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*Virginia Commonwealth University*

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College of Humanities and Sciences
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# Efficacy of a Brief Intervention for Insomnia Among Psychiatric Outpatients

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

by

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Master of Science, Virginia Commonwealth University, 2007
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Acknowledgements

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# Abstract

EFFICACY OF A BRIEF INTERVENTIION FOR INSOMNIA AMONG PSYCHIATRIC OUTPATIENTS

By J. Nile Wagley, Ph.D.

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

Virginia Commonwealth University, 2009

Major Director: Steven J. Danish, Ph.D.
Professor of Psychology, Department of Psychology

Psychiatric patients are particularly affected by symptoms of insomnia. Because insomnia is often secondary to other conditions and was once thought to be less treatable, this condition has received little attention in terms of treatment and research. Additionally, psychiatric patients have typically fewer resources to seek treatment. Generally, insomnia is treated with medications that may have biological side effects and offer little restorative sleep. Behavioral or cognitive interventions have often been overlooked. This experiment uses profile analysis to test the hypothesis that psychiatric outpatients randomized to a treatment group would have decreased levels of sleep difficulties (measured by PSQI) when levels of depression (measured by PHQ-9) were held constant compared to participants in a control/wait group. Also, it was hypothesized that levels of depression



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would decrease in the treatment group when initial levels of sleep difficulties were held constant compared to participants in a wait group. Levels of sleep difficulty were found to be significantly lower in the treatment group than the wait group at post and follow up. Levels of depression were found to be significantly lower in the treatment group than in the wait group at post and follow up. The treatment group received one, 50-minute, individual therapy session that addressed sleep hygiene, stimulus control and sleep restriction activities and also received an additional telephone session two weeks later. These findings suggest that providing behavioral and cognitive interventions may be a feasible alternative to using pharmacological interventions as a first-line treatment for insomnia.



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# Chapter 1

## Introduction

The National Institute of Health reported in 2007 that 40 million Americans would suffer from a chronic sleep disorder in that year (NIH, 2007). The cost to Americans will be $16 billion in direct medical expenses in addition to lost productivity at work and home, automobile accidents caused by drowsy drivers, decreased personal wellness and fewer satisfying social interactions (NIH, 2007).

Once thought of as a dormant or passive state of being, sleep is now understood to be an important part of physical and mental wellbeing, maintenance and growth. This active state of rest and rejuvenation has many facets that, in concert, have specific and necessary functions. The National Institute on Neurological Disorders and Stroke reported that these functions of sleep are not fully understood (NIH, 2007). Better understood are the symptoms of not getting adequate sleep. The severity of these symptoms suggests that sleep is a necessary component of human survival, health and wellbeing.

Unfortunately, millions of people suffer from a lack of sleep each year (NIH, 2007). Problems with sleep or insomnia can be defined in many different ways (Perlis, Jungquist, Smith, & Posner, 2005). The common components of these definitions are threefold and require each of the following: (1) the inability to fall asleep for a period of time over a number of days; (2) the opportunity to fall asleep is present (graduate school



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may preclude insomnia when the opportunity to sleep is not present); and (3) a significant impairment is experienced during the day.

Insomnia has traditionally been treated with medication that can have adverse side effects and may not improve the amount of restful sleep received. Behavioral and cognitive therapies have been used with success. These therapies have been tested in group formats (Jansson & Linton, 2005), within general adult populations (Bélanger, Savard & Morin, 2006), within child populations (Weiss, Wasdell, & Bomben, 2006) and within older populations (Rybarczyk, Stepanski, Fogg, Lopez, Barry, & Davis, 2005). However, the effects of such interventions are not fully understood, especially with populations such as those with co-morbid diagnoses of depression and other psychological conditions.

A population that is particularly affected by symptoms of insomnia are psychiatric patients (Dashevsky & Kramer, 1998). Because secondary insomnia (secondary to other conditions) was once thought to be less treatable, this population has received little attention in terms of treatment and research (Perlis, et al., 2005). They are also less likely to have resources to find treatment for insomnia. When resources are available, psychiatric patients may also be less likely to take advantage of these opportunities (Dashevsky & Kramer, 1998).

This study will evaluate the implementation of a brief CBT intervention for insomnia among psychiatric patients with a co-morbid diagnosis of depression. An assessment will be made as to whether the principles of insomnia treatment, which have been shown to be effective among other populations, can be incorporated into brief interventions among psychiatric populations experiencing depression. The brief



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interventions, if found to be effective, would be helpful to mental health professionals working in medical settings who have limited time and resources to spend with patients. This specifically answers the call of Danish, Forneris & Wilder-Schaaf (2007) to address the context in which health care is delivered. These brief interventions will make treatment modalities available to less advantaged populations who may not have opportunities to receive them elsewhere.



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Chapter Two

*Literature Review*

The following literature review will delineate the relevant issues leading up to the current research problem. It is important to understand the function and architecture of sleep in order to contextualize the problem of insomnia. Also, links between insomnia and depression will be presented followed by a discussion of the current understanding of the various medicinal and cognitive treatments of insomnia.

*Function of Sleep*

There are many hypotheses about the function of sleep. Some believe that the ancient behavior of sleep originally developed in animals to conserve energy or suppress hunger at night when obtaining food is more difficult (Berger & Phillips, 1995). Another possible protective function of sleep is to allow animals to lie still during their most vulnerable hours and hide from predators (Lima & Rattenborg, 2007). Among humans, more popular beliefs and assumptions of the purpose for sleep have been to consider it a venue for the 'resetting of the brain' or simply a chance to rest physically and mentally (Siegel, 2005).

Scientific inquiry has led to evidence that sleep has a more specific and necessary function. Studies have shown that increased REM sleep occurs after learning and that learning decreases after REM deprivation (Peigneux, Laureys, Delbeuck, & Maquet, 2001;



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Stickgold, Hobson, Fosse, & Fosse, 2001), which may suggest a link between sleep and learning.

The brain is not 'hardwired' but pliable. Information stored in the brain is distributed instead of stored in one location. This redundancy creates a robustness that allows information to be retained if a few synapses or neurons are lost. Also, the information is superimposed; meaning one neuron is responsible for more than one piece of information (Delcomyn, 1997). Though the idea that neuroplastic changes are consolidated during sleep is controversial among many (Crick & Mitchison, 1983), it can be deduced that for the brain to store, distribute and then consolidate the amount of information necessary for functioning, a reprocessing and allocating time would be necessary. It has been postulated that REM is the reprocessing and allocating of this information to create an efficient mind. This idea dovetails with the experience that many people have as they sleep and dream of recent and past events.

Siegel (2001) and Vertes and Eastman (2000) argue that the decrease in learning that accompanies losses in sleep is actually a function of a mediating variable, stress. Other studies produce convergent results that begin to suggest that sleep is necessary for some of the neural plasticity of brain functioning (Frank, Issa, & Stryker, 2001; Hobson, 1989).

Hobson and Pace-Schott (2002) have outlined this process in detail on a neurological level. They concluded that the brain is active during waking hours to interact with the world, is less active during non-REM sleep to allow information to be consolidated in the forebrain, and then reactivated during REM sleep to cortically integrate with previously stored information that was consolidated during non-REM sleep. This



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pattern of sleep or sleep architecture is consistent with brain wave activities measured in humans while asleep.

