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THE DISCREPANCY BETWEEN SUBJECTIVE AND OBJECTIVE MEASURES OF SLEEP  
IN OLDER ADULTS RECEIVING CBT FOR COMORBID INSOMNIA

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science  
at Virginia Commonwealth University.

by

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## Abstract

### THE DISCREPANCY BETWEEN SUBJECTIVE AND OBJECTIVE MEASURES OF SLEEP IN OLDER ADULTS RECEIVING CBT FOR COMORBID INSOMNIA

By Hannah G. Lund, B.A.

A thesis submitted in partial fulfillment of the requirements for the degree Master of Science at Virginia Commonwealth University.

Virginia Commonwealth University, 2011

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Clinical research on insomnia has observed that many individuals with this sleep disorder exhibit a significant discrepancy between their subjective reports of symptom severity and objective measures of the same parameters. This study sought to more closely examine this discrepancy by comparing sleep diary estimates with polysomnography data in a population of 60 older adults with comorbid insomnia. Consistent with previous research, results show that participants significantly underestimated sleep efficiency and total sleep time and significantly overestimated sleep onset latency. Participants receiving CBT-I exhibited significantly reduced discrepancy at post-treatment, particularly with regard to sleep latency, compared to those in a Stress Management and Wellness treatment control group. This suggests that the treatment effects observed as a result of CBT can be partly explained by improvements in the accuracy of sleep estimation. Additionally, high discrepancy at baseline was found to be a significant predictor of positive treatment outcome, indicating that sleep misperception is a potential factor in a favorable treatment response to CBT.

## The discrepancy between subjective and objective measures of sleep in older adults receiving CBT for comorbid insomnia

Insomnia is a common sleep disorder characterized by a difficulty in initiating, maintaining, or obtaining sufficient good quality sleep that causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. Prevalence estimates vary greatly but suggest that rates of insomnia in the general population may be as high as 10% to 15%, with 20% to 40% reporting some symptoms of disturbed sleep (Morin, LeBlanc, Daley, Gregoire, & Merette, 2006; Kiley, 1999). Although sleep complaints of this nature are fairly common in most primary care settings and are often regarded as ancillary to other medical conditions, insomnia is a distinct disorder with serious implications that may negatively impact health and impair daily functioning (Gallup Organization, 1995; Dement & Pelayo, 1997; Ohayon, 1997; Simon & VonKorff, 1997). Chronic insufficient sleep has the potential to exacerbate existing health problems and may decrease quality of life in those whom it afflicts (Leger, Guilleminault, Bader, Levy, & Paillard, 2002). Further evidence suggests that symptoms of insomnia may additionally contribute to increased health care costs and utilization, mental illness, work-related accidents, work absenteeism and reduced productivity, increased drug and alcohol use, and higher risk for mortality (Chang, Ford, & Mead, 1997; Livingston, Blizard & Mann, 1993; Vollrath, Wicki, & Angst, 1989; Kripke et al., 1998; Johnson & Spinweber, 1983; Edinger & Wohlgemuth, 1999).

Insomnia that occurs independent of other medical conditions, most often referred to as *Primary Insomnia*, can be difficult to diagnose and treat, however these processes may be complicated further when symptoms of disturbed sleep accompany other medical or psychiatric conditions. Formerly labeled *Secondary Insomnia* due to the original thought that



insomnia symptoms may occur as a direct consequence of or secondary to a separate medical or psychiatric disorder, a 2005 National Institutes of Health (NIH) State of the Science Conference statement suggested that this set of symptoms may be more accurately described by the term *Comorbid Insomnia* (NIH, 2005). Stepanski and Rybarczyk (2006) suggest that the idea of cause and effect implied by the term Secondary Insomnia may be too simple to accurately reflect how insomnia relates to other existing conditions. Although the prevailing treatment approach for insomnia comorbid with other conditions had been to focus the treatment on the primary medical or psychiatric problem, in the past decade treatment guidelines have shifted to behavioral treatment of insomnia as the first step in treatment, whether or not other conditions are present (Stepanski & Rybarczyk, 2006). This demonstrates the growing recognition of insomnia as a distinct condition with serious implications for the physical and mental health of those who suffer from it, especially those with comorbid diagnoses.

Insomnia is of particular concern in the older adult population due to the fact that it is most common, severe, and impairing in adults over 60 years of age (Lichstein, Durrence, & Riedel, 2004; Morgan, 2000; Morphy, Dunn, & Lewis, 2007; Stewart, Besset, & Bebbington, 2006). Insomnia in older adults occurs more frequently in individuals with chronic medical conditions than in those who fall into the healthier segment of this population (Prinz, Williams, & Vitiello, 1990; Williams, Vitiello, Ries, Bokan, & Prinz, 1988). It is estimated that comorbid insomnia may account for approximately 70% of insomnia cases in older adults (Lichstein, 2000). Several common medical illnesses known to be connected with the aging process, including cardiovascular disease, pulmonary disease, and chronic pain conditions caused primarily by arthritis, have been strongly linked to high rates of insomnia

(Jordan et al., 2000; Katz & McHorney, 1998).

One observation in the clinical research that has emerged as a common characteristic of insomnia is the lack of a consistent relationship between subjective reports of sleep and objective measures such as polysomnography. Numerous studies have found that a proportion of insomniacs report sleep problems without exhibiting objective proof of insomnia (Carskadon et al., 1976; Bixler, Kales, & Leo, 1973; Jacobs, Reynolds, Kupfer, Lovin, & Ehrenpreis, 1998; Frankel, Coursey, Buchbinder, & Snyder, 1976; Edinger & Fins, 1995; Rosa & Bonnet, 2000; Venable, Aikens, Tadimeti, Caruana-Montaldo, & Mendelson, 1999; Chambers & Keller, 1993; Mercer, Bootzin, & Lack, 2002). For others, objective measures do reflect problematic sleep but not to the extent that subjective reports suggest. This significant mismatch between individuals' subjective reports of insomnia symptoms and the objective measures of the specific sleep parameters used to identify them is a phenomenon that complicates the question of how to best define and measure insomnia and highlights the complexity and multidimensional nature of insomnia as a disorder. This discrepancy also poses questions about how best to differentially treat insomnia in individuals who do versus do not report an exaggerated subjective impression of their symptoms.

These issues underscore the need for additional research that focuses specifically on the subjective/objective discrepancy in insomnia. A better understanding of this phenomenon may inform our conceptualization and definition of insomnia and may increase our knowledge of what contributes to the development and maintenance of the disorder. These gains would inform insomnia research by assessing the accuracy of subjective measures of sleep, which are solely relied upon to determine criteria for an insomnia

diagnosis in 90% of clinical trials (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). Additionally, such gains would inform clinical treatment of insomnia by providing insight into what should be the primary targets for treatment. Cognitive Behavioral Therapy for Insomnia (CBT-I) has been shown to reduce dysfunctional beliefs and attitudes about sleep as measured by the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale, however the impact of CBT-I on the accuracy of subjective symptom reporting or the discrepancy between subjective and objective measures has not been closely examined (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). Furthermore, it is unknown whether the size of the subjective/objective discrepancy is a predictor of treatment outcome.

Despite the high prevalence of insomnia in the older adult population, concentrated in those with comorbid conditions, few studies to date have examined the subjective/objective discrepancy in this specific population. Research has identified this population to be at particular risk for making inaccurate subjective impressions of their insomnia symptoms due to a higher endorsement of unrealistic sleep expectations or worry over the effect of compromised sleep on daytime functioning (Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). Additional research is needed to gain a clearer understanding of how these cognitions may relate to the discrepancy between subjective and objective measures of insomnia. Identifying cognitive and other correlates of this objective/subjective discrepancy among older adults may improve our understanding of the phenomenon.

The purpose of the proposed study was to examine the discrepancy between subjective and objective measures of insomnia in a sample of 92 older adults who participated in a large randomized controlled intervention study for insomnia that co-occurred with a diagnosis of osteoarthritis (OA), coronary artery disease (CAD) or/and

chronic obstructive pulmonary disease (COPD). Statistical analysis was used to determine whether there exists a significant discrepancy between subjective data collected using participant logs and objective data collected using polysomnography and to examine how this discrepancy might change following eight weeks of treatment with CBT-I as compared to a Stress Management and Wellness (SMW) treatment. Analyses were also conducted to explore possible predictors of the subjective/objective discrepancy (specifically, the endorsement of dysfunctional beliefs and attitudes about sleep and mood or anxiety scores) and to examine the magnitude of the discrepancy as a predictor of treatment outcome.

A secondary purpose of this study was to examine how individuals with insomnia behave when undergoing polysomnographic assessment in the home. Research has identified that nocturnal recording site may impact sleep patterns (Edinger et al., 1997). Additionally, PSG equipment may produce sensory intrusions for some individuals. The potential for sleep to change when one is undergoing polysomnographic assessment in the home has implications for assessing the discrepancy between subjective and objective measures of insomnia and for making conclusions from objective data collected outside the lab. Sleep patterns on nights when participants were undergoing polysomnographic recording in the home were compared to sleep patterns on nights of typical sleep, without the presence of recording equipment, to determine if individuals sleep differently when undergoing PSG assessment.

In the following sections, a review of the relevant insomnia literature will be presented, focusing on studies that have examined or addressed the discrepancy between subjective and objective measures. Thereafter, the following aims will be discussed: 1) to examine the discrepancy between subjective and objective measures of insomnia, which has

been established in adult insomnia populations, in a special population of older adults with comorbid insomnia, 2) to observe whether or not the magnitude of the discrepancy between subjective and objective measures is diminished post-treatment with Cognitive-Behavioral Therapy for Insomnia (CBT-I) compared to those receiving a placebo treatment, Stress Management and Wellness (SMW) therapy, 3) to examine whether scores on the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale, Geriatric Depression Scale (GDS), Profile of Mood States (POMS) scale, and POMS tension subscale predict the size of the discrepancy between subjective and objective measures at pre-treatment, 4) to examine whether change in DBAS score from pre- to post-treatment with CBT-I will predict change in the magnitude of the subjective/objective discrepancy, 5) to examine if the magnitude of the subjective/objective discrepancy prior to treatment with CBT-I is a significant predictor of treatment response, 6) to examine whether change in the discrepancy from pre- to post-treatment with CBT-I is a significant predictor of treatment response, and 7) to examine if participants behave differently in regard to their sleep when undergoing polysomnographic assessment in the home.

### **Review of the Literature**

**Defining Insomnia.** Prevalence rates of insomnia vary widely, with some sources reporting a rate of 2% and others reporting as high 42.5% (Morgan, 2000; Morin, 1993). One likely contributor to this wide variability is the lack of standardized quantitative criteria for defining insomnia (Lichstein et al., 2003). Studies in the field of insomnia research show marked inconsistency in the parameters they use to inform their definition of the disorder, with many relying solely upon a yes or no answer from participants to the question: “Do you have insomnia?” Other studies require a minimum duration or severity of subjective

symptoms, however these standards are inconsistent between studies and markedly few incorporate objective measurements into their diagnostic criteria (Lichstein, 2003). A study by Lichstein and colleagues (2003) reviewed the criteria for defining insomnia in all psychology clinical trials for insomnia during the past two decades and found that fifty-five studies that specified their criteria (90.2%) relied solely on diaries and only five incorporated both diary and polysomnographic data into their definition. The failure to establish consistent criteria for insomnia between studies may lead to unreliable reports surrounding the efficacy of treatments and may contribute to inaccurate epidemiological estimates of insomnia prevalence (Lichstein et al., 2003). This issue is compounded by the fact that the three manuals for diagnosing insomnia, the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association [APA], 2000), the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Second Edition (ICD-10; World Health Organization, 2004), and the International Classification of Sleep Disorders, 2<sup>nd</sup> Edition: Diagnostic and Coding Manual (ICSD2; American Academy of Sleep Medicine, 2005), each report different diagnostic criteria. This, again, brings attention to the marked inconsistencies in the definition of insomnia and complicates the manuals' utility for research and clinical care. The discrepancy between subjective and objective measures of insomnia further complicates this issue. If individuals report subjective symptoms of insomnia without objective corroboration, the question emerges of whether they really have insomnia or if a psychological disorder of perception is to blame for their experience. Without a better understanding of the subjective/objective discrepancy, prevalence rates and efficacy studies of treatments for insomnia will continue to be inconsistent and potentially inaccurate.

