Treating Chronic Insomnia:
A Cognitive-Behavioural Group Therapy Approach

by

Alfonso Marino

A thesis submitted in conjunction with the requirements for the Degree of Doctorate of Education, Department of Adult Education, Community Development and Counselling Psychology
Ontario Institute for Studies in Education of the University of Toronto

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TREATING CHRONIC INSOMNIA:
A COGNITIVE-BEHAVIOURAL GROUP THERAPY APPROACH
Alfonso Marino, Ed.D., 2001
Department of Adult Education, Community Development and Counselling Psychology
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ABSTRACT

Insomnia is the most common of all sleep disorders and its chronic form is associated with significant morbidity. Treatment of insomnia has predominately focused on hypnotic and over the counter-remedies, which do not offer long term relief. Research has shown that individual cognitive-behavioural therapy has been effective in the treatment of insomnia. In comparison, there is limited research which has explored the efficacy of cognitive-behavioural group therapy, in the treatment of chronic insomnia. This study examined the efficacy of cognitive-behavioural group therapy (CBGT) in the treatment of chronic insomnia with 35 participants who took part in CBGT and 35 wait-list control. Measures included the Walter Reed Performance Assessment Battery (PAB), Sleep Diary, Insomnia Severity Index (ISI), the Beck Depression Inventory II (BDI-II) and the Beck Anxiety Inventory (BAI). Outcome assessment was conducted at pre-intervention, as well as two weeks, three months and six months post intervention. CBGT participants were found to score significantly better on all sleep and cognitive performance measures at two weeks post therapy and to show continued improvement up to three months post treatment, when compared to 35 participants (wait list control) who did not receive the CBGT treatment. The study suggests that CBGT may be an effective and cost-efficient alternative to individual cognitive-behavioural therapy in the treatment of chronic insomnia.
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Chapter 1

Introduction

Insomnia is a problem that has affected most individuals at one time or another during their lives. Although the intensity and frequency of insomnia varies, it is a widespread and persistent problem that affects health, mood, performance, and relationships (Lacks & Morin, 1992, Mellinger, Balter & Uhlenhuth, 1985). It is the most common of all sleep disorders and the most frequent health complaint after pain (Morin, 1993). Its prevalence has been reported to be as high as 35% in the general population (Mellinger et al., 1985). Ironically, less than 20% of individuals with insomnia ever discuss it with their doctors (Shapiro, MacFarlane & Hussain, 1994). Insomnia is usually discussed during visits for other medical purposes. Furthermore, 40-60% of individuals with insomnia only acknowledge a sleep problem when specifically asked about it (Dement, 1991). In clinical practice, it has been reported that insomnia sufferers seek treatment after enduring the problem for an average of 12 years (Morin, Sonti, McDonald, & Jones, 1992).

Insomnia can contribute to a diminished quality of life. Increased daytime sleepiness and fatigue can inhibit participation and enjoyment of everyday activities that individuals without insomnia take for granted. There is an increase for accidental physical injury as well as long-term effects on general health and life expectancy (Shaprio et al., 1994). Even so, very few individuals with insomnia choose to do something to treat their problem. It is estimated that 85% of insomnia sufferers remain untreated (Mellinger et al., 1985). What is more astonishing is that individuals with insomnia that do seek help eventually stop as their sleep problem continues (Dement,
1991). The reasons for this could be related to two public misconceptions about insomnia. The first is that insomnia is not really a medical problem and therefore “it will go away on its own”. Secondly, chronic insomnia can only be conquered with the administration of sleeping pills (which are seen by some as dangerous or not helpful) (Dement, 1991). It has been reported that up to 80% of individuals with insomnia treated with medication in the past, re-emerge years later with the same problem (Morin, 1993). This suggests that on its own, medication is not an effective mode of treatment for many individuals with chronic insomnia.

Cognitive-Behavioural therapy (CBT) has been found to be an effective approach to treating insomnia. This psychotherapeutic intervention has been one of the more successful forms of therapy, in that it treats the behavioural and cognitive disruptions associated with insomnia (Espie, 1991; Lacks & Morin, 1992; Morin, Culbert, Kowatch & Walton, 1989). However, to generate further public awareness and trust in this particular intervention, more studies need to be done. For instance, although there is a link between sleep deprivation and impaired cognitive performance (Angus, Heslegrave & Myles, 1985), there are no published studies which have examined cognitive performance of individuals with insomnia following either individual or group CBT. Furthermore, in the area of Cognitive-behavioural group therapy (CBGT), there are few published studies (Jacobs, Benson & Friedman, 1996; Kupych-Woloshyn, MacFarlane & Shapiro, 1993; Morin, Kowatch, Barry & Walton, 1993). The purpose of this study is to examine the efficacy of Cognitive-Behavioural group therapy in treating individuals with chronic insomnia, as measured by cognitive performance, sleep parameters and emotional
well-being. Such a study is in line with the need to find efficient and cost-effective psychotherapeutic ways of dealing with chronic insomnia.

As an alternative to medication, offering short-term group therapy may make individuals with chronic insomnia more open to seek treatment and comply. More focus needs to be placed on non-pharmaceutical interventions. By seeking treatment sooner, insomnia related risk factors associated with health, general well being, work performance and safety of the general public would likely be reduced.

Definition and Diagnostic features of Insomnia

Insomnia is a sleep disorder classified as a “Disorder of Initiating and Maintaining Sleep” (DIMS). It is a complaint of difficulty in sleeping (ICSD, 1990). Insomnia is best understood as a group of related symptoms. The International Classification of Sleep Disorders (ICSD, 1990) and the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV, 1994), define chronic insomnia in terms of the following symptoms: a) subjective complaints of poor sleep, b) difficulties in initiating (sleep onset latency is greater than 30 minutes) and/or maintaining sleep (sleep efficiency, only time asleep divided by time in bed is lower than 85%), c) sleep difficulties are present 3 or more nights per week, d) duration of the problem is greater than 6 months, e) subjective reporting of daytime fatigue, performance impairment or mood disturbances, and f) an impairment in social or occupational functioning.

Insomnia may be manifested in three ways: 1) sleep-onset problems involve sleep latency longer than 30 minutes after turning lights out, 2) sleep maintenance insomnia involves either frequent and/or extended nocturnal awakenings totaling more than 30 minutes of wakefulness after sleep onset, or premature awakening in the morning with
less than 6.5 hours of sleep, finally, 3) mixed sleep-onset and sleep maintenance insomnia involves a combination of difficulties with initiating and sustaining sleep (Morin, 1993).

The severity of insomnia varies from acute to chronic. There are three categories of insomnia: transient, subacute, and chronic. Transient insomnia usually lasts less than one month (Morin et al., 1999). Most individuals experience this type of insomnia at some point in their life. It is usually caused by situational stressors, such as: death of a family member, financial difficulties, or change of job (Morin, 1993). It can also be caused by circadian rhythm disruptions such as, jet lag or shiftwork. Short-term or subacute insomnia, is characterized by the inability to sleep sufficiently for a period of one to six months. Chronic insomnia persists for a length of six months or greater. It often develops from transient insomnia. The effected individual often down plays the insomnia and tries to adapt. Individuals with insomnia mistakenly assume that “it’ll get better on its own”.

Often by treating insomnia in its early stages, the development of chronic insomnia can be prevented (Morin et al., 1999). In most cases, early treatment of insomnia can be combated with medication. However, the longer insomnia is left untreated, the greater the risk that it will become conditioned and chronic.

Etiology of Insomnia.

The challenges involved in the treatment of insomnia may relate to the vast array of causes. The etiological factors that have been attributed to insomnia include: psychiatric disorders (e.g. depression, anxiety, PTSD), stress induced psychophysiological states (conditioned insomnia, bereavement, stress, financial problems), pharmacological substances (alcohol, caffeine, drugs), other sleep disorders
(sleep apnea, periodic leg movements), circadian rhythm disorder (jet-lag, shiftwork), poor sleep hygiene (e.g. irregular sleep and wake up time), and biological conditions (pregnancy, aging) (Yousaf & Sedgwick, 1996). The mechanisms responsible for the development of insomnia are unclear. However, research has suggested that insomnia is a multidimensional problem, that consists of physiological, cognitive, emotional and conditioning variables (Morin, 1993; Morin et al., 1992; Morin et al., 1999).

Autonomic activity must be reduced in order to initiate sleep. A rapid heart rate or muscle tension are examples of factors that can manifest physiological arousal in an individual. When these factors are present, onset of sleep is difficult (Freedman & Sattler, 1982). The aim is to lower the physiological arousal. This can be done by using relaxation techniques (Morin, 1993). Some of these techniques will be discussed later.

Cognitive arousal interferes with the ability to fall asleep. Cognitive activity may be manifested in terms of worry, racing mind, rumination, intrusive thoughts, planning, analyzing, or difficulty in controlling exciting thoughts (Morin et al., 1999). Intrusive thoughts need to be curtailed as they are often associated with negative sleep cognitions. For example, a fear of sleeplessness or performance anxiety (Morin, 1993).

Affect can influence cognitions, through beliefs, expectations and attributions. This can contribute to insomnia. Individuals with insomnia usually possess unrealistic expectations about sleep requirements and the consequences associated with insomnia (Morin, 1993). They may attribute the problem to external factors, such as biochemical imbalances. They may see sleep as something that is uncontrollable, and consequently may think their problem cannot change. Such dysfunctional beliefs contribute to emotional distress and inevitably insomnia (Morin, 1993).
The psychological profile of individuals with insomnia, indicates that these are individuals with higher levels of anxiety, dysphoria, worry or somatized tension (Freedman & Sattler, 1982). This psychological makeup may predispose individuals with insomnia to heightened affective responses to their poor sleep and consequently influence dysfunctional sleep cognitions (Freedman & Sattler, 1982). Individuals with insomnia may be more emotionally reactive to stress. They may have less resources to cope, and consequently internalize conflict and ruminate about what they should have done or said in a given situation (White & Nicassio, 1990). Normal sleepers are more adaptive to such situations, they usually can communicate more assertively, and consequently can go to bed with no trouble (White & Nicassio, 1990).

The ability to fall asleep is influenced by principles of classical and operant conditioning (Bootzin, 1985). On the other hand, these conditioning principles can contribute to difficulties in initiating and maintaining sleep. This theory postulates that for good sleepers, stimuli such as the bed, bedroom, or bedtime can become cues for sleep onset (Bootzin, 1985). However, if the bedroom becomes paired with activities incompatible with sleep, a negative association may develop. An individual who worries at bedtime about projects, problems at work, finances or their inability to fall asleep, eventually associates this particular time with frustration and sleeplessness (Morin, 1993; Sloan, Hauri, Bootzin, Morin, & Stevenson, 1993). This particular stimuli can lead to a conditioned arousal, which may be manifested in terms of physiological, cognitive, or emotional responses. All of which prevent sleep onset (Morin, 1993; Sloan et. al., 1993).
Other Sleep Disorders associated with Insomnia.

In treating insomnia, it is important to assess whether the problem is the result of another sleep disorder. A complaint of poor sleep related to insomnia, could be the result of sleep apnea, nocturnal myoclonus (Restless Legs Syndrome), or Fibrositis Syndrome (Hauri, 1982). Using cognitive-behavioural therapy under such circumstances would not be beneficial. Often treating the other sleep disorder is sufficient to cure the insomnia symptoms. Therefore, it is relevant to discuss other sleep disorders associated with insomnia.

Sleep apnea is a sleeping disorder where a patient (usually overweight) stops breathing many times during nocturnal sleep (300 to 500 times per night) (Mendelson, 1987). This usually leads to the symptom of excessive daytime fatigue. If not dealt with appropriately, sleep apnea could lead to death (Mendelson, 1987). There are three kinds of sleep apnea: central, obstructive, and mixed. Central sleep apnea occurs when the patient falls asleep and the diaphragm stops moving. The brain fails to send impulses through the nerves which control the movement of the diaphragm. The result is that the patient stops breathing, until he or she is awakened gasping for air (Mendelson, 1987). These awakenings are numerous throughout the night and can contribute to poor sleep. Obstructive sleep apnea occurs when there is a loss of tone in the muscles of the tongue, throat and larynx during sleep. The result is the blockage of the air flow (Mendelson, 1987). During an obstructive episode, the diaphragm continues to contract rhythmically against a closed air way, until finally an awakening occurs and normal breathing is resumed. Mixed sleep apneas is a combination of both central and obstructive apnea. Sleep apnea usually precludes restorative sleep. The sleep is disturbed to such an extent, the patient may feel they are not sleeping.

Nocturnal myoclonus is described as repetitive movements of the lower extremities during sleep, usually consisting of stereotyped leg muscle jerks (Moore & Gurakar, 1988). Nocturnal myoclonus takes place mostly during non-rapid eye
movement (NREM) and is associated with light sleep. It is usually associated with restless legs syndrome as well. This is characterized by painful tingling or crawling sensations in the legs when a individual tries to sleep or is resting for long periods of time. The individual is usually awakened by the pain and feels the urge to alleviate it by movement, whether it be by standing, walking, or sitting up (Moore & Gurakar, 1988). The sensations usually disappear after the above strategies are used. The pain temporarily subsides, but returns shortly after.

Fibrositis is a syndrome that is characterized by high amplitudes of alpha waves that intrude an individuals sleep pattern (Hauri, 1982; Moldofsky & Scarisbrick, 1976). These alpha intrusions frequently mix into other sleep wave stages and create mini arousals for the individual. Persons displaying such features often feel that they did not sleep and consequently do not feel restored. The individual usually complains of stiffness in the morning, as well as aches and pains (Hauri, 1982; Moldofsky & Scarisbrick, 1976).

Sleep

Individuals with insomnia are individuals who have impaired sleep. To truly appreciate this impairment, it is essential to understand what normal sleep is and the purpose it serves. A normal sleep cycle in good sleepers consists of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. A normal sleeper usually experiences a sleep latency between 15 to 20 minutes after going to bed. The individual enters NREM sleep starting with stage 1. This is the transition from wakefulness to sleep and makes up about 5% of sleep. Stage two is the next stage of sleep. It makes up 50% of sleep. Stages 3 and 4 make up approximately 20-25% of sleep. These stages are known as deep sleep and serve a restorative purpose. After stage 4, the individual will briefly enter stage 2 again, followed by REM. This is the stage where dreams are usually
abundant. It makes up 20% of sleep. The entire cycle takes approximately 90 minutes, and repeats four to six times during a normal sleep period (Shapiro et al., 1994).

In comparison to normative sleep, individuals with insomnia differ in terms of sleep latency, architecture, maintenance, quality and quantity. Individuals with insomnia typically have excessive amount of stage 2, an average of 20 minutes less of REM sleep, less deep sleep (stages 3 and 4) and more transitions between stages (Taub, 1978). Individuals with insomnia experience greater periods of wakefulness after sleep onset, lower sleep efficiency and longer NREM cycles (Shapiro et al., 1994; Taub, 1978). Consequently, individuals with insomnia do not get sufficient restorative sleep. The ramifications of this will be discussed later.

As stated previously, sleep serves a restorative purpose, both physiologically and psychologically (Flanigan & Shapiro, 1992). More specifically, NREM sleep restores physical energy and REM sleep deals with cognitive functioning (Morin, 1993). The restorative nature of NREM sleep is apparent when considering the high incidence of cellular and hormonal activity that occurs during this period. The highest peak of cell division (mitoses) occurs during NREM sleep, and the release of approximately 80% of daily growth hormones coincides with deep sleep (stages 3 & 4) (Flanigan & Shapiro, 1992). While sleeping, the human body consumes less oxygen to conserve (Flanigan & Shapiro, 1992). Sleep provides the function of producing cells, releasing hormones and conserving energy. When an individual gets an adequate amount of sleep, these functions contribute to feelings of health and alertness. These functions are adversely effected when individuals do not receive an adequate amount of sleep. Deprivation of REM sleep has demonstrated an adverse effect with memory consolidation. This
suggests that REM sleep has a functional role in the retention of material that has been learned during wakefulness (Morin, 1993).

Circadian Rhythms

Insomnia can be manifested by circadian rhythm disorders, such as jet-lag or shiftwork. Insomnia can also contribute to a synchronization of the circadian rhythm. Physiological and biological activities (body temperature, chemical/hormonal secretion, etc.) may become misaligned or suppressed because the insomniac is awake when he or she should be asleep. When assessing insomnia it is important to assess what effects it may have on the circadian rhythm, or conversely, what effects the circadian rhythm has on insomnia.

Human beings are a rhythmic species, experiencing cycles every 24 hours (Shaprio et al., 1994). These 24 hour cycles are known as circadian rhythms or more commonly known as one's biological clock (Flanigan & Shaprio, 1992). In determining an individual's circadian rhythm, a particular variable (i.e. body temperature, hormone secretions) must be measured repeatedly at different points of the day. When measured, systematic changes can be noted from one time of the day to another. These changes are consistent during a 24 hour period. For example, when one is asleep and body temperature is taken, it usually goes down during the night and early morning. This cycle repeats over a 24 hour period.

The purpose of this internal clock is to prepare the body and brain for sleep and active wakefulness at different times of the day (Flanigan & Shaprio, 1992). Moreover, the biological clock lowers body temperature, heart rate and blood pressure and controls the excretion of hormones like melatonin, which help induce sleep (Monk, 1987). The
biological clock also protects our sleep by suppressing hunger and renal and bowel functions, permitting longer and more consolidated sleep with minimal disruptions (Monk, 1987). Combined, these factors produce a high quality of sleep.

When the biological clock becomes misaligned, due to jet-lag or shiftwork, sleep onset is difficult. A good sleep is important for it enables individuals to feel refreshed, energetic and vigilant, aiding with daily functioning (Monk, 1987). The inability to fall asleep at a “normal” time and obtain an adequate amount of sleep, can contribute to physical and psychological health difficulties, as well as negative safety and financial issues.

Consequences of Insomnia

As was discussed above, good sleep serves a restorative purpose. It is apparent that individuals with insomnia do not get good sleep. Unfortunately, this contributes to poor physical and mental health, accidents, and negative economic factors. Discussing these consequences in detail highlights the importance of treating insomnia.

Physical Health

Long-term epidemiological studies have shown that insomnia is directly related to the development of heart disease, high blood pressure, diabetes, and stroke (Ford & Kamerow, 1989; Knutsson, Akerstedt, Jonsson, & Orth-Gomer, 1986). Individuals with insomnia are at higher risk for becoming ill than are good sleepers. Men and women who report no trouble with sleep, have the lowest mortality rates for ischemic heart disease, cancer and stroke (Knutsson et al., 1986; Stoller, 1994). More than 50% of those suffering from insomnia reported two or more health problems during a year, compared with normal sleepers (Stoller, 1994). It is likely that insomnia is both a cause and effect
of poor health, the person with insomnia becomes trapped in a cycle of pain or illness that interrupts sleep, and simultaneously, the lack of sleep compounds the disability. The mechanism by which insomnia affects illness and mortality is not clear. However, as previously discussed, sleep is said to serve a restorative function. Deprivation of this restorative process may impair longevity (Hammond, 1964).

Another common complaint associated with insomnia is gastrointestinal dysfunction or stomach problems (Monk & Folkard, 1992). Insomnia can cause: increased appetites, decreased appetites, constipation, diarrhea, indigestion, and peptic ulcers. The biological clock has important interactions with eating and voiding patterns. Part of the function of the circadian rhythm, is to suppress appetite so that restful sleep may be obtained. When insomnia desynchronizes the circadian rhythm, digestion becomes disrupted (Monk & Folkard, 1992). Moreover, when unable to sleep during the night, the digestive system is often unable to adjust and remains active during the cycles of day and night. The general predisposition of the digestive system is to be resting during one phase of the circadian cycle. In essence, what happens is that the entire digestive system is active all of the time, eventually leading to the gastrointestinal difficulties (Monk & Folkard, 1992).

Further to difficulties with ischemic heart disease, cancer and stroke, individuals with insomnia may also experience: depression, anxiety, and social and domestic stressors.

**Depression.**

Depressive disorders have a strong relationship with insomnia (Soldatos, 1994). Within depressed populations, epidemiological studies have revealed insomnia to be
twice as prevalent in the depressed population than in non-depressed individuals, likewise in the elderly population, and up to three times as prevalent among depressed adolescents (Soldatos, 1994). Two types of insomnia are observed in mood disorders including difficulties initiating sleep and early morning insomnia with excessive daytime sleepiness or frequent napping which is common among young individuals with bipolar illness during the depressive phase. Up to 80% of depressed individuals report their sleep to be non-restorative. They also experience fatigue and tiredness during the day along with concerns of the quality and quantity of their sleep, which may include fragmented sleep and early morning awakenings (Van Moffaert, 1994). Like anxiety, it is not known if depression causes insomnia or if insomnia is the cause of depression. However, improvement in insomnia appears to correspond to improvements in depression, suggesting a common pathogenesis. One study demonstrated that individuals who had insomnia had a slightly higher risk of developing major depression compared to individuals without insomnia (Reynolds, 1989). The onset of depression was 40 times more likely to occur when insomnia was unresolved (Reynolds, 1989). These findings suggest that early treatment of insomnia could reduce the incidence of future psychiatric disorders. This has potential economic gains whereby extensive hospitalization may be avoided with the early detection of sleep disturbances. Correct treatment strategies which directly address the sleep disturbance are likely to avoid the development of depression if the sleep disruption is prevented from developing into a chronic state. Two studies demonstrated that a reduction of depressive and anxious symptoms paralleled sleep improvements (Espie, Lindsay, Brooks, Hood, & Turvey, 1989; Jacobs, Benson, & Friedman, 1993). Jacobs and colleagues (1993) reported significant reductions on the
Center for Epidemiological Study of Depression scale, and on the Spielberger State-Trait Anxiety Inventory with insomnia treatment. Espie and colleagues (1989), demonstrated similar reductions of psychological symptoms with insomnia treatment.