*Sleep Architecture*

Sleep may have been considered by some to be a passive state of rest (Siegel, 2005). However, sleep is now understood to be an active state with specific frequencies of brainwaves that denote defined stages of sleep (Pinel, 1992). These stages, along with tools used for detecting these stages will be described below.

Three fundamental measures are used to define stages of sleep through the detection of brainwaves. Electroencephalogram (EEG) is used as a gross measure of electrical activity emitted by the brain. Electromyogram (EMG) is used to measure muscle tone. Electro-oculogram (EOG) is used to measure eye movement. These measures are collectively called polysomnography (PSG) (Dement, 1978). Advances in quantitative electrophysiology, the advent of functional neuroimaging and the ability to record the waking and sleep of subjects outside the sleep laboratory, have expanded the information we can gather about brain activity during sleep (Hobson & Pace-Schott, 2002).

Perlis (2005) has described the four fundamental types of EEG activity measured in sleep research. These are beta, alpha, theta and delta waves. Beta waves are associated with wakefulness. They range from about 15 to 35 hertz (Hz) and are the least consistent in their pattern because of spontaneous wakeful activities such as sensory, motor and cognitive tasks. Beta waves are the highest in frequency (more waves per millisecond) and lowest in amplitude (lower peaks and troths).



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Alpha waves become predominate during wakefulness that is more relaxed. These waves are more synchronous with decreased frequency and increased amplitude. Activities that promote alpha waves such as meditation and relaxation may lead to positive health benefits.

The first stage of sleep is characterized by theta waves. These waves are measured by EEG and are slower in frequency and greater in amplitude than alpha waves. Deeper stages of sleep are characterized by delta waves. They are the slowest in frequency and largest in amplitude of brain waves (Perlis et al., 2005).

Pinel (1992) presented an overview of the states of sleep. The first four stages of sleep are known as non-REM sleep and correspond with the four types of brain waves described above. Stage 1 Sleep is the first stage of sleep and also the stage of sleep that occurs when one is aroused from any of the other stages. A person awakened in this stage of sleep may feel as if they were not yet asleep. A person in Stage 1 Sleep may have slow and rolling eye movements. Stage 2 Sleep is deeper than stage one and characterized by delta waves. One may spend up to 50% of a normal sleep period in Stage 2 Sleep. Stage 3 and Stage 4 Sleep are made up of delta wave sleep. These stages are similar and only discriminated by slight variations in the amount of delta waves present.

REM sleep is a fifth stage of sleep that occupies between 15% and 25% of a normal sleep cycle. It is known as the deepest stage of sleep and is characterized by rapid eye movement and paralysis of voluntary muscles.

Stages 1 through 4 usually last 5 to 15 minutes each. These Non-REM stages usually progress in the order of stage 1, 2, 3, 4, 3, 2, REM. Disturbance from any of these



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stages results in a return to Stage 1 Sleep. These various waves organized within stages make up a circadian rhythm. This rhythm is controlled by hormones and influenced by external factors such as light and temperature. This rising and falling rhythm dictates the optimal time for falling and staying asleep (Lack & Wright, 2007). This concert of internal and external factors making up a circadian rhythm has a high inertia and resists change. It has been shown that adjusting to as many of these dictating factors as possible can, in many cases, shift this circadian rhythm when needed. For example, a strong influencing factor to this rhythm is bright morning light. Lack, Wright and Paynter (2008) demonstrated that this morning light can not only reduce sleep latencies and frequency of nocturnal awakenings but can actually adjust the circadian rhythm measured by internal human melatonin release.

The above stages describe the shape of sleep and can be measured objectively by polysomnography. Subjective self-reported estimates such as sleep latency, number of nocturnal awakenings and sleep efficiency are also used to measure sleep and are as useful as more objective measures. Sleep continuity is a measure of how efficiently and effectively these stages are occurring. The five variables of sleep continuity are Sleep Latency (SL), Frequency of Nocturnal Awakenings (FNA), Wake After Sleep Onset (WASO) time, Total Sleep Time (TST) and Sleep Efficiency (SE%) (Perlis, et al., 2005).

*Consequences of Sleep Deprivation*

Because of gender differences, individual sleep-need variances, sleep-need differences among age groups and other variables, it is difficult to predict how much sleep any one individual needs (Perlis, et al., 2005). In humans, too little sleep results in



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drowsiness, reduced levels of concentration and impaired memory and physical functioning (Morin, 1996). More prolonged deprivations from sleep result in hallucinations and mood fluctuations. Rats have a normal life span of 2 to 3 years. Death occurs after 5 weeks if deprived of REM. Total deprivation of sleep from rats results in a more speedy death of only 3 weeks (NIH, 2007).

*Insomnia*

Many in the medical field such as the World Health Organization (1993) define insomnia in the International Classification of Disease (ICD-10) as a problem initiating and/or maintaining sleep for three or more nights a week (WHO, 1992). It may also be a complaint of non-restorative sleep for the same period of time and must be associated with daytime distress or impairment.

The American Psychiatric Association (1994) differentiates sleep problems that are an entity unto themselves, such as primary insomnia, and sleep problems that are a result of some other medical or psychiatric condition (secondary insomnia). In the DSM-IV-TR, insomnia is defined as an independent condition causing significant distress and daytime impairment. It is signified by a predominant complaint or difficulty initiating or maintaining sleep or a failure to achieve non-restorative sleep (APA, 1994).

The American Academy of Sleep Medicine has defined primary insomnia by focusing on the cause of the condition. The International Classification of Sleep Disorders–Revised defines insomnia by how it is initiated and maintained (ASDA, 1997). The term psychophysiologic insomnia is a learned sleep-preventing association that results in complaints of not being able to sleep and feelings of distress during wakeful hours. This



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somatic association is signified by increased muscle tension, rapid heart rate, sweating and other nervous symptoms while attempting to sleep (ASDA, 1997). This definition focuses on the classical conditioning that takes place between bedtime routines, environments and thoughts about sleep.

Beyond these definitions, other signifiers of specificity could be used to classify different types of unwanted arousal from sleep. *Initial insomnia* refers to problems falling asleep. *Middle insomnia* or *sleep maintenance* refers to prolonged periods of wakefulness after falling asleep but before morning. *Terminal insomnia* describes the condition of awakening too early and being unable to fall back to sleep (Perlis, et al., 2005).

Arbitrary distinctions in classifying the duration of insomnia separate acute and chronic insomnia. Acute insomnia is usually considered some sleep condition lasting less than six months. Acute insomnia usually has an etiologic underpinning such as stress, pain or substance abuse. Chronic insomnia lasts for over six months and its causes may be more amorphous (Rajput & Bromley, 1999).

Frequency and intensity of insomnia varies among sufferers of this condition (Rajput & Bromley, 1999). Many who suffer from insomnia do not have symptoms every night. When these symptoms occur, they may occur to different degrees. One may report that he is having daytime distress from a lack of sleep and is experiencing initial insomnia for up to 20 minutes, four times a week. Compared to some, these symptoms may be considered mild. Some have suggested that though somewhat arbitrary, cutoff rates for classifying primary, middle or terminal insomnia should be 30 minutes or greater (Lichstein et al., 2002). This distinction is based on the level of typical complaints that



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arise in population studies. Therefore, this is a definition based on what is acceptable among a culture.