**Discrepancy/Concordance Research.** Much of the literature on the discrepancy between subjective and objective measures of insomnia suggests that insomniacs report more delayed sleep onset latency, more frequent nocturnal awakenings, and lower total sleep time than what objective parameters show (Carskadon et al., 1976; Frankel et al., 1976). Other studies, however, have shown that there is wide variability in how accurately insomniacs perceive their sleep (Libman, Creti, Levy, Brender, & Fichten, 1997; Edinger & Fins, 1995; Vanable et al., 1999). Edinger and Fins (1995) found that while most insomniacs underestimated the amount of sleep they were getting, a substantial number produced estimates that were congruent with PSG, and nearly 20% actually overestimated total sleep time. Carskadon and colleagues (1976) similarly reported that in their study, most patients underreported sleep time and overestimated sleep latency, but that 20% made congruent estimates of TST and 12% made substantial overestimates. These findings suggest that subjective reports of insomnia are variable between individuals and show that many insomniacs are inaccurate in their estimates of sleep disturbance.

The presence of a subjective complaint of insomnia in individuals who exhibit objectively measured adequate sleep has been labeled by some as “subjective insomnia” or “paradoxical insomnia,” and in some cases has been suggested to be a transitional state between normal sleep and objectively verifiable insomnia (American Academy of Sleep Medicine [AASM], 2005; Dorsey & Bootzin, 1997; Salin-Pascual, Roehrs, Merlotti, Zorick, & Roth, 1992). Others have debated whether insomnia should be divided into two subtypes; “subjective” and “objective,” to indicate the measurement method by which they meet criteria for insomnia and suggest it is possible that these subtypes may differ in their EEG sleep pathology, etiologies, or courses over time (Edinger & Krystal, 2003). The term “sleep

state misperception” has also been used to classify individuals who complain of marked insomnia without objective corroboration and this is considered a diagnosable disorder by the International Classification of Sleep Disorders (ICSD) that is endorsed by an estimated 10-25% of insomnia patients (Vanable et al., 1999). The ICSD suggests that the disorder develops as a result of a marked over concern with the inability to sleep that leads to a vicious cycle of poor sleep (AASM, 2005). Although there is debate as to whether the mismatch between subjective reports and objective measures of insomnia indicates a distinct subtype of insomnia, this phenomenon is frequently discussed as a generic characteristic of insomnia. Some view sleep state misperception as the extreme end of a continuum of insomnia, with pure insomnia (both subjective and objective agreement) as the other extreme; however, rates of pure sleep state misperception, distinguished by a subjective complaint without any suggestion of insomnia by objective measures, are reported to be fairly low (Reynolds, Kupfer, Buysse, Coble, & Yeager, 1991; Edinger & Fins, 1995). Most studies suggest that patients typically fall somewhere in between the two extreme endpoints, showing some objective corroboration of insomnia symptoms but reporting a disproportionately severe subjective complaint.

Research on the discrepancy between subjective and objective measures of insomnia has suggested multiple variables that may help to explain why the discrepancy exists. Several studies examining personality correlates have found that overreporting of insomnia symptoms is associated with greater neuroticism and hypochondriasis on measures such as the Minnesota Multiphasic Personality Inventory (MMPI; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989; Corsey, Buchsbaum, & Frankel, 1975; Kales, Caldwell, Preston, Healey, & Kales, 1976; Tan, Kales, Kales, Soldatos, & Bixler, 1984). These studies



provide evidence that personality traits may contribute to the symptom exaggeration exhibited by some individuals with insomnia. Over-activation of EEG activity, or brain activity during sleep, has also been identified as a correlate of exaggerated subjective symptoms (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Studies have established that insomniacs are physiologically hyperaroused prior to sleep onset and/or during sleep, which may interfere with their ability to initiate or maintain sleep and has been shown to induce intrusive cognitions around the time of sleep onset (Monroe, 1967; Freedman & Sattler, 1982; Lichstein & Rosenthal, 1980; Mitchell & White, 1977). Other studies using newer methods of quantifying sleep EEG have provided evidence that diminished delta and greater alpha, sigma, and beta EEG spectral power in NREM sleep may be an objective physiologic correlate of subjective sleep complaints in individuals whose traditionally scored EEG profiles do not corroborate these complaints (Krystal, Edinger, Wohlgemuth, & Marsh, 2002). It is possible that some individuals with insomnia possess physiological characteristics that make them prone to overestimate their sleep disturbances; however this suggestion is still under investigation.

Mood and anxiety factors have also been found to relate to the subjective/objective discrepancy. Rosa and Bonnet (2000) found that individuals complaining of insomnia without objective corroboration showed higher anxiety and more negative mood compared with those who did not report insomnia complaints. The authors suggest that their symptoms may be the result of a state of tension and anxious hyperarousal rather than sleep debt and imply that treatment for insomnia should be focused on reducing this anxiety and hyperarousal rather than on objective sleep. In a study by Edinger and colleagues (2000), individuals complaining of insomnia without objective corroboration reported more

depressed mood, anxiety, and dysfunctional sleep-related cognitions as compared to those whose objective measures agreed with their subjective complaints.

Sleep-related cognitions have been the focus of related studies, whose findings have established that insomniacs report more dysfunctional sleep-related cognitions than non-complaining individuals (Morin, 1993; Edinger et al., 2000). Many insomniacs have catastrophic expectations about the impact of poor sleep, which may play an important role in the exaggeration of self-reports of sleep disturbance and ultimately in the maintenance of the disorder (Perlis et al., 1997). One example of a dysfunctional belief about sleep is an individual's belief that he or she must sleep through the night without any awakenings in order to feel sufficiently refreshed the next day. This thought is problematic because nocturnal awakenings are a normal part of sleep and unrealistic expectations about this aspect of normal sleep may promote worry and rumination that could in fact promote more time spent awake at night (Kaplan, Talbot, & Harvey, 2009). Other dysfunctional beliefs may take the form of more general concerns about the long-term negative consequences of chronic insomnia. Researchers have found that these beliefs predict a chronic pattern of poor sleep, even when arousal, depression, anxiety, and beliefs about short-term consequences of insomnia are controlled for (Jansson & Linton, 2007). Other studies exploring the psychological aspects of insomnia have suggested that maladaptive beliefs and cognitions about sleep may induce sleep-related arousal, which has been identified to be a potential contributing factor in the development and maintenance of insomnia (Morin, 1993).

Research has also demonstrated a connection between sleep-related cognitions and treatment outcome after cognitive-behavioral therapy for insomnia (CBT-I), which has been identified as the most effective treatment for chronic insomnia (Espie, 1999; Espie, Inglis, &

Harvey, 2001). It has been hypothesized that CBT-I reduces sleep-related arousal by changing maladaptive beliefs and attitudes that are believed to maintain the arousal (Morin, 1993). The results of a study by Espie, Inglis, and Harvey (2001) demonstrated that higher scores on the Dysfunctional Beliefs and Attitudes about Sleep scale, suggesting stronger endorsement of beliefs about the negative long-term consequences of insomnia, were associated with positive treatment response. This counterintuitive finding suggests that dysfunctional beliefs play a potentially significant role in determining treatment outcome and provides evidence that CBT intervention can effectively change these beliefs. A study by Edinger and colleagues (2008) indicated that patients with a high degree of unhelpful sleep cognitions were more likely to benefit from CBT-I than patients who had lower levels of unhelpful sleep related thoughts. The authors concluded that pre-therapy sleep cognitions could be useful in identifying which patients would benefit most from CBT-I treatment, which has a cognitive treatment component specifically included to combat faulty sleep-related beliefs. Misperception of sleep such that an individual reports significantly more disturbed sleep than what objective measures show could also be considered a dysfunctional belief or maladaptive pattern of thinking. For this reason it could be hypothesized that the subjective/objective discrepancy will be a correlate of score on the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) Scale and may therefore be a predictor of treatment response to CBT, as well.

**Polysomnography in the Home versus in the Lab.** Polysomnography (PSG), which has been established as the primary objective measure for most sleep disorders, involves connecting electrodes with leads to various parts of the scalp, face, torso, and limbs to record eye movements, oxygen intake, snoring, facial movement, limb movement, lung

expansion, and EEG activity associated with specific stages of sleep (Bae & Avidan, 2008). PSG has traditionally been performed in sleep laboratories under the direction of sleep technologists who monitor the individual during nocturnal recording.

Considering the high laboratory costs and the potential for the laboratory sleep setting to alter an individual's normal sleep patterns and satisfaction with their sleep, ambulatory PSG conducted in the home of the sleeper has been proposed as a more cost effective and valid measure of everyday sleep patterns. For normal sleepers the familiar cues of the home sleeping environment provide a sense of stability that facilitates the sleep process (Edinger et al., 2001). Sleeping in the lab, therefore, without these predictable cues may adversely affect the sleep of these individuals unless they are given numerous nights to adjust to the setting. Alternatively, for insomniacs the cues associated with the home environment may serve as conditioned stimuli that produce arousal at bedtime and ultimately perpetuate sleep disturbances. Polysomnographic recordings in the lab may underestimate insomniacs' sleep problems at home because the cues that facilitate arousal and problematic sleep are absent, leading them to experience fewer sleep disturbances and report higher satisfaction with their sleep (Edinger et al., 2001).

It is also reasonable to suggest that the absence of sleep technologists during home assessment may promote differences in sleep behavior relative to the laboratory sleep. Despite instructions given by the sleep technologist to not change their sleep habits on the night of PSG measurement, individuals undergoing assessment in their home setting may potentially discontinue recording upon waking due to the discomfort of the PSG equipment, therefore truncating their night's sleep. Research in insomnia has recognized how the sleep setting may affect individuals' sleep but there has been little specific focus on how sleep

behavior may change when an individual is undergoing PSG assessment at home. Although individuals are advised to maintain the standards adhered to in the lab, the recording conditions cannot always be standardized or controlled.

### **Statement of the Problem**

**Rationale.** Research has established that many insomniacs report subjective symptoms of insomnia that are greater in severity than what is demonstrated by objective measures, namely polysomnography; however few studies have focused this research in the older adult population, for whom insomnia is disproportionately prevalent. Also, few studies have examined the discrepancy in depth by questioning how it responds to treatment with cognitive-behavioral therapy or how dysfunctional beliefs about sleep or depression and anxiety scores may predict the magnitude of the discrepancy. This study will contribute to the literature by studying the subjective/objective discrepancy in a unique sample of older adults with comorbid insomnia and by taking one step beyond much of the existing discrepancy literature to assess its connection with dysfunctional attitudes about sleep and response to the cognitive-behavioral treatment designed to address them. Because few treatment studies have examined sleep using both subjective and objective measures and considered the potential for misperception, this study makes a unique contribution to the field of insomnia research. Additionally, since this study includes individuals who underwent polysomnographic assessment at home, rather than in the lab, we will be able to examine whether individuals discontinued recording early to avoid prolonging the discomfort and inconvenience of being tethered to electrodes and other monitoring devices.

**Study Aims.** The aims of the current study were: 1) to examine the discrepancy between subjective and objective measures of insomnia, which has been previously

established in adult insomnia populations, in a special population of older adults with comorbid insomnia, 2) to observe whether or not the severity of the discrepancy between subjective and objective measures is diminished post-treatment with Cognitive Behavioral Therapy for Insomnia (CBT-I) compared to those receiving a placebo treatment, Stress Management and Wellness (SMW) therapy, 3) to examine whether scores on the DBAS scale, Geriatric Depression Scale (GDS), Profile of Mood States (POMS) scale, and POMS tension subscale predict the size of the discrepancy between subjective and objective measures at pre-treatment, 4) to examine whether change in DBAS score from pre- to post-treatment with CBT-I will predict change in the magnitude of the subjective/objective discrepancy, 5) to examine if the magnitude of the subjective/objective discrepancy prior to treatment with CBT-I is a significant predictor of treatment response, 6) to examine whether change in the discrepancy from pre- to post-treatment with CBT-I is a significant predictor of treatment response, and 7) to examine if participants behave differently in regard to their sleep when undergoing polysomnographic assessment in the home.

**Hypotheses.** Based on the aforementioned literature and in consideration of the study aims, the following hypotheses were proposed:

1) As has been demonstrated in adult insomnia populations, there will be a significant discrepancy between subjective (diary) and objective (PSG) measures of insomnia in the combined sample of older adults with comorbid insomnia at baseline.

2) The magnitude of the discrepancy between subjective and objective measures will be significantly diminished post-treatment with Cognitive Behavioral Therapy for Insomnia (CBT-I) but not post treatment with Stress Management and Wellness (SMW) therapy, suggesting that CBT-I improves insomniacs' ability to accurately report sleep symptoms.

3) DBAS, GDS, and POMS scores will significantly predict the magnitude of discrepancy between subjective and objective measures at pre-treatment, demonstrating that dysfunctional beliefs about sleep, depression, and tension (i.e. anxiety) are correlates of the subjective/objective discrepancy.

4) Change in DBAS score from pre- to post-treatment with CBT-I will predict change in the magnitude of the subjective/objective discrepancy, demonstrating a relationship between dysfunctional beliefs about sleep and the subjective/objective discrepancy.