**Anxiety.**

Anxiety holds a significant bearing on the onset of insomnia. Anxiety is usually accompanied by numerous physical and psychological symptoms including autonomic hyperactivity (tachycardia and palpitations, urinary frequency, diarrhea, dry mouth, sweating, flushing, cold hands), backache, shortness of breath, hyperventilation, difficulty swallowing, edginess, startle response, fatigueability, numbness, headache, inflexible muscles and muscle tension. Psychological symptoms include anxious worry, feelings of dread, a sensation of going crazy, hypervigilance and insomnia. These symptoms may be present through the day and increase just prior to going to bed. Individuals with chronic insomnia are usually concerned over sleep performance and their ability to function the following day with lack of adequate sleep. When anxiety leads to poor sleep a cycle may develop wherein the worry over sleep in itself prevents sleep and becomes a self-fulfilling prophecy with the potential of developing into an overwhelming situation (Shapiro et al., 1994).

Approximately 10% of individuals with insomnia have the problem secondary to anxiety or panic disorder. Other studies have estimated values as high as 37% for those individuals severely troubled by insomnia, 19% of those with less severe insomnia and 14% of those who had been troubled by insomnia some time in the past (Mellinger et al. 1985). Sleep and vigilance problems are often included in the diagnostic criteria for anxiety disorders. This is due to the assumption that if anxiety causes sleep disturbances
it is also believed that sleep deprivation may produce symptoms which may be considered as anxiety. Problems in defining anxiety disorders are complicated by the coexistence of other mental disorders and/or addictive disorders. When symptoms overlap with anxiety and other disorders, the attribution to one specific disorder is difficult and the "chicken and egg" conundrum develops. More than 70% of individuals diagnosed with generalized anxiety disorder are easily fatigueable, report having difficulty concentrating, are irritable and also have insomnia with a tendency towards chronic hyperarousal (Bourdet & Goldenberg, 1994).

In the long-term, it appears that unresolved insomnia is a risk factor in the development of both anxiety disorders and depression. One year follow-up studies revealed a significant increase in the number of anxiety and depressive diagnoses for those whose insomnia remained unresolved than for those whose insomnia did resolve (Soldatos, 1994). Depression, anxiety and related conditions (tension, psychic distress) are quite common within the population of people with insomnia. According to several epidemiological studies, 25% to 42% of individuals with insomnia are diagnosed with various forms of anxiety (including phobias, obsessive-compulsive disorder and panic disorder) (Ford & Kamerow, 1989; Soldatos, 1994). Anxiety disorders, therefore, are more prevalent psychological diagnoses associated with insomnia compared with depression (Ford & Kamerow, 1989; Soldatos, 1994).

**Social & Domestic Issues.**

Insomnia can lead to disruptions in social and family life. Very often fatigue may inhibit active participation in family and social events. Irritability may contribute to communication break downs. As a result of these factors, individuals with insomnia may
be faced with an inability to provide adequate levels of companionship, support and protection. Furthermore, intimate and sexual roles with a partner may also suffer (Mott, 1965). The individual must deal with the often overwhelming stress of balancing many problems (i.e. biological clock factors, sleep factors and social/domestic factors).

The social ramifications of family, friends, employers and educators can create severe strains. For example, individuals with excessive daytime sleepiness (EDS), are often perceived as lazy because of their excessive fatigue during the day and evening. Before being diagnosed, such individuals often come to believe that they are lazy. This often results in low self-esteem, alienation from family and immense problems with school and employment (Sleep\Wake Disorders Canada, 1993). Insomnia may lead to a high incidence of worrying about threat of job dismissal, reduced earning capacity or reduced opportunity for promotion (Broughton & Ghanem, 1976). Not surprisingly, researchers have found high levels of stress amongst individuals with insomnia (Coloquhoun & Rutenfranz, 1980). Prolonged exposure to stress can endanger an individual’s health and well-being by disrupting physiological rhythms and increasing anxiety (Broughton & Ghanem, 1976).

Insomnia may also have a role to play with alcoholism. The rate of alcoholism among individuals with insomnia is twice that of good sleepers (Kales, Kales & Bixler, 1984). Some individuals with insomnia use alcohol as a hypnotic. Although alcohol may help with sleep onset, it fragments sleep quality and contributes to sleep maintenance problems. One study demonstrated not only that insomnia may precede the development of alcohol abuse, but also suggested that treatment of insomnia may reduce the risk of developing alcohol abuse (Ford & Kamerow, 1989).
The physical and mental ailments resulting from insomnia can be serious. Research has shown that sleep duration (5 hours or less) is a better predictor of mortality than a history of diabetes, heart disease, stroke or high blood pressure (Kripke, 1983). Another study demonstrated that even when factors such as physical health were controlled, insomnia was still a predictor of death (Wingard & Berkman, 1983). This particular study demonstrated that sleeping fewer than six hours a day carried the same mortality risk as physical inactivity and high alcohol consumption (Wingard & Berkman, 1983). However, this is only accounting for mortality due to illness. The rate of injury or mortality of individuals with insomnia increases when motor vehicle and industrial accidents are considered.

**Accidents.**

Literature in the area of sleep disorders and sleep deprivation, strongly supports that as people become more tired or sleepy for whatever reason, their ability to function mentally may become impaired (Angus et al., 1985; Monk, 1991a; Monk, 1991b; Reite, Nagel & Ruddy, 1990; Thorpy, 1988). The effects of this cognitive impairment are detrimental. Insomnia may hamper the ability to drive a vehicle safely. Individuals with insomnia are reported to have vehicle accident rates three times higher than the general population (Wake Up America, 1993). In a review of transportation safety issues, sleepiness has been reported as a major factor in many catastrophic accidents (Monk, 1991a). Severe motor vehicle accidents caused by individuals with daytime fatigue are frequent (Monk, 1991a). This poses hazards on the road and endangers the lives of the affected individuals, of other motorists and pedestrians.
The Space Shuttle Challenger explosion, Exxon Valdez grounding, and the Three Mile Island and Chernobyl nuclear power plants' near meltdown, were all disasters that occurred due to human error. In all cases, employee fatigue and sleep deprivation, were main contributing factors to the accidents (Folkard & Totterdell, 1993). Industrial accidents resulting from sleepiness or fatigue are not uncommon (Folkard & Totterdell, 1993). One study reported that 53% of industrial accidents are caused by excessive daytime fatigue (Lavie, 1981). The rate of work related accidents among individuals with insomnia is estimated to be 1.5 times greater than the general population (Lavie, 1981). In some cases, these individuals are in positions that require high levels of vigilance and alertness (e.g., truck drivers, air traffic controllers, train engineers, heavy equipment operators) (Reite, Nagel & Ruddy, 1990). A study demonstrated the negative ramifications of being fatigued when in a position of responsibility. The study assessed the effects of sleepiness on nurses (Gold, 1992). It was reported that as a result of poor sleep, nurses were fatigued and experienced twice the amount of accidents or errors than nurses who had been sleeping well. Errors were often serious, such as giving patients the wrong medication or not giving them medication at all (Gold, 1992).

Studies have shown that sleep loss and fatigue contribute to decrements in performance and subsequently work productivity (Angus et al., 1985; Dinges, 1987). For example, one study reported that sleep loss significantly decreased response speed on both auditory and visual tasks (Dinges, 1987). Another study demonstrated that auditory vigilance and subjective assessment of fatigue, sleepiness and mood, deteriorated during prolonged wakefulness (Angus et al., 1985). Both studies demonstrated that sleep loss
caused participants to become sleepy. Consequently, this sleepiness resulted in decrements on performance tasks.

**Economic.**

There are many economic consequences as a result of insomnia. In the United States, conservative estimates of the total annual direct and indirect costs of insomnia, has been calculated at $92 to $107 billion (Wake Up America, 1993). Much of these costs are not related to the treatment of insomnia, but rather are a direct result of lack of physician and public knowledge, sleep laboratories and specialists (Stoller, 1994).

There are many direct and indirect costs associated with insomnia. Direct costs of insomnia include: prescription and non-prescription medications, physician visits, physician training and knowledge, psychologists, hospitalization, and medical insurance (Stoller, 1994). Indirect costs include: loss of productivity due to missed work, decreased work performance, and accidents related to the side effects of drug treatment (Stoller, 1994).

A recent study investigated the annual costs of sleep related accidents in the United States (Leger, 1994). This study calculated total costs of sleep related accidents to be between $43 to $56 billion. It is estimated that $29 to $38 billion is spent for sleep related motor vehicle accidents, $10 to $14 billion for accidents at work, $2 to $3 billion for home related accidents and $1 to $2 billion for public accidents (Leger, 1994).

Due to the increased morbidity and mortality associated with insomnia, as well as an increased rate of serious accidents, individuals with insomnia have a higher dependence on the medical care system than individuals without insomnia (Stoller, 1994). Hospitalization for individuals with chronic insomnia is twice the rate of
individuals without insomnia (Kales et al., 1984). The estimated economic cost of this hospitalization in the United States is $25 billion (Wake Up America, 1993).

It would appear from the literature that the consequences of insomnia are numerous and problematic. The ramifications of insomnia are far reaching, effecting not only the individual, but society as a whole. For the insomniac, sleep is impaired. As a result, health and well-being are compromised, social and domestic life is disrupted, and safety and work performance becomes compromised. Insomnia can contribute to motor vehicle and industrial accidents. This compromises general public safety and increases the likelihood of environmental problems. All of which contribute to the spending of massive tax dollars. In order to reduce the negative consequences associated with insomnia, it is imperative to assess and treat it in the most efficient and cost-effective manner.

Measures of Insomnia and Fatigue

Measures of insomnia and fatigue include self-report measures of sleep disruption, such as the sleep diary or Insomnia Severity Index, and performance batteries which assess individuals’ cognitive functioning.

Sleep Diary

The majority of treatment studies (90%) have relied on daily sleep diaries to document outcome (Morin et al., 1999). Daily self-monitoring of specific sleep parameters (e.g., sleep latency, sleep efficiency and duration) have proven to be similar to polysomnography results (Morin et al., 1999). Sleep diaries provide sleep clinicians with pertinent information about the individual’s sleep-wake cycle. A typical sleep diary includes the following: documenting the time of retiring at night, subjective sleep latency,
the duration of sleep and wake episodes during the night, the length of the final awakening, and the time of arising in the morning (Thorpy, 1988). It can be used to document the pattern of sleep and wakefulness over a period of two weeks. This can help to provide a baseline once treatment is initiated with an individual with insomnia, to see if any progress has occurred. The following are measures derived from sleep diaries which have been used to estimate the clinical significance of sleep improvements in treatment studies (any one of the indicators imply a clinically significant improvement): 1) sleep onset latency: a 50% reduction on the main target symptom; (2) latency duration: an absolute value of sleep onset latency falling near or below the 30-35 minutes criteria typically used to define insomnia; and (3) sleep efficiency: the proportion of patients whose sleep efficiency moved from a dysfunctional to normative level (i.e. > 80%) (Morin et al., 1999).

A number of studies have been conducted which measured the efficacy of insomnia treatment by using a sleep diary (Lacks, 1991; Morin et al., 1999; Murtagh & Greenwood, 1995). One study demonstrated that approximately 50% of individuals treated for sleep onset insomnia with cognitive-behavioural interventions met criterion of meaningful clinical improvements as described above (Murtagh & Greenwood, 1995). Another study demonstrated a modest 30 minute increase in total sleep duration following an individual cognitive behavioural intervention. Total sleep increased from six hours to six and half hours, whereas the control groups sleep only increased by 4 minutes (Morin et al., 1999). In addition, one other study (Lacks, 1991) showed that a behavioural group intervention reduced sleep onset latency from 72 minutes at baseline to 40 minutes after a five week treatment, and further decreased to 36 minutes at a three
month follow-up, and finally continued to decrease to 30 minutes at a one year follow-up. The study demonstrated that 39% of participants met clinical significance for improved sleep latency. This figure increased to 49% at the three month follow-up.

**Insomnia Severity Index (ISI).**

There are numerous self-report measures that have been developed for the evaluation of insomnia. However, few have been validated specifically as screening or outcome measure for insomnia (Bastien, Vallieres & Morin, 1999). The ISI (Bastien et al., 1999; Morin, 1993) is a measure that yields a quantitative index of sleep impairment (for a detailed description of the ISI, refer to the "Method" section). Research has demonstrated the ISI as a reliable self report instrument in evaluating perceived sleep difficulties, and as a valid measure in detecting the efficacy of treatment outcome (Bastien et al., 1999).

**Performance Assessment Battery.**

Studies have been done with performance tests, to assess the functional capacity of individuals who have experienced sleep loss (Angus et al., 1985; Dinges, 1987; Johnson et al., 1998; Spiegel et al., 1998; Thorne et al., 1983). Such performance tests have been found to be sensitive to sleep loss (Angus et al., 1985; Dinges, 1987; Johnson et al., 1998; Spiegel et al., 1998; Thorne et al., 1983). For example, one study demonstrated that throughout 54 hours of sleep loss, participants progressively did worse on performance tasks (Dinges, 1987). The study demonstrated a significant decrease in response speed on both auditory and visual tasks. Another study, conducted by Angus and colleagues (1985) also used performance tasks to measure sleepiness. Individuals who were sleep deprived and inactive for 60 hours were asked to complete subjective
assessments of fatigue, sleepiness and mood every three hours. They also performed an auditory vigilance task every six hours and completed a cognitive test battery every twelve hours. The results demonstrated decrements in performance as a result of sleep loss. A study by Spiegel and colleagues (1998) explored the alterations of performance with partial sleep loss for several consecutive days (which is the type of sleep deprivation experienced by individuals with insomnia). The study also examined performance after individuals were able to resume an extended duration of sleep (approximately 8 hours). Participants sleep restriction varied from 4 hours per night to 9 hours per night, over 16 nights. Performance deteriorated over the 6 nights of sleep debt, but steadily improved over 7 days as sleep duration increased to 8 hours per night. The study demonstrated that reduced sleep contributed to decrements in performance, and that with the absence of sleep loss, performance began to improve within days. These finding substantiate the recuperative nature of sleep. Johnson and colleagues (1998) found similar findings in their study which explored the effects of partial sleep deprivation and psychomotor vigilance. The study demonstrated that restricting participants sleep to 3, 5 or 7 hours per night for seven nights significantly impaired alertness and vigilance. The data also showed that when sleep was increased back to baseline duration (approximately 9 hours), performance decrements were reversed and stabilized over the week. Another study by Thorne and colleagues, (1983) demonstrated that sleep deprivation impairs alertness, cognitive performance, and mood. The study found that when individuals were deprived of sleep over a 72 hour period of time, performance on the Serial Addition/Subtraction degraded over time. Mental abilities declined by 25% for every 24 hours participants
were awake. His research showed that during sleep deprivation, overall performance typically declines.

Sleep deprivation studies such as the ones above, demonstrated that, although participants did not have a sleeping disorder, their sleep deprivation caused them to become sleepy. Consequently, this sleepiness resulted in decrements with performance tasks. Many other studies have been conducted showing significant adverse effects on performance during sleep loss (Babkoff, 1985; Carskadon & Dement, 1979).

Approaches to the Treatment of Insomnia

The most common approaches to the treatment of insomnia include psycho-pharmacological treatments and cognitive-behavioural interventions.

Psycho-Pharmacological Treatments.

Short-term trials of sleep promoting medication, has been reported to be of benefit for certain types of insomnia (Gillin & Byerley, 1990). For instance, in cases of situational stressors (e.g., death of loved one, surgery), acute insomnia may show relief with the use of sleep medication (Morin, 1993). Short term use of hypnotics may also be helpful in treating jet lag. Medication can also be of benefit when treating insomnia associated with other sleep disorders (sleep apnea, restless legs or periodic leg movements), or an acute medical condition (pain), or various psychiatric disorders (Morin, 1993). In such cases, medication should be used only for a short duration and should not exceed more than two doses per week, in order to avoid habituation (NIH, 1984). Hypnotic therapy should be initiated at low doses to minimize adverse effects and prevent tolerance and addiction (Czeisler & Richardson, 1991). Sleep medication does have limitations and a variety of side effects.
The prescription of medication is the most commonly applied approach to treatment of insomnia by physicians. However, different reports have reported limitations and side effects of some hypnotics (Morin et al., 1992). Certain factors should be considered when using pharmacological therapy in treating insomnia. The potential costs and benefits of medication use must be addressed for both long and short-term gains. The long term effects of medication are not well researched. Several difficulties that can arise from medication use either during the course of treatment or after its discontinuations include: alteration of sleep stages, daytime residual effects (cognitive and psychomotor impairment), rebound insomnia, and dependence (Van Brunt, Riedel & Lichstein, 1996).

Continual or prolonged use of hypnotics can produce a number of problems which may outweigh the benefits of the hypnotic. Tolerance commonly develops with the use of barbiturates, chlordiazepoxide, and barbiturate-like agents where individuals with insomnia eventually need to increase their dose to gain the same effect. Physical dependence can occur with the use of barbiturates and less commonly with benzodiazepines. It is preferable to take the hypnotic every third or fourth night or to have a “drug holiday” every three or four weeks to avoid dependence. Oversedation occurs when hypnotics are combined with alcohol or taken in higher doses than necessary (Seyone & Shapiro, 1995). Rebound insomnia is particularly common in the elderly who tend to take medication on a long term basis and are therefore at greatest risk. Discontinuation may be followed by numerous symptoms including anxiety, depression and worsening of the sleep disruption beyond levels previously experienced (Lader, 1994).
Chloral hydrate, commonly prescribed in the 1970s has serious adverse central nervous system (CNS) and cardiovascular reactions with a high fatality rate when used with alcohol. Rapid tolerance can develop within two weeks (Hussain & Shapiro, 1996).

Barbiturates can be effective in reducing sleep onset latency and nocturnal awakenings, however, side effects include impairments in cognitive and motor functioning. Tolerance is rapid and profound withdrawal symptoms can occur (Hussain & Shapiro, 1996). Benzodiazepines have contributed to several problems including: tolerance to the drugs, withdrawal effects, physical and psychological dependence and abuse. Cognitive and neuromotor impairments have been detected. Discontinuation of the drug has lead to the following symptoms: severe insomnia rebound, agitation, restlessness, hypervigilance, photophobia, anxiety, panic, and seizures (Hussain & Shapiro, 1996).

Various studies using psychotherapy, pharmacotherapy or a combination of both, have demonstrated that psychotherapeutic interventions worked best for chronic insomnia (Hauri & Wisbey, 1993; Morin et al. 1992). As noted earlier, medication was effective in instances that involved transient or situational insomnia, but proved to be less effective than behavioural therapy after a one year follow-up (Hauri & Wisbey, 1993). Chronic insomnia requires a therapeutic intervention aimed at the perpetuating factors. The use of sleeping medication alone is limited to dealing with chronic insomnia and is usually not successful (Morin, 1993).

In a study that evaluated the acceptance of psychological and pharmacological therapies for chronic insomnia, it was reported that individuals with insomnia were more accepting of psychological interventions rather than pharmacological interventions
(Morin et al., 1992). Regardless of how effective a given treatment is, its' acceptance by individuals with insomnia determines how clinically useful it is (Morin et al., 1992). According to this study, the effectiveness of medication needs to be re-evaluated in terms of its side effects, how costly it is, and how socially desirable it is. Despite of the widespread prevalence of sleep complaints, it is estimated that 85% of insomnia sufferers remain untreated (Mellinger et al., 1985). If the present findings generalize to these individuals, it is plausible to assume that their failure to seek treatment may result from the expectation that a drug is the only treatment modality currently available for insomnia. Instead of being prescribed a sleeping pill, a large segment of these people may elect to continue to endure insomnia (Morin et al., 1992). This is apparent when considering that most individuals with insomnia wait an average of 12 years before seeking assistance.

As previously mentioned, some studies have combined behavioural and pharmacological therapies in an attempt to treat insomnia (Hauri, 1997; McClusky, Milby, Switzer, Williams & Wooten, 1991; Milby, Williams, Hall, Khuder, Wooten, 1993). The assumption would be that the bio-behavioural approach should hypothetically maximize a favorable outcome by utilizing the immediate curative effects of medication and the longer lasting effects from cognitive-behavioural interventions. One study (McClusky et al., 1991) compared using triazolam versus a regiment of stimulus control and relaxation training, over a 3 week period. Both treatments demonstrated similar improvements at posttreatment (mean sleep latencies of 36 minutes). However, those participants using trizolam improved faster after the first week of treatment, whereas the participants practicing stimulus control and relaxation began to show a greater
improvement one month following their treatment and their improvement was sustained over time. Another study (Hauri, 1997) compared individuals who used a combination of trizolam and cognitive-behavioural therapy interventions, with individuals who were solely exposed to behavioural strategies. At a 10 month follow-up, participants who were treated only with cognitive-behavioural therapy techniques fared better than those who combined a regimen of trizolam and cognitive-behavioural therapy interventions (Hauri, 1997).

The findings suggest that hypnotic drugs may produce faster sleep improvements, especially in the first few days of treatment, compared to cognitive-behavioural therapy methods. Therapeutic gains in the intermediate term (four weeks), suggest that cognitive-behavioural therapy interventions and pharmacological treatment are comparable. The long term (6-24 months) effects of medication versus cognitive-behavioural therapy suggest that cognitive-behavioural therapy modalities retain their clinical benefits, whereas individuals on medication return to baseline conditions (McClusky et. al., 1991). Furthermore, evidence available suggests that individuals consuming both hypnotic drugs and cognitive-behavioural therapy do not retain their clinical gains at follow-up as well as those individuals receiving cognitive-behavioural therapy alone (Hauri, 1997).

