a follow-up phone call. Sessions 3 and 5 were limited to 45 minutes and included a review of the participant's sleep diary, a discussion of whether any changes to the participant's treatment plan were warranted, a discussion aimed at addressing problems the participant was experiencing in treatment implementation, and an educational component (Rybarczyk et al., 2005). Follow-up phone calls (sessions 2 and 4) were limited to 20 minutes and consisted of reviewing the components introduced in previous sessions, discussing the patient's successes or struggles with the treatment, and addressing these problems through problem-solving and reassurance. Session 2 was typically scheduled for one week after the first session but was permitted to be postponed by a week or two, if necessary. This second session was a follow-up phone call made by the doctoral student and involved providing support to participants as they underwent sleep restriction and answering any patient questions or concerns.

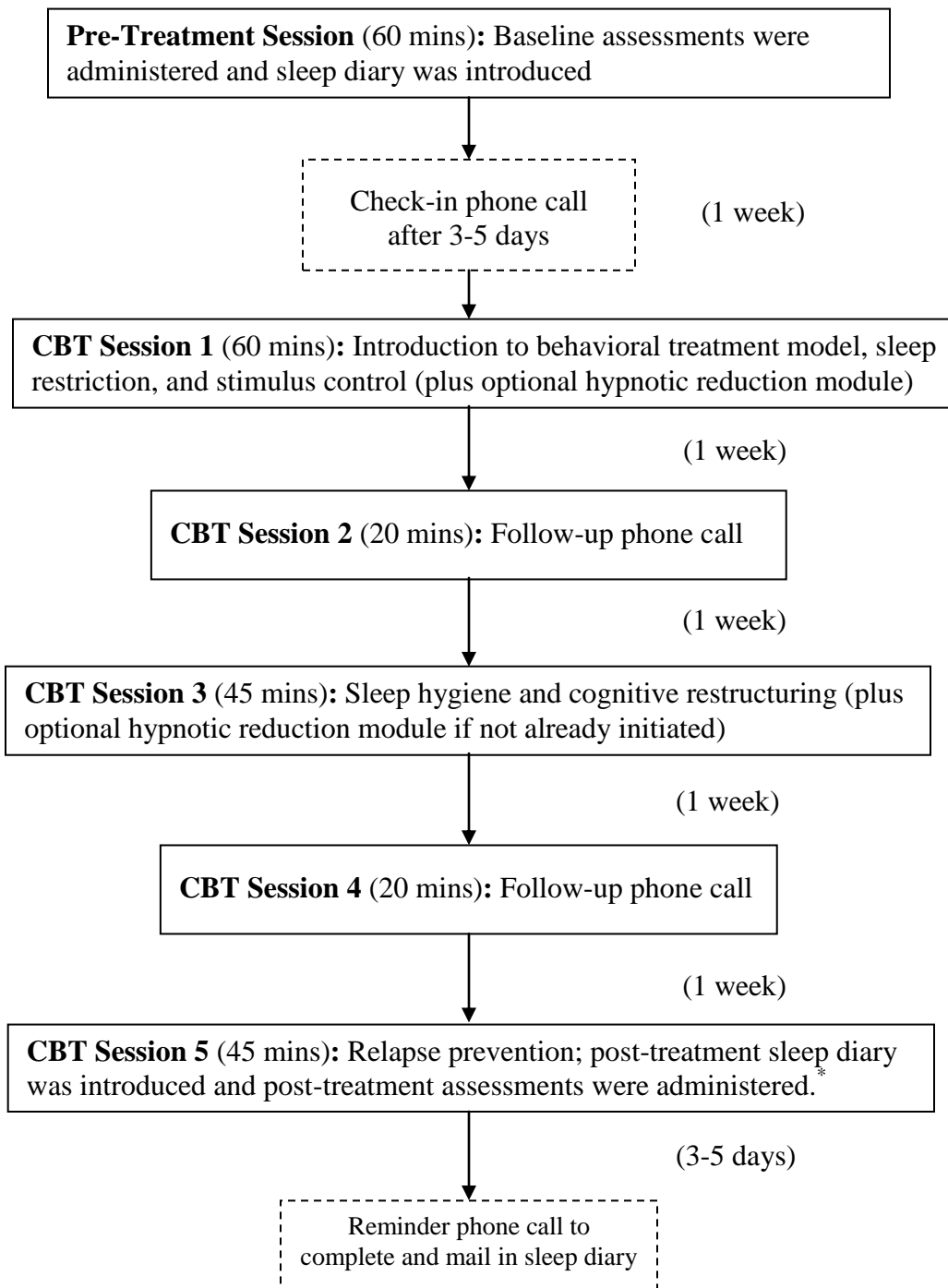
Session 3 was an individual meeting with the doctoral student held at VCU Outpatient Psychiatry that occurred one week after the previous phone session. This session was permitted to be postponed by a week or two, if a patient was unable to make it on schedule. In this third session, sleep hygiene and cognitive restructuring were introduced and the doctoral student checked in on the participant's sleep to determine whether any adjustments to the participant's sleep restriction schedule were warranted. Sleep hygiene was introduced to the patient through verbal discussion of basic guidelines and a handout of tips for the patient to take home. Some examples of sleep hygiene guidelines included: a) get up at the same time each day, seven days a week; b) make sure your bedroom is comfortable and free from light and noise; and c) cut down of caffeine products, especially later in the day. Cognitive restructuring involved an introduction to the concept of how thoughts affect feelings and behaviors and a discussion of common beliefs

about sleep and sleep-related cognitions that may play a role in the maintenance of insomnia. Common maladaptive beliefs about sleep medication were also discussed and the doctoral student provided evidence against such beliefs. Each patient was encouraged to practice identifying and disputing catastrophic sleep-related thoughts through modeling from the doctoral student and a worksheet that guided the patient through this process. In session 3, participants were again offered the optional medication reduction module along with information about the consequences of long term hypnotic use and the evidence for behavioral treatment. As in session 1, of all participants completing the CBT-I treatment, none were interested in coming off their medication. As a result of this fact, the effectiveness of CBT-I paired with a medication taper schedule could not be assessed in this study.

Session 4 occurred one week after the previous session but, again, was permitted to be postponed by a week or two, if a patient was unable to make it on schedule. This session was a follow-up phone call made by the doctoral student and again involved providing support to participants as they underwent sleep restriction and answering any patient questions or concerns. Session 5, the final session, was an in-person meeting with the doctoral student at VCU Outpatient Psychiatry. In this session, techniques for relapse prevention were introduced and the participant was given a sleep diary to complete for post-treatment assessment for a period of one week. Relapse prevention techniques included a review of the behavioral model of insomnia and a discussion of what steps the patient could take to reduce the risk of relapse. The doctoral student also discussed with the patient what to do if insomnia recurs. Also during this session, post-treatment assessments were administered and collected. Patients were given a self-addressed, stamped envelope and were instructed to mail in the sleep diary using this envelope.

Patients received a reminder call approximately five to seven days after session 5 to encourage return of the sleep diary.

All participants who completed the CBT-I treatment were given two-month follow-up assessments via mail and were asked to return these in an enclosed self-addressed, stamped envelope. The TAU group completed questionnaires before and after the five-week TAU period and were offered the opportunity to cross over to the CBT-I treatment group once the initial treatment phase was completed.



**Post-treatment sleep diary was mailed in by each participant using a self-addressed, stamped envelope provided to him or her in Session 5.*

Figure 2. CBT-I Intervention Flow Chart

Pre-Treatment Session: Schedule of Tasks

Patient introductions
Preliminary assessment of eligibility
Brief introduction to study rationale
Discussion of patient questions/concerns
Assessment of patient interest in study
Introduction to and signing of the consent form
Administration of baseline assessments
Introduction to sleep diary

CBT-I Session 1: Schedule of Tasks

Review and summarize sleep diary
Introduce behavioral model of insomnia
Provide rationale for CBT-I as alternative to pharmacotherapy
Provide rationale for sleep restriction
Calculate total sleep time
Set prescribed sleep window
Introduce stimulus control
Set strategy

- How to stay awake until prescribed bed time
- How to respond to nighttime awakenings

If pt is interested in hypnotic reduction module, set tapering schedule based on psychiatrist recommendations

CBT-I Session 2 (Phone Call): Schedule of Tasks

Review of patient's experience employing components from session 1
Discussion of successes/challenges
Problem-solve challenges
Reinforce patient engagement/participation

CBT-I Session 3: Schedule of Tasks

Review and summarize sleep diary
Assess treatment gains and compliance
Determine if changes to patient's treatment plan are necessary

- Increased/reduced restriction of sleep, as appropriate
- Increased reduction of hypnotic medication, if pt has elected to taper

Introduce sleep hygiene
Introduce cognitive restructuring techniques
Cognitive restructuring exercise
If pt is interested in hypnotic reduction module and has not yet completed it, set tapering schedule based on psychiatrist recommendations

CBT-I Session 4 (Phone Call): Schedule of Tasks

Review of patient's experience employing components from sessions 1&3
Discussion of successes/challenges
Problem-solve challenges
Reinforce patient engagement/participation

CBT-I Session 5: Schedule of Tasks

Review and summarize sleep diary
Assess treatment gains and compliance
Determine if changes to patient's treatment plan are necessary

- Reduced restriction of sleep, as appropriate
- Increased reduction of hypnotic medication, if pt has elected to taper

Discuss relapse prevention
Administer post-treatment assessments
Post-treatment sleep diary given to patient

Figure 3. Session-by-Session Schedule of Tasks

Response to Slowed Recruitment and Disinterest in Medication Reduction

When recruitment slowed considerably about eight or nine months into the study despite consistent recruitment efforts, it appeared likely that those patients in the clinic who were eligible for and interested in the study had already been recruited. To understand other potential contributing factors to the lower than expected enrollment, a survey was created to gather information from providers in the clinic who were responsible for referring patients to participate. The doctoral student was present for two or three scheduled psychiatry clinic blocks per week, and could take a very active role in encouraging recruitment during these clinics; however, several providers in the other clinic blocks only learned of the study through limited interactions with the doctoral student, through recruitment flyers in their mailbox, or through word of mouth within the clinic. The survey was designed to briefly assess basic attitudes about the effectiveness of behavioral treatment for insomnia compared to sleep medications. As a result of the lack of participant interest in reducing sleep medication through the optional medication reduction module as part of the CBT-I treatment, additional questions were added to the survey to assess provider prescription practices, attitudes about sleep medication, and the frequency with which the providers advised their patients to consider tapering their sleep medication in the context of this study. Fourteen psychiatry attendings and residents who serve as providers in the clinic were contacted via email and asked to complete the survey. Eight providers completed it. See below for more information about the survey and see survey results beginning on page 71.

Measures

Only self report measures were used in this study. This is common in the insomnia literature (Smith et al., 2005). All measures are included in the Appendix.

Participant Information/Demographic Measure. A paper-and-pencil questionnaire was completed by all participants to collect data on basic demographic information such as age, gender, race, socioeconomic status, and years of education. Relevant medical information was also collected through the demographics questionnaire, such as number and quantity of sleep and non-sleep related medications, as well as comorbid psychological and medical diagnoses. With permission from the IRB, information from participants' medical records was used to confirm or supplement what was provided by participants. Date of first hypnotic use and length of time taking hypnotic medications was also collected. Duration of insomnia was determined using a single self-report question presented verbally to the patient: "For how long have you experienced at least three episodes of disturbed sleep per week along with daytime consequences such as fatigue, irritability, or difficulty concentrating?" An episode was defined as a 30-minute or longer SOL, WASO totaling at least 60 minutes, or TST of less than 6.5 hours per night.

Sleep diaries. Sleep diaries were used to track participant sleep parameters at pre-treatment, post-treatment, and at two-month follow-up. The diaries were paper-and-pencil records adapted from Morin (1993) that were completed by participants each morning for one week prior to treatment, during the duration of the CBT-I treatment phase, and for one week at the end of treatment. Participants were asked to record bedtime, rise time, SOL, awakenings (quantity and duration), time in bed, length and frequency of naps, and any medication used for sleep. Sleep efficiency was also calculated by dividing time spent asleep by time spent in bed and then multiplying this number by 100 to get a percentage. As a complement to this information, participants were also rated the quality of their sleep and how refreshed or exhausted they felt the morning after.

Insomnia Severity Index (ISI). The ISI is a seven-item measure that yields a global score of sleep impairment (Bastien, Vallieres & Morin, 2001). Respondents are asked to rate the severity of sleep disturbance, the degree of interference with daily functioning, how noticeable their impairment is to others, their level of distress in response to sleep disturbance, and overall satisfaction with sleep. Ratings are made on a 1 to 5 scale, with higher scores indicating greater levels of impairment. The ISI has good internal validity and appropriate test–retest reliability over a two-week interval (Bastien et al., 2001).

Patient Health Questionnaire-9 (PHQ-9). The PHQ-9 (Kroenke, Spitzer, & Williams, 2001) is a self-administered version of the depression module from the PRIME-MD, a diagnostic instrument for common mental disorders. This brief nine-item self-report measure is designed to access depression symptoms and functional impairment for diagnostic and treatment monitoring purposes. Scale items correspond to the diagnostic criteria for major depressive disorder in the Diagnostic and Statistical Manual - Fourth Edition, Text Revision (DSM-IV-TR; American Psychological Association, 2000). The PHQ-9 has been shown to be a valid and reliable measure, with diagnostic validity comparable to the original clinician administered instrument (Kroenke et al., 2001).

Generalized Anxiety Disorder 7 (GAD-7). The GAD-7 (Spitzer, Kroenke, Williams, & Löwe, 2006) is a brief, seven-item self-report measure designed to be a screening tool that assesses symptoms of generalized anxiety. The GAD-7 was developed from the diagnostic criteria for generalized anxiety disorder in the DSM-IV-TR (American Psychological Association, 2000). This scale has strong reliability and criterion validity for identifying probable cases of GAD (Spitzer et al., 2006).

Short Form-36 Health Survey Questionnaire. The SF-36 (Medical Outcomes Trust, 1992) is a 36-item assessment designed to measure health status and outcomes from the patient's point of view. It is administered via interview by a clinician (either in person or via telephone) and covers eight health concepts, including physical functioning, role limitations due to physical health problems, bodily pain, general health, vitality (energy/fatigue), social functioning, and role limitations due to emotional problems. The SF-36 has been shown to meet stringent criteria of reliability and validity (Brazier et al., 1992). Eight scale scores inform two summary scores, a physical health component score and a mental health component score.

Provider Survey. Near the conclusion of the study recruitment period, an 11-item online survey was administered to providers at the clinic from which the study participants were recruited. This brief survey was intended to collect information from providers about their attitudes about behavioral treatment versus medication, in general and within their specific patient population. It additionally gathered information about medication prescription practices. The voluntary and anonymous survey was administered via Google Forms. A link to the survey was circulated via email to the 14 psychiatry residents and attendings along with information about the voluntary, anonymous nature of the survey, including the fact that no sensitive or identifying information would be collected.

Data Analysis

Statistical analyses were performed using IBM SPSS Statistics - Version 21 software. As stated previously, intent-to-treat analyses were used for this study; meaning that a "last observation carried forward" method was used to address instances of missing data. For two participants who completed the CBT-I treatment but failed to return their post-treatment sleep diary, their most recent treatment sleep diary (e.g. diary completed during week 4 of treatment)

was used in place of post-treatment sleep diary data. For one participant in the CBT-I group and one participant in the TAU group who both failed to submit any data following the baseline assessments and sleep diary before dropping out of the study, their baseline data was used in place of post-treatment data. This conservative approach to analyzing treatment effects was used in order to limit attrition bias and to strengthen any conclusions that can be made about the intervention (Kazdin, 2003).

To standardize effects for the purpose of interpretation, Cohen's d will be reported as a measure of effect size. As a general guideline, the cutoff is $d = 0.20$ for a small effect size, $d = 0.50$ for a medium effect size, and $d = 0.80$ for a large effect size (Cohen, 1988).

Outline of Analyses by Hypothesis

Hypothesis 1. To test the first hypothesis, that the treatment group would show significantly improved sleep after five sessions of CBT-I compared to the TAU group, repeated measures multivariate analysis of variance (MANOVA) was used. In this analysis, the independent variable (IV) was group (CBT-I treatment vs. TAU) and the repeated factor was time (baseline and post-treatment). The dependent variables (DVs) used to measure improved sleep were total score on the ISI and the four sleep parameters measured by the sleep diary: Sleep efficiency (SE), total sleep time (TST), sleep onset latency (SOL), and wake after sleep onset (WASO). Follow-up repeated measures analyses of variance (ANOVAs) were conducted to assess condition by time interactions for individual sleep variables.