The definitions of insomnia presented in the ICD-10, DSM-IV-TR and ICDS-R are subjective definitions. It allows room for the patient to define her or his own complaint. The subjective components, daytime distress and non-restorative sleep, are helpful because patients can determine if this condition is distressing. Yet, the lack of objectivity can make research in the area less precise. Though this problem is addressed to some extent by distinguishing between primary and secondary insomnia, it is often the case that similar diagnoses are made for very different conditions (Rajput & Bromley, 1999). Restless leg syndrome, sleep apnea, learned sleep-preventing associations, newborn babies in the home, loud neighbors and nocturnal pets may all results in non-restorative sleep and daytime impairments.

The different views of the definition of insomnia have been presented above. Objective and technical views may be helpful in understanding the mechanisms of the condition. More global and patient-centered views may be sufficient in many cases for treating insomnia since it often is the patient's experience of this condition that is being treated.

*Depression and Insomnia*

Five of the nine criteria for major depressive episode in the DSM-IV-TR are related to, highly correlated with or are insomnia itself. Of these nine criteria for depression, insomnia is the most prevalent (Perlis, Giles & Buysse, 2007). Recent studies (National Institute of Health, 2005) suggest that insomnia and depression are more than correlates



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and have more than a simple cause and effect relationship. In fact, these two conditions appear to have a reciprocal relationship in which one or the other may influence, perpetuate, exacerbate or maintain the other (Turek, 2005). A recent pilot study (Taylor, Lichstein & Weinstock, 2007) that treated a small number of participants with a CBT intervention for insomnia revealed that mild symptoms of depression decreased in all participants and were maintained for three months. It is interesting to note that no interventions for depression were administered in this study but the symptoms of depression responded to the CBT intervention for insomnia.

*Treatment of Insomnia with Medication*

Medication has been a common intervention for treating insomnia (Holbrook, Crowther, Lotter, Cheng & King, 2000). Benzodiazepines have been prescribed often for reducing symptoms of insomnia because of their sedative effect. Accompanying this effect are hypnotic, anxiolytic, anticonvulsant, muscle relaxant and amnesic properties. This psychoactive drug slows the central nervous system and make sleep more likely. However, because this drug is habit- forming and comes with many side effects, it should not be a first choice for long-term treatment of insomnia (Espie, 1991). Newer drugs such as Ambien and Sonata are benzodiazepine receptor agonists. Some of the newer drugs are more specific in that they are helpful for falling asleep whereas others are helpful for staying asleep. However, because of side effects and often nominal increases in restorative sleep, they still should not be prescribed without first trying non-chemical strategies and considering the risks (Glass, Lanctôt, Hermann, Sproule, & Busto, 2005). The outcome of these medications is often a hypnotic forgetting of the stress of the restless night without



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adding natural restorative sleep. This effect can be helpful over short time periods after considering the risk of side effects. Genevie`ve Belleville and Charles M. Morin (2008) demonstrated that chronic users of benzodiazepines that stopped using the medication for insomnia experienced fewer symptoms of insomnia, anxiety, distress and increased levels of self-esteem after controlling for readiness to change, current stage of change, levels of psychological distress, health and self-efficacy.

*Cognitive and Behavioral Treatment of Insomnia*

Effective non-medicinal treatments of insomnia will be presented below. It will be shown that several techniques exist that are effective in reducing patients' symptoms of insomnia without the risk of medicine-induced side effects.

Dashevsky and Kramer describe progressive muscle relaxation training, imagery training, biofeedback, and hypnosis as having been used as relaxation treatments for insomnia. Sets of structured exercises are used to teach a person to relax and reduce tension in order to fall asleep. Some medical conditions, medications or psychological conditions could render this treatment useless (1998).

Stimulus control is a method of overcoming cues in the bedroom that contribute to insomnia through classical conditioning (Bootzin, 1972). These methods of establishing good sleep hygiene include using the bed and bedroom only for sleep or sex. This includes avoiding watching television, reading or working in the bedroom. Stimulus control therapies work best when a regular sleep and wake schedule can be established and when daytime napping is avoided. The stimulus control method discourages people from lying in



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bed beyond 20 minutes if not yet asleep to avoid establishing or strengthening a tie between wakefulness and the bed (Bootzin, 1972).

Sleep restriction is a method of reducing symptoms of insomnia by reducing the amount of wakeful time in bed. Some assume that more time in bed will result in more time asleep. However, this time may not be restful sleep or may augment symptoms of insomnia as described above. In Sleep Restriction, sleep logs are used to estimate the time in bed and the time asleep. Once a baseline is formed, a limited amount of time in bed is prescribed which is usually the average time logged sleeping plus 30 minutes. At first, the patient becomes more tired during the day. If daytime napping is avoided, nighttime sleep becomes more consolidated and restful. Time-in-bed prescriptions are increased by 10 or 15 minutes while sleep is relatively good but daytime tiredness continues. Time-in-bed prescriptions are decreased when sleep is non-restful (Spielman, 1987). The goal is to increase the amount of efficient sleep in bed. Sleep pressure is maintained by restricting sleep until the body and mind learn to sleep efficiently.

Cognitive behavioral therapy (CBT) for insomnia addresses irrational beliefs about sleep that are counterproductive to sleep. Examples of common and erroneous beliefs are that everyone should have 8 hours of sleep each night and that sleepless nights will render a person incapable of functioning the next day. Both of these beliefs can increase insomnia symptoms by increasing tension and anxiety. Neither of these beliefs is true. In addition, CBT is aimed at modifying maladaptive sleep habits, reducing autonomic and cognitive arousal and educating patients about healthier sleep hygiene practices (Morin, 1993).



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Evidence for the efficacy of CBT for insomnia is strong. Meta-analysis studies suggest that CBT interventions produce reliable and durable changes in the sleep of patients with chronic insomnia (Morin, Hauri, Espie, Spielman, Buysse & Bootzin, 1999). Reductions in symptoms of insomnia through CBT have been shown to last up to 2 years (Morin, Colecchi, Stone, Sood & Brink, 1999). CBT has been shown to be superior to pharmacological treatments in a meta-analysis study by having more sustained improvements in sleep patterns of participants (Smith, Perlis, Park, Smith, Pennington, Giles & Buysse, 2002) and has been shown to be preferred by patients (Morin, Gaulie, Barry, Kowatch, 1992).

These behavioral interventions have been effective but often not efficient. They take six to twelve sessions and require the expertise of a psychologist trained in CBT interventions for insomnia. Nurses have been given brief training for CBT interventions for insomnia. Subsequently, the interventions delivered by these nurses to participants in a randomized medical trial (Espie, Fleming & Cassidy, 2008) had similar success rates among some participants at reducing symptoms of insomnia as interventions delivered by psychologists. This suggests that efficient interventions may be effective in reducing symptoms of insomnia.