Cognitive-Behavioural Treatment of Insomnia.

Studies have shown that CBT is effective in treating individuals with chronic insomnia (Espie, 1991, Lacks & Morin, 1992, Morin, Culbert, Kowatch & Walton, 1989). One such study involved implementing individual CBT over a period of eight weeks (Espie, 1991). Individuals with chronic insomnia were seen for one hour per week, and were subjected to a variety of cognitive techniques (e.g. cognitive
restructuring), behavioural techniques (e.g. sleep restriction) and psychoeducational techniques. Levels of fatigue, depression, anxiety and sleep efficiency were taken before the CBT interventions and after the CBT interventions. The results indicated marked improvements in all aforementioned parameters (Espie, 1991). Another study looked at 100 individuals (64 women and 36 men) with chronic insomnia over a five year period (Lacks & Morin, 1992). This study involved providing CBT treatment (relaxation techniques, sleep restriction, cognitive restructuring) on an individual basis. The format was short-term and structured. The mean number of therapy sessions was 7.8 conducted over 14.3 weeks. These individuals with insomnia were assessed with a nocturnal polysomnography, sleep diary and a clinical interview. The results indicated an overall reduced time to sleep induction, increased sleep maintenance and increased sleep efficiency (Lacks & Morin, 1992).

Morin (1993) states that 70-80% of treated insomniacs are better off than untreated ones. The magnitude of improvement can be as high as 60% after individual treatment has been implemented (Morin et al., 1992). Research has demonstrated that with CBT treatment, total sleep duration is increased by at least 30 minutes, from an average of 6 hours to 6.5 hours, whereas controls only improve by four minutes (Morin et al., 1999). Furthermore, sleep onset latency is reduced from an average of 60-65 minutes at baseline to approximately 35 minutes at posttreatment, whereas control participants’ sleep latency, is reduced by an average of only 8 minutes (Morin et al., 1999). In addition, a study using the Insomnia Severity Index (ISI) demonstrated that participants with a “moderate level” of insomnia at baseline, improved to a “subthreshold level of insomnia” at 3 months posttreatment, following a CBT intervention (Bastien et al.,
1999). Other research has shown that 50% of individuals treated for sleep onset insomnia with cognitive-behavioural interventions meet criterion of meaningful clinical improvements (sleep onset < 30-35 minutes) (Murtagh & Greenwood, 1995).

Research has also demonstrated that the durability of sleep improvements is well maintained at short (3 month) and intermediate (6 month) range follow-up assessments (Morin et al., 1994; Murtagh & Greenwood, 1995). The average duration of follow-up assessments for treatment efficacy was six months (Morin et al., 1994). Such follow-up studies have shown that 49% of individuals demonstrate reliable changes in their sleep and that 63% have at least a 50% decrease in their insomnia complaints. One study found that sleep onset latency at posttreatment was 37 minutes and continued to improve at the six month mark, to 33 minutes (Morin et al., 1994). In addition, another study demonstrated that total sleep time increased from 349 minutes at baseline to 378 minutes at posttreatment, and continued to improve to 396 minutes at 3 months follow-up (Murtagh & Greenwood, 1995). Morin and colleagues (1999) suggested that because CBT treatments are typically implemented in brief periods of time (6-8 sessions), participants usually begin to fully integrate the newly learned clinical procedures 2-3 months following the last session. Therefore, it is quite normal to see participants continuing to make significant improvements 2-3 months following treatment. To summarize, individual CBT has been found to be effective in the treatment of chronic insomnia and to be associated with maintenance of treatment gains for at least 6 months (Morin et al., 1994; Murtagh & Greenwood, 1995).

The Application of Cognitive-Behavioural Therapy to the Treatment of Insomnia.

Conceptualizing insomnia from a cognitive-behavioural framework is necessary
when attempting to treat this sleep disorder with CBT. Chronic insomnia is a multidimensional problem. It reflects an interaction of physiological, cognitive and emotional arousal, as well as conditioning variables (Morin, 1993). The stimulus-organism-response-consequence model, is derived from social learning theory (Haynes & O’Brien, 1990) and it provides a useful conceptual framework for examining the interrelationships among organismic, temporal and environmental variables (Haynes & O’Brien, 1990; Morin, 1993).

Hyper-arousal is the central feature of insomnia. Arousal regulates the balance between sleep and wakefulness. Therefore, when arousal is present, sleep may be inhibited. Different stimulus conditions can heighten emotional, physiological and cognitive arousal of an individual above a critical threshold, causing it to interrupt the natural sequence of relaxation, drowsiness, and sleep onset (Morin, 1993). For example, after a few episodes of sleepless nights, a person may come to associate certain bedtime routines and bedroom surroundings as stimuli that cause worries, apprehension and fear of being unable to sleep. The amount of time that it takes for this conditioning process to develop, varies from person to person. Daily events or interpersonal conflicts that are frustrating or problematic may activate arousal for some individuals which is taken to the bedroom, and consequently prevents sleep. They may remain worked up at bedtime as they ruminate over the daily events, which then fuels arousal and amplifies the conditioning process (Morin, 1993).

When people experience sleeplessness, responses may include worries over sleep loss, ruminations about their performance the following day and muscle tension. In addition to these reactions, there is a tendency to try harder to go to sleep, which in itself
enhances performance anxiety (Morin, 1993). Excessive arousal causes perceptual distortions of elapsed time, which further accentuate subjective sleep difficulties and distress. Eventually, the sleep drive becomes compelling enough that it overrides all these competing factors.

The next day consequences involve fatigue, mood disturbances (irritability), social discomfort, and performance impairments. These perceived sequelae, whether accurate or amplified, only remind an individual of how miserable sleep was on the preceding night and trigger further dysfunctional cognitions about oneself and about sleep (Morin, 1993). Over time, a sense of learned helplessness becomes ingrained and individuals with chronic insomnia come to believe that their insomnia is uncontrollable, unpredictable, and solely attributable to external causes (Morin, 1993). Inevitably, these negative self-statements set the individuals up for a chain reaction of emotional upset, more cognitive arousal, and further sleep disturbances.

In order to cope with insomnia, people may develop maladaptive sleep habits, such as: excessive time spent in bed, irregular sleep-wake schedules, and daytime napping (Morin, 1993). Although these coping strategies may temporarily minimize sleep loss, over the long run they interfere with the synchronizing effect of a regular and constrained sleep-wake rhythm. Transient use of hypnotic medications may also improve sleep, but with long-term use it eventually becomes part of the problem. Cognitive distortions (e.g., faulty beliefs about sleep promoting practice, unrealistic expectations, misattributions of the causes of insomnia, and amplifications of its consequences) produce emotional distress and aggravate the insomnia problem further (Morin, 1993).
Chronic insomnia is not a disorder that develops overnight. In most cases it is preceded by situational insomnia that is brought on by stressful life events, but fails to improve after the stressors are removed (Morin, 1993). Consequently, it can evolve in a gradual fashion, where the individual becomes increasingly consumed by the sleep problem and by its presumed impact on daytime functioning. Whether the problem is transient in nature or develops into full-fledged chronic insomnia, depends on how the individual perceives and appraises the sleep difficulty from its onset. For example, the individual who is subjected to a few nights of poor sleep, but is able to continue with his or her usual routine without worrying about it, is not likely to develop persistent or chronic insomnia. On the other hand, someone who becomes overly concerned and anxious after a few nights of sleeplessness and begins to catastrophize over the negative consequences on daytime functioning, is likely to enter a vicious cycle of insomnia, emotional and cognitive arousal, and further sleep disturbances (Morin, 1993). Excessive rumination about sleeplessness quickly becomes the centre of the individual’s preoccupations. Performance impairment or mood disturbances during the day tend to be exclusively attributed to poor sleep. Apprehension builds up in the evening, and as bedtime approaches, fearfulness of being unable to sleep becomes magnified. Following a poor night’s sleep, not only does the individual worry about the previous night, but he or she already anticipates the next one with apprehension (Morin, 1993). Hence, insomnia becomes a self-fulfilling prophecy.

As can be seen from this model, there is a bi-directional influence between causes and consequences of insomnia and it becomes very difficult to disentangle their causal relationships (Haynes & O’Brien, 1990). This conceptual framework has several
implications for the management of chronic insomnia. To begin with, learned behavioural and cognitive responses play a major contributory role in maintaining insomnia. Treatment should thus focus not so much on uncovering the precipitating events as on altering its perpetuating conditions. The primary targets for intervention are therefore the maladaptive sleep habits and dysfunctional sleep cognitions.

Cognitive therapy consists of identifying the clients dysfunctional sleep cognitions, challenging their validity, and replacing them with more adaptive substitutes through the use of restructuring techniques. The primary goal of cognitive therapy is to guide clients to re-evaluate the accuracy of their thinking about sleeplessness, its causal factors, and presumed consequences. The implementation of cognitive therapy for the treatment of insomnia is primarily based on cognitive restructuring techniques such as reappraisal, reattribution, and decatastrophizing (Beck & Weishaar, 1989). The client is guided to re-examine the validity of his or her beliefs, and to reframe and replace them with more adaptive substitutes (Morin, 1993). Describing the relationship between cognitions, affect and behaviour is necessary in order to establish a conceptual framework. This can be done by using hypothetical situations either related or unrelated to sleep disturbances. For example, the client may be asked to think of a situation that made him or her angry or sad, and to verbalize a self-statement to accompany that emotion. Dysfunctional sleep cognitions normally consist of: 1) unrealistic sleep expectations, 2) misconceptions of the causes of insomnia, 3) misattributions or amplifications of its consequences, 4) diminished perceptions of control and predictability of the sleep process, and 5) faulty beliefs about sleep-promoting practices (Morin et al., 1999). These dysfunctional sleep cognitions can be further explored by
introducing a series of vignettes that illustrate the clients' underlying maladaptive information processing. Once identified, these cognitions need to be explored and their validity needs to be challenged. Adaptive and rational substitute cognitions should be explored with the client. This can be achieved by using reattribution, hypothesis testing, reappraisal, and decatastrophizing techniques (see Methods section for more detail) (Morin et al., 1999).

In addition to work on dysfunctional cognitions, behavioural treatment components involve the alterations of temporal, contextual and behavioural factors. The intention is to ensure that the timing of sleep is set according to circadian principles (temporal), in an environment that is conducive to sleep (contextual), while maladaptive sleep habits (behavioural) are being modified (Morin, 1993). Behavioural treatment focuses on modifying maladaptive behavioural practices that perpetuate insomnia. The goal of the behavioural therapy module is twofold: 1) to strengthen the association between sleep behaviours and stimuli such as the bed, bedtime, and the bedroom surroundings; and 2) to consolidate sleep over shorter periods of time actually spent in bed (Morin, 1993). Stimulus control therapy is used in an attempt to attain the first goal. The second goal is pursued through the implementation of sleep restriction. In both cases, the rationale for using such procedures is that sleep is a behaviour that is susceptible to conditioning processes which are governed by environmental and temporal stimuli. When these stimulus conditions lose their association to sleep the foundation for chronic insomnia begins to take form. More specifically, an individual who is exposed to transient insomnia resulting from stressors such as marital conflicts or personal losses, may begin to resume normal sleep patterns once the stressors fade away. However, some
individuals may develop negative responses to stimuli that are normally conducive to sleep (e.g. bed, bedtime, bedroom). Bedtime or the bedroom may have been stimuli which were once associated with relaxation and sleep, but with repeated occurrences of sleeplessness a conditioning process evolves which contributes to arousal and further sleep disturbances.

The objective of stimulus control therapy is to re-develop or condition an association between sleep and the conditions in which it normally occurs in (Morin, 1993). Individuals with insomnia are instructed to go to bed only when drowsy. Eliminating the propensity for staying in bed awake, eliminates potential periods of time where the individual may begin to ruminate, worry and engage in internal monologues which are incompatible with relaxation and sleep, and can strengthen maladaptive association between the bedroom and sleeplessness. Therefore, the individual with insomnia is instructed to leave their bedroom if they cannot fall asleep within 20 minutes. They should engage in non-stimulating activities which are monotonous or boring and only return to bed when drowsy. This procedure is repeated until they actually fall asleep within 20 minutes.

Sleep restriction limits the amount of time spent in bed to actual sleep time. Individuals with insomnia tend to spend excessive amounts of time in bed in an attempt to make up for a sleepless night. Sleep restriction incorporates a formula for calculating a sleep efficiency. Sleep efficiency is calculated by dividing total sleep time by total time in bed and multiplying the ration by 100. The goal with sleep restriction is to increase an individual’s sleep efficiency to above 85%. The sleep restriction strategy would for example, suggest to an individual who sleeps for 4 out 8 hours in bed, to sleep only four
hours. When his or her sleep efficiency is above 85%, their sleep time (or “sleep window”) increases by an additional 15 minutes. This procedure continues until the desired sleep time is reached (Morin, 1993). The importance of a sleep diary is apparent when implementing sleep restriction, as it provides an approximate duration of sleep time and time in bed, in order to calculate the sleep efficiency.

An integral component of CBT in the treatment of insomnia is relaxation training. Arousal interferes with sleep. Physiological activity must be reduced in order to initiate sleep. Physiological arousal can be manifested by a rapid heart rate or muscle tension brought on by frustration or anxiety over not being able to sleep (Freedman & Sattler, 1982). Similarly, cognitive arousal may be manifested through worry, rumination, intrusive thoughts and planning (Morin et al., 1999). Cognitive arousal often become associated with negative sleep cognitions. The objectives of relaxation techniques are to distract the individual with insomnia from focusing on intrusive and disruptive thoughts and to help lower physiological arousal. Learning to focus one’s attention on relatively pleasant, monotonous internal sensations may be incompatible with worrisome thoughts and images that prevent sleep onset (Borkovec, 1982). Relaxation techniques such as: progressive muscle relaxation, guided imagery, deep breathing and autogenic training are all common techniques used in an attempt to reduce individuals physiological and cognitive arousal in relation to insomnia (Morin, 1993; Morin et al., 1999).

Finally, the CBT approach to the treatment of chronic insomnia also employs an educational component, mainly teaching basic sleep hygiene principles. Different lifestyle and environmental factors that can be counter-productive to obtaining a good night sleep are described and discussed. Diet, nutrition, exercise, alcohol, caffeine
consumption, cigarettes, napping, light, noise and temperature are examples of things that clients should be informed about (for more detail about sleep hygiene see Manual section). Poor sleep hygiene can often hinder therapeutic progress and complicate the client's insomnia.

Studies have demonstrated that a multifaceted approach to treating insomnia (as described above) is significantly more effective than no treatment (Edinger & Stout, 1985; Jacobs, Benson, & Friedman, 1993). The best outcomes from multicomponent interventions have been reported when sleep restriction and/or stimulus control procedures were integrated with other methods such as cognitive restructuring, sleep hygiene and relaxation methods (Jacobs et al., 1993). Effective clinical management of insomnia will often involve a combination of treatment procedures (Jacobs et al., 1993).

**Cognitive-Behavioural Group Treatment of Insomnia.**

There is considerable research on using individual cognitive-behavioural therapy with individuals that have insomnia (Sloan et al., 1993). However, the focus of this study is to assess the effectiveness of CBT in a group setting for individuals with insomnia.

Group therapy has been known to be effective in working with a variety of psychiatric populations, in particular with psychosomatic disorders (Stein, 1971). However, with the exception of a few studies (Jacobs et al., 1996; Kupych-Woloshyn et al., 1993; Morin et al., 1993), there appears to be a void in the literature as to the effectiveness of Cognitive-behavioural group therapy in working with individuals with insomnia, particularly when assessing cognitive performance.

A study by Morin and colleagues (1993) demonstrated that late-life insomnia could be effectively treated with cognitive-behaviour group therapy (CBGT). Treatment
was effective in reducing sleep latency and early morning awakenings, as well as increasing sleep efficiency and these gains were maintained at 3 month and 12 month follow-ups. Twenty-four participants with late-life insomnia were randomly assigned to an immediate treatment group or a wait-list control group. Treatment consisted of an 8 week intervention aimed at changing maladaptive sleep habits and altering dysfunctional beliefs about sleeplessness. Measures included an overnight polysomnography, sleep diaries, the Beck Depression Inventory (BDI), the State-Trait Anxiety Inventory (STAI) and the Profile of Mood States (POMS). No performance measures were used. Participants continued using sleep medication during the study. All participants were over the age of 60, with a mean age of 67.1 years. Participants receiving the CBGT intervention reduced their sleep latency from 39.6 minutes at baseline to 20.6 minutes at the first posttreatment follow-up, 21.3 minutes at the 3 month follow-up and 22.4 minutes at the 12 month follow-up. Total sleep duration increased from 328.3 minutes at baseline to 341.4 minutes at the first posttreatment follow-up, 364.4 minutes at the 3 month follow-up and 393.8 minutes at the 12 month follow-up. Finally, sleep efficiency improved from 68.5% at baseline to 82.8% at the first posttreatment follow-up, 81.1% at the 3 month follow-up and 83.6% at the 12 month follow-up. A significant reduction of depressive scores as measured by the BDI was also detected in the cognitive-behavioural therapy treatment group.

Another study found that using a group intervention along with cognitive-behavioural and educational techniques, was helpful in improving both sleep and mood of individuals with insomnia (Kupych-Woloshyn et al., 1993). The study involved 30 individuals with insomnia who were treated in four groups of six to eight people each for
a period of eight weeks. The groups received cognitive-behavioural interventions that involved sleep restriction, cognitive restructuring and relaxation techniques. Measures included sleep diaries and the Beck Depression Inventory. No performance measures were used. There were no follow-up assessments and no control group was used. Based on participants' subjective reports, the study showed that group members benefited from the cognitive-behavioural interventions.

A study by Jacobs and colleagues (1996) suggested that following a CBGT approach for the treatment of insomnia, participants' sleep significantly improved. The group approach included: sleep restriction, stimulus control, relaxation techniques and cognitive restructuring. In total 102 participants were included in the study. Sessions were provided on a weekly basis for 10 weeks. Cognitive performance was not assessed and no control groups were used. Sleep parameters were assessed through retrospective measures. Participants also continued using sleep medication during the study. The results of the study demonstrated that 58% of participants reported significant sleep improvements, 33% moderate and 9% slight improvement. A six month follow-up showed that 90% of participants maintained their sleep improvements.

In addition, a study by Lacks (1991) demonstrated that behaviour therapy was successful in treating individuals with insomnia in a group setting. While this study did not assess cognitive-behavioural therapy, it implied that the psychotherapeutic dynamics of group therapy were effective in the treatment of insomnia (see below for more detail). The behavioural approach was based on stimulus control therapy. The study involved 400 participants over an eight year period. Participants were randomly assigned into groups of 5-7 people and administered five sessions of behavioural therapy. Measures
included sleep diaries. No performance measures were used. The study demonstrated that a behavioural group intervention reduced sleep onset latency from 72 minutes at baseline to 40 minutes after a five week treatment. Sleep latency further decreased to 36 minutes at a three month follow-up and continued to decrease to 30 minutes at a one year follow-up. The study demonstrated that 39% of participants met criteria for significant improvement in sleep latency, that is a sleep latency lower than 30 minutes. This figure increased to 49% at the three month follow-up.

The Application of Group Therapy to the Treatment of Insomnia.

The authors (Kupych-Woloshyn et al., 1993; Lacks, 1991) reported that the therapeutic factors of group therapy were important because of the following principles: a) the group setting helped demonstrate to the participants that they were not suffering in isolation and that their problems were not unique; b) inter-individual differences became points of discussion, thereby excluding the notion of a singular recipe for sleep; c) validation and acceptance by the group allowed individual group members to move beyond suffering in isolation; d) a presentation of a variety of points within the group provided individuals with a possibility of several hypotheses to explain their own sleep problem; and e) provision of some structure provided a secure environment which facilitated self-disclosure in the members.

Individuals with insomnia may be more emotionally reactive to stress. They may have less resources to cope and communicate. Consequently, they internalize conflict and ruminate about what they should have done or said in a given situation (White & Nicassio, 1990). By learning to communicate and interact with other group members,
they may minimize future conflict and rumination. The expression of emotion enhances the development of cohesiveness and simultaneously enhances well being (Yalom, 1975).

Instillation of hope is important in order to keep the client in therapy. Seeing other group members get better can be inspiring especially after dealing with insomnia for years (Morin, 1993).

The essence of universality can alleviate clients' experience of being different and socially isolated. The group gives an opportunity for clients to speak about their concerns while providing validation (Yalom, 1975). Individuals with insomnia may need to speak with others about how insomnia has effected their lives.

Providing information to clients in a group regarding the process of the illness (insomnia) can help the group bind together. The group can then discuss the information together. Direct advice from other group members is received more readily. After the process, rather than the content, is valued when clients connect through mutual interest and caring (Yalom, 1975). Learned behavioural techniques which are reinforced by group therapists tend to show the greatest improvements compared to those who learn the techniques on their own. Perhaps this is because model clients who diligently adhere to the clinical procedures can prove strong allies to the therapist in convincing other clients to comply with the prescribed regimen (Lacks, 1991).

Altruism allows clients to receive through giving. Since many clients with insomnia have long considered themselves as burdens, the experience in finding that they are important to others through providing reassurance, suggestions, insight and similar problems may boost their self-esteem. Clients who have completed therapy commonly report other members as being pivotal to their improvement (Yalom, 1975).
Development of socializing techniques is common to all therapy groups. Members are encouraged to provide feedback to others regarding maladaptive behaviours (Yalom, 1975). Imitative behaviours allow members to observe others revealing private matters and coping with similar problems (Yalom, 1975). As a result, social learning and altruism reduce social anxiety and enhance self-esteem (Yalom, 1975).