5) The size of the subjective/objective discrepancy prior to treatment with CBT-I will be a significant predictor of treatment response.

6) Change in the magnitude of the subjective/objective discrepancy from pre- to post-treatment will significantly predict change in sleep efficiency, indicating that improvement in the accuracy of one's perceptions of sleep is a predictor of treatment response.

7) Sleep measures will indicate that participants will sleep differently on the two PSG nights compared to the full two weeks of sleep diary data due to sensory intrusions and a disruption of their sleep routine. Specifically, they will spend less time in bed overall and demonstrate longer sleep onset latency, shorter total sleep time, and more nocturnal awakening time.

## **Method**

### **Participants**

The current study is a secondary analysis of data collected from a previously conducted randomized clinical trial funded by an R01 grant from the National Institutes of Health (NIH). The original study compared Cognitive-Behavioral Therapy for Insomnia (CBT-I) to a Stress Management and Wellness (SMW) therapy placebo condition for older

adults with comorbid insomnia (Rybarczyk et al. 2005). The study protocol was approved by the Rush University Medical Center Institutional Review Board (IRB) and participants involved gave written informed consent prior to enrollment. Inclusion criteria for participation in the study consisted of: a) 55 years of age or older, b) have had at least three episodes of insomnia per week for at least 6 months, c) suffer from daytime consequences of insomnia such as fatigue or trouble concentrating, and c) suffer from OA, CAD, or COPD. Exclusion criteria included: a) diagnoses of restless leg syndrome, sleep apnea, or other sleep disorders, b) consumption of more than a standard dose of sleep medication, c) medical conditions that are highly to likely to cause sleep disturbances such as diabetes or Parkinson's disease, and d) self report of major psychiatric disorder or history of psychiatric hospitalization.

Participants were recruited between January 2001 and October 2003 via flyers left in medical offices and senior centers and letters sent in the mail to addresses provided by doctor's offices or disease related support organizations. Those who expressed interest in participating in the study were first screened by telephone to determine eligibility for participation using the above listed inclusion and exclusion criteria.

Participants who were identified through telephone screening as eligible for participation were subsequently required to complete one night of polysomnographic assessment in the home to screen for sleep disorders such as sleep apnea or periodic leg movement disorder (PLMD). This assessment also allowed participants one night of acclimation to the PSG technology to promote some level of desensitization to the equipment. Due to an increased prevalence of sleep apnea and PLMD in older adults, exclusion thresholds were set so that individuals with mild indications of these sleep



disorders could be included in the study as long as neither could be considered the primary cause of insomnia (Ancoli-Isreal, Kripke, & Klauber, 1991a, Ancoli-Isreal, Kripke, & Klauber, 1991b). To screen for more severe cases of sleep apnea and PLMD, a board-certified sleep specialist reviewed the polysomnographic results for indications of sleep apnea and PLMD and participants who endorsed moderate to severe cases of either of these sleep disorders were subsequently excluded from the study. The exclusion cutoff for the apnea/hypopnea index, which identifies the severity of sleep apnea based on the total number of breathing cessations (apneas) and partial obstructions (hypopneas) occurring per hour of sleep) was  $> 15$ . For the periodic leg movement index used in PLMD, the exclusion cutoff was  $> 30$ . This index identifies the severity of PLMD based on the number of period limb movements per hour of sleep. Potential participants were also given the Mini-Mental State Examination (MMSE) during the initial home visit, on which they were required to score at least a 24 (Folstein, Folstein, & McHugh, 1975). This was administered to screen out high levels of psychiatric illness that could confound the treatment effect. Four hundred sixty-two individuals contacted study staff and met the inclusion criteria for age, medical diagnosis, and level of insomnia. Of these, 149 were excluded due to diagnosis of a primary sleep disorder other than insomnia and 135 were excluded due to a diagnosis of a comorbid medical or psychiatric condition that may complicate insomnia treatment.

Ninety-two participants completed the original study. These participants were randomly assigned to either a CBT-I or a Stress Management and Wellness (SMW) treatment group using a block randomization procedure. No differences in age, gender, education, race, distribution of the targeted chronic illnesses, number of other chronic illnesses, or sleep medication usage were found between the groups (all  $ps \geq .10$ ). The intervention programs

were matched in as many characteristics as possible, including location (an academic medical center in downtown Chicago), experience level and skill of instructors, amount of group discussion and question-and-answer time, course materials, and refreshments. The interventions were conducted in eight 2-hour classes held on a weekly basis and were administered in 22 separate groups. The average group size was five people. Transportation was paid for by the study for the purpose of encouraging participation of individuals at all income levels.

Two clinical psychologists with extensive experience in conducting cognitive-behavioral therapy for Insomnia (CBT-I) led the CBT-I sessions. Each session included a didactic presentation, a question-and-answer period, and a review of each individual's sleep log, followed by a group discussion to address any problems encountered during implementation of the techniques at home. With the exception of an added relaxation training component, the CBT-I treatment intervention followed Morin's (1993) insomnia treatment protocol, which includes individual modules for stimulus control, sleep restriction, cognitive restructuring, and sleep hygiene.

The Stress Management and Wellness (SMW) treatment adapted from Rybarczyk and colleagues (2001), consisted of didactic presentations and corresponding skill training lessons covering the following six topics: 1) the mind/body relationship; 2) modifying self-talk for the reduction of stress and anxiety; 3) effective communication and assertiveness; 4) problem-solving and goal setting; 5) nutrition; and 6) exercise for individuals with chronic conditions. A physician with extensive training and speaking experience regarding mind/body health covered topics 1 and 2 over four separate class sessions. Topics 3 and 4 were covered by a clinical psychologist and topics 5 and 6 were covered by an expert

nutritionist and exercise physiologist. Of the 44 SMW participants who completed treatment, 26 continued to have sleep problems and were subsequently offered the opportunity to participate in the CBT treatment after the posttreatment assessment.

Measures of sleep were collected using sleep diaries completed by participants over a period of two weeks at pre-treatment (within a one month period prior to treatment) and post-treatment (during a 1-month period after treatment ended). Data was also collected at one-year follow-up; however, this data was not included in the secondary analysis. On two nights during each of these two-week periods, participants also underwent two nights of home polysomnographic (PSG) assessment. Collecting both diary and PSG measures of sleep on the same night allowed for direct comparison of subjective and objective data.

The present analysis is based on data from a subset of the participants from the original study. Participants from the original study were excluded based on three criteria: 1) was a crossover from SMW to CBT, 2) did not meet criteria for insomnia during the two-week period at baseline (specifically, sleep onset latency < 30 minutes, total sleep time > 390 minutes, or wake time after sleep onset < 60 minutes), and 3) significant missing data (diary or PSG) that would compromise statistical analysis. Participants excluded for missing data were missing two or more nights of data as collected by diary and/or polysomnography. Seventeen participants were excluded for missing two or more nights of data as collected by diary and/or polysomnography. For the 11 participants who were missing only a single night of either diary or PSG data, the decision was made to impute data from the previous or subsequent night of sleep to the missing cell rather than to exclude the participant from the analysis.

Of the participants from the original study, 62 were identified as meeting criteria for insomnia and having complete data for the variables of interest. Two of these participants were excluded due to extreme values that were highly likely to be erroneous. Of the remaining 60 participants who were included in this secondary analysis, 18 were male and 42 were female. The mean age of the participants was 69.17 (SD=8.9) and 44 were Caucasian, 13 were African American, 2 were Hispanic, and 1 was Asian. The average education level was 15.02 years (SD=3.2). There were no significant differences between the two treatment conditions in baseline demographic data, number of chronic illnesses, or baseline sleep efficiency (all  $ps > .13$ ; see Table 1).

Table 1.

*Demographics and Other Baseline Characteristics by Treatment Group*

	CBT-I (n = 33)	SMW (n = 27)
Gender	21 female	21 female
	12 male	6 male
Mean Age (SD)	70.73 (9.2)	67.26 (8.2)
Mean years of Education (SD)	15.51 (4.0)	14.43 (1.7)
Race	73% Caucasian (24) 21% African-American (7) 3 % Hispanic (1) 3% Asian (1)	74% Caucasian (20) 22% African-American (6) 4% Hispanic (1) 0% Asian (0)
Chronic Illnesses	6 COPD 17 CA 14 OA	4 COPD 10 CA 17 OA
Mean Number of Chronic Illnesses (SD)	1.12 (.33)	1.15 (.36)
Mean Baseline Sleep Efficiency (%;SD)	69.69 (13.34)	67.72 (13.45)

Secondary analysis of this data set qualified for exemption by Virginia Commonwealth University's Institutional Review Board under the study title, "The discrepancy between subjective and objective measures of insomnia in older adults with comorbid insomnia" (IRB# HM13113).

## **Measures**

**Sleep log.** Sleep logs, paper-and-pencil records of nightly sleep patterns, were completed by participants each morning for two weeks at pretreatment (during the month prior to treatment), two weeks following posttreatment (during the month after treatment ended), and weekly during the CBT class. Participants were asked to record sleep latency, total sleep time, awakenings (quantity and duration), time in bed, length and frequency of naps, and any medication used for sleep (see Appendix 1 for measure).

**Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS).** The DBAS scale (Morin et al., 1993) is a 30-item scale designed to measure various beliefs, attitudes, expectations, and attributions about sleep and insomnia. These cognitions involve five conceptually-derived scales that can be used on their own or combined for a global score: misattributions or amplification of the consequences of insomnia, diminished perception of control and predictability of sleep, unrealistic sleep expectations, misconceptions about the causes of insomnia, and faulty beliefs about sleep promoting practices. Items include a 100-mm visual analog scale, with "strongly disagree" and "strongly agree" descriptors at each end of the scale. The DBAS has demonstrated adequate reliability and validity (Morin et al., 1993).

**Geriatric Depression Scale (GDS).** The GDS (Brink et al., 1982) was developed specifically for use with older adults and is comprised of 30 items in a simple "yes/no"

answer format. None of the items reflect the somatic and vegetative aspects of depression, which reduces the possible confounds of depressive and age-related medical illness symptoms.

**Profile of Mood States scale (POMS).** The POMS (McNair, Lorr, & Droppleman, 1971) is a 65-item self-report measure of affective states for the past week. Patients are asked to rate their mood on a 5-point Likert scale ranging from not at all to extremely. The instrument consists of six subscales for the dimensions of vigor, tension, depression, anger, fatigue, and confusion. It has good test–retest reliability, predictive construct validity, and concurrent validity (McNair et al., 1971). The POMS total mood disturbance score, which includes all but the Vigor subscale, will be used as an overall indicator of distress and the Tension subscale will be used as a measure of anxiety.

## **Procedure**

**Polysomnographic Procedures.** All PSGs were conducted in the subjects' homes. Each study included standard placement of electrodes for continuous monitoring of the central and occipital electroencephalograms (EEG), electrooculogram (EOG), submental electromyogram (EMG) and one chest lead for cardiac rhythm (EKG), nasal airflow measured with a pressure transducer, oral respiration measured with a thermistor, and bilateral leg (anterior tibialis) electromyograms. Sleep recordings were made using the Compumedics P-Series Sleep Monitoring System (Compumedics, Australia). All electrode impedances were less than 10,000 ohms. Sleep studies were conducted during each subject's typical sleep period based on self-report.

Each PSG recording was archived to optical disk and scored manually in 30-second epochs according to standard criteria (Rechtschaffen & Kales, 1968). Records were coded

so that scorers remained blind to the experimental conditions. The inter-rater reliability was maintained at 90% or higher. Apnea episodes were defined by the standard criteria of 10 seconds or longer of no nasal or oral airflow. Hypopnea episodes were defined as a period greater than 10 seconds with airflow 50% or less of baseline amplitude and accompanied by oxygen desaturation of 3% or greater.

**Sleep Parameters.** Four standard parameters that measure different aspects of impaired sleep were collected using the two measurement types (sleep log, polysomnography) and compared at four time points (two at pre-treatment and two at post-treatment). These parameters included: 1) Sleep efficiency (SE), 2) Sleep onset latency (SOL), 3) Total sleep time (TST), and 4) Wake time after sleep onset (WASO). Sleep efficiency (SE), defined as the ratio of time spent asleep to time spent in bed, is a reliable composite index of sleep disturbance severity and measure of sleep improvement (Gagné & Morin, 2001). It is obtained by dividing the total amount of time spent asleep by the total amount of time spent in bed per night and multiplying the result by 100. A cut off of 85% is traditionally used to distinguish good sleepers from bad sleepers. Sleep onset latency (SOL) refers to the time between the point when an individual tries to sleep and the point when sleep is actually initiated and encephalographic patterns of sleep first develop. Total sleep time (TST) is defined as the amount of time spent asleep. Wake time after sleep onset (WASO) is defined as the amount of time spent awake after sleep is initiated.