The preceding review of literature details the current understanding of the need for and the function of sleep. The shape of sleep (sleep architecture) and consequences of disruptions in this architecture were presented. Prolonged disruptions may lead to medicinal or cognitive treatments of insomnia with varying effects. Evidence for the efficacy and advantage of cognitive and behavioral treatments was presented and shown



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to be strong.

*Problem*

A population that is particularly effected by symptoms of insomnia is psychiatric patients. Because secondary insomnia (secondary to other conditions) was once thought to be less treatable, this population has received little attention in terms of treatment and research (Perlis, et al., 2005). This population is also less likely to have resources to find treatment for insomnia. When resources are available, psychiatric patients may be less likely to take advantage of these opportunities.

*Hypothesis*

Psychiatric outpatients participating in a brief intervention for insomnia will have significantly lower PSQI scores (a global measure of sleep) at post and follow up compared to a control group not receiving the intervention.

Also, psychiatric outpatients participating in a brief intervention for insomnia will have significantly lower PHQ-9 scores (a measure of depression) at post and follow up compared to a control group not receiving the intervention.



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# Chapter Three

# Method

*Participants*

Thirty-four volunteer adult outpatients of a psychiatric hospital housed inside a medical school were recruited as participants. This clinic serves individuals with a full range of socio-demographic characteristics but consumers of services tend to have lower incomes because of the training component present in the clinic. Participants accepted into this study scored 5 or greater on the PSQI and 10 or greater on the PHQ-9. This indicates that the population is made up of participants with significant co-morbid symptoms of depression and insomnia.

The mean age of participants was 44.9 years of age with a range of 21 to 62 years. Females made up 73.5% of the study group, whereas males accounted for the remaining 26.5%. Self-reported racial make-up of the study population indicated that 55.9% of the population identified themselves as African-American and 44.1% identified themselves as Caucasians. Some 20.5% of the group reported that they did not complete high school; 61.9% of participants did complete high school. The remaining 17.7% of responders completed college or a post-graduate degree. Table 1 below describes the participants according to their randomly assigned group.



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Table 1

*Description of participants by group*

| | Treatment | Wait |
|---|---|---|
| Gender | | |
| Female | 18 | 7 |
| Male | 6 | 3 |
| Mean Age in Years | 43 | 48 |
| Race | | |
| Caucasian | 16 | 3 |
| African-American | 8 | 7 |
| Marital Status | | |
| Married | 7 | 0 |
| Single | 8 | 6 |
| Separated or Divorced | 9 | 4 |
| Education | | |
| Completed High School or Less | 10 | 5 |
| Some College or More | 14 | 4 |

*Measurement*

*Pittsburgh Sleep Quality Index* (PSQI; Smyth, C., 2003). The PSQI is a 9-item self-report measure of insomnia. This global sleep index discriminates between good and poor sleepers (Smith & Wegener, 2003). The questionnaire measures seven different sleeping



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areas including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime dysfunction over the previous month. Scores are based on a 0 to 3 Likert-type scale in which '3' signifies poorer sleep. Global scores range from 0 to 27 and are the result of an algorithm that weighs index scores to compute an outcome. Poor sleep quality is operationalized by a global score of 5 or greater.

Internal consistency has been demonstrated to be high with overall Cronbach's alpha of the seven component scores equaling .83 (Smith & Wegener, 2003). Test-retest reliability has been demonstrated by t-test comparisons that failed to show significant differences between time one and 28 days later (Smith & Wegener, 2003). The product-moment correlation between the two times was .85 $p < 0.001$. Criterion validity has been established (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) showing that the PSQI can distinguish good from poor sleepers as accurately as can the integration of an expert administered clinical exam, physical exam and polysomnographic testing. The PSQI has been shown to be able to assess change after an intervention. In clinical trials of CBT interventions, the PSQI scores significantly decreased in participants who participated in the intervention (Currie, Wilson, Pontefract, DeLaplante, 2000).

*Patient Health Questionnaire - 9* (PHQ-9; Spitzer, Kroenke, & Williams, 1999). The PHQ-9 is the depression module of the broader Patient Health Questionnaire (PHQ). The PHQ-9 assesses each of the 9 DSM-IV diagnostic symptoms of depression with Likert-type ratings on a "0" (not at all) to "3" (nearly every day) scale (Range = 0-27). Psychometric qualities of the PHQ-9 have been assessed in multiple, large-scale studies



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involving primary care (Spitzer, Kroenke, & Williams, 1999), ob/gyn (Spitzer et al., 2000), and in a general, non-medical population (Martin et al., 2006). The PHQ-9 demonstrated high internal reliabilities (.89 and .86), forty-eight hour test-retest reliability (.84), and good evidence of criterion validity by having a reliable inverse relationship with health-related quality of life measures (Kroenke, Spitzer, & Williams, 2001). The PHQ can be meaningfully applied as a diagnostic proxy for depression (score > or = 10 has 88% sensitivity and 88% specificity), and provides a continuous measure of severity (Total Score). The PHQ – 9 has been found to be a practical and responsive tool for pharmacologic treatments for depression among men and women (Löwe, Schenkel, & Carney-Doebeling, 2006).

*Procedure*

Passive recruitment occurred by means of a sign display in the reception area of a psychiatric outpatient clinic. Volunteers consented after being informed of the details and limits of their involvement in this study. After consent was given, the participants completed the PSQI and PHQ-9 while waiting for his or her psychiatric appointment. Any participant returning a PSQI with a global score greater than 5 and also reporting PHQ-9 scores of 10 or above was randomly assigned to either the treatment or control group. To obtain sufficient power to detect differences between the two groups, twenty-four participants were randomly assigned to the treatment group and the remaining ten participants were randomly assigned to the control group. Power estimates were conducted to determine the size of the sample (Cohen, 1988). Four interrelated variables contributed to this calculation. The ratio of any three of these variables (power, effect size, alpha and



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sample size) can determine the fourth variable. Because these variables can not be known before the experiment takes place, an estimate of three of these variables can be set and used to estimate the fourth. In this case, sample size was determined by estimating the other three. With estimates of medium effects size (.4), alpha set to $p < .05$ and power levels estimated at .80, SAS (FPOWER) was used to estimate the appropriate number of participants needed.

*Treatment group.* Participants in the treatment group received the brief insomnia intervention immediately after giving informed consent. This included a 60-minute CBT session (described below and in Appendix A) and a take-home pamphlet reviewing essential points of the session. Two weeks following the first session of the intervention, a phone interview occurred and focused on resolving any concerns pertaining to recommendations given in the first session and addressing any questions about the treatment. Two weeks following the second session, the post measures (PSQI and PHQ-9) were mailed to each treatment participant with a self-addressed and stamped envelope. Four weeks following this measurement, the follow up measures (PSQI and PHQ-9) were again mailed to each treatment participant with a self-addressed and stamped envelope. After three days of mailing, each measurement packet sent was followed by a phone call to encourage the return of the materials. Any participant who did not return data after two weeks of mailing received a phone call to ask for oral report of the data. Non-returned or reported packets constituted missing data which was not analyzed .