To assess for treatment effects in the combined sample of those randomized to the CBT-I group and those initially randomized to TAU who later crossed over to receive CBT-I, paired t -tests were used. Pairs were created between each sleep variable at baseline and at post-treatment to determine any changes observed after participants completed the CBT-I treatment. Only those

who completed the five-session treatment were included in this analysis (n=14). One participant who was randomized to CBT-I but dropped out of the study after the first CBT-I session was excluded from this analysis, as was one crossover who also attended only one CBT-I session before dropping out.

Hypothesis 2. To test the second hypothesis, that the CBT-I group would show reduced hypnotic use compared to the TAU group after treatment with CBT-I, repeated measures analysis of variance (ANOVA) was used. In this analysis, the IV was group (CBT-I treatment vs. TAU) and the repeated factor was time (baseline and post-treatment). The DV was the number of days of sleep medication use in the last week (assessed by one item on the sleep diary).

Hypothesis 3. To test the third hypothesis, that the CBT-I group would show reduced psychiatric symptoms (depression and anxiety) and improved health-related quality of life after treatment compared to the TAU group, repeated measures multivariate analysis of variance (MANOVA) and repeated measures analysis of variance (ANOVA) were used. First, a MANOVA including GAD-7 and PHQ-9 scores as DVs was conducted. For this analysis, the IV was group (CBT-I treatment vs. TAU) and the repeated factor was time (baseline and post-treatment).

Because SF-36 Physical component scores and SF-36 Mental component scores were not significantly correlated, there was no justification to include these DVs together in a MANOVA. Instead, these two variables were assessed as individual DVs in two separate Repeated Measures ANOVAs. In both analyses, the IV was group (CBT-I treatment vs. TAU) and the repeated factor was time (pre-treatment and post-treatment). These analyses were followed by repeated measures ANOVAS with each of the eight SF-36 scale scores; vitality, physical functioning,

bodily pain, general health functioning, physical role limitation, emotional role limitation, social functioning, and mental health.

To assess for treatment effects in the combined sample of those randomized to the CBT-I group and those initially randomized to TAU who later crossed over to CBT-I, paired t-tests were used. Pairs were created between GAD-7, PHQ-9, and SF-36 variables at baseline and post-treatment to determine any changes observed after participants completed the five-week treatment. Only those who completed the five-session treatment were included in this analysis (n=14). One participant who was randomized to CBT-I but dropped out of the study after the first CBT-I session was excluded from this analysis, as was one crossover who also attended only one CBT-I session before dropping out.

Hypothesis 4. To assess longer term outcomes, repeated measures ANOVAs were used. These analyses determined whether there was a significant difference between sleep parameters, use of sleep medication, psychiatric symptoms, and quality of life at baseline, post-treatment, and 2-month follow-up in the CBT-I group, with crossovers included. To assess sleep outcomes, the IV was time (baseline, post-treatment, and follow-up) for each ANOVA. DVs included: ISI score, sleep efficiency (SE), total sleep time (TST), sleep onset latency (SOL), and wake after sleep onset (WASO), days of medication use per week (as reported on the sleep diary), PHQ-9 score, GAD-7 score, and SF-36 component and scale scores.

Results

Hypothesis 1

Change in Sleep Variables by Group. To test the first hypothesis, that the treatment group would show significantly improved sleep after five sessions of CBT-I compared to the TAU group, repeated measures MANOVA was used. Prior to the primary analysis, data were

checked for statistical assumptions, including normality, linearity, and multicollinearity. Outliers were identified by examining boxplots and creating standardized z-scores for both baseline and post-treatment variables. Initially, most variables were significantly skewed or kurtotic (values outside the range of -1 to 1); however, after addressing several outliers, the assumption of normality was no longer violated. Data points with z-scores greater than 1.96 ($p < .05$) were changed to the next closest value that was under the cut-off from the sample for that variable. This method maintains the shape of the sample distribution without distorting the data (Tabachnik & Fidell, 2007) and was determined to be the most appropriate way of addressing outliers for this data set given the small sample size. Outliers were detected and changed for 12 total data points within the variables of baseline ISI Score ($Z = -2.99$), baseline sleep onset latency ($Z = 2.38$; $Z = 3.02$), baseline total sleep time ($Z = -2.65$), baseline wake time after sleep onset ($Z = 3.35$), baseline sleep efficiency ($Z = -2.87$), post-treatment ISI Score ($Z = -2.21$), post-treatment sleep onset latency ($Z = 2.04$; $Z = 2.99$), post-treatment total sleep time ($Z = -1.98$); post-treatment wake time after sleep onset ($Z = 4.49$), and post-treatment sleep efficiency ($Z = -2.89$).

Scatterplots of the dependent variables were eyeballed for linearity and due to the linear relationship observed between the data points, this assumption was determined to be met. Multicollinearity was examined prior to analysis by running bivariate correlations between the variables. Because two variables correlated highly ($r > .8$, all $ps < .05$) with several other variables, multicollinearity was violated and Box's M Test of Equality of Variances could not be calculated in SPSS. As a result, the variables TST and SE were removed from the MANOVA and were only examined individually through repeated measures ANOVAs. After running the MANOVA, the assumption of homogeneity of variance-covariance matrices was checked using

Box's M test of equality of covariance matrices. This test was significant ($p < .05$), suggesting a violation of this assumption. As a precaution, Pillai's Trace will be the test statistic reported because of its known robustness to violations of the assumption of homogeneity of variance/covariance matrices when sample sizes are relatively equal (Field, 2009). To test for equality of error variances, Levene's test was used. Results were nonsignificant (all $ps > .05$) for all variables, suggesting no violation, with the exception of one (post-treatment ISI Total Score; $p < .05$). Despite this exception, because the sample sizes are nearly equivalent between groups, there is little concern for violation of this assumption.

Results of the MANOVA showed a significant time by group interaction across the three sleep variables included in the analysis (ISI Score, SOL, WASO), Pillai's Trace = .222, $F(1, 17) = 4.85$, $p < .05$. Follow-up repeated measures ANOVAs were conducted to assess for condition by time interactions for the sleep variables included in the MANOVA individually, as well as to assess the two sleep variables not included in the previous analysis, sleep efficiency (SE) and total sleep time (TST). The two intent-to-treat dropouts were included in these analyses. Prior to the analyses, the assumption of independence of observations was confirmed to be met. Sphericity was not violated in any of the ANOVAs conducted, as confirmed by the results of Mauchly's test of sphericity (all Epsilons = 1). Results of the ANOVAs showed a significant time by group interaction for SE, Pillai's Trace = .337, $F(1, 17) = 8.64$, $p < .01$. See Figure 4. Marginally significant time by group interactions were found for ISI Score, Pillai's Trace = .207, $F(1, 17) = 4.43$, $p = .050$; for SOL, Pillai's Trace = .152, $F(1, 17) = 3.04$, $p = .099$; and for TST, Pillai's Trace = .173, $F(1, 17) = 3.64$, $p = .076$. See Figures 5, 6, and 7. No significant time by group interaction was found for WASO ($p = .850$). See Figure 8.

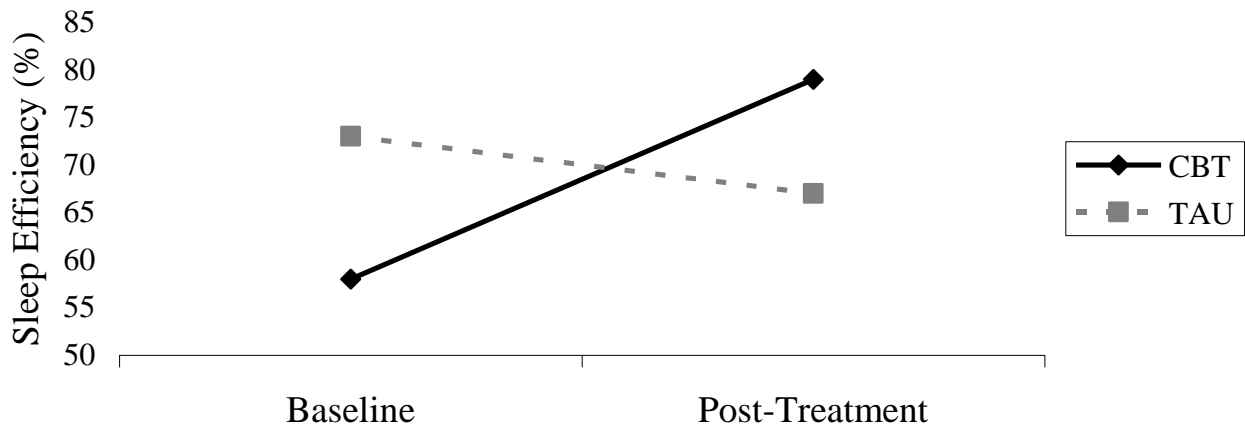


Figure 4. Change in Sleep Efficiency (%) from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19; Outliers Adjusted)

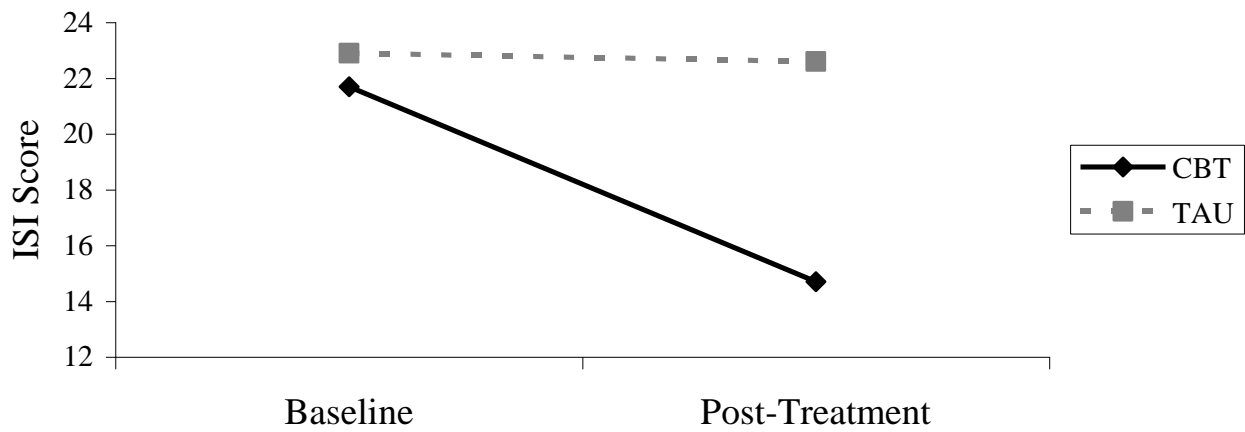


Figure 5. Change in Insomnia Severity Index (ISI) Score from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19; Outliers Adjusted)

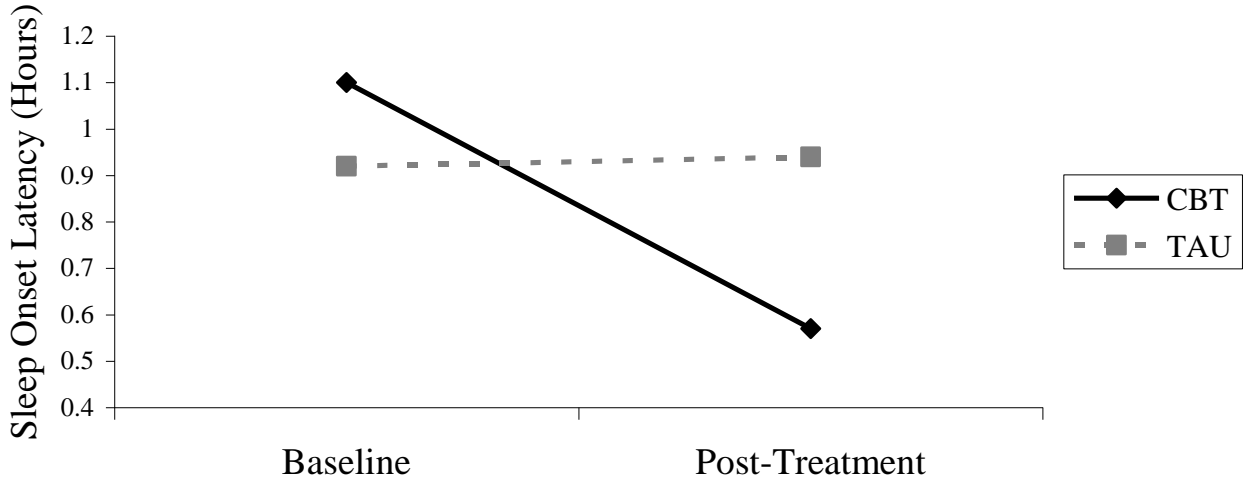


Figure 6. Change in Sleep Onset Latency (Hours) from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19; Outliers Adjusted)

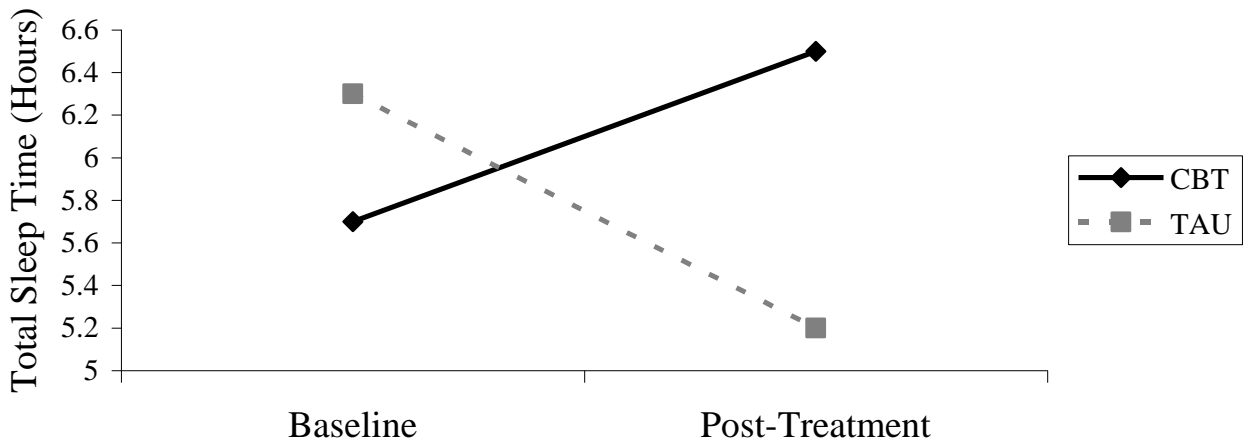


Figure 7. Change in Total Sleep Time (Hours) from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19; Outliers Adjusted)

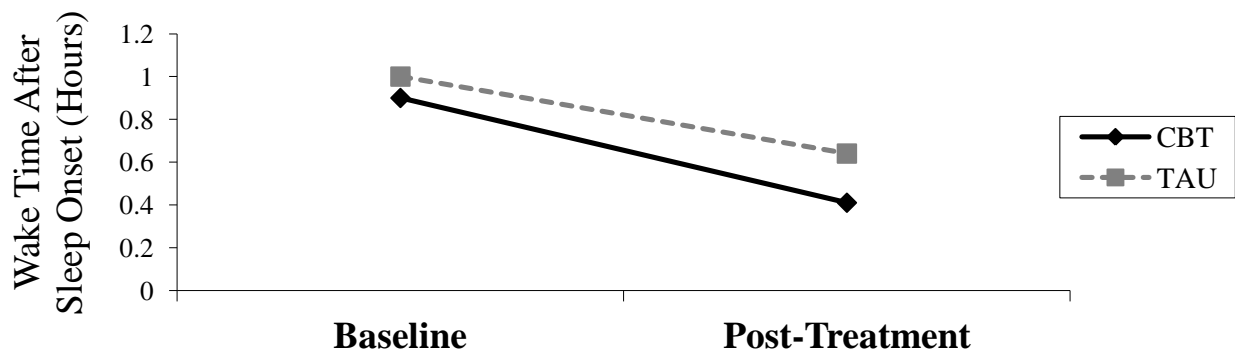


Figure 8. Change in Wake Time After Sleep Onset (Hours) from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19; Outliers Adjusted)

Table 6.