**Data Analysis.** Statistical analyses were performed using PASW Statistics 18 software (formerly SPSS for Windows). Paired t-tests between sleep diary estimates and polysomnographic measures of sleep were used to determine the presence of a significant discrepancy between subjective and objective measures of sleep efficiency, sleep onset

latency, total sleep time, and wake time after sleep onset. Multiple analysis of variance (MANOVA) was used to analyze differences in the magnitude of this discrepancy between treatment groups at pre- versus post-treatment. Based on the aforementioned hypotheses, regression analyses were also conducted to analyze predictors of the magnitude of or changes in the discrepancy and to examine the relationship between the discrepancy and measures of treatment response. Finally, paired t-tests were used to compare sleep behaviors on PSG nights with sleep behaviors on nights with no polysomnographic assessment. In order to complete these analyses, two nights of data for each sleep parameter (measured by either sleep diary or polysomnography) were averaged together at each time point to create a pre- and post-treatment mean. By calculating the difference between sleep diary and polysomnography means, variables were created to capture the magnitude of the discrepancy at pre-treatment and post-treatment as well as the difference between these. Whereas this study sought to examine the pattern of underestimating sleep (reporting greater deficits than objective measures corroborate), a proportion of the participants showed a positive rather than negative bias (reporting fewer deficits than were observed using objective measures). For the purposes of this study and to avoid analyses using negative numbers, only “negative discrepancy” was examined (i.e., diary data that reported a lower sleep efficiency than what was recorded by PSG). Average discrepancy at pre-treatment and post-treatment were calculated by subtracting the diary estimate from the PSG estimate and any negative numbers (indicating positive rather than negative bias) were substituted with “0.” Conversions of positive bias were only used for regression analyses looking at predictors of discrepancy in sleep efficiency. It should be noted that no conversions were done for the MANOVAs used to test the first two study hypotheses. Among the 60 study participants, there were only 19



instances of significant positive discrepancy ( $> 5\%$  difference). That was in contrast to 56 instances of significant negative discrepancy ( $<5\%$ ) and 45 instances of neutral estimation (within  $\pm 5\%$ ).

## Results

### Discrepancy between subjective and objective measures

To test the first hypothesis, that there would be a significant discrepancy between subjective and objective measures of insomnia, paired t-tests were used. Pairs were created between sleep diary and polysomnography variables for each parameter (SE, SOL, TST, WASO) at baseline. Prior to the analysis, statistical assumptions of normality and homogeneity of variance were checked. Normality was assessed by examining skewness and kurtosis statistics. Due to positive skew observed for sleep onset latency and wake time after sleep onset variables, both sleep diary and polysomnography variables for these parameters were transformed. Wake time after sleep onset variables were transformed using square root transformations and sleep onset latency variables were transformed using log transformation due to more severe skew. Levene's test was used to assess homogeneity of variance and nonsignificant results for this preliminary analysis indicated no violation of this assumption.

Results of the paired t-tests show that significant differences were observed at baseline between sleep diary estimates and polysomnography measures for sleep efficiency,  $t(59) = -5.33, p < .001$ ; sleep onset latency,  $t(59) = 7.17, p < .001$ ; and total sleep time,  $t(59) = -5.06, p < .001$ ; with sleep diaries indicating more severe sleep disturbance than polysomnography. These results provide support for the first hypothesis; however, results of the paired t-test for wake time after sleep onset were not significant ( $p = .29$ ). This indicates the absence of a significant discrepancy between measures of wake time after sleep onset at

baseline and fails to provide support for the first hypothesis. See Table 2 for baseline means and standard deviations for all participants. Norms from a published study of 20 healthy older adults using home polysomnography are also included (McCall, Erwin, Edinger, Krystal, and Marsh, 1992).

Table 2.

*Sleep Diary and Polysomnography Norms and Means for Each Parameter at Baseline*

	LOG		PSG		NORM
	Mean	SD	Mean	SD	Mean
Sleep Efficiency (%)	68.19	18.63	78.07	10.14	84.6
Sleep Onset Latency (minutes)	44.62	42.7	14.22	14.49	12.9
Total Sleep Time (minutes)	315.85	97.47	371.52	68.84	372.0
Wake Time after Sleep Onset (minutes)	70.45	64.3	58.91	38.37	52.0

As illustrated in Figure 1, below, the range of discrepancy between subjective and objective measures of sleep efficiency is reduced for those who have high sleep efficiency. With greater time spent awake between bedtime and wake time (i.e. longer sleep onset latency and more awakenings after sleep onset), there is more opportunity for misperception to occur, resulting in greater discrepancy.

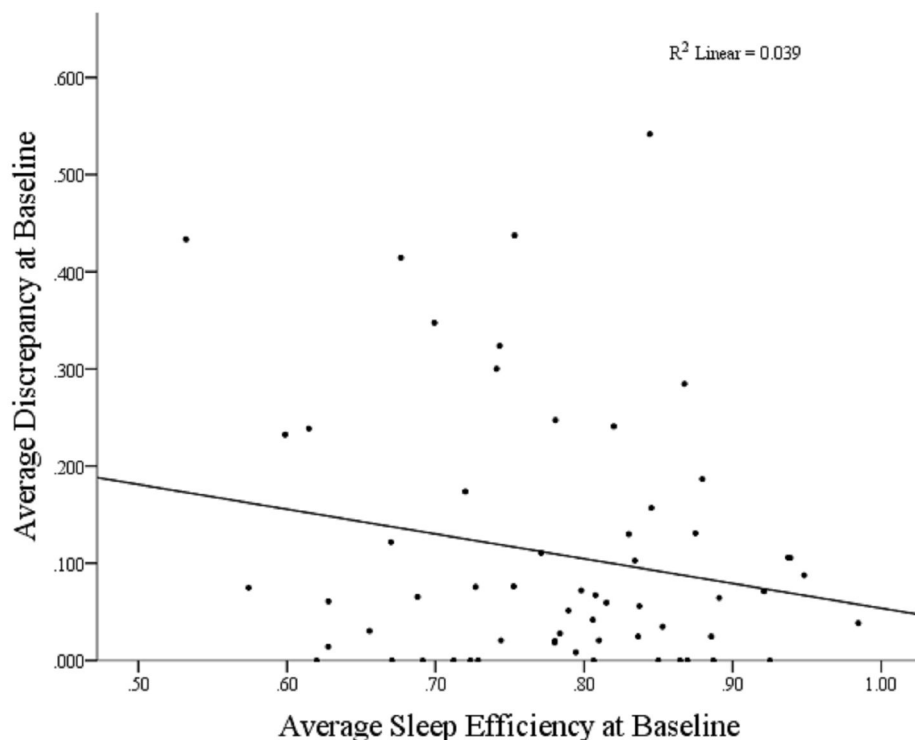


Figure 1. Relationship between Subjective/Objective Discrepancy and Sleep Efficiency

### Change in discrepancy following treatment

To test the second hypothesis, that this discrepancy would significantly decrease following treatment with CBT-I but not with SMW, multivariate analyses of variance (MANOVAs) were used. One MANOVA was conducted for each parameter (sleep efficiency, sleep onset latency, total sleep time, and wake time after sleep onset), with within-subjects factors consisting of the parameter measured during two nights at each time point assessed by either sleep diary or polysomnography and class group entered as the between-subjects factor.

Prior to the primary analysis, data were checked for statistical assumptions, including normality, linearity, homogeneity of variance, and homogeneity of covariance matrices.

Univariate and multivariate outliers were identified using mahalanobis distance and by

examining boxplots. Although several were identified, these were judged to be real values of the population and were thus included in the analysis. Variables were also checked for normality using skewness and kurtosis statistics. Values greater than 1 were observed for sleep efficiency, sleep latency, and wake time after sleep onset variables, indicating that the assumption of normality was violated. To address this, affected variables were subsequently transformed. Square root transformations were applied to all sleep efficiency and wake time after sleep onset variables. Due to more severe skew, sleep onset latency variables were transformed using log transformations. Following these adjustments, skewness and kurtosis were reduced and normality for these variables was significantly improved. 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. Correlation coefficients were all under .65, suggesting that there were no significant problems with multicollinearity. After running the MANOVAs for each sleep parameter, the assumption of homogeneity of variance-covariance matrices was checked using Box's M Test of Equality of Covariance Matrices. This test was nonsignificant for sleep efficiency, indicating that the assumption was met, but was significant for sleep onset latency, total sleep time, and wake time after sleep onset. Because sample sizes between groups are nearly equal, this poses little threat to the assumption; however, Pillai's Trace will be the test statistic reported for all MANOVAs because of its known robustness when sample sizes are equal (Field, 2009). To test for equality of error variances, Levene's test was used. Significant results of this test for sleep efficiency measured by log at two time points suggested possible violation of the assumption; however, because Box's M Test was

nonsignificant, this is not a concern. Levene's test was also significant for one sleep latency and one wake time after sleep onset variable; however, because the sample sizes are nearly equivalent between groups, there is little concern for violation of this assumption.

To more meaningfully interpret patterns observed in the participants' sleep, original variables (before transformations were conducted to make data acceptable for use with parametric tests), are provided in Tables 3.1 - 3.4. As previously mentioned, two nights of data were averaged together to create a single mean score for each measurement type at each time point. MANOVA results showed significant differences between measures of sleep efficiency by group and across time, as evidenced by a significant measure\*time\*group effect, Pillai's Trace = .07,  $F(1,58) = 4.37$ ,  $p < .05$ , multivariate  $\eta^2 = .07$ . Both groups underestimated sleep efficiency at baseline. The CBT group underestimated sleep efficiency by an average of 9% and the SMW by an average of nearly 10.9%. After treatment, discrepancy in the CBT group was 3%, whereas discrepancy in the SMW group was 7.6%. The CBT group diminished their discrepancy by an average of 6%, while discrepancy in the SMW group was reduced by an average of only 3% (see Figure 2). Interestingly, positive discrepancy was observed for the CBT group as a whole at post-treatment, indicating that participants overestimated rather than underestimated sleep efficiency; however, follow-up analysis revealed that this post-treatment discrepancy was nonsignificant. A post-hoc paired t-test was conducted for the CBT group following the MANOVA to compare measures of sleep efficiency at post-treatment and results of this analysis show that subjective and objective measures were not significantly different,  $t(32) = 1.869$ ,  $p = .071$ .

Table 3.1.

*Sleep Efficiency Means at Pre- and Post-Treatment by Measurement Method*

<b>SLEEP EFFICIENCY</b>			
<b>Pre-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	.6969	.1335
2-day Diary	33	.7036	.1469
2-day PSG	33	.7939	.0956
<b>Pre-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	.6772	.1345
2-day Diary	27	.6553	.2255
2-day PSG	27	.7645	.1076
<b>Post-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	.8547	.0778
2-day Diary <sup>a</sup>	33	.8304	.1021
2-day PSG	33	.8007	.1117
<b>Post-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	.7388	.1409
2-day Diary	27	.6981	.1729
2-day PSG	27	.7739	.1046

<sup>a</sup> Paired t-test comparing 2-day sleep diary to 2-day PSG; *not significant*

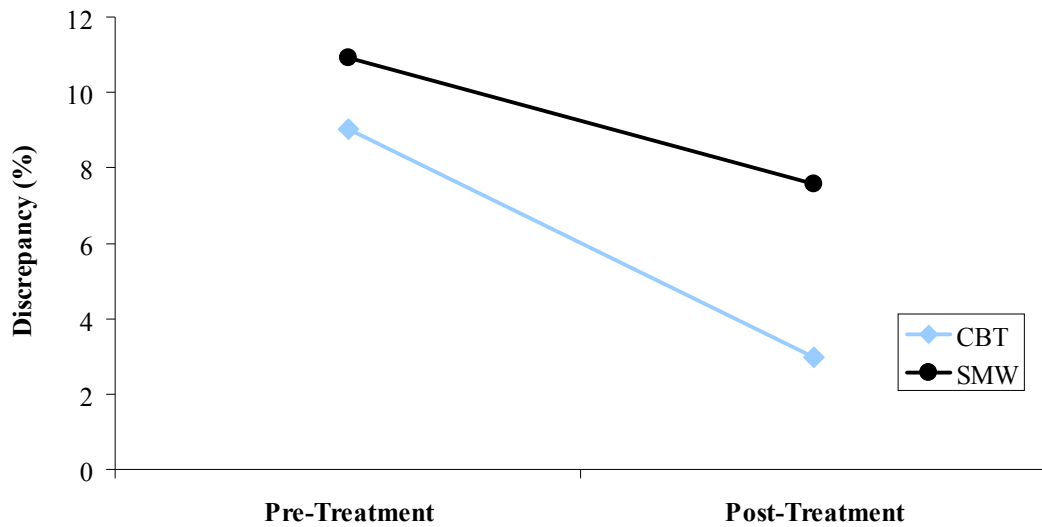


Figure 2. Discrepancy between Subjective and Objective Measures of Sleep Efficiency by Group at Pre- and Post-Treatment

MANOVA results showed significant differences between measures of sleep onset latency by group and across time, as evidenced by a significant measure\*time\*group effect, Pillai's Trace = .14,  $F(1,58) = 9.15$ ,  $p < .01$ , multivariate  $\eta^2 = .14$ . At baseline, the CBT group overestimated sleep onset latency by an average of around 35 minutes, while the SMW group overestimated by an average of 24.8 minutes. After treatment, the CBT group significantly closed the gap between subjective and objective measures of sleep onset latency, reducing their discrepancy to an average of just under 3 minutes; an improvement of 32 minutes. The SMW group, however, only reduced discrepancy to an average of 16 minutes; an improvement of just 8.7 minutes (Figure 3). A post-hoc paired t-test was conducted for the CBT group following the MANOVA to compare measures of sleep onset latency at post-treatment. Results of this analysis show that subjective and objective measures were not significantly different,  $t(32) = 1.308$ ,  $p = .20$ .