It was anticipated that some participants would become unavailable for various reasons during the study at a rate no larger than the number who would drop out of such a



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treatment for insomnia in a non-study intervention. Reasonable efforts were made to assist these participants to remain in the study such as being sensitive to individual circumstances and inquiring by way of telephone to determine if any resolvable concern existed.

*Control group.* Participants in the control group were provided pre-measurements (PSQI and PHQ-9) as described above at their initial visit. Two weeks later, they received a phone call reminding of their participation in the study and that they would receive the measures in two weeks. Two weeks following the phone call, post measures (PSQI and PHQ-9) were mailed to each control participant with a self-addressed and stamped envelope. Four weeks following this measurement, follow up measures (PSQI and PHQ-9) were again mailed to each control participant with a self-addressed and stamped envelope. Finally, the control-group participants were scheduled for an in-person appointment to receive the treatment protocol after their last measurement was received. Any participants not completing all measurements were offered the treatment protocol.

*Intervention*

The intervention was a two-session treatment that included one 60-minute in-person session and follow up session via telephone. See Appendix A for details. This protocol was modeled after a design that was tested and found to have consistent positive effects in producing reductions in wake time after sleep onset and reductions in insomnia symptoms among veterans (Edinger, & Sampson, 2003). In addition, each treatment-group participant during the CBT session was given a brochure detailing important points of the session (Appendix B).



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*Analysis*

Data were inspected for outliers. Measures of normality (skewness and kurtosis) were evaluated. Homogeneity of variance, linearity and multicolinearity of the dependent variable were evaluated to insure assumptions of the statistical method were not violated.

Profile analysis was used to test the null hypothesis that global post and follow-up sleep measures were not significantly different from baseline sleep measures while controlling for initial levels of depression. Profile Analysis was then used to test the null hypothesis that global post and follow-up depression measures were not significantly different from baseline depression levels while controlling for initial levels of sleep difficulties.

Graphical analysis was conducted to determine direction of differences if the null hypothesis is rejected. To demonstrate significance, the null hypothesis must be rejected at the $p < .05$ level.



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Chapter Four

Results

Prior to analysis, the dependent variables for sleep difficulties and depression (PHQ-9 initial, PHQ-9 post, PHQ-9 follow-up, PSQI initial, PSQI post and PSQI follow-up) were examined through SPSS (statistical package for the social sciences) for accuracy of data entry, missing values and fit between their distributions. Only one missing value was discovered that could not be recovered which resulted in the loss of the only dropped data in this study. Results of measures of skewness revealed that all instances of the dependent variables fell within 2-times the standard error of the mean which indicated a significant level of skewness did not exist. Measures of kurtosis showed that neither significant flatness nor steepness existed among the outcome variables. Each measure fell within 2-times the standard error of the mean for kurtosis.

The dependent variables were examined for univariate and multivariate outliers. The conservative estimate of 2 standard deviations of the mean was used as a criterion to identify univariate outliers. No instance among the variables fell outside of this criterion. Mahalanobis distances were computed by linear regression. No value within these distances exceeded the criterion value indicating that no multivariate outliers existed among the dependent variables, $c^2(6, N = 33) = 16.81, p > .05$.

A profile analysis was performed comparing the profile of the level of sleep difficulties of the treatment group with the profile of the level of sleep difficulty of the control group over the time of the three measurement points with initial levels of



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depression held constant for all participants. The group by time interaction for level of sleep difficulty was statistically significant which indicates a deviation from parallelism of the two independent groups, $F(2, 33) = 5.29$, $p < .05$, partial $\eta^2 = .43$. Sleep difficulty scores among the treatment group changed over time in comparison to and independently from the wait group.

Table 2

*Descriptive statistics for dependent variables by treatment group and wait group*

| | Treatment Group | | | Wait Group | | |
|---|---|---|---|---|---|---|
| | M | SD | SE%* | M | SD | SE%* |
| PSQI Initial | 13.7 | 5.0 | 65.1 | 14.78 | 3.38 | 69.9 |
| PSQI Post | 11.8 | 5.0 | 74.5 | 14.3 | 3.50 | 69.2 |
| PSQI Follow up | 9.42 | 5.67 | 74.8 | 15.6 | 3.20 | 68.4 |
| PHQ-9 Initial | 15.75 | 7.29 | | 19.3 | 4.03 | |
| PHQ-9 Post | 10.88 | 7.25 | | 19.3 | 3.47 | |
| PHQ-9 Follow up | 9.58 | 6.62 | | 19.1 | 3.9 | |

* Average Sleep Efficiency derived from reported time asleep divided by time spent in bed



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Figure 1 below plots the profiles of sleep difficulties for the two groups over the time of the three measurement points. Whereas there was no significant difference between the treatment and wait control group before the intervention, sleep difficulty scores declined for the treatment group at four weeks and eight weeks whereas the wait group remained relatively stable over the same times. Thus, there was a decrease in sleep problems while controlling for initial levels of depression over time in the treatment group that was not observed in the wait group.



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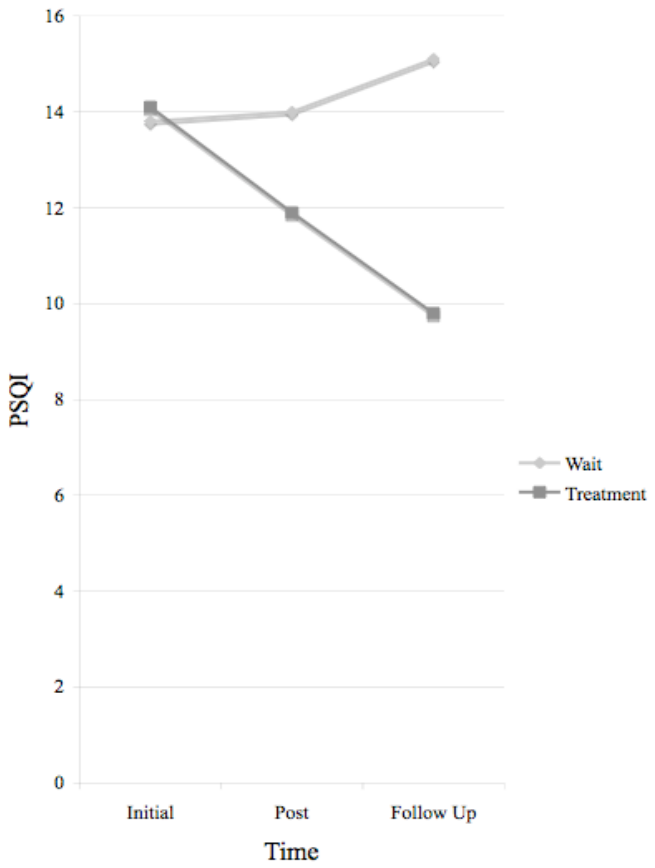


*Figure 1:* This chart demonstrates the estimated marginal means of participants reported levels of difficulties sleeping at initial, post and follow up measurements.

As demonstrated above, multivariate analysis detected average improvements in sleep between treatment and wait groups. Case wise analysis also demonstrated differences



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in sleep between these groups. Of the treatment group participants, 87% improved in measures of sleep. Of the control group, only 30% had improvement in sleep. These improvements were demonstrated by a marginally small rise in sleep scores. Seventy percent of this group remained the same in their level of sleep difficulty or got worse over the course of the study.