Means (Standard Deviations) and Effect Sizes of Sleep-Related Variables in CBT-I and Treatment as Usual Groups (n=19; Outliers Adjusted)

	Baseline M (SD)		Post-Treatment M (SD)		Effect Size (d)
	CBT	TAU	CBT	TAU	
Insomnia Severity Index (ISI) Score*	21.7 (3.8)	22.9 (3.2)	14.7 (8.5)	22.6 (3.1)	1.05
Sleep Onset Latency (SOL)*	1.1 (1.1)	0.9 (1.0)	0.6 (0.6)	0.9 (0.7)	0.80
Total Sleep Time (TST)*	5.7 (2.2)	6.3 (1.4)	6.5 (2.6)	5.2 (2.2)	0.89
Wake Time After Sleep Onset (WASO)	0.9 (0.5)	1.0 (0.4)	0.4 (0.4)	0.6 (0.4)	0.09
Sleep Efficiency (SE)*	58.0 (22.9)	73.4 (10.3)	79.1 (21.0)	66.8 (21.7)	1.37

* Indicates significant ($p < .05$) or marginally significant ($p < .10$) time by group interaction.

Change in Sleep Variables within the CBT-I Treatment Group with Crossover

Participants. By combining the CBT-I group with those who crossed over to the CBT-I condition after completing the treatment as usual period, treatment effects are able to be more closely examined in a slightly larger sample. Doing so maximizes the ability of the analysis to detect clinically important change. Only those who completed the five-session treatment were included in this analysis (n=14). Results of paired samples t-tests showed a significant change

from baseline to post-treatment for ISI Score, $t(13) = 3.43, p < .01$; sleep efficiency, $t(13) = -3.61, p < .01$; sleep onset latency, $t(13) = 3.25, p < .01$; and wake time after sleep onset, $t(13) = 2.30, p < .05$. No significant differences between baseline and post-treatment data were found for total sleep time ($p = .39$). Figure 9 shows the improvement in sleep efficiency within individual cases across treatment.

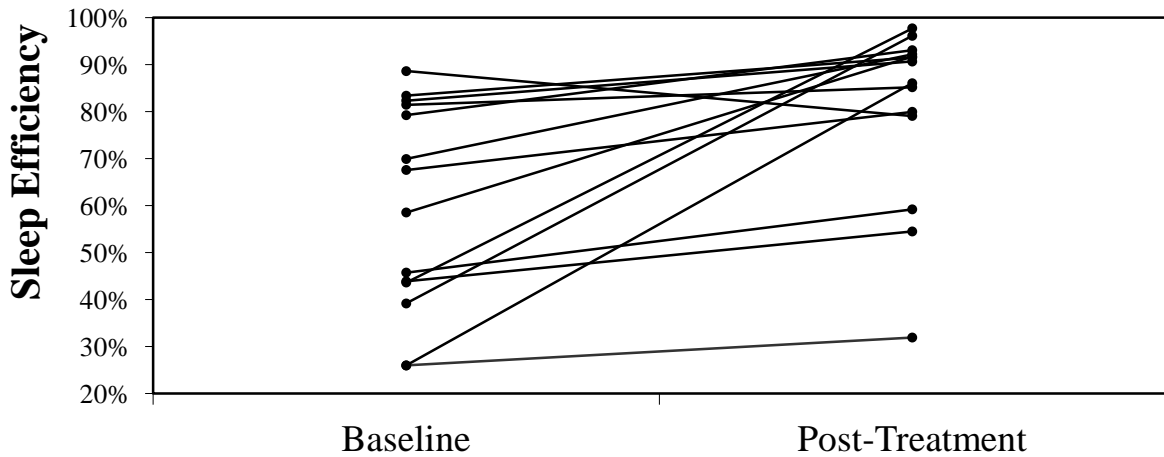


Figure 9. Change in Sleep Efficiency within Individual Cases from Baseline to Post-Treatment in CBT-I Treatment Group with Crossovers (n=14; Outliers Adjusted)

Table 7. Sleep Diary Variable Means (Standard Deviations) and Significance Levels for Combined Crossovers and CBT-I Treatment Group at Baseline and Post-Treatment (n=14; Outliers Adjusted)

	Baseline	Post-Treatment	p-value	Effect Size (d)
Insomnia Severity Index (ISI) Score*	21.8 (3.7)	14.9 (7.6)	0.004	1.00
Sleep Onset Latency (SOL)*	1.1 (1.1)	0.5 (0.6)	0.006	1.15
Total Sleep Time (TST)	5.4 (2.1)	5.9 (2.4)	0.39	0.24
Wake Time After Sleep Onset (WASO)*	0.8 (0.5)	0.4 (0.4)	0.039	0.62
Sleep Efficiency (SE)*	59.7 (22.0)	80.6 (19.1)	0.003	0.97

* Indicates significant change from baseline to post-treatment ($p < .05$).

Hypothesis 2

Change in Use of Sleep Medication by Group. To test the second hypothesis, that the CBT-I group would show reduced hypnotic use compared to the TAU group after treatment with CBT-I, repeated measures ANOVA was used. Prior to the analysis, assumptions of normality, linearity, and independence of observations were checked. Outliers were identified by examining boxplots and creating standardized z-scores for the baseline and post-treatment variable of number of days of medication use per week. As a result of high skewness and kurtosis, outliers were adjusted by calculating z-scores for each data point and replacing all values with z-scores greater than 1.96 ($p < .05$) with the next closest value under the cut-off from the sample for that variable. Outliers were detected and changed for three data points for baseline medication use ($Z = -1.98$; $Z = -1.98$; $Z = -2.97$) and three data point for post-treatment medication use ($Z = -2.15$; $Z = -2.15$; $Z = -3.16$). Sphericity was not violated, as confirmed by the results of Mauchly's test of sphericity (all Epsilons = 1). Results of the ANOVA showed no significant time by group interaction for use of sleep medication between baseline and post-treatment ($p = .429$).

Hypothesis 3

Change in Depression, Anxiety, and Health-Related Quality of Life by Group. To test the third hypothesis, that the CBT-I group would show reduced psychiatric symptoms (depression and anxiety) and improved health-related quality of life after treatment compared to the TAU group, repeated measures MANOVA and repeated measures ANOVA were used. Prior to the analyses, data were checked for statistical assumptions, including normality, linearity, and multicollinearity. After examining boxplots and creating standardized z-scores for all variables, no outliers were identified. Skewness and kurtosis values were within the acceptable range of -1

to 1. Prior to the MANOVA, scatterplots of the dependent variables were eyeballed for linearity and due to the linear relationship observed between the data points, this assumption was determined to be met. Multicollinearity was examined prior to the analysis by running bivariate correlations between the variables. Correlations between the variables were significant and high enough to warrant them being used together in the MANOVA but not so high that the assumption of multicollinearity was presumed to be violated ($r < .8$). After running the MANOVA, the assumption of homogeneity of variance-covariance matrices was checked using Box's M test of equality of covariance matrices. This test was nonsignificant ($p = .550$), suggesting no violation of this assumption. To test for equality of error variances, Levene's test was used. Results were nonsignificant (all $ps > .05$), suggesting no violation.

Results of the MANOVA showed no significant time by group interaction ($p = .388$). See Figures 10 and 11 for changes in mean GAD-7 score and mean PHQ-9 score between the two groups across treatment.

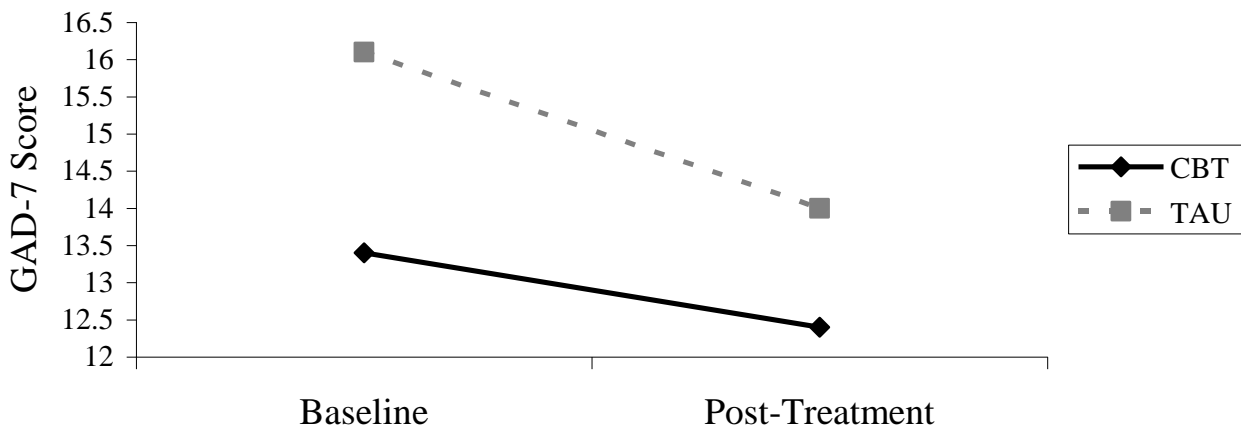


Figure 10. Change in GAD-7 Score from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19)

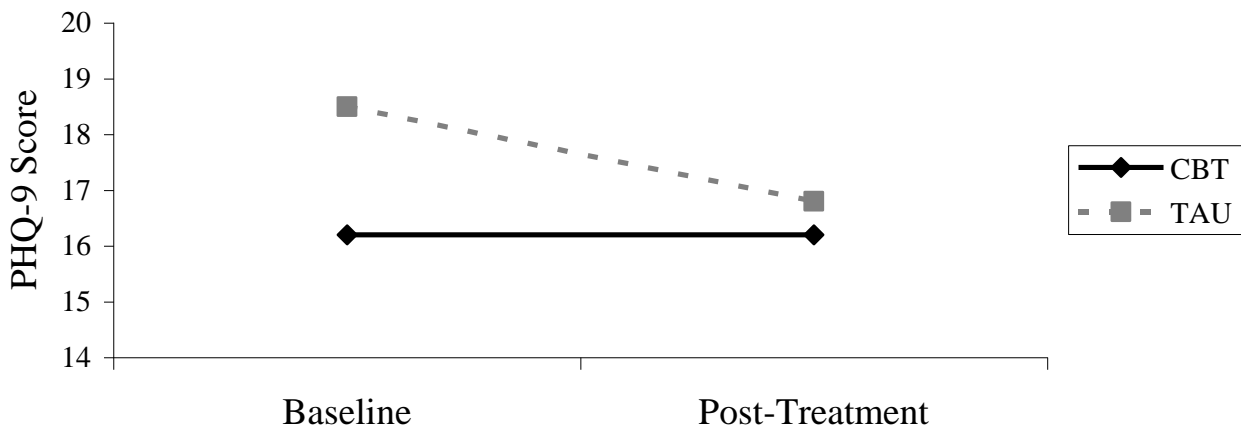


Figure 11. Change in PHQ-9 Score from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (n=19)

Prior to the ANOVAs with SF-36 component scores as DVs, the assumption of independence of observations was confirmed to be met. Sphericity was not violated in either ANOVA, as confirmed by the results of Mauchly's test of sphericity (all Epsilons = 1). Results of the first ANOVA, which included the SF-36 physical component score as the DV, showed no significant time by group interaction ($p = .425$). Similarly, no significant time by group interaction was found for the second ANOVA, which included the SF-36 mental component score as the DV, ($p = .263$). See Figures 12 and 13 for changes in mean SF-36 physical component score and mean SF-36 mental component score between the two groups across treatment.

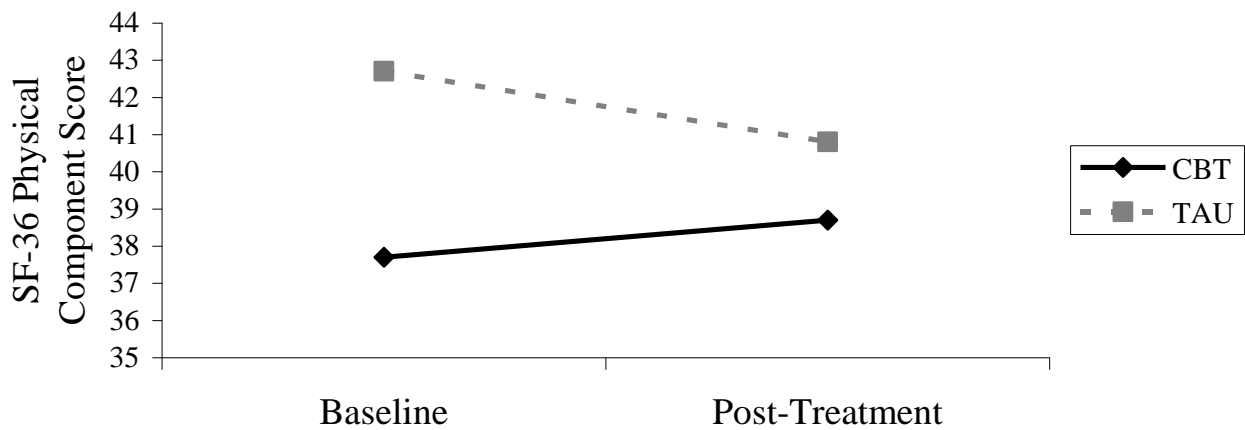


Figure 12. Change in SF-36 Physical Component Score from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (Higher Scores Reflect Higher Ratings of Quality of Life; n=19)

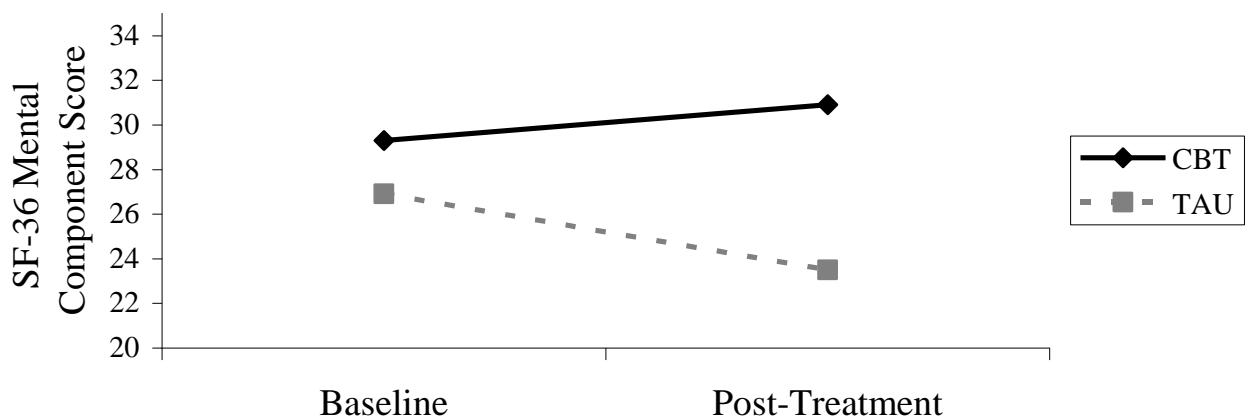


Figure 13. Change in SF-36 Mental Component Score from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (Higher Scores Reflect Higher Ratings of Quality of Life; n=19)

Table 8.

Means (Standard Deviations) of Depression, Anxiety, and Health-Related Quality of Life Variables in CBT-I and Treatment as Usual Groups (n=19)

	Baseline M (SD)		Post-Treatment M (SD)	
	CBT	TAU	CBT	TAU
PHQ-9 Score	16.2 (7.9)	18.5 (4.8)	16.2 (7.5)	16.8 (5.4)
GAD-7 Score	13.4 (6.4)	16.1 (4.8)	12.4 (6.6)	14.0 (4.2)
SF-36 Physical Component Score	37.7 (10.1)	42.7 (13.2)	38.7 (11.9)	40.8 (13.1)
SF-36 Mental Component Score	29.3 (10.6)	26.9 (6.7)	30.9 (11.8)	23.5 (5.9)

Repeated measures ANOVAs with each of the eight SF-36 scale scores (vitality, physical functioning, bodily pain, general health functioning, physical role limitations, emotional role limitations, social functioning, and mental health functioning) were also conducted. Prior to the ANOVAs, the assumption of independence of observations was confirmed to be met. Sphericity was not violated in any ANOVA, as confirmed by the results of Mauchly's test of sphericity (all Epsilons = 1). Results showed no significant time by group interactions (all p s > .05), with the exception of a marginally significant time by group interaction for the social functioning scale, $F(1, 17) = .157, p = .093$, multivariate $\eta_p^2 = .157$. See Figure 14.