Table 3.2.

*Sleep Onset Latency Means at Pre- and Post-Treatment by Measurement Method*

SLEEP ONSET LATENCY			
<b>Pre-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	51.52	57.37
2-day Diary	33	50.00	45.96
2-day PSG	33	15.05	17.53
<b>Pre-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	38.30	27.70
2-day Diary	27	38.04	38.15
2-day PSG	27	13.20	9.81
<b>Post-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	19.76	16.85
2-day Diary <sup>b</sup>	33	16.89	12.98
2-day PSG	33	14.00	13.42
<b>Post-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	33.30	30.51
2-day Diary	27	30.70	23.87
2-day PSG	27	14.57	12.24

<sup>b</sup> Paired t-test comparing 2-day sleep diary to 2-day PSG; *not significant*



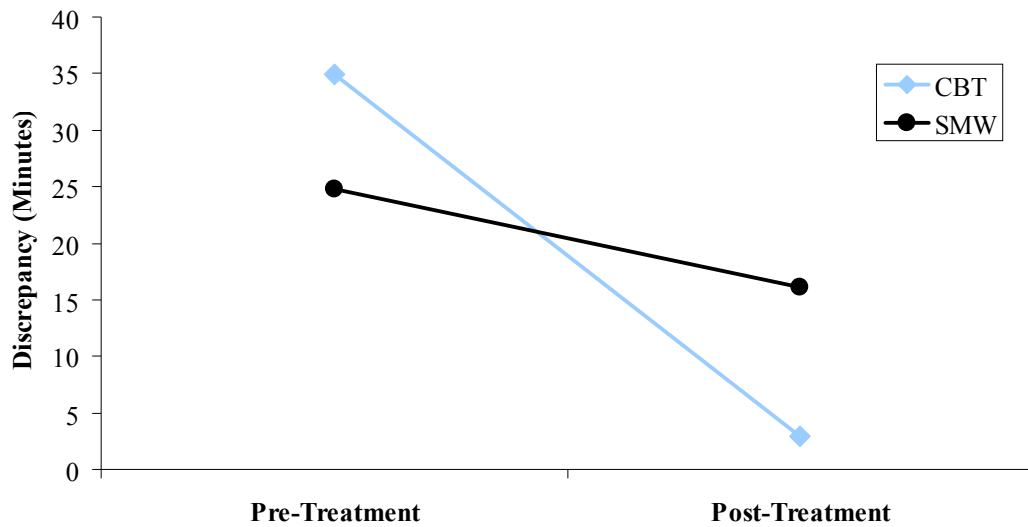


Figure 3. Discrepancy between Subjective and Objective Measures of Sleep Onset Latency by Group at Pre- and Post-Treatment

MANOVA results showed marginally significant differences between measures of total sleep time by group and across time, as evidenced by a nearly significant measure\*time\*group effect, Pillai's Trace = .06,  $F(1,58) = 3.92$ ,  $p = .052$ , multivariate  $\eta^2 = .07$ . Both groups underestimated total sleep time at baseline. The CBT group underestimated total sleep time by an average of around 47 minutes, while the SMW group underestimated by an average of 65 minutes. After treatment, discrepancy between subjective and objective measures of total sleep time was diminished to an average of 7 in the CBT group; an improvement of 40 minutes. A post-hoc paired t-test was conducted for the CBT group following the MANOVA to compare measures of total sleep time at post-treatment. Results of this analysis show that subjective and objective measures were not significantly different,  $t(32) = -7.13$ ,  $p = .48$ . For the SMW group, discrepancy actually increased to nearly 73 minutes; an increase of 8 minutes when compared to the pre-treatment

discrepancy (Figure 4). The marginal nature of the measure\*time\*group effect may be a result of the lack of significant change in discrepancy observed from pre- to post-treatment in the SMW group.

Table 3.3.

*Total Sleep Time Means at Pre- and Post-Treatment by Measurement Method*

TOTAL SLEEP TIME			
<b>Pre-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	320.94	59.02
2-day Diary	33	318.18	75.68
2-day PSG	33	365.83	72.08
<b>Pre-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	315.70	75.77
2-day Diary	27	313.02	120.38
2-day PSG	27	378.46	65.31
<b>Post-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	358.88	55.12
2-day Diary <sup>c</sup>	33	342.08	56.68
2-day PSG	33	349.19	59.65
<b>Post-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	354.74	66.16
2-day Diary	27	327.02	71.73
2-day PSG	27	399.78	62.26

<sup>c</sup>Paired t-test comparing 2-day sleep diary to 2-day PSG; *not significant*

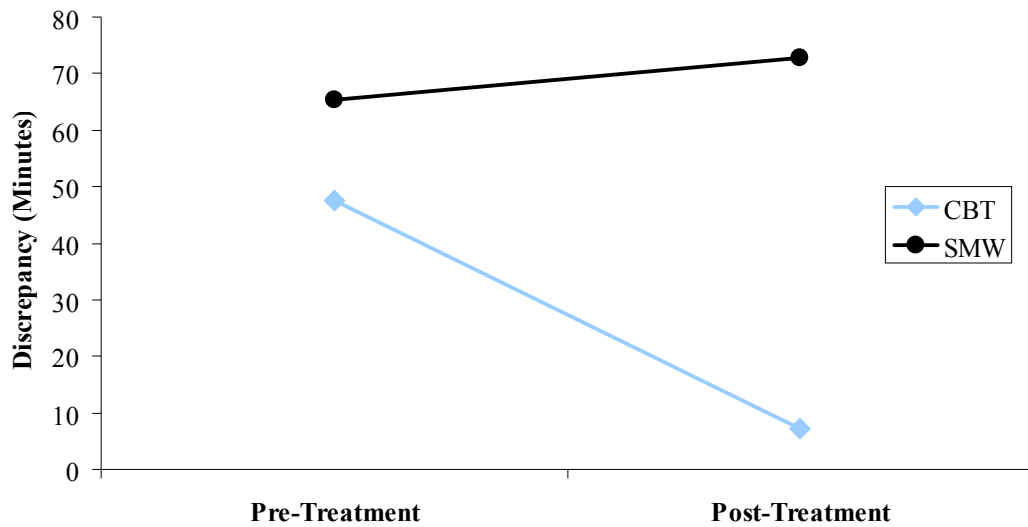


Figure 4. Discrepancy between Subjective and Objective Measures of Total Sleep Time by Group at Pre- and Post-Treatment

MANOVA results showed marginally significant differences between measures of wake time after sleep onset by group and across time, as evidenced by a nearly significant measure\*time\*group effect, Pillai's Trace = .06 ,  $F(1,58) = 3.89$ ,  $p = .054$ , multivariate  $\eta^2 = .06$ . Both groups overestimated wake time at baseline; although not by a statistically significant amount. The CBT group overestimated wake time after sleep onset by an average of only 6 minutes, while the SMW group overestimated by an average of 18 minutes. After treatment, discrepancy in the CBT group actually increased from an average of only 6 to an average of 24 minutes; however, it was noted that they moved from overestimating to underestimating. Discrepancy in the SMW group diminished from 18 minutes to just over 14 minutes; an improvement of nearly 4 minutes. A post-hoc paired t-test was conducted for the CBT group following the MANOVA to compare measures of wake time after sleep onset at post-treatment. Results of this analysis show that subjective and objective measures were

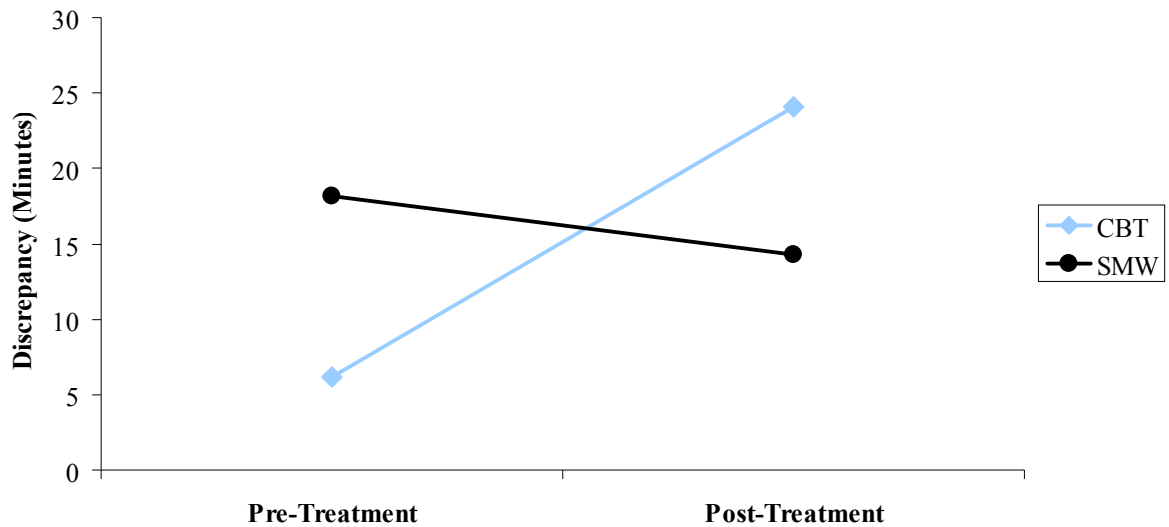
significantly different,  $t(32) = 3.93, p < .001$ . The marginal nature of the measure\*time\*group effect may be a result of the switch from overestimating at pre-treatment to significantly underestimating at post-treatment observed for the CBT group.

Table 3.4.

*Wake Time after Sleep Onset Means at Pre- and Post-Treatment by Measurement Method*

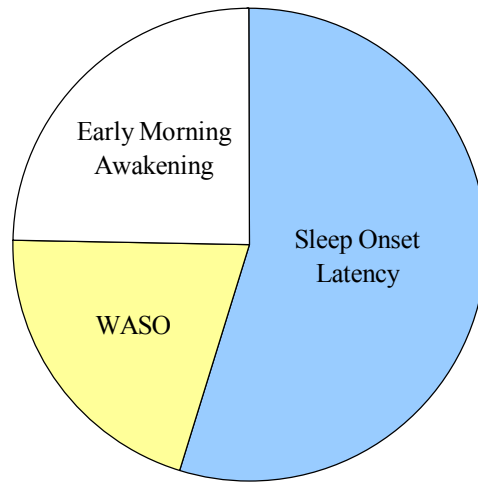
WASO			
<b>Pre-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	51.82	42.79
2-day Diary	33	62.67	46.95
2-day PSG	33	56.52	39.91
<b>Pre-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	71.33	42.04
2-day Diary	27	79.96	80.61
2-day PSG	27	61.83	36.95
<b>Post-Treatment</b>			
CBT Group	N	Mean	SD
14-day Diary	33	20.55	18.23
2-day Diary <sup>d</sup>	33	29.82	34.18
2-day PSG	33	53.87	38.91
<b>Post-Treatment</b>			
SMW Group	N	Mean	SD
14-day Diary	27	54.30	39.71
2-day Diary	27	74.04	67.43
2-day PSG	27	59.77	41.32

<sup>d</sup>Paired t-test comparing 2-day sleep diary to 2-day PSG;  $p < .001$



*Figure 5.* Discrepancy between Subjective and Objective Measures of Wake Time After Sleep Onset by Group at Pre- and Post-Treatment

Sleep efficiency is obtained by dividing total sleep time by total time in bed. Total sleep time, in turn, is obtained by subtracting three separate variables from total time in bed: sleep latency time, awake time after sleep onset, and time awake in the morning prior to getting out of bed (early morning awakening). Thus, it is possible to calculate what percentage of the discrepancy in sleep efficiency was attributed to each of these three variables. Figure 6 illustrates the approximate percentage from each of these categories.