Facilitating the dynamics of group therapy for individuals with chronic insomnia require that the client move from a passive mode of functioning to a problem solving mode (Lacks, 1991). A therapist/facilitator should have a sense of humor to lighten up the proceedings and rapidly develop rapport with group members (Lacks, 1991). Modeling self-disclosure, openness and problem solving will also help clients. The therapist needs to be a directive group leader who gives feedback, reinforces participants efforts and encourages further problem solving (Lacks, 1991).

In addition to the therapeutic benefits of group therapy, there are also economic benefits (Lacks, 1991). Group therapy allows for more clients to be treated within a limited time (Kadis, Krasner, Winick & Foulkes, 1963). For therapeutic settings that are not subsidized, group therapy allows for lower fees than individual therapy. In a setting where clients do not have to pay for treatment, group therapy can substantially reduce a waiting list (Kadis et al., 1963). This benefits the clients’ and also reduces the economic burden that hospitals and other mental health institutions are faced with.

Hypotheses

Chronic insomnia is a relatively prevalent condition with multiple health and behavioural consequences to affected individuals and to society. Sleep cognitions and maladaptive sleep behaviours can be influencing factors in the etiology of insomnia.
Individual cognitive-behavioural therapy has been found to be effective in the treatment of chronic insomnia.

To date, few studies have employed a group approach to cognitive-behaviour therapy with people suffering from insomnia (Jacobs et al., 1996; Kupych-Woloshyn et al., 1993; Morin et al., 1993). However, these studies do present with a number of methodological limitations.

For example, a study conducted by Jacobs and colleagues (1996) was able to demonstrate that CBGT treatment was effective in improving participants' sleep. However, participants did use sleeping medication while simultaneously receiving CBT, which confounds the results of the study. Another limitation was that no control groups were used, making it impossible to determine whether gains were due to the therapeutic treatment. Finally, only subjective outcome measures were used.

Another study (Kupych-Woloshyn et al., 1993) assessed CBGT as an effective treatment for insomnia based on participants' subjective reports. The study, however, had methodological limitations as well. To begin with, the inclusion criteria for participants was too broad. Individuals experiencing insomnia as a result of other sleep related disorders were included in this study. Using CBT with individuals who are experiencing insomnia due to sleep apnea, for example, may be inappropriate. The sleep apnea should be dealt with first, to assure patients' safety. It is likely that if this is done, the insomnia would also dissipate. Similarly, as was described above, participants using sleeping medication while simultaneously receiving CBGT, were also included in the study. In addition, no control groups were used and only subjective outcome measures were administered. Furthermore, no follow-up assessments were taken.
A study by Morin and colleagues (1993) showed that the CBGT intervention improved participants' sleep latency, duration and efficiency. The study had fewer methodological limitations when compared to the two studies above. The study did however consist of a small sample size (N = 24). It also was not representative of the general population, as the CBGT treatment focused on late-life insomnia and was implemented to an older sample (average age = 67.1 years). Participants also continued to take medication during the study.

A study by Lacks (1991) measured the effectiveness of behavioural group treatment to insomnia based on sleep diaries. While not assessing cognitive-behavioural therapy per se, the study suggested the effectiveness of a group approach to insomnia.

Despite the reported biases or flaws in aforementioned studies, they were useful models in exploring the contribution of group therapy to the treatment of insomnia.

The purpose of this research was to examine the efficacy of cognitive-behavioural group therapy in the treatment of chronic insomnia, using both objective and subjective measures and a control group. Such a study is in line with the need to find effective and cost-efficient psychotherapeutic ways of dealing with insomnia.

It was hypothesized that after a cognitive-behavioural group therapy intervention, participants would demonstrate an improvement in cognitive performance (memory, spatial abilities, logical reasoning & concentration), sleep parameters (total sleep time, sleep onset & sleep efficiency) and a decrease in sleep impairment. Furthermore, it was hypothesized that both depressive and anxiety levels would also decrease. More specifically, the following hypotheses were made:
1. It was predicted that participants that received CBGT treatment would demonstrate significant improvements in their cognitive performance, following their participation in the CBGT. It was expected that scores on the Logical Reasoning, Serial Reaction Time and Manikin tasks would demonstrate statistically significant improvements at the post treatment assessment time (2 weeks post treatment). It was also hypothesized that treatment gains would be maintained throughout the follow up period. It was also hypothesized that the no treatment control groups performance would deteriorate over the duration of the study.

2. It was predicted that participants in the treatment groups would demonstrate discernible improvements in relation to sleep parameters (sleep latency, sleep efficiency & sleep duration) at the first follow-up measurement (2 weeks post treatment) and continue to improve to at least the second measurement milestone (3 months post treatment). Furthermore, in relation to meeting sleep diary criteria for significant sleep improvements (i.e. sleep latency < 35 minutes, or sleep efficiency greater than 80%), it was expected that 40-60% of participants who received CBGT treatment would demonstrate such clinical improvements. It was also predicted that average sleep duration would increase by at least half an hour, and that subjective sleep impairment measures (ISI) would also improve. No such changes were hypothesized for the no treatment control groups.

3. It was predicted that participants in the treatment groups would demonstrate significant improvements in relation to depressive and anxiety levels while no such changes were hypothesized for the no treatment control groups.
4. It was predicted that group members would subjectively assess the multi-faceted treatment as helpful in dealing with their insomnia and that they would rate the different components of the CBGT (i.e. guided imagery, progressive muscle relaxation, sleep hygiene, stimulus control, sleep restriction and cognitive restructuring) as helpful.

5. It was predicted that group members would find different therapeutic components of group therapy as helpful in dealing with their insomnia. These include: catharsis, instillation of hope, universality, altruism, imitative behaviour, group cohesiveness and interpersonal learning.
Chapter 2

Method

Participants

A sample of 70 individuals (35 experimental group and 35 wait list control) with chronic insomnia were recruited from the University of Toronto Centre for Sleep and Chronobiology at The Toronto Hospital/Western Division. The age of participants ranged between 26 to 61. All participants were diagnosed with psychophysiological insomnia. Diagnoses were made through the use of a semi-structured clinical interview (Appendix A) by a referring sleep specialist. Participants were informed that a study was being conducting on a cognitive-behavioural group therapy approach to insomnia on a volunteer basis, and that their access to the usual treatment would not be affected by not participating in the study. The referring sleep specialists then provided participants with an Information Sheet (Appendix B). If interested they were invited to contact the researcher.

Inclusion Criteria

All participants were diagnosed with Psychophysiological Chronic Insomnia. Participants had a sleep efficiency (time asleep divided by time in bed) of less than 85%. Participants also had difficulty sleeping at least three times per week for a minimum of 6 months. Participants also expressed a willingness to participate in a group therapy format. All participants spoke English and had at least a high school education. All participants were not interested in use of medication for the treatment of their insomnia, often because they have not found medication helpful.
Exclusion Criteria.

Individuals who had insomnia associated with other primary sleep disorders (e.g. sleep apnea) were not included in the study. Individuals who were using or would be using medication to treat their insomnia were also excluded from the study. Finally, drug dependence and severe psychiatric disorders (such as schizophrenia or psychotic disorders) were also excluding factors, in that the group dynamics could have been negatively affected by the presence of such participants.

Treatment Protocol

Cognitive-Behavioural Group Treatment.

A manual of cognitive behavioural group therapy was developed and used by this researcher. The manual consisted of specific treatment interventions for chronic insomnia (Appendix C). Each group consisted of five participants (seven treatment groups and seven control groups). Sessions were 1.5 hours in length, once per week, and lasted eight weeks. Supervision was provided by Dr. Sheldon Shaul, a psychiatrist and sleep specialist. The groups were facilitated by the author of this dissertation. For the purpose of supervision and adherence to the manual, all group sessions were audio taped with participants’ consent. Presentation of information, group discussion, problem-solving approaches, relaxation techniques and cognitive-behavioural strategies were implemented within the dynamics of group work. The interventions utilized are outlined in the Manual (Appendix C). The goals of the treatment groups included the provision a forum for the sharing of problems and solutions among people suffering from chronic insomnia, identifying and overcoming barriers to healthy sleep, altering dysfunctional cognitive attitudes, examining sleep hygiene factors, and teaching relaxation techniques.
**Outcome Measures**

Outcome measures were administered two weeks prior to treatment, two weeks following treatment, three months following treatment, and six months following treatment. Outcome measures can be classified into objective and subjective measures.

**Performance Assessment Battery Sub-scales (PAB).**

The Walter Reed Performance Assessment Battery (PAB) was used to assess psychomotor, perceptual and cognitive skills, including: memory, spatial abilities, logical reasoning, and concentration (Thorne et al, 1985). The PAB is used as a valid research device for following changes in performance over periods of time (Thorne et al., 1985). The change in performance can be used to indicate fatigue. The PAB is computer generated, controlled and scored. Test items and visual stimuli are shown to participants on video monitors. Participants respond to the stimuli by pressing one or more keys on a conventional keyboard (Thorne et al., 1985). Many different tasks can be implemented using the PAB, including: the choice reaction time, time estimation, mental arithmetic, and logical reasoning. The PAB has been used in a vast array of studies, including: sleep deprivation, sustained performance, physical fatigue, and jet lag (Angus et al., 1985; Dinges, 1987; Thorne et al., 1985).

Learning effect is curtailed because random changes in the order and pattern of all tasks are automatically controlled by a computer program. Individual tasks are automatically generated, presented, recorded and scored on the PAB. The score produced by the PAB is called the *throughput*, which is a speed-accuracy product. The higher the score, the better the performance.
The test battery that was used in this study was configured to take 15-20 minutes to complete, with a total of three tasks. The three tasks were as follows: the Logical Reasoning, Manikin, and, Serial Addition/Subtraction. When performing these tasks, participants were informed that they should respond as quickly and accurately as possible. For this study, participants had one practice run on the PAB. Results on the second attempt were scored.

The Logical Reasoning test has been shown to be very sensitive to fatigue effects, and includes different reasoning tasks. The letters "AB" or "BA" appear in the centre of the monitor, with a statement about the relationship between the two letters. The statement may or may not be true. The test-retest reliability for this task was found to be .78 (Thorne et al., 1985).

The second task was the Serial Addition and Subtraction. This is an arithmetic task that requires sustained attention and concentration. Two numbers appear in the centre of the monitor, one after the other. The numbers are then followed by a "+" for adding or a "-" for subtracting. The participant either adds or subtracts the numbers accordingly. The answer is then typed in, using the numbered keys on the right side of the keyboard. However, the participant has two important things to note when performing the task. The first is that if any answer consists of two digits, then only the second digit is to be typed in. The second thing that has to be noted is that whenever any response is a negative integer, 10 has to be added to the final answer (Ryman, 1974). The test-retest reliability for this task was found to be .82 (Thorne et al., 1985).

The third and last task in the performance battery was the Manikin. This task assesses spatial abilities (Ryman, 1974). A human figure appears in the centre of the
monitor. The figure is enclosed either by a green circle or a red square. The human figure holds a small green circle in one hand and a small red square in the other. Based on the shape surrounding the figure's body, the participant must indicate which hand the corresponding shape is in. The letter "V" is pushed if the shape is in the left hand or the letter "M" is pushed if the shape is in the right hand. The test-retest reliability for this task was found to be .74 (Thorne et al., 1985).

**Subjective Measures.**

The self report questionnaires package included a sleep diary, Insomnia Severity Index, Beck Depression Inventory II, Beck Anxiety Inventory, as well as a demographic survey and a group evaluation.

1) Sleep Diary:

The sleep diary (Appendix D) can provide extremely useful clinical information. A sleep diary requires daily recording of the following parameters: bedtime, arising time, sleep onset latency, number and duration of awakenings, time of last awakening, naps, meals, snacks, caffeinated drinks, exercise, and any use of sleeping medication. It is simple to use, and its design gives a quick pictorial display of a participant's sleep patterns over a two week period. A clinician can at a glance gain an understanding of the nature, frequency, and intensity of insomnia of nightly variations in sleep schedules and of some common perpetuating factors (e.g. daytime naps). It includes estimates of time in bed, sleep time, and wake time, so that a global sleep efficiency ratio can be calculated. This is obtained by dividing "total sleep time" by "time in bed" and multiplying by 100.

In this study participants completed two weeks of sleep diary information at each assessment time. Treatment efficacy was assessed using the following measures: 1)
participants displaying a 50% reduction on the main target symptom (sleep onset latency); 2) an absolute value of that symptom falling near or below the 30-35 minute criteria typically used to define insomnia; 3) the proportion of patients whose sleep efficiency moved from a dysfunctional to normative level (> 80%) (Morin et al., 1999).

2) Insomnia Severity Index:

The Insomnia Severity Index (ISI) (Morin, 1993) (Appendix D) is a 7 item measure that yields a quantitative index of sleep impairment. The participant provides ratings (on a 5 point Likert scale, “0” = not at all, “4” = extremely) of the severity, degree of interference with daily functioning, level of distress caused by the sleep problem, and satisfaction with current sleep patterns. These subjective ratings provide valuable information on the participant’s perception of his or her sleep problem (Morin, 1993, Bastien et al., 1999). Total scores range from 0 to 28, with high scores indicating greater insomnia severity. A score of 0-7 indicates no clinically significant insomnia, while 8-14 indicates subthreshold insomnia. A score between 15-21 suggests a clinical insomnia (moderate severity) and, finally, a score between 21-28 indicates clinical insomnia (severe) (Bastien, et al., 1999). Internal consistency was found to be 0.74. Concurrent validity was found to be 0.65 when comparing changes over time with ISI and sleep diaries (Bastien et al., 1999). These findings suggest that the ISI is a valid and reliable instrument for assessing insomnia.

3) Beck Depression Inventory-II:

The Beck Depression Inventory-II (BDI-II) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Beck & Steer, 1996) (Appendix D) is a frequently used self-report method of assessing depressive symptomatology. It is a 21 item self-report instrument
that assesses the affective, cognitive, motivational, and physiological symptoms of depression. Item categories include mood, pessimism, crying spells, guilt, irritability, sleep and appetite disturbance, and loss of libido. For each of these categories of symptoms, there is a graded series of four alternative statements, ranging from neutral to a maximum level of severity. The items are scored from 0 to 3, so that the total BDI-II score can range from 0 to 63. Generally, a total BDI-II score of 0-13 indicates a minimal depressed state, 14-19 reflects a mild level of depression, 20-28 reflects moderate depression, and 29-63 indicates a severe level of depression. The test-retest reliability for the BDI-II was found to be 0.93 (Beck & Steer, 1996). Internal consistency was found to be 0.92 (Beck & Steer, 1996). Depressive disorders have a strong relationship with insomnia (Soldatos, 1994). Studies have demonstrated that a reduction of depressive and anxious symptoms paralleled improvements in sleeping patterns (Espie et al., 1989; Jacobs et al., 1993).

4) Beck Anxiety Inventory:

The Beck Anxiety Inventory (BAI, 1993 Edition manual) (Beck, Epstein, Brown & Steer, 1988) (Appendix D) is a 21 item self-report scale used to assess the severity of anxiety in adults and adolescents. It evaluates the affective, cognitive, motivational, and physiological symptoms of anxiety. Descriptive symptom labels include feeling hot, sweaty, scared, afraid of dying, and difficulty breathing. Each symptom is rated on a 4-point scale ranging from 0 to 3. Generally a total BAI score of 0-7 indicates a minimal anxious state, 8-15 reflects a mild level of anxiety, 16-25 reflects moderate anxiety, and 26-63 indicates a severe level of anxiety (Beck & Steer, 1993). The test-retest reliability for the BAI is 0.75 (Beck & Steer, 1993). Internal consistency is 0.92 (Beck & Steer, 1988).
1993). Anxiety holds a significant bearing on the onset and maintenance of insomnia. Therefore, it is important to be able to assess for anxiety as an outcome measure (Espie et al., 1989; Jacobs et al., 1993).

5) Demographic Survey and Group Evaluation:

A demographic survey (Marino, 1998) (Appendix E) was developed for the study. It included information about participants' age, sex, marital status, duration of the insomnia, and previous use of medication or other treatment modalities. The demographic survey was administered at the first assessment time.

The “Evaluation of Group Intervention” survey (Marino, 1998) (Appendix E) was administered in the follow-up questionnaire battery. This survey was used to assess participants' response to the group intervention.

Procedures

Administrative consent from the Toronto Hospital was obtained for this study. Participants attending the Center for Sleep and Chronobiology at The Toronto Hospital due to a sleep disturbance were assessed by Dr. Shaul, a psychiatrist and sleep specialist. Diagnoses were made through a semi-structured clinical interview based on the SCID, a standardized assessment battery (Appendix A). Participants diagnosed with chronic insomnia were invited to participate in an eight week cognitive-behavioural group for insomnia by distributing written information about the study (Appendix B). All participants were informed that their assessment or the provision of the usual treatment protocol at the Sleep Clinic would not be affected in any way, if they chose not to participate or withdraw from the study. Interested participants contacted the researcher via the phone and were provided with a verbal description of the study.
Participants were informed that the purpose of the study was to examine the effects of Cognitive-Behavioural Group Therapy in treating Chronic Insomnia. They were informed that they would be randomly assigned to an immediate treatment group or to a delayed group, which would receive treatment in approximately eight months time. Participants were also informed of the length and duration of the cognitive-behavioural group therapy (1.5 hours per week, for eight weeks). A description of all assessment measures were then provided. Those who were interested in participating in the study signed the consent form (Appendix B) at the pretreatment assessment time. All participants were notified that information discussed in the group sessions would be kept in strict confidence (except if they were in imminent danger of harming themselves or others or indicate that a child is at risk of abuse/neglect at the time of study or if there was a subpoena by the court). They were also informed that all data collected would be assigned a research code number and that their confidentiality would be maintained with respect to any publication or presentation.

Limited by the number of referrals provided, only 10 participants at a time were randomized to either an immediate treatment group, or to a delayed treatment group (5 per group). The delayed treatment group served as an 8 month control group (no treatment). The ten names were chosen randomly (through a lottery system), one at a time, and placed in each group. The subsequent 10 referrals followed the same process; each name chosen randomly and assigned to the immediate treatment group or delayed treatment group. The process was repeated seven times in total.

The study was conducted at the Center for Sleep and Chronobiology at The Toronto Hospital (Western Division). All technical equipment was provided by the sleep
clinic and sessions were held in a conference room at the Toronto Hospital. Sleep disorder consultation was provided by Dr. Shaul. All sessions were audio taped for purposes of supervision by Dr. Shaul and adherence to the manual (Appendix C).

The sample size and group size chosen was based on samples used in other studies that explored the effects of CBT in the treatment of insomnia (Lacks, 1991, Morin et al., 1992, Kupych-Woloshyn et al., 1993). The number of sessions used for this study was also based on standards found in the literature (Lacks, 1991, Morin et al., 1992, Kupych-Woloshyn et al., 1993). In addition, six months was the average period of time that was used for follow-up evaluations (Morin et al., 1999).

**Implementation of Cognitive-Behavioural Group Therapy Intervention.**

A manual (Appendix C) was used when implementing the CBGT. The same manual was implemented in all treatment groups. The treatment agenda was covered during eight weekly groups session. Session one began with a brief repeated introduction of the study and of the limits of confidentiality. Participants were then broken up into sub-groups of two or three members. They were asked to obtain information (name, age, sleep problem, duration of problem) about the other participants. This information was then brought back to the group as a whole. As the facilitator of the group, it was important to model self-disclosure and openness with the participants. Having worked in a sleep clinic for several years, a job that involved shiftwork, this writer was able to disclose sleep related problems that were similar to those conveyed by group members. Participants were encouraged to discuss their sleep problems in their own words and describe how insomnia has effected their lives. Creating a sense of universality was essential in order to help participants feel that they were not isolated in their experiences.
of insomnia. It was also explained to participants that sessions would begin with a “check in” and “self monitoring”, where they discuss how their week had been with respect to their sleep, emotions and physical and cognitive functioning. A review of the sleep diary was also conducted and any questions that participants had were answered. This became an opportunity for other group members to make suggestions or comments to fellow members who may have had a particularly difficult week, thereby making the processes of imparting of information and altruism possible. The diversity of the group and the sleep difficulties they experienced was used to provide participants with possible explanations or rationales to misconceptions they have had with their own sleep problem.

Session one also described the cognitive-behavioural therapy approach. A social learning explanation of insomnia was then provided. Sleep physiology was discussed in order to give participants an idea of what sleep really is, what it looks like and how it changes over time.

Session two began with a “check in” and with a review of the sleep diary. An introduction of the behavioural interventions (stimulus control and sleep restriction) was provided. The behavioural treatment rationale was also provided. The directions for both stimulus control and sleep restriction were also provided as handouts.

Session three had the usual “check in” and review of the sleep diary. Behavioural procedures were reviewed. Feedback pertaining to stimulus control and sleep restriction was discussed. Problems encountered, predominately with adherence, were discussed. The encouragement of problem solving was seen as important in an effort to move the group members from a passive mode of functioning to a more active mode and to create greater cohesiveness amongst the group members. Positive feedback allowed for
constructive changes to be implemented by others and to instill hope. Session three also consisted of a brief introduction and an overview of the relaxation techniques. Relaxation techniques consisted of progressive muscle relaxation, guided imagery, deep breathing and autogenic training. Handouts of these descriptions were provided to participants.

Session four reviewed the usual "check in" procedure and sleep diary, as well as behavioural procedures and relaxation techniques. Cognitive therapy and its rationale were also introduced. Participants' beliefs and attitudes about sleep were identified through non-standardized scales and cognitive vignette handouts (Appendix E). These handouts were explored within the group for various dysfunctional sleep cognitions and misconceptions about insomnia. Once identified, the objective was to challenge these faulty beliefs and cognitive errors and replace them with more appropriate ones.