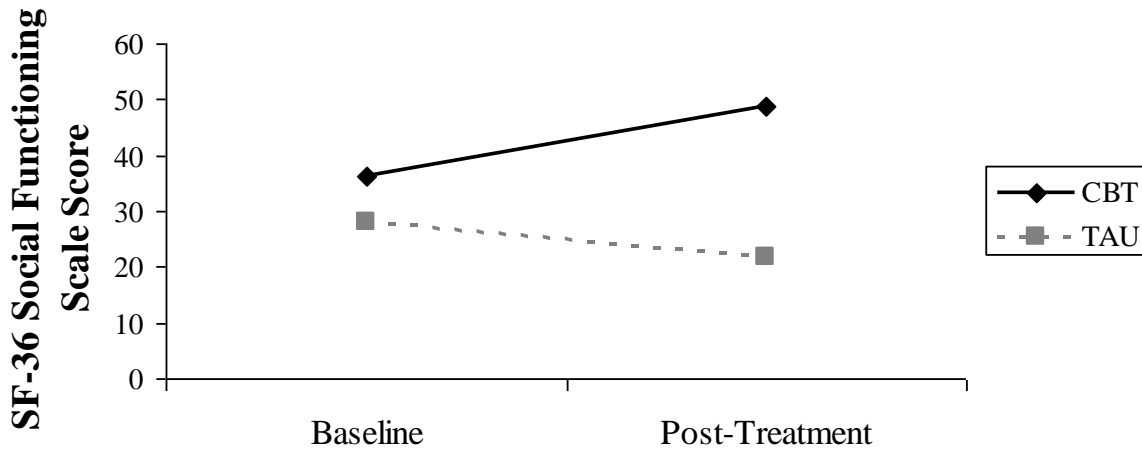


Figure 14. Change in SF-36 Social Functioning Scale Score from Baseline to Post-Treatment in CBT-I and Treatment as Usual (TAU) Groups (Higher Scores Reflect Higher Ratings of Quality of Life; n=19)

Change in Depression, Anxiety, and Health-Related Quality of Life within the CBT-I Treatment Group with Crossover Participants. To examine treatment effects in a slightly larger sample, the CBT-I group was again combined with those who crossed over to the CBT-I condition after completing the treatment as usual period. Combining these participants and examining changes from baseline to post-treatment in only those who received the CBT-I treatment maximizes the ability of the analysis to detect clinically important change. Only those who completed the five-session treatment were included in this analysis (n=14). Results of paired samples t-tests showed no significant differences between baseline and post-treatment scores on the GAD-7 and PHQ-9, nor between baseline and post-treatment SF-36 physical health and mental health component scores (all $ps > .05$). Results of paired t-tests with each of the eight SF-36 scales, including vitality, physical functioning, bodily pain, general health functioning, physical role limitations, emotional role limitations, social functioning, and mental health functioning were nonsignificant (all $ps > .05$), with the exception of the social functioning scale.

A significant difference in this scale score was found between baseline and post-treatment, $t(13) = -7.10, p < .05$. See Figure 15.

Table 9.
Anxiety, Depression, and Health-Related Quality of Life Means (Standard Deviations) and Significance Levels for Combined Crossovers and CBT-I Group at Baseline and Post-Treatment (n=14)

	Baseline	Post-Treatment	<i>p</i> -value
PHQ-9 Score	15.2 (6.8)	14.1 (7.4)	0.611
GAD-7 Score	12.9 (5.7)	11.9 (5.4)	0.453
SF-36 Physical Component Score	38.6 (9.4)	42.2 (13.5)	0.169
SF-36 Mental Component Score	28.9 (8.8)	30.5 (10.4)	0.581

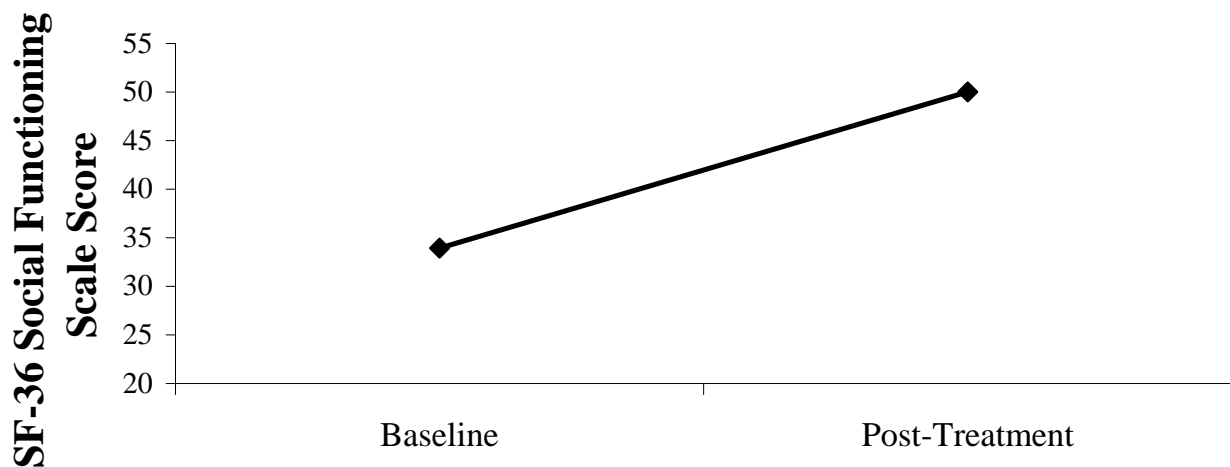


Figure 15. Change in SF-36 Social Functioning Scale Score from Baseline to Post-Treatment in Combined CBT-I and Crossovers Treatment Group (n=14)

Hypothesis 4

To assess longer term outcomes, repeated measures ANOVAs were used. Prior to the analyses, data collected at two-month follow-up were checked for statistical assumptions, including normality, linearity, and sphericity. Outliers were identified by examining boxplots and creating standardized z-scores for both baseline and post-treatment variables. Initially, several variables were significantly skewed or kurtotic (values outside the range of -1 to 1); however, after addressing outliers, normality was significantly improved. Data points with z-scores greater than 1.96 ($p < .05$) were changed to the next closest value that was under the cut-off from the sample for that variable. This method maintains the shape of the sample distribution without distorting the data (Tabachnik & Fidell, 2007) and was determined to be the most appropriate way of addressing outliers for this data set given the small sample size. Outliers were detected and changed for three data points at follow-up within the variables of sleep onset latency ($Z = 2.37$), total sleep time ($Z = 2.19$), sleep efficiency ($Z = -2.07$). The assumption of independence of observations was confirmed to be met. Sphericity was violated for all ANOVAs conducted, as indicated by the results of Mauchly's test of sphericity (Epsilons < 1). In any case where Epsilons were less than 1, a Greenhouse Geisser correction was applied to adjust the degrees of freedom.

Durability of Gains for Sleep Variables at Two-Month Follow-up. Results of the ANOVA for sleep efficiency showed a significant difference between time points, $F(1.69, 11.80) = 11.17, p < .01$. Posthoc tests (Bonferroni correction) showed that CBT-I was associated with an improvement in sleep efficiency from baseline to post-treatment, ($p < .05$), and from baseline to follow-up ($p < .05$). No significant differences were observed from post-treatment to follow-

up assessment ($p = .100$), indicating that treatment gains were maintained two months after the end of treatment.

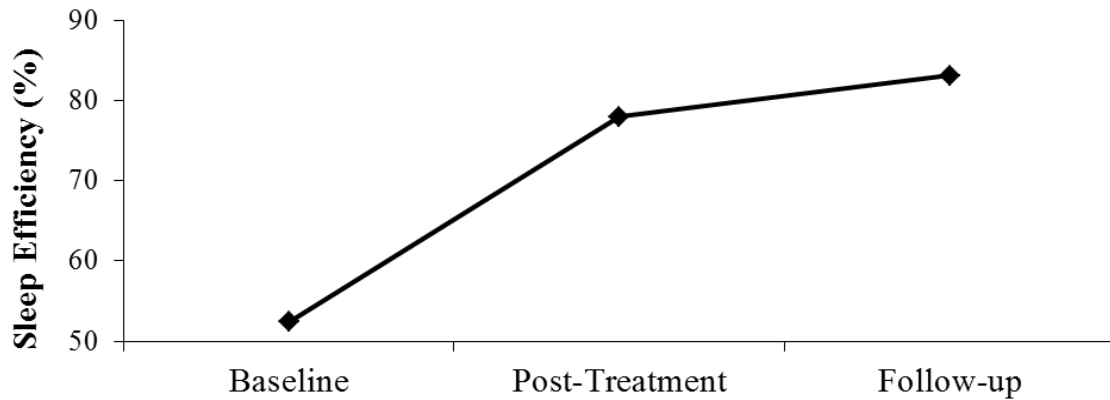


Figure 16. Sleep Efficiency at Baseline, Post-Treatment, and Two-Month Follow-up in CBT-I Treatment Completers (n=8)

Results of the ANOVA for ISI score showed a significant difference between time points, $F(1.28, 8.93) = 5.17, p < .05$. Posthoc tests (Bonferroni correction) demonstrated that CBT-I was associated with an improvement in ISI score from baseline to post-treatment; however, this effect was only marginally significant ($p = .09$). No significant differences were observed from baseline to follow-up ($p = .23$) or from post-treatment to follow-up assessment ($p = .100$). The latter result indicates that treatment gains were maintained at two-month follow-up.

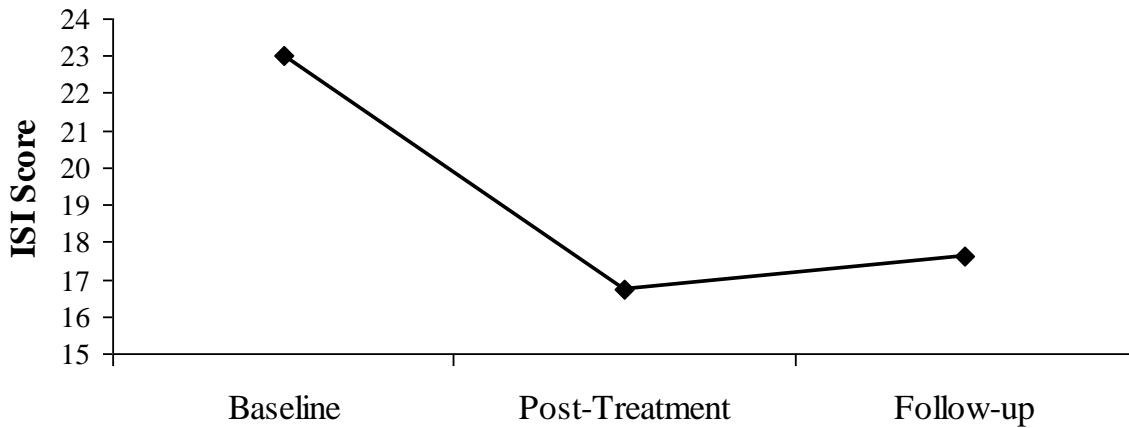


Figure 17. ISI Score at Baseline, Post-Treatment, and Two-Month Follow-up in CBT-I Treatment Completers (n=8)

Results of the ANOVA for sleep onset latency showed a significant difference between time points, $F(1.28, 8.98) = 8.85, p < .05$. Posthoc tests (Bonferroni correction) demonstrated that CBT-I was associated with an improvement in sleep efficiency from baseline to post-treatment, ($p < .05$), and from baseline to follow-up; however, the difference between baseline and follow-up time points was only marginally significant ($p = .06$). No significant differences were found between post-treatment and follow-up ($p = 1.00$), showing that treatment gains were maintained from post-treatment to follow-up assessment.

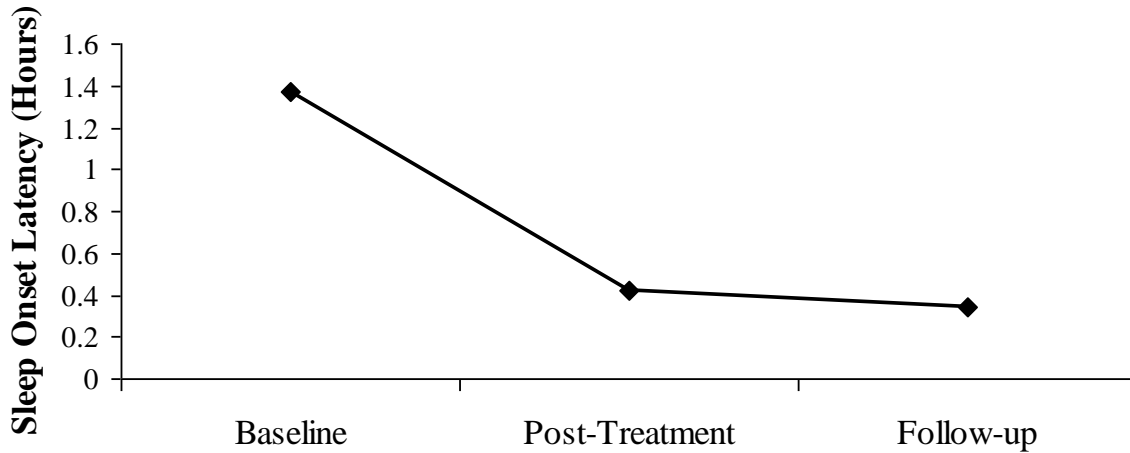


Figure 18. Sleep Onset Latency at Baseline, Post-Treatment, and Two-Month Follow-up in CBT-I Treatment Completers (n=8)

Results of the ANOVA for total sleep time showed no significant difference between time points, $F(1.42,9.91) = 1.60, p = .24$. Mean total sleep time improved minimally from baseline to post-treatment and increased by an average of nearly 60 minutes from post-treatment to follow-up in the sample of those who completed assessments at all three time points (n=8). These differences, however, were not found to be statistically significant.

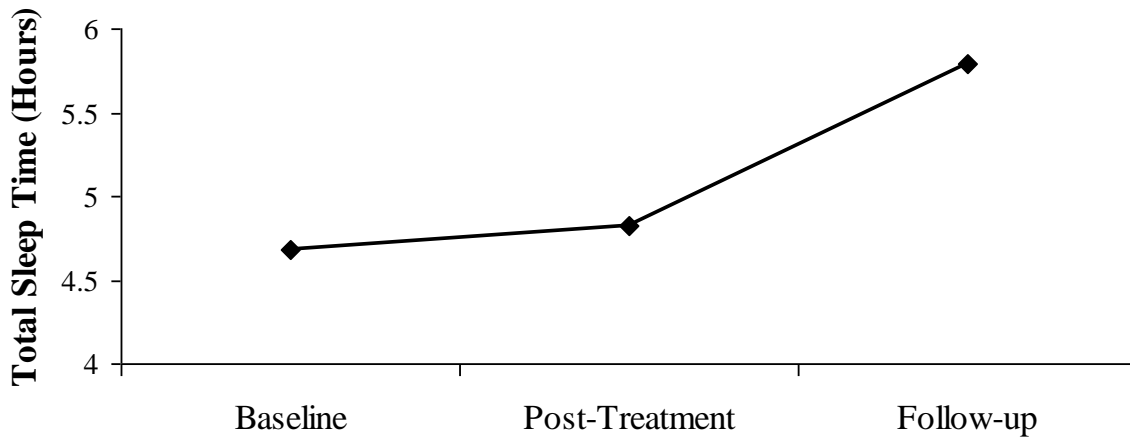


Figure 19. Total Sleep Time at Baseline, Post-Treatment, and Two-Month Follow-up in CBT-I Treatment Completers (n=8)

Results of the ANOVA for wake time after sleep onset showed no significant difference between time points, $F(1.29,9.05) = 1.59, p = .25$. Time spent awake after sleep onset improved by an average of about 20 minutes from baseline to post-treatment; however, this difference was not found to be statistically significant ($p = .70$). No significant difference was found between post-treatment and follow-up WASO ($p = 1.00$), showing that the small gains made in this parameter after treatment with CBT-I were maintained at the two-month follow-up assessment. Wake time increased by an average of less than 2 minutes between post-treatment and follow-up.

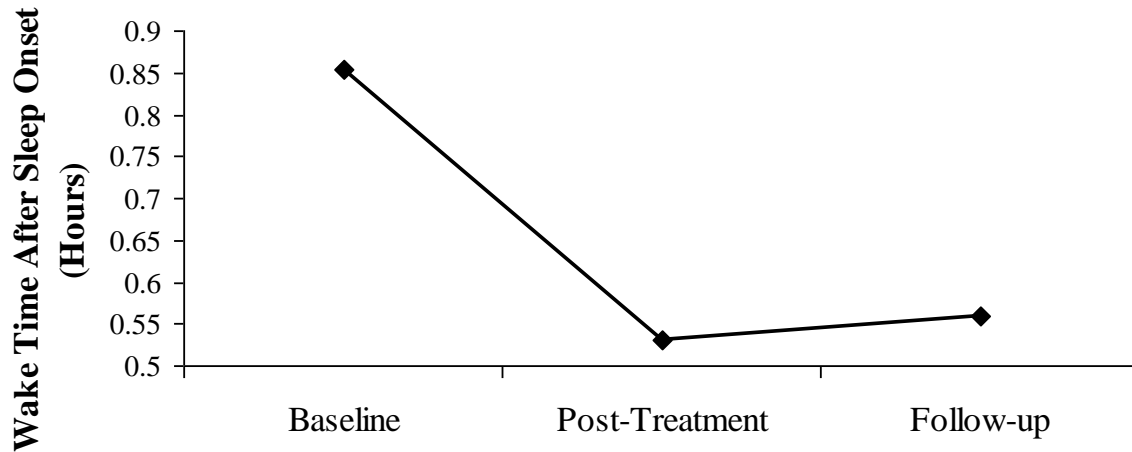


Figure 20. Wake Time After Sleep Onset at Baseline, Post-Treatment, and Two-Month Follow-up in CBT-I Treatment Completers (n=8)

Table 10.