In addition to the comparisons made of sleep, the profile analysis compared the profile of the level of depression of the treatment group with the profile of the level of depression of the wait group over the time of the three measurement points with initial levels of sleep difficulties held constant for all participants. The group by time interaction for level of depression was statistically significant which indicates a deviation from parallelism, $F(2, 33) = 8.34$, $p < .01$, partial $\eta^2 = .37$. Depression scores among the treatment group changed over time in comparison to and independently from the wait group.

Figure 2 plots the profiles of depression levels for the two groups over the time of the three measurement points. Whereas there was no significant difference between the treatment and wait group before the intervention, depression scores declined after treatment at four weeks and eight weeks while the wait group remained relatively stable over the same times. Thus, there was a decrease in depression while controlling for initial levels of sleep difficulties over time in the treatment group that was not observed in the wait group.



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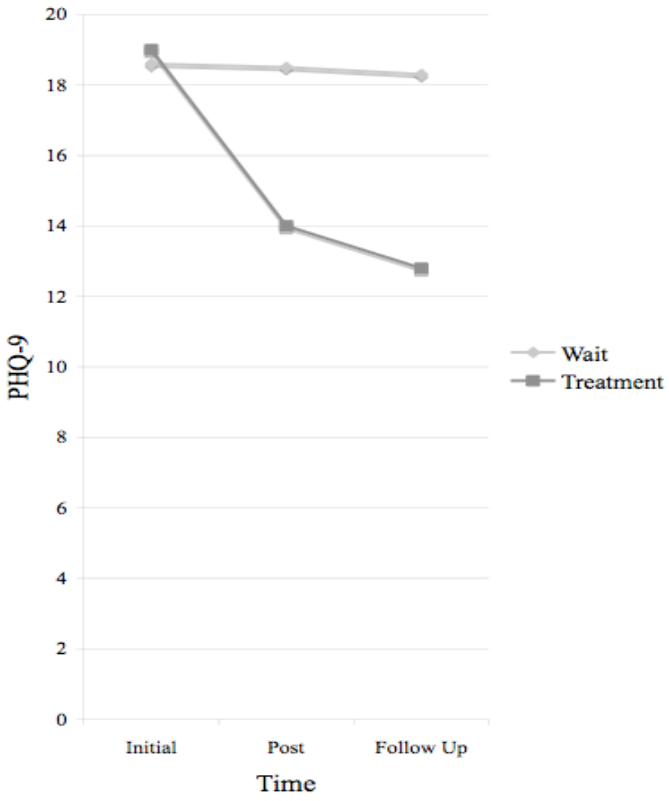


*Figure 2:* This chart demonstrates the estimated marginal means of participants reported levels of depression at initial, post and follow up measurements.



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## Chapter Five

## Discussion

The findings of this research support the two hypotheses proposed. Psychiatric outpatients participating in a brief intervention for insomnia have significantly lower PSQI scores, a global measure of sleep, at post and follow up compared to a control group not receiving the intervention when initial levels of depression are held constant. Also, psychiatric outpatients participating in the same brief intervention for insomnia have significantly lower PHQ-9 scores, a measure of depression, at post and follow up compared to a control group not receiving the intervention when initial levels of insomnia are held constant. The amount of variance accounted for by these variables was small which is expected in such psychological constructs. The condition of insomnia in humans has many contributing factors such as genetics, changing brain and body chemistries, seasons, medications, drugs, mental illnesses, daily stressors, worry and life events, all of which have a different impact on the unique individuals they effect. However, because the factors listed above may be closely correlated with symptoms of insomnia, decreases in sleep difficulties have the potential to have a synergetic effect on many areas of individuals' lives. A small improvement in restful sleep may help improve other areas of difficulty and make improvement in these areas or life more likely. An important area where this synergy was observed in this study is in the reduced levels of depression found after this treatment for insomnia was administered. Contrary to beliefs that insomnia is often secondary to depression, depression may in some cases be secondary to insomnia. Some unresponsive instances of depression may be unresponsive because the root problem, in some of these cases insomnia, is not being addressed.



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These findings have clinical and financial implications. Because medicinal interventions for insomnia often have biological side effects and because they often only offer nominal increases in restorative sleep, they should be prescribed for insomnia after evaluating the possibility of non-chemical strategies. The findings of this study add support to the evidence that has been accrued that indicates that behavioral strategies for insomnia such as CBT are an effective and implementable tool for reducing symptoms of insomnia. At first, prescription of medication seems to be the most efficient treatment for insomnia because it takes little time and physician resources. However, non-chemical interventions increase the chance that individuals experience the benefit of increased restful sleep that is more enduring, has fewer or no biological side effects and is less expensive when considering the price of medication, repeat visits to continue medication, treatment for addiction to medication and reduction in other psychiatric symptoms that come with actual improved restful sleep.

As is the case in many research projects, an equilibrium that ranges from complete laboratory control to real-life validity must be negotiated. This equation can only be balanced by understanding the problem of the research project. In this study, the central question required implementation and evaluation of a real-life intervention for insomnia in a psychiatric outpatient setting. Previous research has addressed the theoretical and more controlled components of this issue in lab settings. Creating a 'real-life scenario' was a principle that guided decisions in designing this study. For example, when deciding on the amount of control to place on the treatment protocol (to use a word-for-word script or to let the intervention be guided by CBT principles), it was decided to follow the path that most



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approximated how service providers in real life would implement such an intervention and use a principle-based protocol. This decision-making was used throughout this project to make this a useful study for those interested in CBT for insomnia at the actual implementation level. Laboratory exactness is not always desirable when this reliability does not exist in the real world.

However, other lines of research would reveal the elements of this intervention that accounts for the actual results. It would be useful to understand the amount of variance that was accounted for by individual differences, the stage of change of the participant or interpersonal factors present in the therapist/participant relationship. Differentiating among the various elements of depression being influenced by this intervention in a factorial analysis of individual symptoms would begin to define the nature of depression and its synergetic relationship to insomnia. It would also reveal the specific elements of the treatment that are necessary which would lead to more efficient, personalized and targeted treatment plans for individuals.

As mentioned above, the CBT treatment for insomnia reduced levels of insomnia and, at the same time, reduced levels of depression. The DSM-IV-TR criterion for Major Depression includes nine elements of depression (five of which must be met to qualify for the diagnosis). Of these nine elements, four have a direct etiology to, are indistinguishable from or are insomnia itself. These elements are anhedonia, psychomotor agitation, fatigue, diminished ability to think or concentrate and insomnia itself. The descriptions of insomnia and depression are so closely related that it is not surprising that an affective treatment for insomnia would also be affective for treating depression. Given the illusive and insidious



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nature of depression, future studies in this area may open new paradigms for understanding depression and may define new treatments that treat the root cause that may be insomnia in some cases.

One limitation of the study is the lack of multiple providers conducting the intervention. One researcher provided the intervention for all participants. In many real-life situations, these sets of principles would be taught to a team of service providers who would each interpret and deliver the intervention with their own style and individuality. An improvement on this study and an area for further study is to examine the implementation of the principles of this intervention for insomnia at the training-of- service-providers level. A study that compared the reliabilities among different deliverers of this intervention to the results of this study would be useful to further understand the actual implementation of such a study at the institution level.