Session five allowed for further exploration of participants' dysfunctional sleep cognitions. Cognitive restructuring techniques such as reappraisal, reattribution, and decatastrophizing were used when exploring their sleep beliefs. A brief introduction to sleep hygiene education was provided in session five along with a handout.

Session six continued to review participants' "check in", as well as the behavioural, cognitive and relaxation technique procedures. Session six also continued to explore sleep hygiene and discussed issues specific to caffeine, nicotine, alcohol and environmental factors.

Session seven was similar to session six in that it consisted of a review of all the behavioural, cognitive, and educational interventions. It also integrated the separate
therapeutic components. Feedback was provided by this facilitator and by other group members.

Session eight covered ways in which members could continue to adhere to treatment and maintain any gains. Relapse prevention was also discussed where members were informed that even with major gains, the inevitability of having occasional poor sleep in not unusual. A “Group Evaluation” (Appendix E) was distributed.

**Statistical Analysis**

The data were analyzed using a mixed multivariate analyses of covariance. For all the measures in the study there was not sufficient statistical evidence to conclude that the assumption of parallelism underlying analysis of covariance was violated. The basic design, applied where the assumption of parallelism was not violated, was of Age as the single covariate, Protocol group (treatment groups/control groups), Replication group nested within Protocol group as a blocking factor, Gender, the interaction between Gender and Protocol group, and Replication group nested within the interaction between Gender and Protocol group, with, time of measurement (baseline/10 weeks/3 months/6 months) as the repeated measures factor. Replication group is a 14 level factor corresponding to the Protocol groups into which the sample was divided as described within the Methods section. Comparisons of individual marginal means were made using T tests with Bonferroni corrected p values.
Chapter 3

Results and Findings

Descriptive Statistics for the Obtained Sample

There were 24 males (34.3%) participating in the study with an average age of 40.3 years, and 46 females (65.7%) with an average age of 42.1 years. Ages ranged from 26 to 61. Most participants were married and completed university or college. Only a minority of the sample (less than 25%) had insomnia for less than 4 years. No significant differences were found between the treatment and control groups. No participants were using medication during the study (see Table 1).

Table 1
Comparison of demographic distributions in the Protocol Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Category</th>
<th>Protocol Group</th>
<th>( \chi^2 )</th>
<th>df</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Treatment</td>
<td>Control</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>13 (37.1)</td>
<td>11 (31.4)</td>
<td>0.25</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>22 (62.9)</td>
<td>24 (68.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marital Status</td>
<td>Single</td>
<td>12 (34.3)</td>
<td>4 (11.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Married</td>
<td>16 (45.7)</td>
<td>22 (62.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Living with Partner</td>
<td>5 (14.3)</td>
<td>4 (11.4)</td>
<td>6.34</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Separated or Divorced</td>
<td>2 (5.7)</td>
<td>5 (14.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Educational Attainment</td>
<td>High School</td>
<td>5 (14.3)</td>
<td>6 (17.1)</td>
<td>0.11</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>University or College</td>
<td>30 (85.7)</td>
<td>29 (82.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep Problems</td>
<td>Falling Asleep only</td>
<td>10 (28.6)</td>
<td>9 (25.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Staying Asleep only</td>
<td>14 (40.0)</td>
<td>13 (37.1)</td>
<td>0.26</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Both Falling and Staying Asleep</td>
<td>11 (31.4)</td>
<td>13 (37.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of Insomnia</td>
<td>1-3 years</td>
<td>8 (22.9)</td>
<td>9 (25.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4-6 years</td>
<td>12 (34.3)</td>
<td>15 (42.9)</td>
<td>1.01</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>At least 7 years</td>
<td>15 (42.9)</td>
<td>11 (31.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous assistance with sleep</td>
<td>Medical Doctor or prescription drugs</td>
<td>22 (62.9)</td>
<td>25 (71.4)</td>
<td>0.59</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>13 (37.1)</td>
<td>10 (28.6p)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Numbers in parentheses represent percentages of the Protocol group.
There were 47 participants who previously used medication to treat their insomnia. Participants that had previously used medication to treat their insomnia, all reported that it (medication) provided only temporary relief of their insomnia symptoms. Furthermore, a tolerance developed when medication was used for prolonged periods of time (greater than 3 months). This was the main reason why participants discontinued the use of medication. The average duration of insomnia reported by participants was seven years.

T-tests were used to compare treatment and control groups on all objective and subjective outcome measures. No significant differences were found (see Table 2).

Table 2

Comparison of Treatment and Control Groups at Baseline

<table>
<thead>
<tr>
<th>Measure</th>
<th>Treatment M</th>
<th>Treatment SD</th>
<th>Control M</th>
<th>Control SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logical</td>
<td>14.16</td>
<td>5.28</td>
<td>15.60</td>
<td>5.70</td>
<td>1.10</td>
<td>0.28</td>
</tr>
<tr>
<td>Serial RT</td>
<td>64.09</td>
<td>8.80</td>
<td>63.34</td>
<td>9.51</td>
<td>0.34</td>
<td>0.74</td>
</tr>
<tr>
<td>Manikin</td>
<td>17.24</td>
<td>1.99</td>
<td>17.94</td>
<td>2.15</td>
<td>1.42</td>
<td>0.16</td>
</tr>
<tr>
<td>Sleep Latency</td>
<td>58.60</td>
<td>14.03</td>
<td>60.62</td>
<td>15.16</td>
<td>0.58</td>
<td>0.57</td>
</tr>
<tr>
<td>Sleep Efficiency</td>
<td>49.07</td>
<td>4.66</td>
<td>47.17</td>
<td>5.04</td>
<td>1.64</td>
<td>0.11</td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>3.60</td>
<td>0.33</td>
<td>3.46</td>
<td>0.35</td>
<td>1.74</td>
<td>0.19</td>
</tr>
<tr>
<td>ISI</td>
<td>25.25</td>
<td>1.31</td>
<td>25.97</td>
<td>1.42</td>
<td>2.18</td>
<td>0.14</td>
</tr>
<tr>
<td>BDI-II</td>
<td>24.76</td>
<td>3.08</td>
<td>24.98</td>
<td>3.32</td>
<td>0.29</td>
<td>0.77</td>
</tr>
<tr>
<td>BAI</td>
<td>24.50</td>
<td>2.22</td>
<td>25.35</td>
<td>2.40</td>
<td>1.54</td>
<td>0.13</td>
</tr>
</tbody>
</table>

Note: Both groups consist of p=35 subjects.

Performance Assessment Battery Outcome of the Treatment and Control Groups

On all measures of the PAB, the treatment groups changed differently than did the control groups: Logical Reasoning ($F (3, 129) = 150.76, p < .001$), Serial Addition and Subtraction ($F (3, 129) = 219.92, p < .001$) and Manikin ($F (3, 129) = 139.22, p < .001$) (see $T \times P$ factor in Table 3).
As can be seen in Figures 1-3, the treatment groups had better average *throughput* scores on the Logical Reasoning, Serial Addition/Subtraction Reaction Time and Manikin tasks when compared to the control groups. Figures 1-3 demonstrate that the treatment groups continued to show improvement in their performance from the initial assessment time up to the 3 month follow-up period. Furthermore, the control groups' performance appears to deteriorate up until at least the 3 month follow-up mark. The MANCOVA Group factor across all assessment times was found to be significantly different between the treatment and control groups.
Figure 1

Logical Reasoning

Throughput

TIME

Pre 10 wks 3 mths 6 mths

control

treatment

Figure 2

Serial Addition/Subtraction RT

Throughput

TIME

Pre 10 wks 3 mths 6 mths

control

treatment
Table 4 details means, standard deviations and results of the analysis of covariance for each PAB measure. With regard to performance on the Logical Reasoning task, the treatment groups' average level of performance improved from baseline to initial post treatment measurement at the 10 week mark (\(t\) (129) = 14.01, \(p < 0.005\)). There was also statistical evidence of further improvement at the 3 month mark (\(t\) (129) = 5.14, \(p < 0.005\)). No discernible change in the treatment groups' performance was found beyond the 3 month mark. The level of performance for the control groups appeared to get worse at the 10 week point (\(t\) (129) = 3.16, \(p < 0.05\)). Furthermore, there was further significant change at the 3 month mark (\(t\) (129) = 3.36, \(p < 0.05\)), and continued deterioration at the 6 month mark (\(t\) (129) = 4.67, \(p < 0.005\)).
Similarly, the treatment groups performance on the Serial Addition/Subtraction improved from baseline to initial post treatment measurement at the 10 week \( (t (129) = 14.75, p < 0.0005) \). There was further improvement at the 3 month mark \( (t (129) = 14.70, p < 0.0005) \) and no significant changes with the treatment groups performance beyond the 3 month mark. The level of performance for the control groups appeared to get worse at the 10 week mark \( (t (129) = 3.16, p < 0.05) \), at the 3 month mark \( (t (129) = 3.36, p < 0.05) \) and 6 month mark \( (t (129) = 4.67, p < 0.005) \).

Finally, the treatment groups performance of the Manikin improved from baseline to initial post treatment measurement at the 10 week \( (t (129) = 14.40, p < 0.005) \). There was also a discernible change at the 3 month mark \( (t (129) = 8.55, p < 0.005) \). No further change was found beyond the 3 month point. The level of performance for the control groups appeared to deteriorate beyond the 3 month point \( (t (129) = 6.22, p < 0.005) \).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>10 Weeks</th>
<th>3 Months</th>
<th>6 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Control</td>
<td>Treatment</td>
<td>Control</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Treatment</td>
<td>Control</td>
</tr>
<tr>
<td>Logical Reasoning</td>
<td>14.16</td>
<td>15.60</td>
<td>14.53</td>
<td>19.49</td>
</tr>
<tr>
<td></td>
<td>(5.28)</td>
<td>(5.70)</td>
<td>(6.08)</td>
<td>(5.93)</td>
</tr>
<tr>
<td>Serial Add/Sub</td>
<td>64.09</td>
<td>63.34</td>
<td>77.61</td>
<td>56.07</td>
</tr>
<tr>
<td></td>
<td>(8.00)</td>
<td>(9.51)</td>
<td>(11.31)</td>
<td>(13.59)</td>
</tr>
<tr>
<td>Manikin</td>
<td>17.24</td>
<td>17.94</td>
<td>17.11</td>
<td>23.03</td>
</tr>
<tr>
<td></td>
<td>(1.99)</td>
<td>(2.13)</td>
<td>(3.11)</td>
<td>(3.05)</td>
</tr>
</tbody>
</table>

Note: Values within parentheses represent Standard Deviation.

**Table 4**

**Means and Standard Deviations and Analysis of Covariance for the three PAB measures**

**Sleep Measures Outcome**

On all measures of sleep and fatigue the treatment groups changed differently than did the control groups: Sleep Diary Latency \( (F (3, 126) = 52.11, p < .001) \), Sleep Diary Efficiency \( (F (3, 129) = 55.78, p < .001) \), Sleep Diary Duration \( (F (3, 129) = 58.75, \)
p < .001) and the Insomnia Severity Index (F (3, 129) = 138.38, p < .001) (see T x P factor in Table 5).

Table 5

**Multivariate Analysis of Covariance for Sleep Measures**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sleep Diary Latency</th>
<th>Sleep Diary Efficiency</th>
<th>Sleep Diary Duration</th>
<th>Insomnia Severity Index</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (A)</td>
<td>1</td>
<td>0.30</td>
<td>1.77</td>
<td>0.57</td>
<td>0.97</td>
</tr>
<tr>
<td>Protocol Group (P)</td>
<td>1</td>
<td>29.27***</td>
<td>158.93***</td>
<td>112.22***</td>
<td>124.94***</td>
</tr>
<tr>
<td>Replication Group (Protocol Group)</td>
<td>12</td>
<td>0.21</td>
<td>0.57</td>
<td>0.55</td>
<td>0.17</td>
</tr>
<tr>
<td>Gender (G)</td>
<td>1</td>
<td>0.51</td>
<td>0.17</td>
<td>0.11</td>
<td>0.48</td>
</tr>
<tr>
<td>P x G</td>
<td>1</td>
<td>1.17</td>
<td>0.01</td>
<td>0.11</td>
<td>0.12</td>
</tr>
<tr>
<td>R (P x G)</td>
<td>10</td>
<td>0.84</td>
<td>0.59</td>
<td>0.73</td>
<td>0.67</td>
</tr>
<tr>
<td>S x R (P x G) within Group error</td>
<td>43</td>
<td>(584.87)</td>
<td>(130.55)</td>
<td>(1.53)</td>
<td>(22.21)</td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time (T)</td>
<td>3</td>
<td>2.98*</td>
<td>2.10</td>
<td>2.08</td>
<td>0.82</td>
</tr>
<tr>
<td>T x A</td>
<td>3</td>
<td>1.57</td>
<td>0.05</td>
<td>0.11</td>
<td>2.76*</td>
</tr>
<tr>
<td>T x P</td>
<td>3</td>
<td>52.11***</td>
<td>55.78***</td>
<td>58.75***</td>
<td>138.38***</td>
</tr>
<tr>
<td>T x R (P)</td>
<td>36</td>
<td>1.35</td>
<td>0.20</td>
<td>0.25</td>
<td>0.21</td>
</tr>
<tr>
<td>T x G</td>
<td>3</td>
<td>0.49</td>
<td>0.03</td>
<td>0.06</td>
<td>0.15</td>
</tr>
<tr>
<td>T x P x G</td>
<td>3</td>
<td>0.27</td>
<td>0.21</td>
<td>0.11</td>
<td>0.26</td>
</tr>
<tr>
<td>T x R (P x G)</td>
<td>30</td>
<td>0.55</td>
<td>0.24</td>
<td>0.26</td>
<td>0.78</td>
</tr>
<tr>
<td>T x S x R (P x G) within Group error</td>
<td>129</td>
<td>(33.85)</td>
<td>(43.96)</td>
<td>(0.31)</td>
<td>(1.82)</td>
</tr>
</tbody>
</table>

Note: S = subjects. Values within parentheses represent mean square errors.

*p < .05  **p < .01  ***p < .001.

As can be seen in Figures 4-7, at all different times of measurement the treatment groups were found to be sleeping better and reporting less fatigue. Figures 4-7 demonstrate that the treatment groups continued to show improvement in their sleep from the initial assessment time up to the 3 month follow-up period. The control groups sleep appears to remain unchanged throughout the entire duration of the study.
Table 6 details means, standard deviations, and results of the analysis of covariance between the treatment and control groups. With regard to the results on the Sleep Diary Latency, the treatment groups average latency to sleep onset improved from baseline to initial post treatment measurement at the 10 week mark ($t$ (129) =13.21, $p < 0.001$) and further improved as of the second post treatment measurement taken at the 3 month mark ($t$ (129) = 6.88, $p < 0.001$). There were no discernible changes beyond the 3 month mark. The sleep latency for the control group appeared to be constant throughout the 6 month duration of the study.

Furthermore, the treatment groups Sleep Diary Efficiency increased from baseline to initial post treatment measurement at the 10 week mark ($t$ (129) = 14.29, $p < 0.005$). There was a further increase to the 3 month mark ($t$ (129) = 7.94, $p < 0.005$). Beyond the 3 month mark there was no discernible change in the treatment groups Sleep Efficiency. There were no statistically significant changes to the control groups Sleep Efficiency throughout the duration of the study.

In addition, the treatment groups Sleep Diary Duration rose from baseline to initial post treatment measurement at the 10 week mark ($t$ (129) = 5.63, $p < 0.005$). Sleep duration continued to increase further between the 10 week and 3 month and no significant changes occurred to Sleep Duration beyond the 3 month mark. The control groups had no discernible changes throughout the entire period of the study.
Finally, the treatment groups results on the Insomnia Severity Index (ISI) showed an improvement in participants’ severity of insomnia from baseline to initial post treatment measurement at the 10 week mark ($t(129) = 19.12, p < 0.001$), and further improvement at the 3 month point ($t(129) = 7.88, p < 0.001$). There were no further discernible changes among the treatment groups beyond the 3 month mark.

The control groups results indicate no discernible changes throughout the duration of the study.

Table 6

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
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<th></th>
<th></th>
<th></th>
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<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Control</td>
<td>Treatment</td>
<td>Control</td>
<td>Treatment</td>
<td>Control</td>
<td>Treatment</td>
<td>Control</td>
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<td>F</td>
<td>P</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep Diary</td>
<td>58.60</td>
<td>60.62</td>
<td>40.21</td>
<td>57.98</td>
<td>32.33</td>
<td>56.75</td>
<td>32.14</td>
<td>57.57</td>
<td>52.11</td>
<td>&lt;.0005</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Latency</td>
<td>(14.03)</td>
<td>(15.16)</td>
<td>(10.97)</td>
<td>(11.86)</td>
<td>(13.89)</td>
<td>(15.02)</td>
<td>(14.94)</td>
<td>(16.20)</td>
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</tr>
<tr>
<td>Sleep Diary</td>
<td>49.04</td>
<td>47.17</td>
<td>64.67</td>
<td>48.98</td>
<td>76.11</td>
<td>47.58</td>
<td>75.97</td>
<td>47.63</td>
<td>55.78</td>
<td>&lt;.0005</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Efficiency</td>
<td>(4.66)</td>
<td>(5.03)</td>
<td>(6.56)</td>
<td>(7.09)</td>
<td>(10.45)</td>
<td>(11.29)</td>
<td>(10.29)</td>
<td>(11.19)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep Diary</td>
<td>3.60</td>
<td>3.46</td>
<td>5.16</td>
<td>3.46</td>
<td>5.97</td>
<td>3.40</td>
<td>5.81</td>
<td>3.41</td>
<td>58.75</td>
<td>&lt;.0005</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Duration</td>
<td>(0.33)</td>
<td>(0.35)</td>
<td>(0.67)</td>
<td>(0.73)</td>
<td>(1.03)</td>
<td>(1.11)</td>
<td>(1.02)</td>
<td>(1.12)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ISI</td>
<td>25.25</td>
<td>25.97</td>
<td>17.34</td>
<td>25.21</td>
<td>15.98</td>
<td>25.31</td>
<td>15.85</td>
<td>25.47</td>
<td>138.38</td>
<td>&lt;.0005</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1.44)</td>
<td>(1.42)</td>
<td>(2.88)</td>
<td>(3.12)</td>
<td>(3.03)</td>
<td>(3.28)</td>
<td>(3.22)</td>
<td>(3.48)</td>
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</tr>
</tbody>
</table>

Note: Values within parentheses represent Standard Deviation.

Depression and Anxiety Outcome

On both measures of depression and anxiety the treatment groups changed differently than did the control groups: the Beck Depression Inventory-II ($F(3, 129) = 123.67, p < .001$), and the Beck Anxiety Inventory ($F(3, 129) = 259.98, p < .001$) (see $T \times P$ factor in Table 7).
As can be seen in Figures 8 and 9, at all times later than the baseline measurement the treatment groups were found to be less depressed (BDI-II) and anxious (BAI) than the control groups. The treatment groups appeared to show improvement up until the 6 month mark, whereas the control groups depression and anxiety levels remained relatively unchanged during the entire study.
Figure 8

**Beck Depression Inventory II (BDI-II)**

![Graph showing changes in Beck Depression Inventory II scores over time.]

- **Estimated Marginal Means**
- **TIME:** Pre, 10 wks, 3 mths, 6 mths
- **Control** indicated by squares
- **Treatment** indicated by asterisks

Figure 9

**Beck Anxiety Inventory (BAI)**

![Graph showing changes in Beck Anxiety Inventory scores over time.]

- **Estimated Marginal Means**
- **TIME:** Pre, 10 wks, 3 mths, 6 mths
- **Control** indicated by squares
- **Treatment** indicated by asterisks
Table 8 describes the means and standard deviations of treatment and control groups for the BDI-II and BAI. The treatment groups scores on the BDI-II decreased from baseline to initial post treatment measurement at the 10 week mark (t (129) =18.05, p < 0.001). A further decrease in the scores took place at the 3 month mark (t (129) =7.35, p < 0.001), and continued to decrease at the 6 month point (t (129) =7.53, p < 0.001). The control group had no discernible changes regarding scores on the BDI-II throughout the entire duration of the study.

Finally, the treatment groups results on the BAI suggest a significant decrease to the initial post treatment measurement at the 10 week mark (t (129) =20.84, p < 0.001). Also, at the 3 month mark there was evidence that the treatment groups level of anxiety decreased (t (129) = 8.86, p < 0.001). In addition, there was a discernible change at the 6 month mark (t (129) = 7.42, p < 0.001). There were no distinguishable changes in the control groups throughout the duration of the study.

Table 8

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>10 Weeks</th>
<th>3 Months</th>
<th>6 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Control</td>
<td>Treatment</td>
<td>Control</td>
</tr>
<tr>
<td>BDI-II</td>
<td>24.76</td>
<td>24.98</td>
<td>18.97</td>
<td>25.07</td>
</tr>
<tr>
<td></td>
<td>(3.08)</td>
<td>(3.32)</td>
<td>(3.46)</td>
<td>(3.74)</td>
</tr>
<tr>
<td>BAI</td>
<td>24.50</td>
<td>25.35</td>
<td>18.80</td>
<td>25.86</td>
</tr>
<tr>
<td></td>
<td>(2.22)</td>
<td>(2.34)</td>
<td>(2.92)</td>
<td>(3.26)</td>
</tr>
</tbody>
</table>

Note: Values within parentheses represent Standard Deviation.

Table 9 describes the frequency and percentages of treatment group members that did not meet clinical criteria for chronic insomnia on the measures of Sleep Diary Latency, Efficiency and Duration. At the 10 week measurement time, 22.9% of the subjects in the treatment groups did not meet the conditions of clinical significance for sleep diary sleep latency. At all measurement times beyond the 3 month mark,
approximately 55% of the subjects in the treatment groups did not meet the conditions of clinical significance for sleep diary latency.