Means (Standard Deviations) of Sleep Diary Variables for Combined Crossovers and CBT-I Group at Baseline, Post-treatment, and Two Month Follow-up (n=8)

	Baseline	Post-Treatment	Follow-up	Effect Size (Baseline to Follow-up)
Insomnia Severity Index (ISI) Score	23.00 (3.8)	16.75 (7.1)	17.63 (8.2)	0.87
Sleep Onset Latency in hours (SOL)	1.37 (1.1)	0.42 (0.6)	0.34 (0.3)	1.42
Total Sleep Time in hours (TST)	4.68 (2.1)	4.83 (2.0)	5.79 (1.7)	0.53
Wake Time After Sleep Onset in hours (WASO)	0.85 (0.5)	0.53 (0.5)	0.56 (0.3)	0.49
Sleep Efficiency % (SE)	52.36 (20.5)	78.00 (0.2)	83.12 (9.9)	1.49

Durability of Gains for Depression, Anxiety, and Health-Related Quality of Life at Two-

Month Follow-up. A repeated measures ANOVA was used to test the durability of gains in social functioning as measured by the SF-36. Of all scores on the GAD-7, PHQ-9, and SF-36, this was the only score that was significantly improved from baseline to post-treatment. Results showed a statistically significant difference between time points, $F(1.71,12.0) = 7.53, p < .01$.

Posthoc tests (Bonferroni correction) demonstrated that CBT-I was associated with a marginally

significant improvement in SF-36 social functioning scale score from baseline to post-treatment, ($p = .05$); however, a significant decrease was seen in this score from post-treatment to follow-up ($p < .05$). Therefore, the gains in social functioning seen immediately after treatment with CBT-I concluded were not maintained in this sample of eight treatment completers at two-month follow-up.

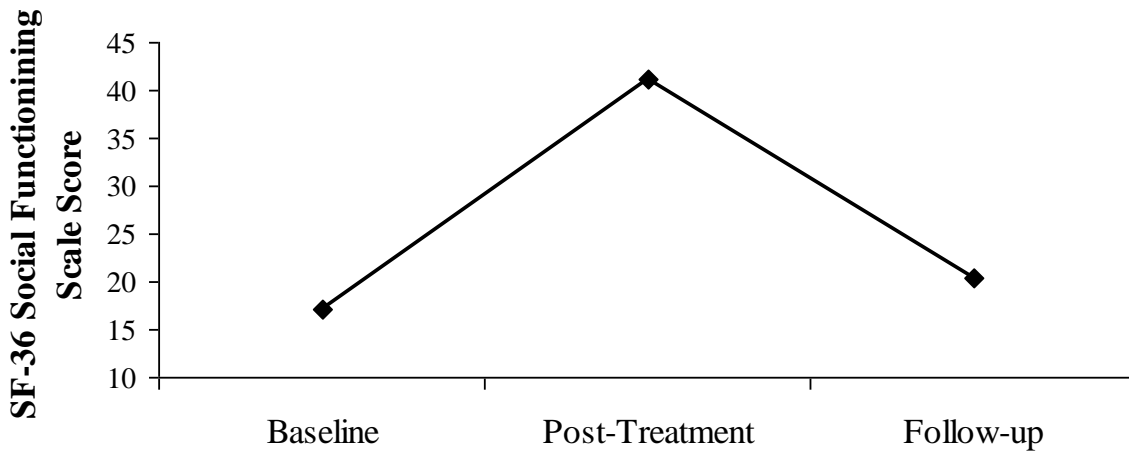


Figure 21. SF-36 Social Functioning Scale Score at Baseline, Post-Treatment, and Two-Month Follow-up in CBT-I Treatment Completors (n=8)

Repeated measures ANOVAs were used to assess longer term outcomes in medication use, PHQ-9 score, GAD-7 score, and other SF-36 scores; however, no significant changes were found between time points (all $ps > .05$).

Provider Survey

Results of the anonymous online survey completed by providers in the clinic (n=8) are summarized by question in the figures below.

CBT-I is an effective way to treat insomnia (n = 8)

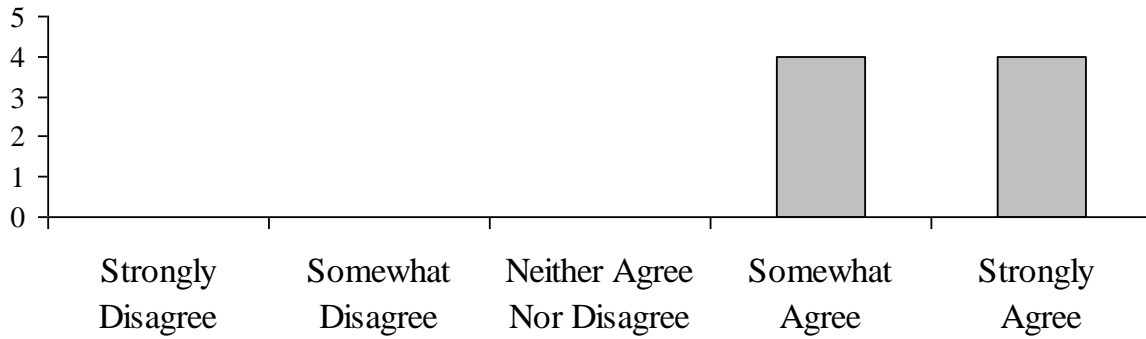


Figure 22. Provider Responses to Question 1: CBT-I is an Effective Way to Treat Insomnia (n=8)

Behavioral strategies for treating insomnia are too demanding for most of my psychiatric patients (n = 8)



Figure 23. Provider Responses to Question 2: Behavioral Strategies for Treating Insomnia are Too Demanding for Most of My Psychiatric Patients (n=8)

When treating patients with chronic insomnia, I commonly prescribe sleep medications for longer than one month (n = 8)

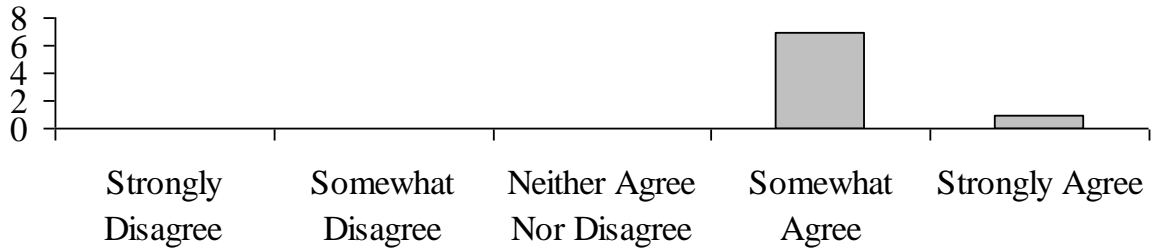


Figure 24. Provider Responses to Question 3: When treating patients with chronic insomnia, I commonly prescribe sleep medications for longer than one month (n=8)

Medication for sleep is more effective in the long term than are behavioral treatments such as CBT-I (n = 8)

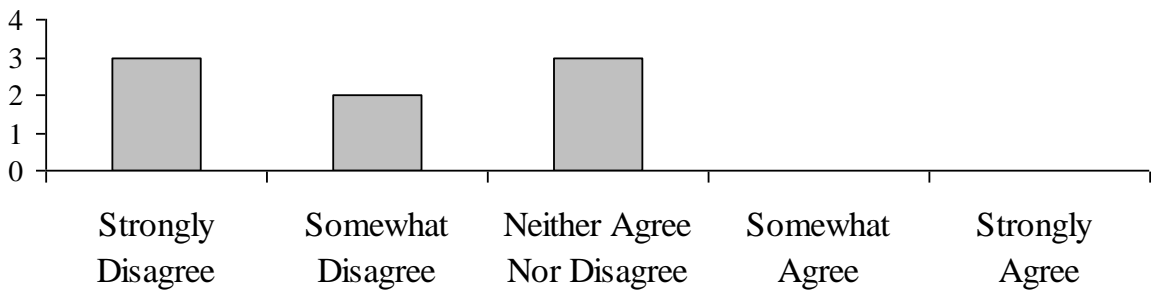


Figure 25. Provider Responses to Question 4: In general, medication for sleep is more effective in the long term than are behavioral treatments such as CBT-I (n=8)

Chronic use of medications for sleep is problematic and should be avoided at all costs (n = 8)

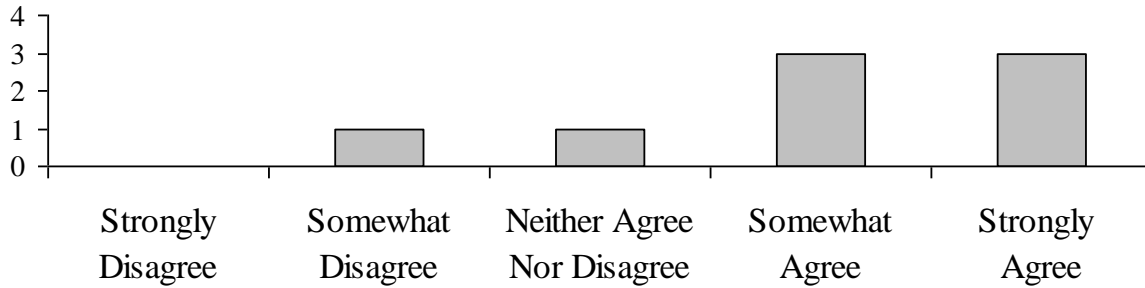


Figure 26. Provider Responses to Question 5: Chronic use of medications for sleep (e.g. sedative hypnotics) is problematic and should be avoided at all costs (n=8)

If a patient with insomnia is seeing little benefit from his/her sleep medication but asks me to continue prescribing it, I often do so (n = 8)

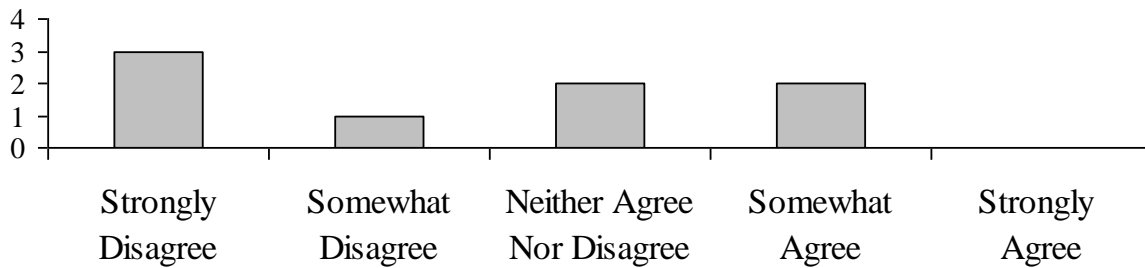


Figure 27. Provider Responses to Question 6: If a patient with insomnia is seeing little benefit from his/her sleep medication but asks me to continue prescribing it, I often do so (n=8)

Tapering my patients off their sleep medication leads to problematic side effects (n = 8)

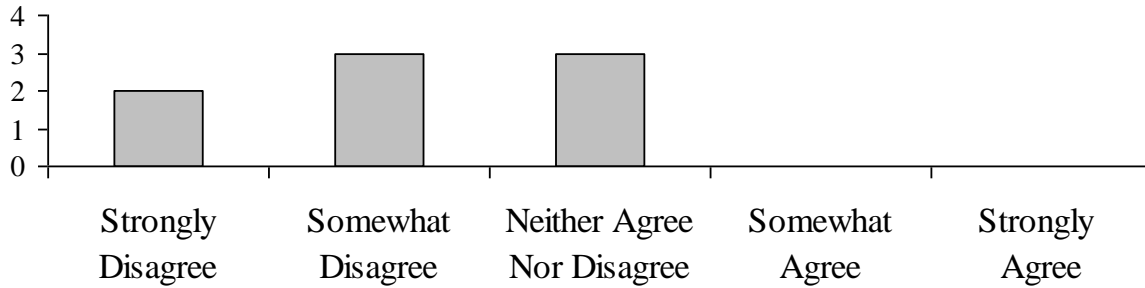


Figure 28. Provider Responses to Question 7: Tapering my patients off their sleep medication leads to problematic side effects (n=8)

Sleep medications are appropriate for short term use but should not be prescribed for chronic insomnia (n = 8)

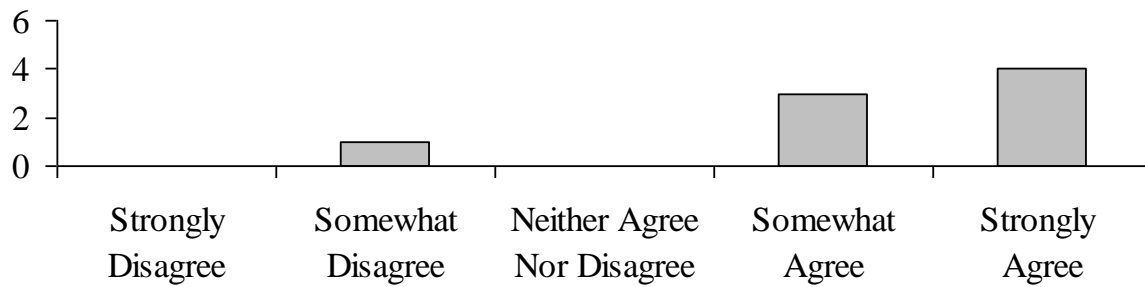


Figure 29. Provider Responses to Question 8: Sleep medications are appropriate for short term use but should not be prescribed for chronic insomnia (n=8)

Most of my patients with insomnia are not interested in behavioral treatments (such as CBT-I) and prefer medication (n = 8)

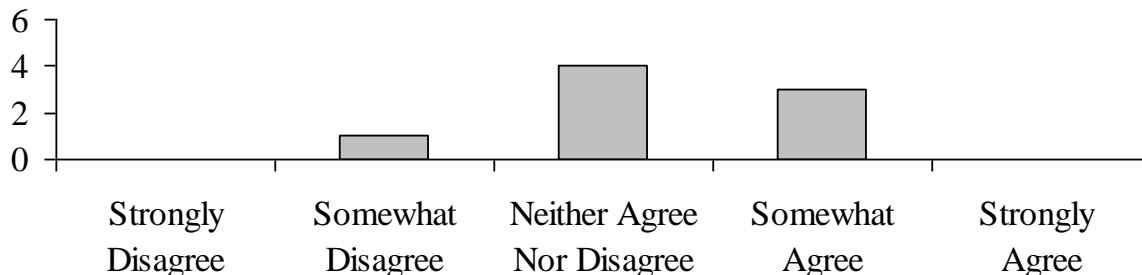


Figure 30. Provider Responses to Question 9: Most of my patients with insomnia are not interested in behavioral treatments (such as CBT-I) and prefer medication (n=8)

When discussing treatment options with patients who have insomnia, how often do you mention behavioral treatments (such as CBT-I) in addition to medication (n = 8)

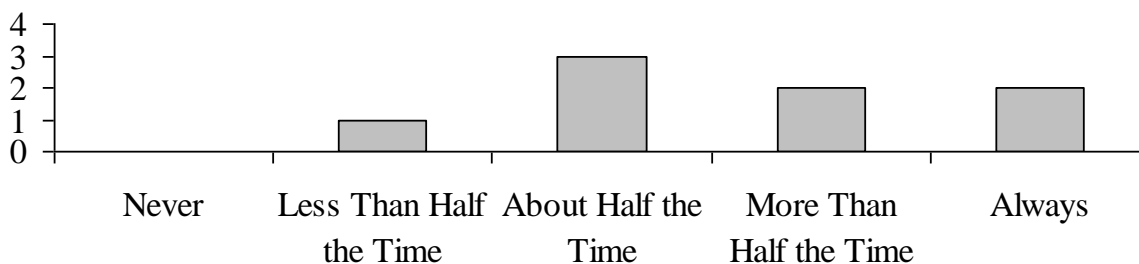


Figure 31. Provider Responses to Question 10: When discussing treatment options with patients who have insomnia, about how often do you mention behavioral treatments (such as CBT-I) in addition to medication? (n=8)