Several principles were followed in collecting data for this project that were not only important for data integrity in evaluating the findings but also translated into principles that can be used to make this intervention effective in real-life situations. Personal attention to individuals' unique needs, sound record keeping and follow-up tracking in this study resulted in very low instances of missing data and high rates of participant follow-through, which will be delineated below.

It was assumed in this study that there is not one intervention that will 'fix' all problems with sleep for everyone. Therefore, diversity among individuals was not only assumed but addressed and valued. The protocol for intervention outlines this by providing room for building a relationship of trust with each participant and making space for the



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participant to ask questions. This stance engenders trust in the participant that his or her service provider is invested in understanding his or her uniqueness, invites the participant to be invested in the process and would account for a certain amount of variance when implementing such interventions. For example, in the study a man from a Southeast Asian island enrolled in the study after moving to the area without his wife who remained in her country. The man was surprised in the planned post-treatment phone call when the researcher asked about the pending status of his wife's travel to the states. He later, at follow-up, confessed that he was less invested in the study until he learned that the clinic was not only interested in his data but also interested in him as a person. In a post-study questionnaire, he said this made him want to fulfill his agreement to implement the principles of the intervention for the full time of the study. This personal attention not only contributed to more complete data collection, but also is an example of a clinical treatment principle that is part of the real-life nature of psychological interventions.

Second, sound and efficient record keeping is necessary to keep up with the information described above. Process notes that were short and contained pertinent information were kept after any interaction with participants. These notes were written immediately after each interaction and completed in less than 60 seconds each. They contained a sentence describing what transpired and a sentence describing what should be followed up on in the next interaction. Also, a free online calendar program was used without any identifying information about participants to make precise implementation of the components of the intervention possible. The simple calendar program could be accessed and edited by any member of the research team. Upon admission to the study, an



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assigned number was given to the participant and entered into the calendar. The calendar was programmed to automatically schedule each step of the intervention for the individual and track returned data.

Knowing exactly which data had not been returned on time made the principle of follow-up tracking possible. When data were not received, the researcher could immediately call and resolve any concern that was preventing the return of the data. Often, the participant had simply forgotten and was happy to drop the envelop in the mail. Other instances involved questions about procedure that were easily explained. This technique was instrumental in compiling a complete data set with only one missing response in the whole study. Additionally, the tracking systems resulted in greater statistical power to detect and lower the probability of Type II error as well as address the real-life problem of high drop-out rates among clients of low SES outpatient clinics.

In this study, a real limitation that existed was the low effect size. Ideally, a set of principles for decreasing sleep problems and co-morbid depression could be implemented in outpatient clinics and cure insomnia for anyone participating in the study. In reality, only a small amount of variance can be attributed to any psychological intervention. Other real factors such as an individual's ability and willingness to change, timing, external resources and internal resources influence the efficacy of the intervention. Marginal means of the dependent variables in the results of this study showed average decreases in sleep problems and levels of depression for those receiving the insomnia treatment, which is an accepted form of statistical analysis for psychological studies. However, this form of analysis does not take into account the reality of the human condition. Instead, it lumps all



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participants together in respective groups and shows that when looking at averages between groups, this intervention creates some change in one group compared to another. Theoretically, a few really large effect sizes among individuals in one group could create the illusion that that group is unique because of the treatment, when in reality, most participants in the group did not benefit. However, a case-by-case analysis of this data shows that most individuals in the treatment group had some improvement in sleep difficulties and depressive symptoms and few participants got worse. This is evidence that though effect size is low when all change is averaged within groups, the intervention itself is helpful in reducing these symptoms for enduring amounts of time for most people to varying degrees depending on other, less controllable factors.

These other factors can be better accounted for in future evaluations of similar CBT interventions for insomnia by following recommendations (Davidson, Feldman-Stewart, Brennenstuhl and Ram, 2007) in a qualitative study that incorporated participant feedback of roadblocks to effective treatment of insomnia. Feedback included the findings that many patients wanted more recognition of the reality of sleep difficulties by health care providers. They felt that insomnia was not regarded as a legitimate medical complaint. These same patients felt that not enough information was provided about sleep difficulties and options for treatment. In the present study, this information about options was provided by way of take-home brochure. See Appendix B.

Recent research has already been conducted that explores these other factors that reduce the effect size of insomnia treatments. Specifically, Vincent, Lewycky, and Finnegan (2008) found that those who comply with sleep restriction and stimulus control



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interventions almost always make improvements in symptoms of insomnia. The next logical question, which was addressed in the same study, showed that those who do not comply with sleep restriction and stimulus control are those who perceive barriers to compliance such as boredom and annoyance with the treatment. Addressing these concerns of individuals in treatment for insomnia may increase the average effects size of such treatments. Others, such as Constantinio, Manber, Ong, Kuo, Huang, and Arnow (2007) have recently shown that other common factors including patient expectation and therapeutic alliance are effective at distinguishing those who receive benefits from therapy and those who do not. Patients who perceived their therapist as critically confrontive or low in affiliation were less likely to gain benefits in restful sleep after CBT interventions. Attention to these factors will raise the probability of increased average effects sizes in such treatment protocols.

Adequate information is currently available to make sound recommendations for the treatment of insomnia in many populations. The next step is implementation. Research that demonstrates real-life, cost-effective, successful implementation models of insomnia treatments in many settings will be most important in decreasing costs and side effects associated with automatic reliance on pharmaceuticals as a primary treatment for insomnia. Being able to efficiently teach the treatment principles of insomnia to health care providers at many levels and through many modes such as video, web-based training and literature will decrease the negative outcomes of poor sleep and may help decrease stress that contributes to symptoms associated mental illnesses such as depression and anxiety.



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Appendix A

Brief Insomnia Protocol for Treatment

Tasks:

- Overview of brief CBT for insomnia
- Bedtime habits
- Stimulus control
- Sleep restriction strategies
- Take-home sleep log and summary of session

1. Introduce self and study. Build report and gain useful information by expressing genuine interest in some details of participant's life.

2. Explain overview of treatment
    - Time (meet for one hour and follow up with three phone calls)
    - Process by which we can train our bodies to sleep better without using drugs
    - Will be asked to do several things for several weeks which will result in better sleep
3. Bedtime Habits
    - Explain how certain things can hinder sleep
    - Using caffeine, nicotine or alcohol near bedtime (reduce as much as possible)
    - Exercising fewer than four hours before bedtime
    - Discuss each with participant

4. Stimulus control
    - Avoid all activities in bed to sleep and sex (no TV, working, computers, reading, resting, listening to music, etc)

5. Sleep restriction activities
    - No napping during the day (discuss implications; give time for questions and concerns)
    - Set regular time for going to bed and waking up

6. Wrap up
    - Present summary inquire about additional questions and concerns, give contact information, express confidence in participant and program



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Appendix B

Sleep Brochure for Treatment Intervention page # 1

**Quick Reminders for Better Sleep:**

- Avoid caffeine completely or at least after dinner time.
- Avoid vigorous exercise two hours before bedtime.
- Avoid nicotine two hours before bedtime.
- Avoid alcohol use two hours before bedtime.
- Avoid heavy meals two hours before bedtime.
- Have a regular bed time and wakeup time each day when possible.
- Begin a relaxing bedtime routine 30 minutes before bedtime.
- Do your best to create a relaxing and comfortable bedroom atmosphere.
- Do not lay in bed awake for long periods of time. After 20 minutes, leave your bedroom and do something relaxing until you feel sleeping again.
- Avoid daytime naps.
- Do your best to follow these guidelines as often as possible. Don't stress out if you are unable to do them all.
- Remember that it may take some time before you see your sleep improve. Keep trying and be hopeful.