At the 10 week measurement time, 14.3% of the subjects in the treatment groups did not meet the conditions of clinical significance (indicative of insomnia) for sleep diary efficiency. At all measurement times beyond the 3 month mark, approximately 40% of the subjects in the treatment groups did not meet the conditions of clinical significance (indicative of insomnia) for sleep diary sleep efficiency.

As was hypothesized, the intervention is associated with an improvement in sleep time duration which is still detectable at six months after the intervention. At all measurement times beyond the 10 week mark, 100% of treatment groups members did not meet conditions significant of insomnia based on sleep diary duration. It was observed that the treatment groups experienced a 2.2 hour increase in sleep time duration on average between baseline and the six month measurement, \( t(129) = 12.54, p < .0005 \).

Contrary to the treatment groups, the control groups met conditions of clinical significance (indicative of insomnia) on all sleep parameter measures (sleep latency, efficiency and duration) from baseline to the 6 month follow-up point (Appendix F).

Table 9

<table>
<thead>
<tr>
<th>Variable</th>
<th>Clinical Status</th>
<th>10 weeks</th>
<th>Measurement Time</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>3 months</td>
<td></td>
</tr>
<tr>
<td>Sleep Diary Latency</td>
<td>Normal range</td>
<td>8 (22.9)</td>
<td>20 (57.1)</td>
<td>19 (54.3)</td>
</tr>
<tr>
<td></td>
<td>Clinical range</td>
<td>27 (77.1)</td>
<td>15 (42.9)</td>
<td>15 (45.7)</td>
</tr>
<tr>
<td>Sleep Diary Efficiency</td>
<td>Normal range</td>
<td>5 (14.3)</td>
<td>14 (40.0)</td>
<td>13 (37.1)</td>
</tr>
<tr>
<td></td>
<td>Clinical range</td>
<td>30 (85.7)</td>
<td>21 (60.0)</td>
<td>22 (62.9)</td>
</tr>
<tr>
<td>Sleep Diary Duration</td>
<td>Normal range</td>
<td>35 (100)</td>
<td>35 (100)</td>
<td>35 (100)</td>
</tr>
<tr>
<td></td>
<td>Clinical range</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

Note: Numbers in parentheses represent percentages of the treatment groups.
Table 10 describes the frequency and percentages of the treatment groups clinical status regarding the severity of insomnia as measured by the ISI. At baseline, most (97.1%) of the treatment groups suffered from severe insomnia as measured by the Insomnia Severity Index (ISI). In contrast, 85.7% of the treatment groups had attained moderate to subthreshold levels of insomnia by the 10 week mark, as measured by the ISI. This trend did not continue over the remaining measurements.

Unlike the treatment groups, no consistent changes were found throughout the duration of the study with regard to the frequency and percentages of the control groups clinical status based on the ISI (Appendix F).

Table 10

Summary of Frequency and Percentages of Treatment Groups Clinical Status regarding the Insomnia Severity Index (ISI)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Clinical Status</th>
<th>Baseline</th>
<th>10 weeks</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subthreshold</td>
<td>-</td>
<td>5 (14.3)</td>
<td>10 (28.6)</td>
<td>10 (28.6)</td>
<td></td>
</tr>
<tr>
<td>Insomnia Severity Index</td>
<td>Moderate</td>
<td>1 (2.9)</td>
<td>25 (71.4)</td>
<td>21 (60)</td>
<td>22 (62.9)</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>34 (97.1)</td>
<td>5 (14.3)</td>
<td>4 (11.4)</td>
<td>3 (8.6)</td>
</tr>
</tbody>
</table>

Note. Numbers in parentheses represent percentages of the treatment groups.

Table 11 describes the frequency and percentages of the treatment groups clinical status regarding depression and anxiety as measured by the BDI-II and BAI, respectively. At baseline, most of the treatment groups (97.1%), suffered from moderate depression, as measured by the Beck Depression Inventory-II (BDI-II). There was a change at the 10 week mark, where 74.3% of the treatment groups revealed a mild level of depression, as measured by the BDI-II. These findings remained relatively stable throughout the duration of the study.

In reference to the Beck Anxiety Inventory (BAI), between the 10 week and 3 month marks the number of subjects who had attained mild anxiety levels as indicated by the BAI,
rose 6 fold from its prior level. Over the course of the intervention, 67% of the treatment groups who had severe levels of anxiety had attained less than severe levels of anxiety.

The control groups did not demonstrate any consistent changes during the study with regard to the frequency and percentages of their clinical status on the BDI-II and BAI (Appendix F).

Table 11

**Summary of Frequency and Percentages of Treatment Groups Clinical Status regarding Depression (BDI-II) and Anxiety (BAI)**

<table>
<thead>
<tr>
<th>Clinical Status</th>
<th>Measurement Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Minimal</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>34 (97.1)</td>
</tr>
<tr>
<td>Severe</td>
<td>1 (2.9)</td>
</tr>
</tbody>
</table>

Note. Numbers in parentheses represent percentages of the treatment groups.

**Observed Preferences for Components of the CBGT Intervention**

Table 12 describes the frequency and percentages of the treatment groups evaluation of whether they found the interventions listed below helpful in dealing with their insomnia.

**Guided Imagery.**

The preferences expressed for guided imagery appeared stable across measurement times in the study. Roughly 23% of participants who received the CBGT intervention found guided imagery very helpful, while the other 77% of participants found guided imagery only somewhat helpful.

**Progressive Muscle Relaxation.**

Across the period of the study, it is apparent that there was a constant decline in the portion of participants who found progressive muscle relaxation (PMR) to be very helpful. However, all participants feel that PMR is at least somewhat helpful.
Breathing Exercises.

The preferences expressed for breathing exercises appeared stable across measurement times in the study. Approximately, 14% of participants who received the CBGT intervention found breathing exercises very helpful, while the other 86% of participants found breathing exercises only somewhat helpful.

Autogenic Training.

Throughout the study, it appears that there is a constant decline in the portion of participants who found autogenic training to be somewhat helpful. However, a majority of participants never felt that autogenic training was helpful.

Sleep Restriction.

There appears to be a trend towards higher levels of participant positive evaluation for sleep restriction. At the 6 month follow-up point, approximately 66% of treatment groups members found sleep restriction as somewhat helpful and 34% found it as very helpful. By the end of the study, no participants felt that sleep restriction was not helpful.

Stimulus Control.

Participants evaluation of stimulus control appeared to be constant over the period of the study. Approximately 66% found stimulus control somewhat helpful and 20% found it very helpful. However, approximately 14% found stimulus control not helpful at all.

Sleep Hygiene.

The preferences expressed for sleep hygiene appeared stable across measurement times in the study. Approximately, 60% of participants found sleep hygiene as somewhat helpful and 40% found it as very helpful.
Cognitive Restructuring.

It appears that participants rated cognitive restructuring positively, with approximately 55% rating the technique as very helpful at three and six months, compared to 46% at the 10 week mark.

Sleep Physiology.

Approximately 74% of participants found sleep physiology information as very helpful at the 10 week mark. This rating steadily declined by the six month follow-up measure, with only roughly 54% rating it as very helpful.

Table 12

Summary of Frequency and Percentages of Treatment Groups Evaluation of whether they found the interventions listed below helpful in dealing with their insomnia

<table>
<thead>
<tr>
<th>Clinical Status</th>
<th>Measurement Time</th>
<th>10 weeks</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Not at all</td>
<td>Somewhat</td>
<td>Very much</td>
<td>Not at all</td>
</tr>
<tr>
<td>Interventions</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Guided</td>
<td>1</td>
<td>25</td>
<td>9</td>
<td>27</td>
</tr>
<tr>
<td>Imagery</td>
<td>(2.9)</td>
<td>(71.4)</td>
<td>(25.7)</td>
<td>(77.1)</td>
</tr>
<tr>
<td>Progressive</td>
<td>-</td>
<td>24</td>
<td>11</td>
<td>28</td>
</tr>
<tr>
<td>Muscle Relax</td>
<td>(68.5)</td>
<td>(31.4)</td>
<td>(80.0)</td>
<td>(20.0)</td>
</tr>
<tr>
<td>Breathing</td>
<td>-</td>
<td>29</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>(82.9)</td>
<td>(17.1)</td>
<td>(85.7)</td>
<td>(14.3)</td>
</tr>
<tr>
<td>Autogenic</td>
<td>27</td>
<td>8</td>
<td>-</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>(77.1)</td>
<td>(22.9)</td>
<td>(82.9)</td>
<td>(17.1)</td>
</tr>
<tr>
<td>Sleep</td>
<td>12</td>
<td>18</td>
<td>5</td>
<td>23</td>
</tr>
<tr>
<td>Restriction</td>
<td>(34.3)</td>
<td>(51.4)</td>
<td>(14.3)</td>
<td>(5.7)</td>
</tr>
<tr>
<td>Stimulus</td>
<td>10</td>
<td>19</td>
<td>6</td>
<td>23</td>
</tr>
<tr>
<td>Control</td>
<td>(28.6)</td>
<td>(54.3)</td>
<td>(17.1)</td>
<td>(14.3)</td>
</tr>
<tr>
<td>Sleep Hygiene</td>
<td>-</td>
<td>20</td>
<td>15</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>(57.1)</td>
<td>(42.9)</td>
<td>(60.0)</td>
<td>(40.0)</td>
</tr>
<tr>
<td>Cognitive</td>
<td>-</td>
<td>19</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>Restructuring</td>
<td>(54.3)</td>
<td>(45.7)</td>
<td>(42.9)</td>
<td>(57.1)</td>
</tr>
<tr>
<td>Sleep Physiology</td>
<td>-</td>
<td>11</td>
<td>26</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>(31.4)</td>
<td>(74.3)</td>
<td>(42.9)</td>
<td>(57.1)</td>
</tr>
</tbody>
</table>

Note. Numbers in parentheses represent percentages of the treatment groups.
Table 13 describes the frequency and percentages of treatment groups evaluation of whether they found factors of group therapy as helpful in dealing with their insomnia. A majority of participants (62-97%) rated the following factors as intensely helpful, in dealing with their insomnia: expressing my concerns and feelings, instillation of hope, my problems and feelings are not unique, getting advise from other members, feeling of belonging, helping others, and learning from watching others.

Table 13

<table>
<thead>
<tr>
<th>Clinical Status</th>
<th>Measurement Time</th>
<th>10 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Expressing my concerns &amp; feelings</td>
<td>Instillation of Hope</td>
</tr>
<tr>
<td>Minimally</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(2.9)</td>
<td></td>
</tr>
<tr>
<td>Moderately</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>(34.3)</td>
<td>(28.6)</td>
</tr>
<tr>
<td>Intensely</td>
<td>22</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>(62.9)</td>
<td>(71.4)</td>
</tr>
</tbody>
</table>

Note. Numbers in parentheses represent percentages of the treatment groups.
Chapter 4

Discussion

Prior to discussing these results further, it is of relevance to mention the limitations of the study. The main limitation of the study relates to the nature of the control group. This control group was a no treatment control group. Gains made in the treatment groups may relate to non-specific therapy components of being in the therapy group, rather than specifically to the CBGT components. Group sessions following an alternative therapeutic approach may have been preferable in assessing the specific effectiveness of CBGT. In addition, a longer term follow-up would have yielded important information about the longer-term maintenance of therapeutic gains. Another limitation of the study relates to the participant selection. The process of referral to the study disallowed keeping a complete record of what percent of eligible patients seen at the sleep clinic chose to participate in the study or how representative the study sample was of the clinic patient population. In addition, research diagnoses were based on a semi-structured rather than a fully structured interview guide and no inter-rater reliability measures were employed. The researcher, however, did repeat the semi-structured evaluation and constantly agreed with the psychiatrists' diagnosis.

Taking the above limitations into consideration, the purpose of this study was to implement and evaluate cognitive-behavioural group therapy (CBGT), for the treatment of psychophysiological insomnia. Specifically, the study evaluated whether the implementation of the CBGT intervention would improve participants’ quality of life in the following areas: cognitive performance, sleep and fatigue, and emotional well-being. All objective (PAB) and subjective measures were found to be statistically significant.
Significant gains were found on all objective and subjective measures during the treatment period with maintenance of therapeutic gains during the follow-up period. CBGT was found to be effective in improving both cognitive performance and sleep. The CBGT also appeared effective in ameliorating emotional well-being, indicated by a decrease in depression and anxiety levels.

The results indicating improvement in sleep and emotional well-being replicate similar findings in other studies which employed an individual cognitive-behaviour approach (Espie, 1991; Espie et al., 1989; Jacobs et al., 1993; Lacks & Morin, 1992; Morin et al., 1989; Murtagh & Greenwood, 1995), as well as studies which implemented a group therapy approach (Jacobs et al., 1996; Kupych-Woloshyn et al., 1993; Lacks, 1991; Morin et al., 1993). There are no published studies concerning improvement with regard to cognitive performance, following either an individual CBT or CBGT intervention for people with insomnia. This is one of the first studies to explore this specific area. The results of this research demonstrated statistically significant differences in cognitive performance between members of the CBGT treatment groups and members of the control groups. These findings are in line with the results of studies that demonstrated that sleep deprivation was shown to accompany lowered cognitive performance (Angus et al., 1985; Johnson et al., 1998; Spiegel et al., 1998; Thorne et al., 1983).

Cognitive Performance

As was hypothesized, participants' cognitive performance, as assessed by the Walter Reed Performance Assessment Battery (PAB), was found to show statistically significant improvements up until the 3 month follow-up period. Morin and colleagues
(1999) suggested that because CBT treatments are typically implemented in brief periods of time (6-8 sessions), participants usually begin to fully integrate the newly learned clinical procedures 2-3 months following the last session. Therefore, it is quite usual to see participants continuing to make significant improvements 2-3 months following treatment. At all different times of measurement subsequent to the CBGT intervention, the treatment groups were found to have higher throughput (see Method section for definition) with regard to cognitive performance when compared to the control groups.

The control groups performance deteriorated over the duration of the study, as their sleep remained impaired. This was not surprising since the control groups lacked treatment for insomnia, thereby maintaining a level of sleep deprivation and consequently increased impairment in performance. That is to say, the more sleep deprived an individual remains, the more likely their performance is to deteriorate (Johnson et al., 1998; Spiegel et al., 1998; Thorne et al., 1983).

The fact that the treatment groups demonstrated improved cognitive performance is not surprising given that they also demonstrated improvement in their sleeping patterns. As previous research has demonstrated, sleep deprivation adversely affects cognitive performance. The observed improvement in cognitive performance of the treatment groups is likely the result of improvement in the quality and quantity of sleep. For example, a study (Angus et al., 1985) demonstrated that individuals who were sleep deprived and inactive for 60 hours and completed assessments of fatigue, sleepiness and mood every three hours, vigilance task every six hours, and a cognitive test battery every twelve hours, were found to exhibit decrements in their performance. Furthermore, another study (Spiegel et al., 1998) demonstrated that reduced sleep contributed to
decrements in participants’ performance, and that within days of being able to resume a normal sleep routine, participants’ performance began to show improvements. These findings parallel the results of this research in that participants who have been partially sleep deprived for years exhibited a level of performance that began to steadily improve once their sleep became less restricted. These findings were also substantiated by a study conducted by Johnson and colleagues (1998), where they were able to show that partial sleep deprivation over several days negatively affected participants psychomotor vigilance. The study demonstrated that when sleep was increased back to baseline duration (approximately 9 hours), performance decrements were reversed and stabilized over the week. These studies also demonstrated that as sleep was progressively deprived over time, participants’ level of performance continued to deteriorate. These findings parallel the results that were obtained from this study, where the no treatment control groups performance progressively deteriorated up until the 6 month follow-up period.

Sleep Parameters

In conjunction with the assessment of cognitive performance, this study assessed sleep parameters associated with the sleep diary in keeping with the practice in the research literature (Espie, 1991, Lacks, 1991, Lacks & Morin, 1992, Morin, Culbert, Kowatch & Walton, 1989, Morin et al., 1993; Murtagh & Greenwood, 1995), along with the assessment of additional sleep parameters associated with insomnia (Insomnia Severity Index).

As was hypothesized, at all different times of measurement subsequent to the CBGT intervention, the treatment groups always exhibited less severe symptoms of insomnia, relative to the control groups, as measured by the levels of their sleep
parameters (sleep latency, sleep efficiency, sleep duration, and insomnia severity). These results parallel the effects of individual and group cognitive-behaviour therapy reported in numerous previous studies (Kupych-Woloshyn et al., 1993; Lacks, 1991; Morin et al., 1993; Morin et al., 1994, Morin et al., 1999; Murtagh & Greenwood, 1995). Furthermore, in keeping with the literature, it was expected that approximately 40-60% of the participants who received CBGT treatment would demonstrate clinical improvements with their sleep (Jacobs et al., 1996; Lacks, 1991; Morin et al., 1994, Murtagh & Greenwood, 1995). The magnitude of improvement has been reported to be as high as 60% after individual treatment (Morin et al., 1992). The aforementioned expectations were met in this study.

The treatment groups exhibited clinically significant improvements in sleep latency, as was hypothesized. As was the case with cognitive performance, it was observed that the proportion of participants exhibiting clinically significant improvements in sleep latency stabilized at the 3 month measurement and beyond. This may be because, as previously described, the Cognitive-Behavioural treatment was implemented over a relatively brief period (8 weeks) and therefore likely required more time to fully integrate the newly learned clinical procedures (i.e. relaxation techniques, sleep restriction, stimulus control, etc.). The results of this study (57.1% of participants with clinical significant improvements in sleep latency) are similar to those of Murtagh and Greenwood (1995) study, where a 50% improvement was found among study participants. A study by Lacks (1991) demonstrated that 39% of individuals who were treated for insomnia through behaviour group therapy met clinical significance for improved sleep latency. This figure increased to 49% at the three month follow-up.
assessment. Another study (Morin et al., 1993) demonstrated that following a CBGT approach to treat late-life insomnia, participants sleep latency improved from 39.6 minutes at baseline, to 20.6 minutes at the first posttreatment follow-up and remained relatively stable up until the 3 month (21.3 minutes) and 12 month (22.4 minutes) follow-up periods.

The treatment groups exhibited an increase in the proportion of participants who failed to meet the clinical sleep efficiency standards for insomnia, as was hypothesized. Specifically, the clinical standard for insomnia, based on sleep efficiency, is a level below 80%-85%. As was the case with sleep latency, it was observed that the proportion (approximately 40%) of participants exhibiting clinically significant improvements in sleep efficiency stabilized at the 3 month measurement and beyond. These findings are consistent with the literature (Morin et al., 1994). Morin and colleagues (1994) demonstrated that 53% of their participants had a sleep efficiency greater than 80%. Another study (Morin et al., 1993) showed that sleep efficiency improved from 68.55% at baseline to 81.12% at the 3 month follow-up and 83.68% at the 12 month follow-up.

There are no criteria for clinically significant improvements in sleep time duration, although, Morin and colleagues (1999) suggest that a half hour increase is a modest improvement. We observed an average 2 hours and 20 minutes increase in sleep time duration six months following the CBGT intervention. On the basis of the published standard for modest improvement, the CBGT intervention appears to be associated with at least a modest improvement in sleep time duration. This improvement parallels similar findings of another study (Morin et al., 1993) which demonstrated that following a CBGT intervention for insomnia, participants sleep duration increased from approximately 5
hours and 15 minutes at baseline to 6 hours and 30 minutes at a 12 month follow-up. Approximately an improvement of 1 hour and 15 minutes.

The treatment groups exhibited statistically significant improvements on all subsequent measurements of the Insomnia Severity Index (ISI), up to three months after the CBGT intervention. These findings, in the context of CBGT, are consistent with recently published research studies on improvements on the ISI in individuals following participation in individual cognitive-behavioural treatment. For example, a study using the Insomnia Severity Index (ISI), demonstrated that participants with a “moderate level” of insomnia at baseline, improved to a “subthreshold level of insomnia” at 3 months posttreatment, following a CBT intervention (Bastien et al., 1999). The present study demonstrated a movement from “severe” insomnia to both “moderate” and “subthreshold” insomnia at the 3 month mark.

**Depression and Anxiety Measures**

Statistical improvements in depression and anxiety were observed to occur between all successive measurements following the CBGT intervention. It was observed that both depression and anxiety, as measured by the Beck Depression Inventory-II (BDI-II) and Back Anxiety Inventory (BAI), respectively, improved within the treatment groups up until the six month mark. In contrast, the average depression and anxiety levels within the control groups, did not change throughout the study. These findings concerning a reduction in depression and anxiety in association with improvements in sleeping patterns are consistent with those in the published literature (Espie et al., 1989; Jacobs et al., 1993; Morin et al., 1993). It was observed that whereas sleep and performance measures improved only up to the three month mark, depression and anxiety
continued to improve up to the six month mark. The continuing improvement in depression and anxiety may be due to positive changes in varied aspects of participants' lives which are enhanced by the improved levels of sleep and performance. The treatment groups exhibited an increase in the proportion of participants with a less severe clinical status of depression and anxiety.

Preferences for Components of the CBGT Intervention

As was predicted, it was found that members of the treatment groups did find the multi-faceted treatment as helpful in dealing with their insomnia. This is in keeping with other studies which have demonstrated that a combined approach is significantly more effective than no treatment (Edinger & Stout, 1985; Jacobs et al., 1993). These studies also demonstrated that the best outcomes from multicomponent interventions included sleep restriction and/or stimulus control procedures, integrated with other methods such as cognitive restructuring and relaxation methods (Edinger & Stout, 1985; Jacobs et al., 1993). These studies demonstrated that effective clinical management of insomnia often involves a combination of treatment procedures.