If you referred patients to the CBT for Insomnia research study, about how often did you speak with them beforehand about coming off their sleep medication? (n = 8)

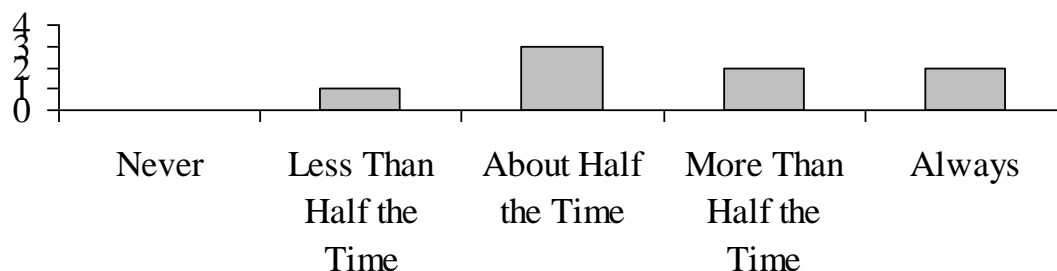


Figure 32. Provider Responses to Question 11: If you referred patients to the CBT-I for Insomnia research study, about how often did you speak with them beforehand about coming off their sleep medication? (n=8)

Discussion

The present study sought to examine the effectiveness of a brief CBT-I treatment for persistent insomnia in chronic users of sleep medication who were receiving mental health treatment in a psychiatric clinic located in an urban academic medical center. Previous research supports the efficacy of CBT-I for comorbid insomnia in patients with psychiatric diagnoses (Lichstein et al., 2000; Manber et al., 2008; Ulmer et al., 2011; Watanabe et al., 2011) and a handful of studies have examined the effectiveness of this treatment (Dashevsky & Kramer, 2008; Perlis et al., 2000; Espie, Inglis, Tessier, et al., 2001; Espie et al., 2007); however, most studies have excluded patients with significant psychiatric symptoms. In a search of the literature, this author could find only one other study to date that was carried out in an outpatient psychiatry setting (Watanabe et al., 2011). The findings from this study in patients recruited from outpatient psychiatric clinics in Japan showed that adding a brief CBT-I intervention to usual care led to significant improvements in sleep and depressive symptoms compared to

treatment as usual. It is difficult to generalize these findings to heterogeneous psychiatric populations, however, due to the fact that patients with psychiatric comorbidities other than depression were excluded from participating and patients with mild, moderate, or partially remitted depression were specifically targeted (Watanabe et al., 2011).

This study sought to include a diverse sample of patients from an outpatient psychiatry clinic, excluding only those whose psychiatric symptoms were so severe that they would not be safe to participate or likely to benefit from CBT-I. The sample that resulted had a range of diagnoses, with 84% having two or more diagnoses and 21% having three or more, including bipolar disorder, PTSD, panic disorder, major depressive disorder, and drug or alcohol dependence in full or partial remission, among others. Thirteen participants (68%) had personality traits suggestive of possible personality disorder, according to their treating psychiatrist, and one participant (5%) had a diagnosis of borderline personality disorder. One hundred percent (100%) of the sample was prescribed at least one psychotropic medication at the time of the study, with 68% prescribed three or more psychotropic medications. The sample was also diverse in education level and socioeconomic status, with 32% reporting less than a high school education and 26% reporting a college education or beyond. Eighty-four percent (84%) of participants were unemployed or on disability and only 16% (3 individuals) were employed at the time of the study. The sample was also relatively medically complex, with all participants reporting at least 1 medical condition and 80% reporting 2 or more comorbid health problems. The combined groups reported an average of 4 comorbid medical conditions, with diagnoses such as coronary artery disease, alcoholic cirrhosis of the liver, COPD, asthma, hepatitis C, hypertension, chronic pain, migraines, peripheral neuropathy, osteoarthritis, and sarcoidosis. By including nearly all patients in the clinic with insomnia who were interested in receiving

treatment, the resulting sample is a more accurate representation of the typical patient population from an outpatient psychiatry clinic. Because the particular clinic from which participants were recruited serves a significant number of underserved groups, this sample may in fact be more medically, psychiatrically, and socially complex than other outpatient clinics.

Participants in this study were randomized to either treatment as usual or to the CBT-I group, in which patients received a five-session intervention for insomnia delivered individually. It was hypothesized that participants receiving CBT-I would show significant improvement in insomnia symptoms, reduced depression and anxiety, and improved health-related quality of life, and that these gains would be maintained at two-month follow-up. It was also hypothesized that participants in the CBT-I group would show reduced use of sleep medication after receiving the medication reduction module as part of the treatment and working to taper down their medication over the course of the study. These hypotheses were based on the supporting literature, which suggests that CBT-I is an effective treatment and may improve other mental health symptoms in addition to sleep (Rybarczyk et al., 2009; Watanabe et al., 2011); although no known studies to date have tested the effectiveness of CBT-I in a diverse, real-world psychiatric population.

The results of the present study largely supported the hypotheses related to sleep-related variables and partially supported the hypothesis related to quality of life, but they failed to support hypotheses related to medication use or anxiety and depression. Results show that CBT-I is an effective treatment in chronic psychiatric patients who are current users of sleep medication and social functioning was improved following CBT-I; however, the treatment was not effective in reducing depression and anxiety. Given the resistance of patients in this sample to reduce their sleep medication, CBT-I was not effective in reducing medication use, as hypothesized; however, it is possible that with changes to the study design, a different result

would be found in future studies. For example, the optional medication reduction module could have been administered in full to all patients as a required part of the first session. Alternatively, the treating psychiatrist and doctoral student delivering CBT-I could have met with the patient together in the first session to discuss reducing sleep medication, thereby providing more active guidance on the issue. This study demonstrates that the treatment approach taken in this study is feasible in complex outpatient psychiatric populations; however, more intensive focus on medication reduction may be warranted in this population for the treatment to effectively reduce patient reliance on sleep medications.

CBT-I Treatment Effects on Self-Reported Sleep

Baseline sleep parameters were comparable to those reported in a recent meta-analysis that compiled results across 14 randomized, controlled studies testing CBT-I in primary insomnia (Okajima, Komada, & Inoue, 2011). Mean sleep efficiency at baseline in participants randomized to CBT-I was 57.2% in the meta-analysis, compared to 58.0% in the present study. Mean total sleep times were 5.3 hours and 5.7 hours, respectively. Small differences were observed in mean sleep onset latency (44 minutes versus 66 minutes, respectively) and wake time after sleep onset (79 minutes versus 54 minutes, respectively).

Results from intent-to-treat analyses in the present study including 11 participants receiving CBT-I and 8 participants receiving treatment as usual showed that the CBT-I group exhibited significant improvement on a global measure of insomnia and on several specific sleep parameters. Sleep efficiency, the parameter known to be a reliable composite index of sleep disturbance severity and measure of sleep improvement (Gagné & Morin, 2001), improved by an average of 21% in the treatment group, with 64% (9 participants) achieving the 85% cut-off for sleep within the normal range after treatment. This is compared to only 7% (1 participant) in the

normal range at baseline. In the treatment as usual group, 0 participants fell in the normal range at baseline; however, 25% (2 participants) did achieve the 85% cut-off by post-treatment. On a global measure of insomnia severity, the Insomnia Severity Index (ISI), the treatment group reduced their mean score from 21.7 (indicating severe clinical insomnia) to 14.7, which falls just outside the range of "subthreshold insomnia" in the moderate severity clinical insomnia range. Scores for the treatment as usual group remained consistent in the severe category, with a mean score of 22.9 at baseline and a post-treatment mean of 22.6. Despite this improvement within the treatment group, only 2 participants (14%) achieved a score equal to or below 7 after treatment, indicative of complete remission of insomnia, and the same number achieved a score at or below 11, the recommended cutoff for identifying cases of insomnia in a clinical population (Morin, Belleville, Belanger, & Ivers, 2011). Although the number of participants achieving these cutoffs in the CBT-I group was small, no participants in the treatment as usual group scored below a 17 (moderate severity clinical insomnia).

Sleep onset latency and total sleep time were also improved for the treatment group; although time by group interactions were only marginally significant between groups. This could be due to the small sample size, which may have limited the ability to detect differences that are small but do exist. Sleep onset latency reduced from over one hour at baseline to 34 minutes at post-treatment, while the treatment as usual group showed a slight increase from 55 minutes at baseline to 56 minutes at post-treatment. Total sleep time improved from 5.7 hours at baseline to 6.5 hours at post-treatment in the CBT-I group, and actually decreased in the treatment as usual group, from 6.3 hours to 5.2 hours. Wake time after sleep onset was reduced in both groups at post-treatment, from 54 minutes to 24 minutes in the treatment group, and from 60 minutes to 38 minutes in the treatment as usual group. The fact that no statistically significant

time by group interaction was found for this variable is likely explained by the unanticipated improvement in this parameter in the treatment as usual group.

Effect sizes in this study are comparable to the other studies that have tested CBT-I for insomnia comorbid with psychiatric diagnoses. The most appropriate comparison studies (Dashevsky & Kramer's [1998] study in patients with psychiatric diagnoses who failed to improve with sleep medication; Watanabe et al.'s [2011] study in psychiatric outpatients with depression) did not include a control group or report within-subjects effect sizes; however, the percentages of improvement in sleep parameters for those receiving treatment were comparable to those found in the present study. Although the former study did not use the ISI as a global measure of insomnia, Watanabe et al. (2011) reported 31% improvement in this measure, whereas this sample showed 32% improvement. Dashevsky & Kramer (1998) and Watanabe et al. (2011) reported 61% and 52% improvement in sleep onset latency, respectively, whereas this sample showed 54% improvement. For total sleep time, the studies reported 11% and 15%, respectively, and although no significant differences were found for this parameter for those receiving CBT-I in the present study, 9% improvement was shown between baseline and post-treatment means. The percentage of improvement in wake time after sleep onset was somewhat lower in the present study as compared to the other two; with 50% reduction in this parameter compared to 68% and 63%, respectively. However, sleep efficiency improved 16% and 17% in the comparison but improved by a superior 21% in this study.

Effect sizes in this study were large for most sleep parameters and in line with the general CBT-I literature. Compared to the results from the recent meta-analysis by Okajima et al. (2011), mean effect sizes after CBT-I were larger for several sleep parameters in the present study. Although no significant time by group effect was found for wake time after sleep onset in

this study, which is in contrast to the medium mean effect size of 0.75 in the meta-analysis, the effect size for sleep onset latency, was nearly double that reported by Okajima et al. (2011; Cohen's d of 0.80 [large effect size] versus 0.44 [small effect size]). No significant effect for total sleep time was found across the primary insomnia studies; however, a marginally significant effect was found in the present study, with a large effect size of 0.89. Similarly, for sleep efficiency, the effect size in this study was 1.37, which is large and considerably higher than the mean reported in the meta-analysis ($d = 0.86$). Within subjects effects were similar between this study and the meta-analysis, with Okajima et al. (2011) reporting 0.67, 0.32, 0.70, and 0.89 as mean effect sizes for sleep onset latency, total sleep time, wake time after sleep onset, and sleep efficiency, respectively, compared to 1.15, 0.24, 0.62, and 0.97. The effect size for sleep efficiency was again larger in the present study compared to the 14 studies included in the meta-analysis, suggesting that CBT-I is a potent treatment even among a chronic psychiatric population.

The considerable change in sleep efficiency observed in this sample's CBT-I group is likely the result of the reduced sleep onset latency and wake time after sleep onset that is anticipated with an effective treatment for insomnia. However, in this chronic psychiatric population, other contributing factors are also worth noting. One change that may partially explain the large improvement in sleep efficiency observed in this study is the sizeable reduction in total time in bed observed across treatment for the CBT-I group. Compared to the treatment as usual group, who reported a mean total time in bed of 8.5 hours per night at baseline and a nearly equivalent 8.4 hours at post-treatment, the CBT-I group reported 9.1 hours at baseline and only 7.9 hours per night at post-treatment. This difference of 1.2 hours observed only in the treatment group suggests that spending less time in bed was one behavioral change made during

the course of treatment. Given that CBT-I involves setting regular bed times and rise times in order to restrict the time spent in bed and teaches sleep hygiene concepts including: "Spend only as much time in bed as you need to feel refreshed," this change is to be expected. However, it is worth highlighting in this particular sample of chronic psychiatric patients who may be more likely to spend excessive time in bed as the result of psychiatric symptoms such as anhedonia and fatigue associated with depression or avoidance associated with anxiety. With 84% of participants unemployed or on disability and 80% reporting at least two comorbid medical conditions, excessive time spent in bed is a probable perpetuating factor for chronic insomnia; however, significant changes were made by the participants in this study's treatment condition in regard to this pattern of behavior. The treatment group also reduced their "early morning awakening," or the amount of time spent in bed after awakening in the morning from 1.1 hours at baseline to less than 20 minutes at post-treatment.

The marginally significant time by group interaction for total sleep time may have fallen short of statistical significance as a result of this study's small sample size; however, within the CBT-I treatment group with crossovers included, total sleep time was the only parameter in which baseline to post-treatment differences were nonsignificant. This is not surprising given the fact that many other studies in the literature report no change in total sleep time despite improvement in other parameters (Morin & Benca, 2012). This is likely explained by the fact that CBT-I works to consolidate sleep so that receivers of the treatment are "more efficient" sleepers who spend less time awake while in bed but does not focus on increasing sleep time as the primary goal. CBT-I initially shortens the "sleep window" but slowly expands the time spent in bed as sleep becomes consolidated. With interventions that are brief, such as the five week CBT-I treatment used in the present study, it may take more time than the treatment period for

sleep to be consolidated to the point that the sleep window can be expanded to its optimal length of time.

Durability of CBT-I Treatment Effects: Sleep Variables

This study included a two-month follow-up period, which allowed for an examination of the treatment's durability across time. The fourth hypothesis predicted that improvements in sleep would be maintained at this assessment, demonstrating the durability of the CBT-I treatment effects. Only 8 of 14 participants who completed CBT-I treatment returned complete follow-up data (57%), which limits the ability to draw conclusions about the durability of CBT-I in this sample. Despite this point, results suggest that sleep gains were largely maintained two months after the end of treatment. Specifically, no significant change from post-treatment to follow-up was observed for sleep efficiency, ISI score, sleep onset latency, wake time after sleep onset, or total sleep time. This is consistent with other studies in psychiatric patients; which show treatment durability for sleep parameters at 1 month (Watanabe et al., 2010), 6 months, and 12 months post-treatment (Dashevsky & Kramer, 1998).

The two-month time frame for the follow-up assessment in this study was decided upon due to time constraints of the doctoral student and because a longer follow-up period was anticipated to result in a lower response rate in this complex population. Results of other studies suggest that sleep gains may be durable up to as long as 12 months after treatment; however, it is uncertain whether this would be true for this relatively more severe psychiatric population. Because all participants in the present study were actively taking a medication prescribed to improve sleep, the treatment effects described above also cannot be assumed to be generalizable to all outpatient psychiatric populations. It is possible that without the addition of pharmacotherapy, the participants' significant level of psychiatric illness may have impeded their

ability to participate in or benefit from the CBT-I intervention. Further studies will need to explore this in larger samples.

CBT-I Treatment Effects on Anxiety and Depression

The third hypothesis predicted significant improvements in anxiety and depression following treatment with CBT-I, in line with previously published studies in comparable samples who found that brief CBT for insomnia led to significantly lower scores on measures of depression (Watanabe et al., 2011; Karlin et al., 2013; Taylor et al., 2007) and reduced symptoms of anxiety related to PTSD (DeViva et al., 2005; Germain et al., 2007; Ulmer et al., 2011). Although very few studies have examined the effect of CBT-I on other anxiety disorders, the evidence for improvement in PTSD was seen as suggestive of improved anxiety in patients undergoing treatment with insomnia.

Results from intent-to-treat analyses including 11 participants receiving CBT-I and 8 participants receiving treatment as usual showed no significant differences in anxiety and depression scores between groups. Baseline to post-treatment differences within the combined CBT-I and crossover group were also nonsignificant. In line with the concept of "behavioral activation" (Ferster, 1973), through which activation strategies are used to treat depression by increasing positive reinforcement from the environment, it would seem plausible that the significant reduction in time spent in bed in the CBT-I group would be associated with improvements in depression. It is a common finding in the sleep literature that improvements in sleep have reciprocal effects for depression (Perlis et al., 2006); however, this finding was not demonstrated in the current study. Participants randomized to CBT-I reported a baseline PHQ-9 mean score of 16.2, which corresponds with moderately severe depression, and this score was not changed at the time of post-treatment assessment. In the treatment as usual group, a higher

mean score of 18.5 was reported at baseline, which also falls into the moderately severe depression category, and this score dropped slightly to 16.8 within the same category at post-treatment. Within the combined CBT-I and crossover treatment group (n=14), the baseline mean of 15.2 dropped slightly to 14.1 following treatment, an improvement of 7%.