*Figure 6.* Percentage of Discrepancy in Sleep Efficiency at Pre-treatment Accounted for by Sleep Onset Latency, Wake Time after Sleep Onset, and Early Morning Awakening

Figure 7, below, illustrates the reduction in discrepancy after treatment with CBT-I and the change in distribution of the discrepancy in sleep efficiency from pre- to post-treatment. The CBT group demonstrated reduced discrepancy for all three parameters included but made particular improvements in their estimates of sleep onset latency.

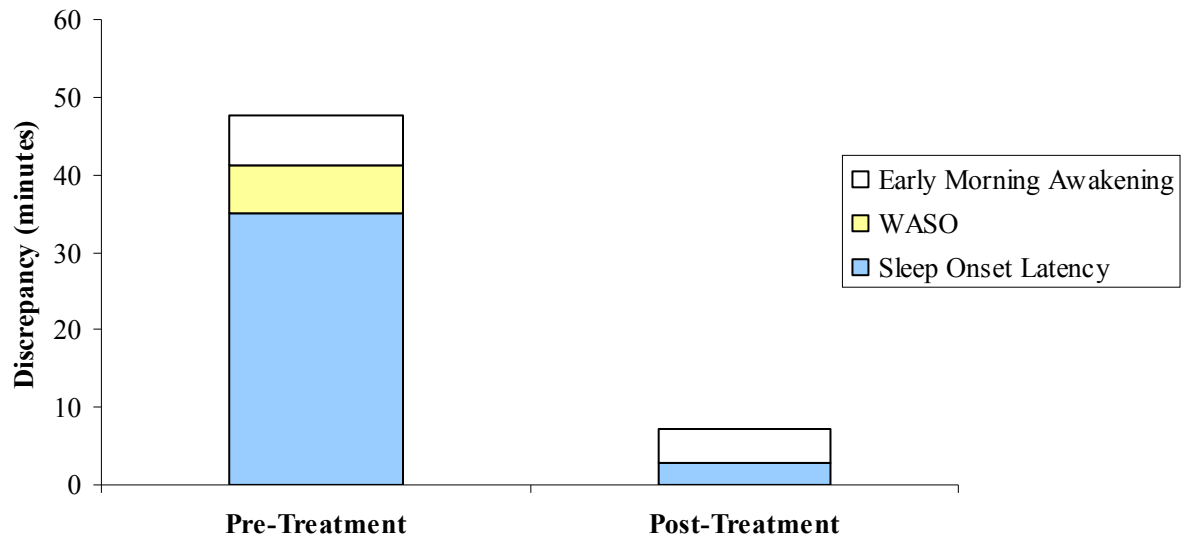


Figure 7. Distribution of Discrepancy in Sleep Efficiency at Pre- and Post-Treatment in the CBT Group

### DBAS, GDS, and POMS scores as predictors of discrepancy

To test the third hypothesis, that Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS), Geriatric Depression Scale (GDS), and Profile of Mood States (POMS) scores would significantly predict the magnitude of negative discrepancy between subjective and objective measures at pre-treatment, multiple regression analysis was used. DBAS total score, GDS total score, POMS total score, POMS tension subscale score, and the four DBAS subscale scores (perceived consequences of insomnia, worry/helplessness about insomnia, sleep expectations, medication) were entered as independent variables and average discrepancy in sleep efficiency at pre-treatment was included as the dependent variable. Because there was no clear a priori hypothesis about the order of the predictors, they were entered simultaneously into a single block.

Prior to the analysis, statistical assumptions were checked, including normality, linearity, multicollinearity, and homoscedasticity. Univariate and multivariate outliers were identified using mahalanobis distance and by examining boxplots. No values were so extreme as to be assumed that they were thought to not be real values of the population and thus all cases were included in the analysis. Normality was assessed by examining skewness and kurtosis statistics. Due to high positive skew observed for POMS total score, POMS tension scale, and average discrepancy, square root transformations were applied to these variables. The residuals scatterplot was 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. Correlation coefficients were greater than .7 for DBAS total score and the four DBAS subscales so the subscale scores were subsequently removed from the analysis. Finally, homoscedasticity was assessed by examining the residuals scatterplot. The band enclosing the residual data points appeared approximately equal in width at all values of the dependent variable, indicating no violation of the assumption.

Results of the multiple regression show that the model was nonsignificant ( $p = .33$ ) and none of the independent variables, individually, were significant predictors of the average discrepancy between measures of sleep efficiency at baseline (all  $ps > .09$ ). This result failed to support the third hypothesis.

### **Change in DBAS score as a predictor of change in discrepancy**

To test the fourth hypothesis, that change in DBAS score from pre- to post-treatment with CBT-I would predict change in the magnitude of the subjective/objective discrepancy, hierarchical multiple regression was used. Change in DBAS score from pre- to post-



treatment was entered as the primary independent variable and average change in the subjective/objective discrepancy for sleep efficiency from pre- to post-treatment was included as the dependent variable. To control for change in sleep efficiency due to treatment effects (due to the known relationship between sleep efficiency and discrepancy illustrated in Figure 1), change in sleep efficiency from pre- to post-treatment as measured by polysomnography was entered into the first block of the model and change in DBAS score from pre- to post-treatment was entered into the second block.

Prior to the analysis, statistical assumptions of normality, linearity, multicollinearity, and homoscedasticity were checked. Univariate and multivariate outliers were identified using mahalanobis distance and by examining boxplots. No values were so extreme that they were thought to not be real values of the population and thus all cases were included in the analysis. Normality was assessed by examining skewness and kurtosis statistics. The change in sleep efficiency variable was slightly positively skewed; however, it was decided that transformation of this variable was not necessary. The residuals scatterplot was eyeballed for linearity and due to the linear relationship observed between the data points, this assumption was determined to be met. Multicollinearity was examined during the analysis using the Tolerance statistic. The value of this statistic was sufficiently high to conclude that this assumption had been met. Finally, homoscedasticity was assessed by examining the residuals scatterplot. The band enclosing the residual data points appeared approximately equal in width at all values of the dependent variable, indicating no violation of the assumption.

The hierarchical multiple regression was first conducted using all cases to examine the predictors after eight weeks of either treatment. Results of the regression show that when

entered alone, change in sleep efficiency significantly predicted change in the subjective/objective discrepancy,  $F(1,57) = 5.22, p < .05; R^2 = .085$ , as expected. Eight point five percent (8.5%) of the variance in change in discrepancy could be predicted by change in sleep efficiency. When change in DBAS score was added, it significantly improved the prediction,  $R^2$  change = .065,  $F(2,57) = 4.85, p < .05; R^2 = .15$ . When controlling for change in sleep efficiency from pre- to post-treatment, change in DBAS score significantly predicted 6.5% of the variance in change in the subjective/objective discrepancy. Together, the two independent variables significantly predicted a total of 15% of the variance in this variable (see Table 4).

Table 4.

*Hierarchical Multiple Regression Analysis Summary for Change in Sleep Efficiency and Change in DBAS Score Predicting Change in the Subjective/Objective Discrepancy*

Variable	B	SEB	$\beta$	$R^2$	$R^2$ change
<b>Step 1</b>				.085	.085
Change in SE	.467	.204	.292*		
Constant	.052	.018			
<b>Step 2</b>				.15	.065
Change in SE	.424	.200	.265*		
Change in DBAS Score	.003	.002	.256*		
Constant	.039	.019			

\* $p < .05$

To assess change in sleep efficiency and change in DBAS score as predictors of change in the subjective/objective discrepancy specifically after treatment with CBT-I, the

hierarchical linear regression was next conducted using only CBT cases. Results show that both change in sleep efficiency and change in DBAS score were not significant predictors of change in the subjective/objective discrepancy, (all  $ps > .31$ ), failing to provide support for the fourth hypothesis.

Returning to the hierarchical regression using all cases, change in DBAS subscale scores were examined as predictors of change in the discrepancy for the purpose of identifying whether the endorsement of particular types of dysfunctional beliefs or attitudes about sleep relate more closely to change in misperception of one's sleep parameters. Change scores (differences observed between pre- and post-treatment) were calculated for each subscale (perceived consequences of insomnia, worry/helplessness about insomnia, sleep expectations, and medication). When entered together into a model predicting change in the subjective/objective discrepancy, both the overall model and individual variables were nonsignificant (all  $ps > .43$ ).

### **Baseline discrepancy as a predictor of treatment response**

To test the fifth hypothesis, that the size of the subjective/objective discrepancy prior to treatment with CBT-I would be a significant predictor of treatment response, hierarchical multiple regression was used. Treatment response was measured as change in sleep efficiency assessed by sleep diary across two weeks at pre- and post-treatment. Average discrepancy between measures of sleep efficiency at pre-treatment was entered as the primary independent variable along with age, number of medications, and number of medical conditions. To control for baseline sleep efficiency (due to the known relationship between sleep efficiency and discrepancy illustrated in Figure 1), baseline sleep efficiency measured by polysomnography was entered into the first block of the model and the other variables

were entered into the second block. Change in sleep efficiency was entered as the dependent variable. Only CBT cases were included in the analysis.

Prior to the analysis, statistical assumptions were checked, including normality, linearity, multicollinearity, and homoscedasticity. Univariate and multivariate outliers were identified using mahalanobis distance and by examining boxplots. No values were so extreme that they were thought to not be real values of the population and thus all cases were included in the analysis. Normality was assessed by examining skewness and kurtosis statistics. Due to high positive skew observed for the discrepancy between measures of sleep efficiency at pre-treatment variable, a square root transformation was applied. The residuals scatterplot was eyeballed for linearity and due to the linear relationship observed between the data points, this assumption was determined to be met. Multicollinearity was examined during the analysis using the Tolerance statistic. The value of this statistic was sufficiently high to conclude that this assumption had been met. Finally, homoscedasticity was assessed by examining the residuals scatterplot. The band enclosing the residual data points appeared approximately equal in width at all values of the dependent variable, indicating no violation of the assumption.

Results of the hierarchical multiple regression showed that discrepancy at pre-treatment was found to be the only significant predictor of treatment response when controlling for baseline sleep efficiency, so the other predictors were subsequently removed from the model. When entered alone, baseline sleep efficiency marginally significantly predicted treatment response,  $F(1,32) = 3.88, p = .058; R^2 = .11$ . When baseline discrepancy was added to the model, it significantly improved the prediction,  $R^2$  change = .207,  $F(2,32) = 7.00, p < .01; R^2 = .32$ . When controlling for baseline sleep efficiency, baseline discrepancy

significantly predicted 20.7% of the variance in fourteen day treatment outcome. Together, the two independent variables significantly predicted a total of 31.8% of the variance in this variable (see Table 5).

Table 5.

*Hierarchical Multiple Regression Analysis Summary for Baseline Sleep Efficiency and Baseline Discrepancy Predicting Treatment Response*

Variable	B	SEB	$\beta$	$R^2$	$R^2$ change
<b>Step 1</b>				.111	.111
Baseline SE	-.372	.189	-.334		
Constant	.453	.151			
<b>Step 2</b>				.318	.207
Baseline SE	-.464	.171	-.416*		
Baseline Discrepancy	.272	.090	.462*		
Constant	.456	.134			

\* $p < .05$

**Change in discrepancy as a predictor of treatment response**

To test the sixth hypothesis, that change in the magnitude of the subjective/objective discrepancy from pre- to post-treatment would significantly predict treatment response to CBT-I, hierarchical multiple regression was used. Treatment response was again measured as change in sleep efficiency assessed by sleep diary across two weeks at pre- and post-treatment. To control for baseline sleep efficiency (again, due to the known relationship between sleep efficiency and discrepancy illustrated in Figure 1), baseline sleep efficiency measured by polysomnography was entered into the first block of the model and change in

the discrepancy between subjective and objective measures of sleep efficiency from pre- to post-treatment was entered into the second block. Change in sleep efficiency as measured by two-week sleep diary was entered as the dependent variable. The analysis was conducted in CBT cases only.

Prior to the analysis, statistical assumptions were checked, including normality, linearity, multicollinearity, and homoscedasticity. Univariate and multivariate outliers were identified using mahalanobis distance and by examining boxplots. No values were so extreme that they were thought to not be real values of the population and thus all cases were included in the analysis. Normality was assessed by examining skewness and kurtosis statistics. Both variables (change in sleep efficiency and change in discrepancy) were slightly positively skewed; however, it was decided that it was not necessary to transform them. The residuals scatterplot was eyeballed for linearity and due to the linear relationship observed between the data points, this assumption was determined to be met. Multicollinearity was examined during the analysis using the Tolerance statistic. The value of this statistic was sufficiently high to conclude that this assumption had been met. Finally, homoscedasticity was assessed by examining the residuals scatterplot. The band enclosing the residual data points appeared approximately equal in width at all values of the dependent variable, indicating no violation of the assumption.