As was hypothesized, the Group Evaluation measure revealed that participants in the treatment groups rated psychotherapeutic factors of group therapy as moderately to intensely helpful in dealing with their insomnia. Participants rated factors such as: catharsis, instillation of hope, universality, interpersonal learning, group cohesiveness, imparting information, altruism, and imitative behaviour as helpful. These factors are pertinent to the principles and findings set forth by Lacks (1991) and Kupych-Woloshyn and colleagues (1993). The group format helps demonstrate to participants that they are not alone in their suffering and that their problems are shared by others. Inter-individual
differences become points of discussion, thereby validly excluding the notion of a singular recipe for sleep. Improvement in some members of the groups instills hope in others.

Implications and Suggestions for Future Research

The study suggest that CBGT may be an effective and cost-efficient treatment approach to chronic insomnia. At the time of cuts to the funding of health services, the provision of cost-efficient treatment becomes imperative.

Further studies of this approach to the treatment of chronic insomnia are therefore indicated. The observed improvement in cognitive performance suggests that beyond the benefit of reduced stress to individuals, effective treatment will benefit the larger society through improved work performance and reduction in insomnia related accidents. Beyond therapy groups to individuals with chronic insomnia, relevant psychoeducational and cognitive information may be shared through brochures and other forums, such as work place workshops.

Further study of the CBGT approach to chronic insomnia may take different directions. A study comparing the effect of CBGT to that of sleep medication will be informative in affecting normative clinical procedure which often involves drug prescription. The combination of CBGT and drug treatment could be compared to CBGT or drug treatment only. Cross validation of the results of the study in different clinic populations is needed as well. A larger scale study with different comparison groups may allow the separation of cognitive behavioural components from the therapeutic factors inherent in group settings.
The interaction of cognitive-behavioural therapeutic components with group therapy components could be studied through process research. This may lead to refinements in the treatment model.

To conclude, the CBGT approach was found to be promising in the treatment of chronic insomnia. Further research with this model may lead to changes common in clinical practice which involves drug prescription or individual treatment.
Reference List


APPENDIX A

INSOMNIA INTERVIEW SCHEDULE

Adapted from Morin (1993)

Insomnia Interview Schedule
### INSOMNIA INTERVIEW SCHEDULE

1. **Nature of Sleep-Wake Problem**

<table>
<thead>
<tr>
<th>Question</th>
<th>No</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have a problem with falling asleep?</td>
<td>No</td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Do you have a problem with staying asleep?</td>
<td>No</td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Do you have a problem with waking up too early in the morning?</td>
<td>No</td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Do you have a problem with staying awake During the day?</td>
<td>No</td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
</tbody>
</table>

2. **Current Sleep-Wake Schedule**

- What is your usual bedtime on weekdays? ______ o’clock
- At what time do you last awaken in the morning? ______ o’clock
- What is your usual arising time on weekdays? ______ o’clock
- Do you have the same sleep-wake schedule on weekends? YES NO
- How often do you take naps (including unintentional naps)? ______ days/week
- Do you ever fall asleep at inappropriate times/places? YES NO
- How many nights/week do you have a problem with falling/staying asleep? ______ nights
- On a typical night (past month), how long does it take you to fall asleep after you go to bed and turn the lights off? ______ hours ______ minutes
- On a typical night (past month), how many times do you wake up during the middle of the night? ______ times
- What wakes you up at night? (pain, noise, nocturia, child, spontaneous)
- On a typical night, how long do you spend awake in the middle of the night (total no. of minutes/hours for all awakenings)? ______ hours ______ minutes
- How many hours of sleep per night do you usually get? ______ hours ______ minutes
3. **Sleep Aids**

In the past 4 weeks have you used sleeping pills? **YES** **NO**

- Which drugs? Prescribed, over-the-counter, or both?
- What dosage?
- How many nights/week?

If no, have you ever?

When did you first use sleep medication?
When did you last use sleep medication?

In the past 4 weeks, have you used alcohol as a sleep aid? **YES** **NO**

- What kind and how many ounces?
- How many night/week?

If no, have you ever?

4. **Sleeping Problem History (onset, course, duration)**

How long have you been suffering from insomnia? ___ years ___ months

- Were there any stressful life events related to its onset (death of loved one, divorce, retirement, medical or emotional problems, etc.)?
- Gradual or sudden onset?
- What has been the course of your insomnia problem since its onset (persistent, episodic, seasonal, etc.)?

5. **Functional Analysis**

- What is your prebedtime routine like?
- What do you do when you can’t fall asleep or return to sleep?
- Is your sleep better/worse/same when you go away from home?
- Is your sleep better/worse/same on weekends?
- What types of factors exacerbate your sleep problem (eg. stress at work, travel plan, etc.)?
- What types of factors improve sleep (eg. vacation, sex, etc.)?
- How concerned are you about sleep/insomnia?
- What impact does insomnia have on your life (mood, alertness, performance)?
- How do you cope with these daytime sequelae?
- Have you received treatment in the past other than sleeping aids?
- What prompted you to seek insomnia treatment at this time?

6. **Symptoms of Other Sleep Disorders**

During a week do you or bed partner notice the following?

- a) Restless legs: Crawling or aching feelings in the legs, (calves) and inability to keep legs still.
- b) Periodic limb movements: Leg twitches or jerks during the night, waking up with cramps in legs.
- c) Apnea: Snoring, pauses in breathing at night, shortness of breath, choking at night, morning headaches, chest pain, dry mouth.
- d) Narcolepsy: sleep attacks, sleep paralysis, hypnagogic hallucinations, cataplexy.
- e) Gastro-esophageal reflux: Sour taste in mouth, heartburn, reflux.
- f) Parasomnias: Nightmares, night terrors, sleepwalking/talking, bruxism.
- g) Sleep-wake schedule disorder, rotating shift or night shift work, jet-lag.

**Diagnostic Impression:**

7. **Medical History/Medication Use**

- Last physical exam: **Weight:** **Height:**
- Current Medical Problems:
- Current Medications:
- Hospitalization/surgery:
8. History of Psychopathology/Psychiatric Treatment

Are you currently receiving psychological or psychiatric treatment for emotional or mental health problems?

Have you or anyone in your family ever been treated for emotional or mental health problems in the past?

Have you or anyone in your family ever been a patient in a psychiatric hospital?

Has alcohol or any drug ever caused a problem for you?

Has anything happened lately that has been especially hard for you?

What about difficulties at work or with your family?

In the last month, has there been a period of time when you were feeling depressed or down most of the day nearly every day? If yes, as long as 2 weeks?

What about being a lot less interest in most things or unable to enjoy the things you used to enjoy? If yes, was it nearly every day?

For the past couple of years, have you been bothered by depressed mood most of the day, more days than not? More than half the time?

Have you ever had a panic attack, when you suddenly feel frightened, anxious or extremely uncomfortable? If yes, 4 attacks within 1 month?

Have you ever been afraid of going out of the house alone, being in crowds, standing in a line, or traveling on buses or trains?

Have you ever been bothered by thoughts that didn’t make any sense and kept coming back to you even when you tried not to have them?

In the last 6 months, would you say that you have been worrying most of the time (more days than not)?

If psychopathology is present, evaluate its onset and temporal course in relation to sleep disturbance.

Does insomnia occur exclusively during the course of anxiety/depression episodes?
APPENDIX B

INFORMATION AND INFORMED CONSENT FORM

1. Information Sheet

2. Informed Consent Form
**INFORMATION SHEET**

**Treating Chronic Insomnia: A Cognitive-Behavioural Group Therapy Approach**

Investigators: Alfonso Marino, M.Ed.*, Dr. Niva Piran* and Dr. Sheldon Shaul**
The Ontario Institute for Studies in Education, University of Toronto*
Department of Psychiatry, The Toronto Hospital/Western Division**

Insomnia is a problem that has affected most individuals at one time or another during their lives. Although the intensity and frequency of insomnia varies, it is a widespread and persistent problem that affects, health, mood, performance, and relationships. For some, insomnia can become a persistent and debilitating problem that impairs one’s quality of life. The purpose of this study is examine the efficacy of Cognitive-Behavioural group therapy in treating individuals with chronic insomnia. Such as study is in line with the need to find psychotherapeutic ways of dealing with insomnia. This research is being conducted as a thesis project in connection with the Ontario Institute for Studies in Education (University of Toronto) and The Toronto Hospital (Western Division).

The treatment involves participation in a group of 4-5 individuals who also have chronic insomnia (there will be no fee for treatment). Issues related to your sleep problem will be discussed and interventions will also be provided, including some homework assignments. Sessions will be once per week, for 8 weeks, and were last 1.5 hours. Since the number of groups that can be run simultaneously is limited, participants will be randomly assigned to either: (1) an immediate treatment group or (2) a delayed treatment group (which will commence six months after the immediate treatment group has been completed, approximately eight months wait in total). If you agree to participate in this study, you will be asked to complete a variety of different tasks that will be presented to you on a computer. You will be required to come to the Sleep Clinic two weeks prior to treatment and will be given 2 sessions on the computer. Each task set will take about 20 minutes to complete. You will not require any computer skills. You will also be given a package of questionnaires to complete. This will take approximately 20 minutes complete. A sleep diary will also be given. This is completed over a two week period and can be returned when treatment begins. The computer tasks, questionnaires and sleep diary will be completed on 3 more occasions: two weeks following the end of treatment, three months follow-up, and six months follow-up. When treatment starts, sessions will be audio taped, strictly for the purposes of supervision and adherence. Tapes will be destroyed when the study is complete.

There are no risks associated with this study. Information discussed in Group sessions will be kept in strict confidence (*except in the event of child abuse or self-harm*). All data collected will be kept confidential and your anonymity with respect to any publication or presentation of this material be maintained. The data will be stored in confidentiality. Only this researcher (Alfonso Marino), and supervisors Drs. Piran and Shaul will have access to the data.

Your participation in this study is completely voluntary and you may discontinue your participation without prejudice and without affecting your medical care. You may withdraw from the study, or may withdraw your data from the study at any time up until the time that the study is published.

If you are interested in participating in the study or would like more information, please contact me.

Alfonso Marino, M.Ed. (Doctorate Candidate)
Department of Counselling Psychology
The Ontario Institute for Studies in Education (University of Toronto)
(416) 603-5667 (Sleep Clinic)
email: amarino@cyceur.ca
OISE Supervisor: Dr. Niva Piran
Toronto Hospital Supervisor: Dr. Sheldon Shaul
INFORMED CONSENT FORM

Treating Chronic Insomnia: A Cognitive-Behavioural Group Therapy Approach

Investigators: Alfonso Marino, M.Ed*, Dr. Niva Piran* and Dr. Sheldon Shaul**
The Ontario Institute for Studies in Education, University of Toronto*
Department of Psychiatry, The Toronto Hospital/Western Division**

I, ____________________________, have been asked to participate in a study being conducted at the Sleep disorders Clinic at the Toronto Hospital/Western Division in association with The Ontario Institute for Studies in Education, University of Toronto.

The purpose of this study is examine the efficacy of Cognitive-Behavioural group therapy in treating individuals with chronic insomnia. Such as study is in line with the need to find psychotherapeutic ways of dealing with insomnia. Information discussed in Group sessions will be kept in strict confidence (*except in the event of child abuse or self-harm*). All data collected will be kept confidential and your anonymity with respect to any publication or presentation of this material were be maintained. The data will be stored in confidentiality. Only this researcher (Alfonso Marino), and supervisors Drs. Piran and Shaul will have access to the data.

I understand that since the number of groups that can be run simultaneously is limited, I will be randomly assigned to either: (1) an immediate treatment group or (2) a delayed treatment group (which will commence six months after the immediate treatment group has been completed, approximately eight months wait in total). I understand that if I agree to participate I this study, I will be asked to complete the following: a variety of different tasks that will be presented to me on a computer, questionnaires and sleep diary. I understand that this information will be collected: two weeks before the treatment, at the end of treatment, 3 months following treatment, and 6 months following treatment. I understand that my participation in this study is completely voluntary and that I may discontinue my participation without prejudice and without affecting my medical care. I understand that I may withdraw from the study, or may withdraw my data from the study at any time up until the time that the study is published. A copy of the signed consent form will be available to me upon request. A summary of the results will be provided to me upon completion of the study at my request. If I desire the results, I will place a checkmark in the space provided below and provide my address.

My signature below indicates that I have read and understood this consent form, I have had all of my questions answered to my satisfaction, and have voluntarily agreed to participate in the study that has been outlined.

Participant's Name (Print)          Participant's Signature

Date                                Witness

__________________________________________________________

___ I would like a summary of the results sent to me at the address provided below.

Address: ____________________________
APPENDIX C

COGNITIVE-BEHAVIOURAL GROUP THERAPY MANUAL

Manual: Coping Strategies & Interventions for Insomnia
TREATMENT MANUAL

Treating Chronic Insomnia: A Cognitive-Behavioural Group Therapy Approach
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**Overview of Treatment Procedures**

**Assessment of Insomnia**
(*all should be administered 2 weeks prior to treatment & 2 weeks, 3 months and 6 months following treatment*)

a) Structured Interview by clinician
b) Sleep Diary
c) Sleep Impairment Index
d) BDI-II
e) BAI
f) Demographic Information
g) Evaluation of Intervention (only to be given after treatment)
h) Walter Reed Performance Assessment Battery (PAB)
Treatment Session 1

1. **Self-monitoring**
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce patient for self-monitoring

2. **Program overview**
   a. Behavioural: Changing maladaptive sleep habits
   b. Cognitive: Reframing dysfunctional beliefs and attitudes
   c. Educational: Sleep physiology and Promoting good sleep hygiene

3. **Agenda of therapy sessions 1-8**

4. **Nature of self-management approach**
   a. Emphasize the notion of self-control and problem solving skills
   b. contrast this approach with dependency upon sleeping pills
   c. Stress the active role of patient in treatment process
   d. Discuss the time-limited format of the intervention program

5. **Social learning explanation of insomnia**
   a. Describe contributing factors: predisposing, precipitating, perpetuating factors
   b. Review conceptual model of insomnia
   c. Relate this model to the patient’s personal sleep problem history

6. Basic facts about sleep and changes in sleep patterns over the life-span
   a) The nature of sleep (stages 1-4 NREM, REM)-handout and overheads were used
   b) Changes in sleep patterns over the course of the lifespan

7. Goal setting- what they hope to get out of group

**Materials:**
- sleep diary
- Handout: Sleep Physiology Booklet and show overhead

**Reminders:** Importance of self-monitoring
Treatment Session 2

1. Self-monitoring
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce patient for self-monitoring

2. Introduction of behavioural (sleep restriction and stimulus control) procedures
   a. Restrict time in bed to ___ hours per night
   b. Optional daytime naps (< 1 hour) no later than 3 p.m.
   c. Go to bed only when sleepy
   d. Get out of bed when unable to fall asleep/return to sleep within 10-20 minutes.
   e. Repeat this procedure as often as necessary
   f. Arise at the same time every morning
   g. Do not use the bed/bedroom for nonsleeping activities

3. Behavioural Treatment Rationale

   Materials:  Sleep diary
               Handouts of stimulus control & sleep restriction procedures (see
                        behavioural section of manual)

   Reminders: Importance of self-monitoring
Treatment Session 3

1. **Self-monitoring**
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce participants for self-monitoring

2. **Review of behavioural procedures and their rationale**
   a. Restrict time in bed to ___ hours per night
   b. Optional daytime naps (< 1 hour) no later than 3 p.m.
   c. Go to bed only when sleepy
   d. Get out of bed when unable to fall asleep/return to sleep within 10-20 minutes.
   e. Repeat this procedure as often as necessary
   f. Arise at the same time every morning
   g. Do not use the bed/bedroom for nonsleeping activities

3. **Review of problems encountered in home practice**

4. **Generation of methods to enhance compliance**
   a. Find activities to engage in when getting out of bed
   b. Identify cues to determine sleepiness and time to return to bed
   c. Use alarm clock to maintain regular arising time
   d. Find competing activities to fight urge to take nap or overwhelming sleepiness before prescribed bedtime
   e. Secure support from spouse/significant others
   f. Remember the time-limited format of program
   g. Pace activity levels and change their timing

5. **Review of homework assignment and sleep window (restriction of time in bed)**

6. **Introduce Relaxation Techniques**
   a. Progressive muscle relaxation
   b. Imagery
   c. Breathing
   d. Autogenic Training

7. **Preview of session 4 - cognitive therapy**

**Materials:**
- Sleep Diary
- Handouts of Relaxation technique procedures for PMR, Imagery, Breathing and Autogenic training.

**Reminders:**
- Importance of self-monitoring
Treatment Session 4

1. **Self-monitoring**
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce participants for self-monitoring

2. **Review of home practice and problems with behavioural procedures**
   a. Restrict time in bed to ____ hours per night
   b. Optional daytime naps (< 1 hour) no later than 3 p.m.
   c. Go to bed only when sleepy
   d. Get out of bed when unable to fall asleep/return to sleep within 10-20 minutes.
   e. Repeat this procedure as often as necessary
   f. Arise at the same time every morning
   g. Do not use the bed/bedroom for nonsleeping activities

3. **Enhancing compliance with treatment requirements**
   a. Find activities to engage in when getting out of bed
   b. Find competing activities to fight sleepiness at inappropriate times
   c. Secure support from spouse/significant others
   d. Use alarm clock to maintain regular arising time
   e. Identify behavioural cues of sleepiness (yawning, heavier eyelids)

4. **Review Relaxation Techniques**
   a. Progressive muscle relaxation
   b. Imagery
   c. Breathing
   d. Autogenic Training

5. **Cognitive Therapy**
   a. Introduce basic principles, rationale, and goals of cognitive therapy (see cognitive therapy section of manual)
   b. Discuss clinical relevance of this framework with regard to insomnia
   c. Select examples of dysfunctional cognitions and look at Beliefs and Attitude Scale as well as Cognitive Vignette handouts
   d. Identify, challenge and replace dysfunctional cognitions
      i) Correct misconceptions about the causes of insomnia
      ii) Alter dysfunctional beliefs about the impact of insomnia
      iii) Modify unrealistic sleep expectations
      iv) Enhance perceptions of control and predictability
      v) Dispel myths about good sleep practices
   e. Generate additional maladaptive self-statements specific to the participants

**Materials:**
Sleep Diary
Beliefs and Attitudes About Sleep Scale & Cognitive Vignettes Handout
Treatment Session 5

1. Self-monitoring
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce participants for self-monitoring

2. Review of home practice and problems with behavioural procedures
   a. Restrict time in bed to ___ hours per night
   b. Optional daytime naps (< 1 hour) no later than 3 p.m.
   c. Go to bed only when sleepy
   d. Get out of bed when unable to fall asleep/return to sleep within 10-20 minutes.
   e. Repeat this procedure as often as necessary
   f. Arise at the same time every morning
   g. Do not use the bed/bedroom for nonsleeping activities

3. Review Relaxation Techniques
   a. Progressive muscle relaxation
   b. Imagery
   c. Breathing
   d. Autogenic Training

4. Cognitive Therapy
   a. Review principles, rationale, and goals of cognitive therapy
   b. Discuss clinical relevance of this framework with regard to insomnia
   c. Select examples of dysfunctional cognitions and look at Beliefs and Attitude Scale as well as Cognitive Vignette handouts
   d. Identify, challenge and replace dysfunctional cognitions
      i) Correct misconceptions about the causes of insomnia
      ii) Alter dysfunctional beliefs about the impact of insomnia
      iii) Modify unrealistic sleep expectations
      iv) Enhance perceptions of control and predictability
      v) Dispel myths about good sleep practices
   e. Generate additional maladaptive self-statements specific to the participants

5. Review of progress and goal attainment

6. Preview of session 6- sleep hygiene education

Materials: Sleep Diary
Beliefs and Attitudes About Sleep Scale
Cognitive Vignettes Handout
Treatment Session 6

1. Self-monitoring
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce participants for self-monitoring

2. Review of home practice and problems with behavioural procedures
   a. Restrict time in bed to ___ hours per night
   b. Optional daytime naps (< 1 hour) no later than 3 p.m.
   c. Go to bed only when sleepy
   d. Get out of bed when unable to fall asleep/return to sleep within 10-20 minutes.
   e. Repeat this procedure as often as necessary
   f. Arise at the same time every morning
   g. Do not use the bed/bedroom for nonsleeping activities

3. Review Relaxation Techniques
   a. Progressive muscle relaxation
   b. Imagery
   c. Breathing
   d. Autogenic Training

4. Cognitive Therapy
   a. Select examples of dysfunctional cognitions and look at Beliefs and Attitude Scale as well as Cognitive Vignette handouts
   b. Identify, challenge and replace dysfunctional cognitions
      i) Correct misconceptions about the causes of insomnia
      ii) Alter dysfunctional beliefs about the impact of insomnia
      iii) Modify unrealistic sleep expectations
      iv) Enhance perceptions of control and predictability
      v) Dispel myths about good sleep practices

5. Sleep hygiene education
   a. Caffeine
   b. Nicotine
   c. Alcohol
   d. Exercise
   f. Noise, light, temperature

Materials: Sleep Diary
          Beliefs and Attitudes About Sleep Scale
          Cognitive Vignettes Handout
          Sleep hygiene handout
Treatment Session 7

1. Self-monitoring
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce participants for self-monitoring

2. Answering questions and resolving problems regarding sleep hygiene principles

3. Brief review and integration of all therapy components

4. Feedback to patient
   a. Provide feedback regarding progress and compliance with treatment
   b. Emphasize specific problem areas that need more attention
   c. Examine process and outcome relationships
   d. Increase time in bed so that it gets closer to baseline values