One possible explanation for the lack of improvement in depression scores is that the depression severity of the sample in this study was higher than in samples tested in other studies. In the study by Taylor et al. (2007), participants had only mild depression on the BDI-II. Similarly, in Perlis et al. (2001), depression scores as measured by the BDI were not in the clinically significant range. In the study by Watanabe et al. (2011), participants entered the study with a moderate mean level of depression, as scored by the Hamilton Depression Scale (HAM-D). The mean depression level of participants in the study by Karlin et al. (2013) was also moderate, according to the Beck Depression Inventory (BDI-II), and in that paper no level of significance was reported to confirm the statistical significance of the six-point pre- to post-treatment score change. With a moderately severe level of depression at baseline, it is possible that this study's sample was severe enough to require a depression-focused behavioral intervention in order to elicit significant changes in depression symptoms. This effectiveness study sought to include as many participants as possible without exclusions to capture as much of a real world sample as possible, and because of this the number and severity of the participants' psychiatric and medical comorbidities may have been considerably greater than those included in the comparison studies. In addition to comorbid conditions, the present study's sample also had a high level of unemployment (84%) and chronic social stressors, which are likely to maintain depression.

Baseline anxiety symptoms were also more severe in the present sample compared to other studies. In a study by Perlis et al. (2001), baseline mean anxiety level as measured by the Beck Anxiety Inventory (BAI) was 7.6, with scores from 0-21 on that measure corresponding to a "very low" level of anxiety. In the current study, mean baseline anxiety on the GAD-7 was 14.5 in the combined sample, which corresponds to moderate symptoms of anxiety. The number and severity of medical and psychiatric comorbidities and chronic social stressors may explain why no improvements were seen in anxiety scores on the GAD-7; however, because the effect of CBT-I on anxiety symptoms has not been a focus of research studies aside from anxiety symptoms in the context of PTSD, it is difficult to have confidence in any proposed explanation. In the present study, both the CBT-I and treatment as usual groups saw slight drops in mean GAD-7 score across treatment. Participants randomized to CBT-I reported a baseline mean score of 13.4, corresponding to a moderate level of anxiety, and at post-treatment the mean score was 12.4 and in the same category. In the treatment as usual group, the mean score at baseline was 16.1 and in the severe anxiety category, whereas at post-treatment the mean score was 14.0 and in the moderate anxiety category. Within the combined CBT-I and crossover treatment group (n=14), the baseline mean of 12.9 dropped only one point to 11.9 following treatment, an improvement of 8%. One of the few CBT-I studies to include measures of anxiety across treatment also reported no significant change after CBT-I, with a baseline score on the State/Trait Anxiety Inventory (STAI) of 39.3 and a post-treatment score of 38.0 (Lichstein et al., 2000). The STAI has a range of scores from 20-80 (with higher scores indicating higher levels of anxiety). Considering the mean score reported and the fact that the sample in Lichstein's (2000) study was not a clinical sample, this may not be a fair comparison to the sample in the present study. Until future studies focus more explicitly on whether anxiety symptoms improve

with CBT-I, it is difficult to propose an explanation for the lack of findings in this sample. It is possible that a more anxiety-specific treatment approach is required to effect significant improvement in anxiety symptoms.

Although anxiety and depression scores were not lowered after treatment with CBT-I despite considerable improvement in sleep, it is possible that these secondary benefits have yet to emerge but will be evident as improvement in sleep is maintained. No significant changes in depression or anxiety score were found at two-month follow-up; however, changes beyond this time point remain unknown.

CBT-I Treatment Effects on Health-Related Quality of Life

The third hypothesis also predicted significant improvements in health-related quality of life following treatment with CBT-I. This hypothesis was based on the findings of previously published studies reporting improvements in the vitality scale (Morgan et al., 2003), physical functioning scale, emotional role limitation, and mental health (Dixon et al., 2006) on the SF-36 after treatment with CBT-I. Another study supported gains on a different measure of quality of life, the WHOQL-BREF (Karlin et al., 2013). Results from intent-to-treat analyses including 11 participants receiving CBT-I and 8 participants receiving treatment as usual showed no significant differences in SF-36 scores between groups, with the exception of a marginally significant time by group interaction for the social functioning scale. Baseline means for the social functioning scale were 36.3 in the CBT-I group and 26.5 in the treatment as usual group (on a scale of 0 - 100, with 100 corresponding to higher quality of life). Following treatment, the CBT-I group mean score improved to 48.9 and the treatment as usual score fell to 21.9. When the CBT-I group was combined with the treatment as usual crossovers (n=4) to create a larger treatment sample, there was a significant difference between baseline and post-treatment scores

on the social functioning scale, with a baseline mean score of 33.9 and a post-treatment mean score of 50.0. One other study found significant improvement in social relationships following treatment with CBT-I, using a different measure of quality of life (WHOQL-BREF; Karlin et al., 2013). Returning to the reduction in total time in bed mentioned earlier, it is possible that reducing excessive time spent in bed in the evenings or the morning could have a positive impact on an individual's social functioning. It is also a possibility that with improved sleep comes improved energy, allowing patients to feel more interested in or more able to seek out social interaction and to participate in social activities.

Durability of CBT-I Treatment Effects: Health-Related Quality of Life

In contrast to the maintained improvement in sleep parameters at two-month follow-up, the improvement in social functioning as measured by the SF-36, was not maintained at this assessment. This suggests that the gain in social functioning was only temporary in this sample of eight treatment completers. It is difficult to explain this finding given that sleep effects were largely maintained at 2-month follow-up; however, fluctuations in other psychiatric, medical, or social factors in these patients' lives may have played a role.

CBT-I Treatment Effects on Sleep Medication Use

The second hypothesis predicted a significant reduction in use of sleep medication in the CBT-I group; however, there was very little change in medication use in either group in this sample. This finding is easily explained by the hesitance to reduce medication that was consistent across participants undergoing CBT-I in this study. Prior to being approached about the study, all CBT-I participants were confirmed by their treating psychiatrist to be safe to taper their sleep medication if they chose to do so; however, this was always brought up as an optional portion of the study that was not a requirement for them to participate. In the first treatment

session, the doctoral student provided brief information about the potential adverse effects of long term use of sleep medications and about the potential benefits of treating insomnia with alternative methods (i.e. behavioral techniques). Each participant was then asked if they were interested in receiving the medication reduction module in that day's session and initiating a taper schedule with assistance and guidance from their treating psychiatrist. In all cases, participants declined this optional session and when offered it again in session 3, they again declined, even though in many cases sleep had improved by this point. Several participants expressed interest in reducing or discontinuing their medication at some point in time but when encouraged to consider doing it in alignment with the beginning of the behavioral techniques and told of the literature supporting this idea, all participants chose against it.

Some hesitance to reduce sleep medication was anticipated. The decision not to only recruit those patients who would commit to reducing their medication during the course of the study was made for fear of limiting the recruitment numbers. However, it is interesting to note that the full sample was uncomfortable with committing to reducing their use. At baseline, 74% of those randomized to CBT-I or treatment as usual (n=19) reported taking sleep medication seven days a week. Within the CBT-I group including crossovers, the percentage of those taking sleep medication on a nightly basis actually increased across treatment, from 64% to 86%. There are a few possible explanations for this result, the first of which may again relate back to the chronic psychiatric and medical issues experienced by the sample. With chronic social stressors, physical symptoms, and psychiatric symptoms to cope with on a daily basis, perhaps the idea of making changes to one's sleep schedule seems doable but trying new behaviors as well as letting go of a medication you have been taking for some time with the belief that it will help make things better for you may seem overwhelming. Similarly, these patients may

experience heightened distress when lying awake in bed unable to sleep, as compared to a sample without such significant psychiatric illness. This distress may fuel maladaptive beliefs about their inability to sleep without sleep medication or about the consequences of chronic insomnia.

The duration of insomnia in this sample may also be a factor. The mean reported duration of insomnia was 17.1 years (205 months) at baseline in the combined sample, which is higher than all durations reported in the meta-analysis referred to previously (Okajima et al., 2011). Of the 10 out of 14 studies that assessed duration of insomnia, 50% reported a mean duration of 10 years or less. With individuals in the present study reporting insomnia symptoms for nearly two decades, on average, despite a mean age of only 50 years (comparable to studies in the meta-analysis), the chronicity of insomnia in this sample may partially explain their dependence on sleep medication.

Duration of insomnia in the present study was also longer than that reported in a study of similar design that examined hypnotic reduction after treatment with CBT-I (Morgan et al., 2003). This study recruited participants with insomnia who were chronic users of sleep medications and gave them the option to reduce their use over the course of CBT-I treatment. Mean age in this study was 63.3 years in the treatment group and mean age at onset of insomnia was 51.3, resulting in a mean duration of insomnia of 12 years. Rate of nightly or "continuous" use of sleep medications was also higher in the present study compared to the study by Morgan et al. (2003); 74% compared to 59.3%, respectively. The chronicity of insomnia is one possible contributing factor; however, another is the fact that participants in the comparison study were patients in primary care clinics and did not have a high rate of psychiatric comorbidities. This may also explain the differences in medication reduction observed between the two studies after

treatment. Participants in the Morgan et al. (2003) study were not required to reduce their medication and sleep needs were prioritized over reducing medication during treatment; however, by 3 months after treatment ended, the rate of continuous users had decreased to 30.3%. Given the similarities between the two study designs, the finding that the rate of nightly users in the present study actually increased after CBT-I points to differences between the two study samples. The chronicity and severity of the psychiatric problems in the present sample may explain why there was lower willingness to reduce the use of sleep medication and a lack of significant change in medication use across treatment with CBT-I. Participants from a non-psychiatric sample may be more motivated to learn to manage their sleep without the chronic use of medication and may possess greater psychological and emotional resources for attempting such a change.

Another possible explanation for the very low rate of interest in the medication reduction module may lie in the prescribing behaviors exhibited and messages sent by the treating psychiatrists. If study participants did not receive the message from their provider that reducing medication would be beneficial to them or that the risks mentioned by the doctoral student are real risks about which they should be concerned, they may not feel comfortable committing to the idea of change. Looking to the results of the provider survey, 4 providers (50%) reported that they spoke with their patients about coming off their medication before referring them to the study more than half the time or always; however, the other 4 (50%) reported doing so half the time or less than half the time. If half the participants enrolled in the study had not heard even a brief recommendation by their provider to consider reducing their sleep medication, it is not surprising that many were hesitant to consider such a change to their medication regimen. Looking further at the survey, when providers were asked if they continue prescribing sleep

medication if requested to by their patient, even if no benefit of the medication is evident, 25% (2 providers) responded with "somewhat agree." Although 4 (50%) responded with "strongly disagree," or "somewhat disagree," it is possible that a culture among some providers to respond to patient requests rather than push to educate about what is in a patient's best interest could affect the way patients respond to a discussion of medication reduction.

If provider beliefs or prescribing behaviors did in fact play a role in this sample's failure to reduce sleep medication, it should be noted that the majority of providers in the clinic were psychiatry residents. It is possible that their beliefs and practices may be shaped by demonstrating the effectiveness of CBT-I in their patients and in the clinic, in general.

Comparison to Previous Study

The fact that our earlier study (Wagley, Rybarczyk, Nay, Danish, & Lund, 2012) was conducted in the same outpatient psychiatric clinic presents an opportunity to compare and contrast sample characteristics and outcomes. The previous study randomized 30 outpatients with insomnia and residual depressive symptoms and other psychiatric comorbidities to either a two-session abbreviated CBT-I intervention or a treatment as usual waitlist control condition. Results showed that those receiving CBT-I exhibited significant improvements on a measure of sleep quality, the Pittsburgh Sleep Quality Index (PSQI), and significant reductions in depression score using the PHQ-9 (Buysse, 1989). Although current use of sleep medication was not an inclusion criterion for the previous study as it was in the current one, 10 of the 30 participants (30%) were taking a prescribed hypnotic at baseline. One interesting difference between this study and the present study is the number of psychiatric medications prescribed. In Wagley et al. (2012)'s sample, the mean number of psychiatric diagnoses was 1.5 and 2.0 in the CBT-I and control groups, respectively, compared to 2.1 in both groups in the present study. Despite these

comparable numbers, participants in the previous study were taking a mean of 1.82 and 1.33 psychotropic medications in the two groups respectively, compared to 2.9 and 3.5, respectively, in the present study. This difference suggests that the sample enrolled in our more recent study may have had a greater general propensity for pharmacological treatments compared to the previous sample. Because participants in the earlier study were self-referred through recruitment procedures carried out in the waiting room, they may have been more inclined to seek out non-medication solutions for their disturbed sleep. An additional point is that only 10% of those randomized to CBT-I in the previous study reported substance abuse, with no participants reporting a history of chemical dependency. This is in contrast to the present study, wherein 36% of those randomized to CBT-I reported a history of drug or alcohol dependence. This illustrates that the sample in the current study may be prone to dependency in general.

Depression levels at baseline were comparable between the two studies, with a mean PHQ-9 score of 16.1 in the CBT-I group in Wagley et al. (2012)'s study and 16.2 in the CBT-I group at baseline in the present study. Neither study showed significant between-group changes in depression after treatment with CBT-I; however, at post-treatment, depression reduced to a PHQ-9 score of 10.9 in the previous study, a significant within-group difference. This can be contrasted with the lack of change in the present study (mean post-treatment PHQ-9 score was 16.2). Given the higher number of psychotropic medications prescribed in the present study's sample, it is possible that they had more complex psychiatric presentations. Other factors may have also contributed to this difference; however, because duration of insomnia, employment status, and number of medical comorbidities were not reported in the previous study, it is impossible to know whether these may have differed between samples.

Study Feasibility

The present study failed to meet its recruitment goals, despite consistent efforts to recruit across an 11-month period. This may be attributable to a lower number of eligible patients than anticipated in the psychiatric clinic wherein this study took place; however, it is also possible that the chronic psychiatric, social, and medical characteristics of the sample resulted in a low number of participants who were willing or able to participate in this research study. The clinic from which participants were recruited was in large part a safety net clinic serving a significant number of underserved groups. Of the 48 patients in the clinic who were identified as likely candidates for the study, 6 declined outright to learn more about the study and 20 who were initially willing to meet with the doctoral student to learn more about the study declined to participate once they learned what was involved. Had those 20 been enrolled in the study, the sample size would have doubled. Of those who provided a reason for declining to participate (n=15), as summarized in Table 4, 33% (five patients) reported that it would be too difficult for them to make it to study visits and 27% (four participants) declined due to chronic life stressors. Other patients' reasons included low reported stress about their sleep problem (27%; 4 patients), perhaps in comparison to the other chronic stressors that they had in their lives; concerns about the treatment not working (7%; 1 patient); and concerns about the low compensation for participation (7%; 1 patient).

When asked to respond to the statement that behavioral strategies are too demanding for most patients in the clinic, only 25% (2 providers) of the treating psychiatrists responded with "somewhat agree," and none responded with "strongly agree." In fact, 37% (3 providers) responded with "somewhat disagree." There is no clear agreement among the providers of these patients about the appropriateness of CBT-I in this psychiatric sample; however, a number of those who dropped out of treatment and provided a reason for doing so (n=4) reported on the

difficulty of keeping up with treatment guidelines and (25%; 1 participant) and the burden of other life stressors and medical issues (25%; 1 participant).

Although 19 participants did complete the study, 26 declined to participate and of those who enrolled in the study, 4 participants dropped out before completing it. Another participant completed the treatment as usual period but dropped out of the CBT-I treatment after crossing over due to a reported inability to keep up with study guidelines. Given that only 39% of those invited to participate and 82% of those who enrolled actually completed the study, it may be that the present study design was too demanding or difficult for this psychiatric population. Perhaps if the number of sessions had been reduced or the time frame relaxed, more participants would have completed the study or been willing to enroll. Alternatively, had a higher level of support been provided from each patient's treating psychiatrist, perhaps this would have facilitated greater recruitment or lower attrition. Greater support provided by the treating psychiatrists may also have been necessary in this population in order to increase the willingness of participants to taper their sleep medication as part of the study.