Results of the regression show that when entered alone, baseline sleep efficiency marginally significantly predicted treatment response,  $F(1,32) = 3.88, p = .058; R^2 = .11$ . When change in discrepancy was added to the model, it significantly improved the prediction,  $R^2$  change = .354,  $F(2,32) = 13.03, p < .001; R^2 = .465$ . When controlling for baseline sleep efficiency, change in discrepancy significantly predicted 35.4% of the variance

in fourteen day treatment outcome. Together, the two independent variables significantly predicated a total of 46.5% of the variance in this variable, setting an upper limit of 53.5% of variance in two-week treatment outcome explained by actual changes in sleep quantity (see Table 6).

Table 6.

*Hierarchical Multiple Regression Analysis Summary for Baseline Sleep Efficiency and Change in Discrepancy Predicting Treatment Response*

Variable	B	SEB	$\beta$	$R^2$	$R^2$ change
<b>Step 1</b>				.111	.111
Baseline SE	-.372	.189	-.334		
Constant	.453	.151			
<b>Step 2</b>				.465	.354
Baseline SE	-.418	.149	-.375*		
Change in Discrepancy	.552	.124	.596**		
Constant	.448	.119			

\* $p < .01$

\*\*  $p < .001$

**Comparison of sleep parameters on PSG nights versus non-PSG nights**

To test the seventh hypothesis, that participants would sleep differently on PSG nights due to sensory intrusions, paired t-tests were used. Pairs were created between two-week sleep diary and two-night PSG variables for each parameter (SE, SOL, TST, WASO). Prior to the analysis, statistical assumptions of normality and homogeneity of variance were checked. Normality was assessed by examining skewness and kurtosis statistics. Due to

positive skew observed for sleep onset latency and wake time after sleep onset variables, both two-week and two-night variables were transformed. Wake time after sleep onset variables were transformed using square root transformations and sleep onset latency variables were transformed using log transformation due to more severe skew. Levene's test was used to assess homogeneity of variance and nonsignificant results for this preliminary analysis indicated no violation of this assumption.

Results of the paired t-tests show that no significant differences were observed between two-week and two-night averages for all parameters (all  $ps > .17$ ), indicating that sleep behaviors on nights when participants are undergoing polysomnographic assessment do not look markedly different from normal nights of sleep. This finding fails to provide support for the seventh hypothesis.

### **Discussion**

The primary purpose of this secondary analysis was to further explore the discrepancy between subjective and objective measures of insomnia as a phenomenon characteristic of those with insomnia by assessing its presence in a sample of older adults with comorbid insomnia enrolled in a treatment study. This study also sought to determine how the discrepancy changes after treatment with Cognitive-Behavioral Therapy for Insomnia as compared to a Stress Management and Wellness control treatment. Additionally, this study sought to examine predictors of the discrepancy as well as the relationship between discrepancy and treatment response and between discrepancy and dysfunctional beliefs and attitudes about sleep.

The paired t-test analyses show that, as hypothesized, there exists a significant discrepancy between subjective and objective measures of insomnia. Table 2 illustrates that



participants underestimated sleep efficiency and total sleep time and overestimated sleep onset latency at baseline. These findings are consistent with the results of previous studies, which found that many individuals with insomnia report more subjective sleep disturbance than what objective measures corroborate (Carskadon et al., 1976; Frankel et al., 1976). However, the difference between subjective and objective measures of wake time after sleep onset was not significant at baseline. This finding suggests that participants in this study, overall, were accurate in their perceptions of this parameter before intervention. In line with what other studies from the literature have found, a number of participants in this study exhibited a positive bias when estimating their sleep, reporting fewer sleep complaints than objective measures showed (Edinger & Fins, 1995; Carskadon et al, 1976). Out of the 60 two-day estimations made at pre-treatment, 4 showed positive bias ( $> 5\%$ ), 35 showed negative bias ( $> 5\%$ ), and 21 were neutral (within  $\pm 5\%$ ). Because this particular study sought to focus on the negative rather than positive bias, correlates and predictors of this positive bias were not examined. It is recommended that this positive form of bias be further explored in future studies.

By examining the discrepancy for individual sleep parameters, a picture emerges of which parameters were most affected by misperception in this sample and which parameters were most affected by CBT-I intervention. At baseline, as seen in Figure 6, more than 50% of the discrepancy in total sleep time could be accounted for by discrepancy in sleep onset latency. This indicates that participants most significantly misperceived the length of time it took them to fall asleep at night. Following treatment, the CBT group was observed to have significantly diminished discrepancy as compared to SMW, as illustrated in Figures 2-5. As Figure 7 illustrates, the CBT group made a particularly marked improvement in accuracy of

perception of sleep onset latency, reducing discrepancy by an average of 32 minutes.

Although this group started out with an average discrepancy more than ten minutes higher than the average for the SMW group, they reduced the discrepancy to only 3 minutes after treatment. This was 13 minutes less than the post-treatment discrepancy observed for the SMW group. For sleep efficiency, sleep onset latency, and total sleep time, participants' subjective reports of sleep were not significantly different from objective measures after treatment with CBT, demonstrating that CBT treatment basically eliminated discrepancy in these domains. However, there was an *increase* in discrepancy observed for the parameter of wake time after sleep onset. For this parameter, there was no significant discrepancy at baseline but the CBT group moved to significant underestimation of awakening time at post-treatment.

It is difficult to interpret why this shift to significant underestimation occurred for this sleep parameter, in particular. It may be that wake time after sleep onset is especially difficult to estimate because it occurs after sleep onset and before a final awakening and often involves keeping track of multiple periods of arousal. Despite the relatively low discrepancy observed for this parameter at pre-treatment in this study, it could be that it is particularly vulnerable to a placebo effect based on positive expectancy. The purpose of the placebo treatment group was to control for such effects, but it is not a perfect substitute for a true treatment group. Participants in the CBT group may have still picked up on some type of additional positive expectancy while in treatment that resulted in a greater expectation for improvements in sleep in this group. This bias could be the result of inadvertent influence by study staff or clinicians or differences in plausibility and expectancy that occurred after these

variables were measured and shown to be the same after the first two sessions (Rybarczyk et al., 2005).

As illustrated in Figure 1, lower sleep efficiency at baseline was associated with higher discrepancy due to the fact that more awakening time leads to more opportunities to misjudge one's sleep. Following this logic, it could be suggested that the observed improvement in discrepancy after treatment with CBT-I is the result of an improvement in sleep efficiency. However, because objective measures of sleep efficiency were not observed to change significantly from pre- to post-treatment, this is not a plausible explanation. This fact suggests that CBT had an effect on sleep perception, resulting in more accurate subjective reporting of sleep compared to the SMW treatment condition.

Multiple regression results established that, contrary to the third hypothesis, negative mood, anxiety, and dysfunctional beliefs and attitudes about sleep were not significant predictors of the magnitude of the discrepancy at baseline. This finding goes against what has been established in the literature about mood and anxiety factors as correlates of the subjective/objective discrepancy (Rosa & Bonnet, 2000; Edinger et al., 2000) and what was hypothesized in terms of a relationship between dysfunctional cognitions and sleep misperception. It is possible that the exclusion of those participants with a history of a major psychiatric condition contributed to this lack of significant results; however, similar exclusion criteria existed in the previously conducted studies. Another possibility is that the small sample size included in this study may have limited statistical power, thereby producing a false negative result. Regardless, it is unknown from the results of this study what types of psychological characteristics are associated with the subjective/objective

discrepancy before treatment. More research will need to be conducted to identify clear correlates of this phenomenon.

Although DBAS score was not a significant predictor of the subjective/objective discrepancy at baseline, results of the hierarchical multiple regression using all subjects showed that change in DBAS score from pre- to post-treatment was a significant predictor of change in the discrepancy. This finding provides evidence for a relationship between dysfunctional beliefs about sleep and the subjective/objective discrepancy and suggests that a reduction in these maladaptive thinking patterns correlates with a reduction in negative sleep misperception. Examination of the specific dysfunctional beliefs one might endorse through an additional hierarchical regression with the DBAS subscale scores failed to provide evidence that endorsement of a particular type of belief (e.g. worry about the consequences of insomnia) is associated with change in the discrepancy more so than others. Results of the hierarchical regression using only CBT cases were nonsignificant, indicating that change in DBAS score did not predict change in the subjective/objective discrepancy for the CBT group. However, as shown in Figure 8, below, those participants in the CBT group who showed particularly high discrepancy between subjective and objective measures of sleep efficiency at baseline showed greater reduction in dysfunctional beliefs and attitudes about sleep as compared to those in the CBT group with less discrepancy or those in the SMW group.

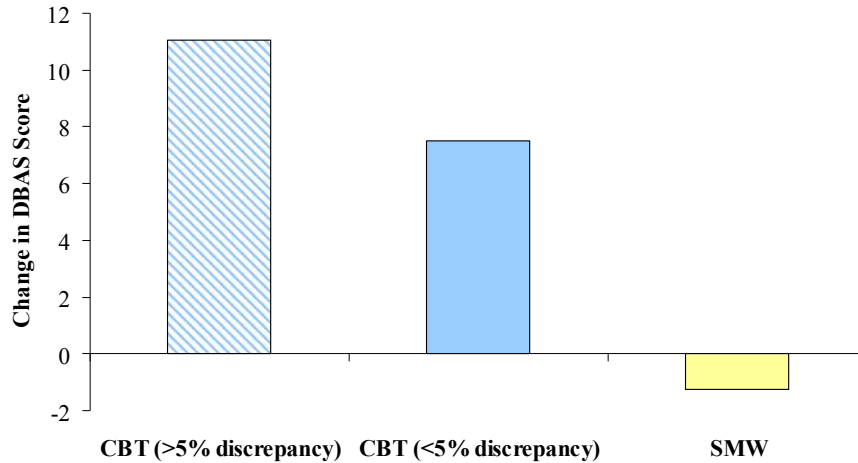


Figure 8. Change in DBAS Score from Pre- to Post-Treatment, by Amount of Baseline Discrepancy and/or Treatment Group

This finding provides evidence to suggest that misperception, as one form of dysfunctional belief, is improved with CBT and suggests that the lack of significant effect in the CBT group may be due to insufficient statistical power due to small sample size ( $n = 33$ ) rather than a true lack of association between these variables.

Results of the hierarchical multiple regression analysis examining the relationship between baseline discrepancy and treatment response provide support for the fifth hypothesis and suggest that individuals who enter treatment with high subjective/objective discrepancy show significantly better treatment response to CBT-I. This finding parallels those of previous studies reporting a significant relationship between treatment response to CBT and high endorsement of dysfunctional beliefs and attitudes about sleep (Espie, 1999; Espie et al., 2001). Specifically, Espie and colleagues (2001) found that negative beliefs about the long term effects of insomnia prior to treatment were associated with positive treatment response and interpreted this to mean that sleep related thinking errors could be a mediating factor for treatment effects as supported by the fact that DBAS scores changed after CBT intervention.

The results of Edinger and colleagues' 2008 study indicated that patients with a high degree of unhelpful sleep cognitions were more likely to benefit from CBT-I than patients who had lower levels of maladaptive sleep related thoughts. The authors concluded that pre-therapy sleep cognitions could be useful in identifying which patients would benefit most from CBT-I treatment. If misperception can be conceptualized as a form of dysfunctional belief not captured by the DBAS scale, the results of the current study suggest that high subjective/objective discrepancy prior to treatment may be another useful tool for identifying which patients should be referred for CBT-I. The fact that high pre-treatment discrepancy was predictive of positive treatment response in this sample also identifies misperception as an important target for treatment.

Change in discrepancy from pre- to post-treatment was also found to be a significant predictor of treatment response to CBT-I when controlling for baseline sleep efficiency, providing support for the sixth hypothesis. Results of the hierarchical multiple regression tell us that 35.4% of change in fourteen day treatment outcome can be accounted for by improvement in the subjective/objective discrepancy during the two PSG nights; however, 64.6% of treatment response has yet to be explained. This remaining portion could presumably be accounted for by factors other than changed sleep perception, including "real" changes in sleep. Additionally, the diminished discrepancy between subjective and objective measures of insomnia on PSG nights does not account for all of the significant change observed at post-treatment using two-week sleep diary measures. Polysomnographic measures of sleep efficiency did not even change by a full percentage point after treatment with CBT; however, the two-week sleep diary measures indicate an improvement of nearly 16%. This implies that the two days may not be fully representative of two weeks of sleep

and other factors may be involved. Further research is needed in order to better understand this result.