For any questions about this study please contact
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-or-

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*Getting the Sleep You Need*

MCVHS Sleep Study

*Appendix B continued on next page



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Appendix B (continued)

Sleep Brochure for Treatment Intervention page # 2

**Why is Sleep so Important?**

Our bodies need regular sleep to function properly during the day. Good sleep leads to alertness during the day. We will function and feel better when we get between six and eight hours of sleep each night.

**Why am I having problems sleeping?**

Many things can cause sleep problems. Some have illnesses such as sleep apnea that make it difficult to sleep. Most people with difficulties sleeping can reset their "sleep clock" by making simple adjustments to their bedtime behaviors.

**Is there anything I can do make my sleep better?**

Yes! The ideas in this pamphlet have been helpful to other people in getting better sleep and feeling better during the day.

**Sleep Hygiene**

Here are five easy steps you can take that may improve your sleep immediately:

**1.** Avoid caffeine in coffee, tea, some sodas, energy drinks, chocolate and some pain relievers. Most chocolate has very little caffeine compared with the other items listed above. If you feel you can not avoid caffeine all day long, do not use it after dinner. Caffeine can reduce the quality of your sleep for up to five hours after ingesting it.

**2.** Avoid vigorous exercise two hours before bedtime. Exercise wakes your body up.

**3.** Avoid nicotine two hours before bed time. Nicotine is a stimulant and may reduce your ability to fall asleep.

**4.** Avoid alcohol for two hours before bedtime. Some feel that they can fall asleep more quickly after drinking alcohol. This may be true in some cases. However, alcohol will reduce the overall quality of your sleep and may prevent you from sleeping soundly through the night.

**5.** Avoid heavy meals two hours before bedtime. While you should not go to bed hungry, a large meal near bedtime may upset your stomach and cause poor sleep.

**Your Bedroom Might Be Keeping You Awake!**

Sometimes, a connection forms between our bedroom and being awake. This happens when we do other things in our bed other than sleeping. For example, watching TV, writing bills, reading, talking on the phone or working in bed may train our body to be awake when we lay down to try to sleep. This may have happened to you if you often feel sleepy until the moment your head hits your pillow…then you are instantly awake. Follow these steps to break this connection:

**1.** Set a regular time to wake up each morning and follow it 7 days a week. It is important to follow this wakeup time even if you did not sleep well the night before.

**2.** Begin a bedtime routine of relaxing that begins 3o minutes before you go to bed but does not happen in the bed. This may include taking a relaxing bath, reading a relaxing book, meditation or prayer or enjoying a non-caffeinated warm herbal tea. You should avoid any stressful activity, phone conversations, stimulating TV programs or anything that does not help you feel more relaxed.

**3.** Do your best to make sure your bed and bedroom is comfortable and quiet.

**4.** Relax in bed until you fall asleep. If you feel like you have been in bed for over 20 minutes, do not lie there and try to go to sleep. Get up and do something relaxing until you feel sleepy again. Then, relax in bed for another 20 minutes unlit you fall asleep. Repeat this until you fall asleep.

**Things to remember**

Each of the things described in this pamphlet have helped people in the past get more quality sleep and feel better during the day. Each person is different and these may not all be possible or work for you. Do your best with each one as often as you can. Do not stress out or give up on getting good sleep if you can not do each one perfectly each night. Remember, one night or poor sleep is not awful. You can do okay during the day with poor sleep for one or two nights. Continue to try these things and your sleep may improve gradually over the course of a few days or weeks.



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Appendix C

## Pittsburgh Sleep Quality Index

**Instructions:** The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

### During the past month,

1) When have you usually gone to bed? _____

2) How long (in minutes) has it taken you to fall asleep each night? _____

3) When have you usually gotten up in the morning? _____

4) How many hours of actual sleep did you get that night? (This may be different than the number of hours you spend in bed) _____

| 5) **During the past month, how often have you had trouble sleeping because you...** | Not during the past month (0) | Less than once a week (1) | Once or twice a week (2) | Three or more times a week (3) |
|---|---|---|---|---|
| a) ...cannot get to sleep within 30 minutes | | | | |
| b) ...wake up in the middle of the night or early morning | | | | |
| c) ...have to get up to use the bathroom | | | | |
| d) ...cannot breathe comfortably | | | | |
| e) ...cough or snore loudly | | | | |
| f) ...feel too cold | | | | |
| g) ...feel too hot | | | | |
| h) ...have bad dreams | | | | |
| i) ...have pain | | | | |
| j) ...other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s): | | | | |
| 6) During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep? | | | | |
| 7) During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity? | | | | |
| 8) During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done? | | | | |
| 9) During the past month, how would you rate your sleep quality overall? (circle one) | Very good (0) | Fairly good (1) | Fairly bad (2) | Very bad (3) |



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Appendix D

## PATIENT HEALTH QUESTIONNAIRE (PHQ-9)

**NAME:** _____ **DATE:** _____

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

| | |
|---|---|
| **10.** If you checked off any problems, how *difficult* have these problems made it for you to do your work, take care of things at home, or get along with other people? | **Not difficult at all** ____<br>**Somewhat difficult** ____<br>**Very difficult** ____<br>**Extremely difficult** ____ |

PHQ-9 is adapted from PRIME-MD TODAY, developed by Drs Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke, and colleagues, with an educational grant from Pfizer Inc. For research information, contact Dr Spitzer at rls6@columbia.edu. Use of the PHQ-9 may only be made in accordance with the Terms of Use available at http://www.pfizer.com. Copyright ©1999 Pfizer Inc. All rights reserved. PRIME-MD TODAY is a trademark of Pfizer Inc.



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VITA

James Nile Wagley was born the oldest of four boys to James and Gena Wagley in Metter, Georgia on the 30th of May 1976. His grandmother went to college in Louisiana and worked as a music teacher and advocate for children. His father is now a social worker and his mother is a nurse. This tradition of helping led Nile to study psychology and earn a bachelors degree from The University of Georgia in 2003 where he met and married his wife, Renell. Family is a priority in Nile's life, and his two sons, Athen and Van, were born during the following graduate school years. He received a masters degree in counseling psychology from Virginia Commonwealth University in 2006, and will complete his doctorate in counseling psychology from the same university in 2009. He has often found himself working among under-served populations including at the Center for Psychological Services and Development as and assistant director, at Northwest Georgia Regional Hospital as a therapist, as an undergraduate instructor at VCU, as a teacher at S.PA.RK. alternative school and as a ropes course facilitator among disadvantaged youth.



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