Study Limitations and Directions for Future Research

Ideally, this study would employ both objective (polysomnography, actigraphy) and self-report measures (sleep diaries) of sleep; however, due to costs and feasibility concerns, for this doctoral dissertation study only self-report measures were used. The use of only self-report measures is common in the insomnia literature (Smith et al., 2005); however, all outcome data was subject to bias by the participant reporting the information.

A primary limitation in the present study was the lower than expected recruitment numbers, which led to a small sample size. This may have reduced the ability to detect statistically significant treatment effects. One contributing factor to this may be the study design,

which may have involved a schedule of study visits and time frame that was too demanding for this sample with chronic psychiatric and medical issues as well as ongoing social stressors. Future studies in psychiatric populations may consider reducing the number of sessions to two or three and relaxing the time frame across which participants are expected to make trips to meet in person with the clinician. In this study, two of the five sessions were phone visits in an attempt to reduce demand on participants; however, a significant number of potential participants declined to participate because of the difficulty of finding transportation to the clinic for three study visits.

Changes to the study design could also have improved the rate of medication reduction in this study. Rather than the medication reduction module being an optional treatment component, perhaps if the full module of information was presented to participants in session 1 rather than just brief education on the subject, there would have been an increase in willingness to taper. Additionally, by partnering with the treating psychiatrist to a greater extent and having this provider present a certain amount of information to every patient about the benefits of medication reduction, it is possible that more participants would have been willing to consider initiating a taper schedule as they began treatment. These are considerations to be taken into account for future studies.

Because this study included only participants who were taking sleep medication at the time of the study, the results of this study cannot be generalized to all outpatient psychiatric populations. It may be that the combination treatment of CBT-I and pharmacotherapy for sleep results in improvements in insomnia but that CBT-I alone would not have the same effect. Future studies should consider adding a CBT-I group without the addition of sleep medication as well as a true control group, including those who receive no medication or CBT-I.

Another limitation is that participants were not formally screened for sleep apnea or other sleep disorders as part of the enrollment process. The doctoral student confirmed that all patients did not have exclusionary diagnoses in their medical record prior to approaching them about the study; however, it became known following the study that one participant was later diagnosed with sleep apnea and began treatment with CPAP. If more than one participant had an undiagnosed sleep disorder other than insomnia at the time of the study, this may explain why not all participants experienced the same level of symptom improvement or why some treatment effects were only marginally significant between groups.

A final limitation is that the same individual was the clinician for all study participants as well as the one responsible for the study design, recruitment, and assessment in this study. This leaves room for demand effects to influence participant responses and for characteristics specific to this single clinician to be partially responsible for the study findings.

Future studies should aim for a larger design involving multiple study personnel to increase recruitment efforts, separate assessment responsibilities from the provision of treatment, and include multiple therapists to control for clinician effects. A more orchestrated effort between CBT-I clinicians and treating psychiatrists may also be possible with a larger study staff, which could lead to an increase in participant willingness to taper sleep medications. With a larger sample size, statistical significance for treatment effects should be easier to achieve and with a larger sample it may also be possible to explore differences between subsets of participants, such as those who have been on sleep medications for a very long time compared to those who are relatively new users.

Implications of the Present Study

The present study provides some initial support for the effectiveness of cognitive behavioral therapy for insomnia (CBT-I) in a sample of chronic users of sleep medication in an outpatient psychiatry clinic. Results show that five sessions of individually administered CBT-I led to significant improvements in global insomnia severity, sleep efficiency, sleep onset latency, and wake time after sleep onset, and that gains were maintained two months following the end of treatment. Effect sizes were generally large, illustrating the potency of CBT-I in a psychiatric sample. Additionally, treatment with CBT-I led to improvement in social functioning on a measure of quality of life at post-treatment. CBT-I did not lead to improvements in anxiety or depression, suggesting that in this complex, chronic psychiatric population either more time or more anxiety- or depression-focused treatment is required to elicit improvements in the symptoms of these mental health conditions. To the best of the author's knowledge, this study is the first randomized controlled trial of CBT-I in a hypnotic-dependent outpatient psychiatric sample that included a diverse range of psychiatric comorbidities. By including a varied sample of complex psychiatric patients it serves as an initial effectiveness study for the psychiatric outpatient setting, where treatment refractory insomnia is prevalent. The study findings provide support for a larger randomized effectiveness trial to be conducted in the future in a similar sample.

The present study also provides information about study feasibility that may be useful to those designing and carrying out future studies. Five sessions of CBT-I across five weeks may appear demanding to psychiatric patients with multiple comorbidities and chronic social stressors and significant active guidance from their treating psychiatrist may be necessary for them to consider reducing their use of sleep medications in the context of behavioral treatment.

This study shows that although many patients in an outpatient psychiatric clinic may find that CBT-I or committing to participating in a research study is too demanding given the burden of their other stressors, those who follow through with treatment do experience benefits in sleep and quality of life in the realm of social functioning. Although depression or anxiety were not improved in this sample within the time frame of the study, results show that sleep does improve after CBT-I in those with significant psychiatric illness, providing support for the use of this behavioral treatment in complex psychiatric populations. Given that so few studies have tested CBT-I interventions in real world samples without excluding those with significant psychiatric comorbidities, this study begins to address a gap in the insomnia literature by carrying out this endeavor with the methodological rigor of a treatment as usual control group. Future research should further address this gap by testing CBT-I interventions in larger real-world samples, including a range of psychiatric diagnoses and severity levels. Other studies should focus on identifying factors that predict which “real-world” psychiatric patients may be most likely to undergo behavioral treatment for insomnia as well as which patients are most likely to benefit from CBT-I.

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Appendix A
Study Measures

Participant Information

Telephone number: _____(home) _____(cell)

At which number do you prefer we call you? _____

Mailing address: _____

Age: _____ **Gender (Circle one):** Male Female

Marital Status (Circle one): Married Single
Divorced/Widowed Living with Partner

Ethnicity (Circle one): White, not Hispanic Black, not Hispanic Hispanic/Black
Hispanic, White American Indian/Alaskan Asian
Pacific Islander Unknown/Other

Education (Circle one): Some High School High School Some College
College Graduate School

Height: _____ feet _____ inches **Weight:** _____ lbs

Please list current health conditions:

- | | |
|----------|----------|
| 1) _____ | 5) _____ |
| 2) _____ | 6) _____ |
| 3) _____ | 7) _____ |
| 4) _____ | 8) _____ |

Please list current mental health conditions:

- 1) _____ 3) _____
2) _____ 4) _____

Please list all medications currently taking including sleep-related/non-sleep related medications:

- | <i>Medication</i> | <i>Dosage</i> |
|-------------------|---------------|
| 1) _____ | _____ |
| 2) _____ | _____ |
| 3) _____ | _____ |
| 4) _____ | _____ |
| 5) _____ | _____ |
| 6) _____ | _____ |
| 7) _____ | _____ |
| 8) _____ | _____ |
| 9) _____ | _____ |
| 10) _____ | _____ |

An episode of insomnia is defined as:

1. An inability to fall asleep within 30 minutes
AND/OR
2. Awakening during the night (after falling asleep) for a total of 60 minutes or more
AND/OR
3. Achieving less than 6.5 hours of sleep per night

For how long have you experienced the following symptoms?:

- a) At least three episodes of disturbed sleep per week
AND
- b) daytime consequences such as fatigue, irritability, or difficulty concentrating?

_____ years _____ months

Approximately when did your insomnia begin?

(MM/DD/YYYY): _____

For how long have you been taking hypnotic medication for sleep?

_____ years _____ months

When did you first begin taking hypnotic medication for sleep?

(MM/DD/YYYY): _____

SLEEP DIARY

Date:

10/9

Example

Name: _____

1. Yesterday, I napped from ___ to ___ (note the times of all naps).

*1:50 to
2:30 p.m.*

2. Yesterday, I took ___ mg of medication and/or ___ oz of ___ alcohol as a sleep aid.

*Ambien
5 mg*

3. Last night, I went to bed and turned the lights off at ___ o'clock.

11:15

4. After turning the lights out, I fell asleep in ___ minutes.

40 min

5. My sleep was interrupted ___ times (specify number of nighttime awakenings).

3

6. My sleep was interrupted for ___ minutes (specify duration of each awakening).

*10
5
45*

7. This morning, I woke up at ___ o'clock (note time of last awakening).

6:15

8. This morning, I got out of bed at ___ o'clock (specify the time).

6:40

<i>1:50 to 2:30 p.m.</i>							
<i>Ambien 5 mg</i>							
<i>11:15</i>							
<i>40 min</i>							
<i>3</i>							
<i>10 5 45</i>							
<i>6:15</i>							
<i>6:40</i>							

Insomnia Severity Index

The Insomnia Severity Index has seven questions. The seven answers are added up to get a total score. When you have your total score, look at the 'Guidelines for Scoring/Interpretation' below to see where your sleep difficulty fits.

For each question, please CIRCLE the number that best describes your answer.

Please rate the *CURRENT* (i.e. *LAST 2 WEEKS*) *SEVERITY* of your insomnia problem(s).

Insomnia Problem	None	Mild	Moderate	Severe	Very Severe
1. Difficulty falling asleep					
2. Difficulty staying asleep					
3. Problems waking up too early					

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?

Very Satisfied Satisfied Moderately Satisfied Dissatisfied Very Dissatisfied
 0 1 2 3 4

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?

Not at all Noticeable A Little Somewhat Much Very Much Noticeable
 0 1 2 3 4

6. How WORRIED/DISTRESSED are you about your current sleep problem?

Not at all Worried A Little Somewhat Much Very Much Worried
 0 1 2 3 4

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

Not at all Interfering A Little Somewhat Much Very Much Interfering
 0 1 2 3 4

Guidelines for Scoring/Interpretation:

Add the scores for all seven items (questions 1 + 2 + 3 + 4 + 5 + 6 + 7) = _____ your total score

Total score categories:

0–7 = No clinically significant insomnia

8–14 = Subthreshold insomnia

15–21 = Clinical insomnia (moderate severity)

22–28 = Clinical insomnia (severe)

Used via courtesy of www.myhealth.va.gov with permission from Charles M. Morin, Ph.D., Université Laval

GAD-7

Over the last 2 weeks, how often have you been bothered by the following problems? <i>(Use "X" to indicate your answer)</i>	Not at all	Several Days	More than half the days	Nearly every day
1. Feeling nervous, anxious or on edge	0	1	2	3
2. Not being able to stop or control worry	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it is hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3

2. If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people? (PLEASE CIRCLE)

Not difficult at all

Somewhat difficult

Very difficult

Extremely difficult

PHQ-9

Over the last 2 weeks, how often have you been bothered by any of the following problems? <i>(Use "X" to indicate your answer)</i>	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself – or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed? Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3

2. If you checked off any problem on this questionnaire so far, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people? (PLEASE CIRCLE)

Not difficult at all Somewhat difficult Very difficult Extremely difficult

SF-36(tm) Health Survey

Instructions for completing the questionnaire: Please answer every question. Some questions may look like others, but each one is different. Please take the time to read and answer each question carefully by filling in the bubble that best represents your response.

Patient Name: _____

SSN#: _____ Date _____

Person helping to complete this form: _____

1. In general, would you say your health is:

- Excellent
- Very good
- Good
- Fair
- Poor

2. Compared to one year ago, how would you rate your health in general now?

- Much better now than a year ago
- Somewhat better now than a year ago
- About the same as one year ago
- Somewhat worse now than one year ago
- Much worse now than one year ago

3. The following items are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?

a. Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

b. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf?

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

c. Lifting or carrying groceries.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

d. Climbing several flights of stairs.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

e. Climbing one flight of stairs.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

f. Bending, kneeling or stooping.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

g. Walking more than one mile.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

h. Walking several blocks.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

i. Walking one block.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

j. Bathing or dressing yourself.

- Yes, limited a lot.
- Yes, limited a little.
- No, not limited at all.

4. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

a. Cut down the amount of time you spent on work or other activities?

- Yes No

b. Accomplished less than you would like?

- Yes No

c. Were limited in the kind of work or other activities

- Yes No

d. Had difficulty performing the work or other activities (for example, it took extra time)

Yes No

5. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

a. Cut down the amount of time you spent on work or other activities?

Yes No

b. Accomplished less than you would like

Yes No

c. Didn't do work or other activities as carefully as usual

Yes No

6. During the past 4 weeks, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbors, or groups?

- Not at all
- Slightly
- Moderately
- Quite a bit
- Extremely

7. How much bodily pain have you had during the past 4 weeks?

- None
- Very Mild
- Mild
- Moderate
- Severe
- Very Severe

8. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

- Not at all
- Slightly
- Moderately
- Quite a bit
- Extremely

9. These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the past 4 weeks.

a. did you feel full of pep?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

b. have you been a very nervous person?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

c. have you felt so down in the dumps nothing could cheer you up?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

d. have you felt calm and peaceful?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

e. did you have a lot of energy?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

f. have you felt downhearted and blue?

- All of the time

- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

g. did you feel worn out?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

h. have you been a happy person?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

i. did you feel tired?

- All of the time
- Most of the time
- A good bit of the time
- Some of the time
- A little of the time
- None of the time

10. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting friends, relatives, etc.)?

- All of the time
- Most of the time
- Some of the time
- A little of the time
- None of the time

11. How TRUE or FALSE is each of the following statements for you?

a. I seem to get sick a little easier than other people

- Definitely true
- Mostly true
- Don't know
- Mostly false

Definitely false

b. I am as healthy as anybody I know

- Definitely true
- Mostly true
- Don't know
- Mostly false
- Definitely false

c. I expect my health to get worse

- Definitely true
- Mostly true
- Don't know
- Mostly false
- Definitely false

d. My health is excellent

- Definitely true
- Mostly true
- Don't know
- Mostly false
- Definitely false

Provider Survey

1. Cognitive-Behavioral Therapy for Insomnia (CBT-I) is an effective way to treat insomnia.

- Strongly Disagree
- Somewhat Disagree
- Neither Agree nor Disagree
- Somewhat Agree
- Strongly Agree

2. Behavioral strategies for treating insomnia (beyond basic sleep hygiene) are too demanding for most of my psychiatric patients.

- Strongly Disagree
- Somewhat Disagree
- Neither Agree nor Disagree
- Somewhat Agree
- Strongly Agree

3. When treating patients with chronic insomnia, I commonly prescribe sleep medications for longer than one month.

- Strongly Disagree
- Somewhat Disagree
- Neither Agree nor Disagree
- Somewhat Agree
- Strongly Agree

4. In general, medication for sleep is more effective in the long term than are behavioral treatments such as CBT-I.

- Strongly Disagree
- Somewhat Disagree
- Neither Agree nor Disagree
- Somewhat Agree

Strongly Agree

5. Chronic use of medications (e.g. sedative-hypnotics) for sleep is problematic and should be avoided at all costs.

Strongly Disagree

Somewhat Disagree

Neither Agree nor Disagree

Somewhat Agree

Strongly Agree

6. If a patient with insomnia is seeing little benefit from his/her sleep medication but asks me to continue prescribing it, I often do so.

Strongly Disagree

Somewhat Disagree

Neither Agree nor Disagree

Somewhat Agree

Strongly Agree

7. Tapering my patients off their sleep medication leads to problematic side effects.

Strongly Disagree

Somewhat Disagree

Neither Agree nor Disagree

Somewhat Agree

Strongly Agree

8. Sleep medications are appropriate for short term use but should not be prescribed for chronic insomnia.

Strongly Disagree

Somewhat Disagree

Neither Agree nor Disagree

Somewhat Agree

Strongly Agree

9. Most of my patients with insomnia are not interested in behavioral treatments (such as CBT-I) and prefer medication.

Strongly Disagree

Somewhat Disagree

Neither Agree nor Disagree

Somewhat Agree

Strongly Agree

10. When discussing treatment options with patients who have insomnia, about how often do you mention behavioral treatments (such as CBT-I) in addition to medication?

Never

Less Than Half the Time

About Half the Time

More Than Half the Time

Always

N/A

11. If you referred patients to the CBT for Insomnia research study, about how often did you speak with them beforehand about coming off their sleep medication?

Never

Less Than Half the Time

About Half the Time

More Than Half the Time

Always

N/A

Vita

Hannah Lund Taylor was born in Minnesota in 1983 and she is an American citizen. She graduated from Bates College in 2006 with a bachelor's degree in Psychology. After working for two years as a research coordinator at the Bipolar Clinic and Research Program at Massachusetts General Hospital (MGH) in Boston, Massachusetts, she enrolled in the doctoral program in clinical psychology at Virginia Commonwealth University (VCU) in Richmond, Virginia. Hannah completed her master's degree in 2011 and anticipates graduating with her Ph.D. in Clinical Psychology in 2014. She is currently completing her internship at Warren Alpert Medical School of Brown University.