Results of paired t-tests between two-week sleep diary and the sleep diary for the two PSG-nights for each parameter (sleep efficiency, sleep onset latency, total sleep time, and wake time after sleep onset) showed that sleep does not look markedly different on nights when participants were undergoing home polysomnographic assessment relative to normal nights. Despite the presence of recording equipment, including leads connected to various parts of the body and head, participants did not report longer sleep latencies, more nighttime awakenings, or reduced total sleep time. Despite what was implied by the previous analysis, this suggests that two-night averages of sleep patterns in this study were representative of participants' typical sleep and also provides evidence for the clinical utility of home polysomnographic assessment. Had participants shown evidence of altered sleep patterns, perhaps due to truncating sleep by removing uncomfortable PSG equipment in the morning hours, this would indicate a concern regarding the accuracy of data collected using the method of objective assessment. It would also threaten the generalizability of sleep data from these nights.

It has been established that many insomniacs sleep differently in the lab than they do in their home environment due to the absence of cues that facilitate arousal (Edinger et al., 2001). The results of this study provide evidence to suggest that home polysomnography may have the potential to capture a more accurate picture of an individual's typical sleep behavior while also reducing costs to the patient. This is an important finding given the fact that technological advances in PSG will eventually lead to a more widespread use of home assessment of sleep due to the lower costs. If cost becomes low enough and the feedback

regarding sleep state misperception is shown to improve CBT-I treatment response, PSG may even be justified as a tool for assessment in all cases of insomnia.

In summary, this study sought to more closely examine the discrepancy between subjective and objective measures of insomnia, which has been established as a common characteristic of many insomniacs. After assessing discrepancy between sleep diary and polysomnographic measures of four sleep parameters in a sample of older adults with comorbid insomnia, the results of this study show that many individuals reported subjective complaints of sleep disturbance that were significantly more severe than what objective measures showed. Negative mood, anxiety, and high endorsement of dysfunctional beliefs and attitudes about sleep were not significant predictors of this discrepancy before treatment, as was hypothesized. However, change in dysfunctional beliefs about sleep did significantly predict change in the discrepancy, providing evidence to support that misperception may be one form of dysfunctional belief. Assessing the magnitude of the subjective/objective discrepancy prior to treatment was identified as a potentially useful tool for recognizing patients who are likely to respond well to treatment with CBT-I after high discrepancy was found to be predictive of positive treatment response. An additional finding suggests that a reduction in misperception rather than actual sleep changes accounts for a portion of the treatment response observed after CBT-I. Finally, home polysomnographic assessment was not shown to significantly alter normal sleep behaviors.

One observation of the data regarding discrepancy in this study that is not highlighted in other studies to date is that estimates of sleep onset latency constituted the highest portion of both initial discrepancy and change in discrepancy after CBT. This suggests that the initial transition from wake to sleep is where the highest risk for discrepancy occurs when



recalling sleep the next day, even though this does not constitute the largest portion of self-reported insomnia in this population (30% of awake time was attributed to latency) and other populations (an average of 44% of awake time was attributed to latency in a meta-analysis by Smith et al., 2002). To some extent, this is a logical finding because it is the point of time furthest away from when the diary is completed. Additionally, stage 1 sleep is often difficult for an individual to detect. What an individual reports to be an extended sleep onset latency may actually be light, stage 1 sleep interrupted by an arousal or awakening. Future studies should analyze whether the percentage of stage 1 sleep is maintained longer in the initial period of sleep in those who tend to overestimate the severity of their sleep disturbance. An additional treatment implication from this finding is that CBT-I intervention should attempt to redirect awareness by emphasizing the difficulty that individuals with sleep problems have in assessing when the transition occurred. It also supports the possibility that reductions in anxiety at bedtime will reduce the perceived salience of that period of time during the night, therefore reducing its perceived length.

Perhaps the most intriguing finding from this study is the significant reduction in discrepancy observed after treatment with CBT-I. This suggests that CBT may not only work to diminish maladaptive thinking patterns related to perceived consequences of insomnia, worry about insomnia, sleep expectations, and medication, but may also reduce the misperception of sleep. In fact, using variance data from regression analyses, the increase in accuracy of sleep perception accounted for approximately 32% of the change that occurred in sleep at post-treatment. This finding raises questions not emphasized in the literature thus far about the extent to which improvements in insomnia are largely “real” changes in sleep or merely perceived changes. A number of studies have demonstrated corroborating evidence

for improvements in sleep after treatment but these changes are frequently of a much lower magnitude than self-reported changes occurring over a larger window of time than one or two nights of PSG (Morin et al., 2006). Previous interpretations of this discrepancy have been attributed to differences between home sleep and laboratory sleep (Edinger et al., 2001) and have not been subjected to the same type of analyses performed in this study to our knowledge. Post-hoc analyses using the same methods employed in this study by other investigators who reported both self-report and PSG outcomes from CBT-I would shed further light on this issue and be a valuable contribution to the literature.

Taken together, the findings of this study have implications for our understanding of how CBT-I works and for the discussion of how insomnia is best defined. The significant improvement observed in maladaptive thinking and misperception after treatment with CBT in this study suggests that for those with subjective reports of sleep disturbance that are not objectively corroborated, CBT might work by improving the accuracy of sleep perception more so than changing physiological sleep patterns. From this idea, the question emerges of CBT's effectiveness for those who do present with objectively disturbed sleep. It is reasonable to wonder whether there should in fact be "subjective" versus "objective" subtypes of insomnia, as the literature has debated (Edinger & Krystal, 2003). Subjective insomnia may be an entity distinct from insomnia that is objectively corroborated. Different underlying factors for these two subtypes would necessitate different approaches to treatment in order to produce optimal outcomes. Alternatively, the findings of this study may provide support for the utility of a "sleep-state misperception" subtype of insomnia, which is recognized as a separate diagnosis by the International Classification of Sleep Disorders (AASM, 2005). More research is needed to further explore this possibility.

Despite its strengths, this study was subject to multiple limitations that must be considered. Perhaps the most significant limitation is the absence of analysis of sleep stages or depth of sleep using PSG measures. For the present study it was decided that the focus would be on discrepancy between perception of being asleep and documented sleep of any type. A more refined analysis would take into account the depth of sleep to determine if individuals who perceive themselves to be awake are actually experiencing lower quality or less restorative sleep relative to those who are more accurate in their sleep estimates. This has been suggested by previous investigators (Mendelson, James, Garnett, Sack, & Rosenthal, 1986) along with the possibility that the disorder of “sleep-state misperception” is an inaccurate and pejorative term. Even though some research has failed in its attempt to find EEG abnormalities or lighter sleep in these individuals (Mendelson et al., 1986) it is possible that our EEG scoring methods are not sophisticated enough to detect subtle but true physiological differences in this population. Power density analysis offers some potential for detecting such differences in future studies (Merica, Blois, & Gaillard, 1998). If such were the case, then CBT changes may in fact be leading to subtle improvements in sleep physiology, resulting in an accurate perception of improved sleep. As such, future analyses of the current database would be well advised to include analyses of changes in sleep staging or other EEG changes that may have occurred in the negative bias group.

A second limitation is the small sample size ( $n = 60$ ) utilized in this study. Particularly in the case of the two analyses involving only the CBT group ( $n = 33$ ), this small number of data points may have limited the ability to find significant effects that would have emerged from a larger sample. The multiple regression predicting change in the magnitude of the discrepancy was significant when all participants were included in the analysis;

however, when the analysis was conducted in the CBT group alone, the results did not reach significance. The inherent variability of sleep parameters must also be considered. Sleep onset latency and wake time after sleep onset, in particular, vary significantly both night-to-night and between individuals. As a result of this fact, small sample size in this study led to the appearance of extreme values and data that did not fit a normal distribution. Extreme values that did not appear to be real values of the population were excluded from analyses; however, many extreme values were not excluded due to the fact that significant variation is not unexpected with a sample of individuals with chronic insomnia. Many variables included in the primary analyses were found to be skewed and kurtotic and were subsequently transformed in order to meet the assumptions required by parametric tests. The necessary steps were taken to minimize non-normality; however, some minimal skewness and kurtosis remained for some variables. With a larger sample size, normality would pose less of an issue and the significance and robustness of the findings would have likely been improved.

A third limitation is that of missing sleep diary or polysomnography data in this study. Seventeen participants were dropped from the parent study due to the absence of diary or polysomnography data at either pre- or post-treatment. It is possible that this attrition was non-random and was, in fact, more common among those participants who were better at estimating their sleep. Additionally, eleven participants were missing a single night of either diary or PSG data due to data entry error, PSG technology failure, or errors in participant reporting. Rather than excluding these participants, it was decided that these missing cells would be filled with data from the previous or subsequent night of sleep. While this prevented potential problems due to an even more diminished sample size, sleep parameters

at individual time points were captured by a single night of assessment rather than averaged across two nights, which may limit how representative the data was.

A fourth limitation of the current study is the lack of direct personality measures that may have been useful in determining predictors of the subjective/objective discrepancy. Previous research has suggested that personality factors may be correlated with the tendency to misperceive disturbances in sleep (e.g. neuroticism, hypochondriasis; Corsey et al., 1975; Kales et al., 1976; Tan et al., 1984); however, this study left these factors unexplored.

A fifth limitation involves the level of insomnia severity. In order to be included in the original study, a potential participant had to have had at least three episodes of insomnia per week for at least 6 months, and had to have suffered from daytime consequences of insomnia such as fatigue or trouble concentrating. Individuals with fewer episodes of insomnia or shorter duration of complaints were excluded from participation. Because of this, the individuals who participated in this study suffered from severe insomnia which could limit the generalizability of the study. It is possible that the subjective/objective discrepancy would manifest itself differently, relate differently to dysfunctional beliefs, or respond differently to CBT-I in a less specific or severe population.

A final flaw in the study design was that individuals in the CBT group kept a sleep diary throughout treatment, whereas the SMW group only engaged in required self-monitoring during set periods of time (two weeks) at pre-treatment and post-treatment. This means that one alternative explanation for the findings is that the CBT-I treatment per se was not the factor that led to improved accuracy in sleep estimation but, instead, it was simply caused by what some researchers have termed a "diary-keeping effect" (Franklin, 1981; Engel-Friedman, Bootzin, Hazelwood, & Tsao, 1992, Morin, 1993). Future studies should

control for this threat to internal validity by having control participants also keep a weekly diary during the treatment period.

This study contributes to the existing literature by identifying the subjective/objective discrepancy in a unique sample of older adults with comorbid insomnia and providing evidence to suggest that this discrepancy may be a potential factor in a favorable treatment response to CBT. Despite what is offered by the results of this study, there is more to be learned about the subjective/objective discrepancy. Several directions for future research can be suggested with this objective in mind. As mentioned above, a major limitation of the current study was the small sample size. The literature would benefit from studies including a substantial number of participants to account for the potential for missing data and the variability of sleep patterns that increases the likelihood of non-normality. An additional limitation of this study was the lack of personality variables when examining predictors of the subjective/objective discrepancy. Future studies should consider neuroticism, hypochondriasis, or other personality factors as potential correlates of sleep misperception.

Additionally, physiological correlates identified in previous studies (e.g. EEG over-activation or overall hyperarousal) should be considered in future research endeavors to uncover more about their hypothesized relationship to misperception of sleep. As mentioned previously, future studies should also take into account the depth of sleep to determine if individuals who perceive themselves to be awake are actually experiencing lower quality or less restorative sleep relative to those who are more accurate in their sleep estimates.

Another important future direction is to examine not only "negative bias," or the tendency to misperceive sleep as more disturbed than what objective measures show, but to examine "positive bias," as well. The current study did not consider this phenomenon but future

research should examine correlates and predictors of this tendency and could examine how those with a positive bias respond to CBT. A final idea for future research, in consideration of the proposed "subjective" and "objective" subtypes of insomnia mentioned previously, is to conduct a comparison of individuals fitting these classifications to determine if different variations of CBT-I are more or less successful for the different forms of insomnia. This would improve our understanding of whether some components of CBT-I are most essential for the treatment of subtypes of insomnia in order to improve treatment efficiency and thereby reduce cost. This would also contribute to the ongoing discussion of how insomnia is best defined.

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## Appendix 1



# SLEEP DIARY

Date:

Name: \_\_\_\_\_

10/9

Example

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>							

## Vita

Hannah Gretchen Lund was born on August 8, 1983, in Northfield, Minnesota, and is an American citizen. She graduated from Northfield High School in 2002. She received her Bachelor of Arts in Psychology from Bates College, Lewiston, Maine in 2006 and subsequently worked as a research coordinator at the Bipolar Clinic and Research Program at Massachusetts General Hospital in Boston, Massachusetts for two years. She began the doctoral program in Clinical Psychology at Virginia Commonwealth University in 2008.