5. Review of home practice and problems with behavioural procedures
   a. Restrict time in bed to ____ hours per night
   b. Optional daytime naps (< 1 hour) no later than 3 p.m.
   c. Go to bed only when sleepy
   d. Get out of bed when unable to fall asleep/return to sleep within 10-20 minutes.
   e. Repeat this procedure as often as necessary
   f. Arise at the same time every morning
   g. Do not use the bed/bedroom for nonsleeping activities

6. Review Relaxation Techniques
   a. Progressive muscle relaxation
   b. Imagery
   c. Breathing
   d. Autogenic Training

Materials: Sleep diary
Treatment Session 8

1. **Self-monitoring**
   a. Review sleep diary
   b. Answer questions pertaining to diary only
   c. Reinforce participants for self-monitoring

2. **Brief review and integration of all treatment procedures**

3. **Maintaining treatment gains**
   a. Motivation and commitment
   b. Continued adherence to treatment (making part of lifestyle)
   c. Social support from spouse or significant others

4. **Relapse prevention**
   a. Make distinction among lapse, relapse, and collapse
   b. Discuss the inevitability of having an occasional poor night’s sleep and caution against interpreting this as evidence that chronic insomnia has returned
   c. Identify high-risk situations
      i) Negative emotional states (e.g. stress, anxiety, depression)
      ii) Positive emotional states (e.g. anticipation of a trip, baby)
   d. Give tips for coping with the inevitable
      i) Stay calm- no need to panic, it just makes things worse
      ii) Analyze antecedents or precipitating circumstances
      iii) Reinstate restriction of time in bed and follow stimulus control procedures
      iv) Ask for further help- phone call, booster session
   e. Give tips for coping with daytime sequelae of insomnia
      i) Change the timing of scheduled activities
      ii) Engage in sensory stimulation and time management to increase performance
      iii) Increase tolerance to sleep loss

5. **Review of progress and goal attainment- handout evaluation of group intervention**

*Materials:*
  Sleep diary
  Evaluation of group intervention
**Behavioural Strategies For Sleep:**

Sometimes problems arise with sleep when people engage in activities at bedtime that are incompatible with falling asleep. For example, they use their bedrooms for reading, talking on the phone, watching television, snacking, listening to music, paying the bills, planning the next day's events or worrying. The bottom line is that the bed and bedtime become cues for arousal rather than sleep. As mentioned above, the bedroom becomes a cue for anxiety and frustration when trying to fall asleep. The goal in order to resolve this problem is to help the person to fall asleep quickly and to maintain sleep. This is done by helping the person to associate the bed as a cue for sleep, and to weaken it as a cue for activities that might interfere with sleep. The following technique may help in this process:

*Stimulus Control Training.*

a) Lie down to go to sleep, only when you are sleepy.

b) Don't use your bed for anything except sleep and sexual activity.

c) If you find you cannot fall asleep in about 10 minutes after going to bed, get up and go into another room. Stay up as long as you want, read, listen to relaxing music, do things that are sedentary, then when feeling sleepy return to the bedroom to sleep.

d) Remember, you want to associate your bed with falling asleep quickly. Therefore, if you still cannot fall asleep after trying Rule “C”, repeat it again.

e) Set your alarm and get up at that same time every morning, regardless of how much sleep you got the night before. This were help your biological clock acquire a consistent sleep rhythm.

f) Apply these same rules, if your problem is waking up but not being able to fall back asleep later on in the night.
**Sleep Restriction Therapy**

Many studies have shown that sleep becomes robust once it has been deprived. The objective of Sleep Restriction Therapy is to do just that. For this particular intervention, a Sleep Diary is very helpful. First, estimate the amount of time you think you **sleep** each night, for example, four hours (sleep does not include the amount of time you have stayed in bed trying to sleep, it only includes the time you think you have actually slept). Once you have established your sleep time, restrict your bedtime to that amount of time (i.e. four hours), whether you have slept or not. Once four hours have gone by, get out of bed to start your day. This of course were make you feel tired during the day. However, this is a temporary side effect that is to be expected. Once you feel you have slept for the complete four hours, you may add another 15 minutes to your bedtime. If you continue to sleep better and longer, then continue to add 15 minutes to your bedtime until you reach a desired length of sleep time. However, if you have a poor night’s sleep, you should again **decrease** your total sleep time by 15 minutes, until your sleep improves again.
Relaxation Techniques:

There are many relaxation techniques that may help you fall asleep. You may try experimenting with a variety to see which works best for you. Meditation, yoga, abdominal breathing, autogenic training and progressive muscle relaxation are some forms of relaxation that may help you to fall asleep. Autogenic training were be discussed later. The main form of relaxation that were be discussed here is progressive muscle relaxation. This form of relaxation is rather simple to do and easy to learn. However, do not try this relaxation technique while driving, because it can make you sleepy, and this is not what you are looking for while driving. Before we discuss progressive muscle relaxation, it’s worth discussing how relaxation may help you in falling asleep. According to Borkovec (1982), learning to focus one’s attention on relatively pleasant, monotonous, internal sensations may be incompatible with worrisome thoughts and images that prevent sleep onset. The cognitive re-focusing may be the sleep-inducing mechanism rather than the actual tension that is released. Whatever the case may be, relaxation techniques have proven to help with sleep onset.

1) Progressive Muscle Relaxation- It’s a method of tensing and relaxing various muscle groups throughout the body. Beginning with your head and working down to your toes, contract each muscle group at a time for twenty seconds. Make yourself aware of the sensation, then slowly relax, experiencing the relaxation for another twenty seconds. You then move down to the next muscle group and repeat the procedure. Contract the muscle group for twenty seconds, make yourself aware of the sensation, then slowly relax, experiencing the relaxation for another twenty seconds, then again move down to another muscle group. At the very end once you have done each muscle group, contract
your whole body for 20 seconds, and relax. Do this three times. You should spend about
15-20 minutes doing this exercise while lying in bed before trying to go to sleep. You
can also do this in a chair during the day when feeling tense or stressed.

Remember contract for 20 seconds, then relax for 20 seconds. Begin with the
muscles in:
a) your forehead  f) hands  k) your whole body, 3 times
b) jaw  g) stomach
c) neck  h) upper legs
d) shoulders  i) lower legs
e) arms  j) feet & toes

2) Guided Imagery- various visual relaxation techniques can be used in an attempt to
relax. The body has the ability to react to mental pictures as if they were real. The
following visualization exercises may help one to relax by having an image of oneself in
the various settings, either as yourself or as an inanimate object. The following may be
modified, changed completely or done exactly as described:
a) Seashore: Relax, by breathing deeply, closes eyes, and imagine strolling along a quiet
beach on a balmy day. Imagine wearing a swim suit, and feeling the warm sun on the
skin and the sand between the toes. Smell the fresh sea air and listen to the waves
breaking on the sand. Enjoy all of the soothing surrounding as walk continues down the
beach.
b) Bubbles: Imagine that your thoughts are bubbles, which float up and out of you,
clearing your mind as they go. Or picture bubbles rising to the surface of a glass of soda
water. As the carbonated bubbles reach the surface and burst, release any thoughts you
may have with them. Clear your mind and continue watching the glass. As each bubble
reaches the surface, related it to the letting go of your own bubbling tension. Continue to
release your tensions as you watch all the bubbles escape, until you have relaxed your mind and body and the glass of carbonated water is crystal clear and undisturbed.

c) Escalator: Imagine stepping onto the top of a long, slow-moving escalator. While you slowly ride down this escalator, feel yourself going down, down, down into a deeper and deeper state of relaxation. Allow your entire body to relax as you continue to ride down this escalator.

d) Floating clouds: Explore a feeling of airiness and lightness with this image. Close your eyes and picture a bright, sunny day. The air is still except for a few wispy clouds that glide by. Imagine yourself as one of those white fluffy clouds. Feel the sensation of weightlessness as you allow yourself to drift through the sky. You feel warmed by the sun, and very buoyant. If you have any difficulty with this image, you may first want to visualize yourself in an airplane. Gaze out of your window and see the sea of cotton clouds below you. Imagine yourself safely stepping out of the plane and onto the clouds. You bounce from one mound to another until you land on the one you were become. Imagine lying down on this cloud bed, then allow yourself to become the floating cloud.

e) Hot air balloon: Picture yourself as a colourful inflatable balloon. Mentally inflate yourself with every inhalation. Imagine yourself gently rising to the ceiling, or if outdoors, to the sky. Take in the scenery around you as you gently continue to rise. Notice all the details of your surroundings: the colours, objects, smells, temperature, and so on. You can float as long as you wish, and when you are ready to return, slowly exhale. With each exhalation, gently return to your original position. Take time to enjoy this feeling.
3) **Breathing**—should be in a quiet comfortable room. The practice begins in a sitting posture and progresses to a lying-down position. It takes approximately 20 minutes to complete. In the sitting position, sit in a comfortable chair, preferably one providing good lower back support. Place both feet on the floor, with knees slightly apart. Wear loose clothing so you do not feel constricted. Begin by stroking your abdomen gently to bring your awareness there and to help you let go of tense, tight muscles. Breathe as you stroke, for the next few minutes. You may close your eyes in order to bring awareness to your breathing. What do you feel? As you breath out, make a soft whispered “Haaaa” sound. Allow your shoulders to stay relaxed. Slowly and gently inhale through your nose and exhale through your mouth whispering “Haaaa”. Notice any tension in your abdomen and let it go. The same process is repeated after a few minutes in the lying down position.

In the lying position, choose a comfortable surface such as your bed. Lie on your back with your feet shoulder-width apart and your arms a few inches away from the body. Be sure you are comfortable, try placing one pillow under your knees for the lower back support and one under your head. Then be ready to place a 2 to 5 pound weight on your abdomen. This can be a book or a bag of rice or beans. Begin to take slow deep breaths. The breathing should be effortless.

4) **Autogenic Training**—this is a technique that requires focusing one’s attention on various parts of the body (particularly your limbs), coupled with self-suggestions of heaviness and warmth.
Cognitive Strategies For Sleep:

Principles of CBT- Cognitive-behavioural therapy looks at the relationship between thoughts, behaviours and emotions. Cognitive therapy is an active, directive, time-limited, structured approach. The philosophy of cognitive therapy is largely based on the rationale that individuals affect, or emotions, and behaviour are largely determined by the way in which they structure the world (cognitions). These structures or cognitions are rooted in attitudes or assumptions developed from previous experiences. Alterations in the way someone structures his/her world can lead to subsequent changes in affect and behaviour which can ultimately lead to an improvement in the way he/she functions. A model of CBT, links the environment or ‘Activating’ events to the emotional ‘Consequences’ by the intervening ‘Belief’. Becoming aware of maladaptive beliefs and the negative emotional consequences of these beliefs, can empower clients to actively alter their beliefs which were subsequently alter the emotional consequences.

CBT and Chronic Insomnia- The first step in cognitive therapy is to identify the client’s specific dysfunctional sleep cognitions. As a starting point in therapy a questionnaire on personal beliefs and attitudes about sleep is given to clients. It is used to help identify cognitions to work on during treatment. Once the cognitions have been identified, clients are asked to monitor them at home, both at night and during the day. Cognitions need to be identified, clarified and challenged and their validity needs to be tested. Finally dysfunctional cognitions need to be replaced with more adaptive substitutes. (Sloan et al., 1993)

There are four main targets for cognitive therapy in insomnia. The first is to change the unidimensional explanations of insomnia, for example, ‘insomnia is entirely
due to some chemical imbalance and therefore the only treatment that can work is a sleeping pill', and to give the individual more control over changing the factors that are causing the insomnia. The second is to address some misattributions or amplifications of the consequences of insomnia. Some people feel that whenever they feel bad during the day it is because they have not slept well the night before. This may be true to some extent but it appears to be amplified in chronic individuals with insomnia. The third target is to change unrealistic explanations about sleep, such as the absolute need to have 8 hours of sleep per night in order to function the next day. Finally, the issue of performance anxiety, fear of losing control or the idea that insomnia is destroying one’s life completely, and the learned helplessness that is associated with insomnia, need to be addressed.

Psychophysiological insomnia may be manifested by increased internal physiological arousal and conditioning factors (Hartmann, 1988). The characteristics of internal arousal can include: a tendency to high muscle tension, anxiety about specific daytime events (anxiety in the sense of fear of failure), a fear of letting go in many different senses, obsessional characteristics associated with holding on and an inability to let go of anything, perfectionistic tendencies- a need to do everything right, and personality features of anger and paranoia. Sleep is associated with reduced autonomic activity. Consequently, when physiological arousal is present, sleep becomes impaired.

An important principle in many cognitive therapies is to enlist the patient as “coscientist”. Instead of passively listening to the advice of the experts, the patient should be asked to make and test their own recommendations. In the forum of a group setting this could include the collaboration of other group members who are experiencing
similar problems. Being an active participant can be done by keeping a sleep diary, where the he or she notes how they slept. Using a day log, can help the participant keep track of activities which in their opinion, might be related to sleep quality. Daily events can be rated on a scale from 0 to 10 every night before going to sleep. After the individual has kept the day logs and for a week or two, one can evaluate whether or not there is actually a relationship. Instead of being a helpless victim of insomnia who has to be cured by the therapist, the patient now becomes an active collaborator in the task of finding solutions to a circumscribed problem (Hauri, 1991)

Chronic insomnia is seen as a symptom of a breakdown in coping and in adaptation. Emotional arousal is seen as being precipitated by unexamined, unresolved emotional conflicts. A goal of therapy is to help determine the nature of these conflicts, to correct misperceptions, and to encourage a resolution of the conflict (Hauri, 1991).

Going to bed with a racing mind were almost guarantee you disturbed sleep, if any. This anxiety reinforces that you were not be able to sleep, which then causes further anxiety, which then perpetuates your inability to sleep. It’s a vicious cycle. Cognitive strategies try to help you to deal with your worrisome preoccupations and aim to replace them with calmness. The person who lies in bed and is constantly tossing, turning and cursing that they cannot sleep, and “I’m going to be a wreck tomorrow!”, were only be perpetuating further anxiety, making sleep that much more difficult. Cognitive interventions try to focus on what patients think and tell themselves and correct irrational “catastrophizing” thoughts with calming ones. When going to bed and feeling anxious or having many thoughts race through your mind, try some of the following:
Positive Self-Talk—try to have a positive attitude towards your sleep and try repeating the following points to yourself:

a) I have set aside time to deal constructively with my problems tomorrow.

b) If I awaken early tonight, I were not dwell upon it, but were remain relaxed with my mind in neutral.

c) Nightly arousals are normal.

d) Developing these poor sleep habits took time, so it were also take time for them to return back to normal.
Educational Strategies For Sleep - Sleep Hygiene:

There is no one quick fix solution on how to fall asleep and how to sleep well. Everyone is different. You should be patient and see what works best for you. This may require a little time and some trial and errors. However, it's well worth it once you find the system that best works for you. The following are some suggestions:

- avoid naps, except for a brief 10-15 minutes nap 8 hours after arising
- restrict sleep period to average number of hours actually slept per night in the preceding week. Too much time in bed can decrease quality on subsequent night
- get at least 20 minutes of exercise, 3-5 days per week. It's best to finish exercise 6 hours before bed
- take a hot bath to raise your temperature 2 degrees Celsius for 30 minutes within 2 hours before bedtime. A hot drink, (warm milk) may promote relaxation.
- keep a regular time out bed 7 days a week
- limit exposure to bright light during the night
- do not smoke during the night
- do not smoke after 7 p.m.
- avoid caffeine after 10 a.m..

Heavy smoking can disturb sleep (2 packs a day). Your body reacts to the time of the last cigarette after 3 hours. Nicotine is a stimulant. Therefore, a cigarette before sleeping may awaken you. Quitting smoking all together, may cause some sleep problems due to withdrawal. However, this should subside (Community Health Network, 1984).

- avoid alcohol consumption. Alcohol can fragment sleep. Alcohol is not a good aid to sleep. It may help you to fall asleep, but the sleep is of poor quality and it causes early awakenings

The best sleep happens without sleeping pills. Sleeping pills can provide temporary relief to insomnia, but can have varied short and long term effects. Sleeping pills become less effective with regular use and can become addictive. Try to use them only if you have to.

- Some sleeping pills intensify the effects of alcohol and vice versa. The combination of the two should be avoided

- do not eat or drink heavily for 3 hours before bedtime. A light bedtime snack may help (warm milk). A sudden change in diet can disrupt your sleep. Too heavy or spicy a meal before bed may wake you, and so may hunger. Not eating in the last two to four hours before bedtime may help. Try to stick to normal mealtimes if possible.

- reduce drinking a few hours before bed. The human bladder usually has an emptying frequency with a day rhythm. Therefore, sleep is more likely to be interrupted by the need to urinate. So cutting down the amount of drinking gives your bladder less stimulus to work. Avoid caffeine (coffee, tea, cola, chocolate and some pills) two hours before sleep. Caffeine should be avoided for at least five hours before trying to go to sleep. A warm glass of milk before bed has found to help sleep quality.
• list problems, worries or things that you have to do on a piece of paper, or leave for the following day. Do not bring any concerns into the bed with you.

• keep clock face turned away with alarm set, this avoids excessive worry with regard to total sleep time.

• use bedroom only for sleep, do not work of do other activities that can lead to prolonged arousal.

• keep room dark, quiet, well ventilated and at comfortable temperature. Light from the sun, really creates problems in trying to sleep. Melatonin, which has a circadian rhythm, is a hormone which induces sleep. However, light inhibits this hormone. Therefore, making sure it’s dark in your bedroom and avoiding light were help to fall asleep. Heavy curtains and an eye-mask really helps.

• Fresh air in the bedroom before you sleep should help. Using an air-conditioner if the room gets too hot, cold, humid or dry should be helpful. Sleep is best at about 15 to 18 degrees Celsius

• Beds often deteriorate over time. Upgrade if it’s in poor condition. Its mattress should be firmly sprung, with no sag

• use a bedtime ritual, reading before lights-out may be helpful. Go through the same rituals of going to bed as you usually do before night sleep. Brushing your teeth and putting on sleeping clothes may help you feel ready for sleep. A warm bath or shower may also help you to feel sleepy.

• See whether you need time to wind down after work. It may be helpful to relax if you are usually tense. It may be wise to read or watch TV to calm down.

• Trying to get in the right frame of mind for sleep is important. If you have difficulties falling asleep, try gently music or a relaxation tape or some of the relaxation techniques that were be discussed later in the workshop. Getting angry that you cannot sleep, were only make it more difficult to sleep.

• Working the morning shift may cause some anxiety about waking up in time. This anxiety can prevent you from having a good night’s rest. Making sure that you have a foolproof alarm system can help reduce this anxiety. For example, get an alarm that repeats, or an alarm that increases its volume, or set two alarms, one that runs on batteries and one that you can plug in.

• keeping a record of your sleep, its problems and successes, and of these guidelines, may help you to build up your experience and expertise more quickly. It may allow you to follow in a systematic manner, what needs to be worked on and what is working well for you. You are the best expert on your own sleep.
**Nutrition: Good Nutrition equals Good Sleep:**

Nutrition plays a role in how we feel. Depending on what you eat, you may find you are more alert when working night shifts and that you can sleep better. Having a meal rich in protein boosts concentrations of chemicals in the brain which can stimulate activity. Eating meals rich in carbohydrates increases concentrations of serotonin, a sleep-inducing chemical in the brain. However, there are other vitamins and minerals that one should know about, especially if you are having difficulty sleeping. The following is a description of vitamins and minerals that can help you sleep: Hauri, P.J. (1991). Bootzin, R.R. (1991). Bulletin of European Shiftwork Topics (1991) Monk, H.T. and Folkard, S. (1992)

1. **The B vitamins:** These vitamins regulate the body's use of amino acids, including tryptophan. Some studies have shown that Vitamin B-3, enhances the effect of tryptophan. Tryptophan is one of the 22 amino acids found in protein. Tryptophan is the substance from which the sleep-inducing brain chemical serotonin is made. This vitamin is reported to be effective in alleviating the type of insomnia suffered by people who fall asleep readily but who are unable to fall back asleep after awakening later in the night. The current Recommended Dietary Allowance (RDA) for vitamin B-3, which is found in high-protein foods such as fish, liver, kidney, chicken, peanuts, milk and eggs is 15 milligrams a day. Other research indicates that some sleep problems can arise from a deficiency Inallic acid, which is a member of the Vitamin B family. This can be found in asparagus, brocoli, cauliflower, cabbage, green peas, kidney and lima beans, beets, sweet potatoes, whole-grain cereals and breads, oranges, cantaloupe and organ meats.
The RDA is 180 micrograms per day. B vitamins can be easily leached from our body through cigarette smoking, alcohol, birth-control pills and stress.

2. **Calcium:** This mineral is a natural relaxant that has a calming effect on the central nervous system. Some studies have shown that even a minor calcium deficiency can cause muscle tension and insomnia. Stress also rapidly depletes our bodies of calcium. Therefore, you should take enough calcium daily. The RDA is 800 milligrams daily. If you are allergic to milk or just do not like it, try supplemental forms of calcium, like mustard greens, dandelion greens, broccoli, spinach and sardines.

3. **Magnesium and potassium:** Magnesium (which is found in potatoes, whole-grain bread, milk, meat, fish, poultry, eggs, dark green leafy vegetables and citrus fruits), is a natural sedative. Studies have shown that magnesium deficiency can cause insomnia. The RDA is 280 milligrams a day. Potassium (which is found in meat, milk, potatoes, bananas, oranges, apricots), in combination with magnesium has also been found effective in alleviating chronic fatigue.

4. **Zinc:** A deficiency in zinc can contribute to insomnia. This mineral can be found in oysters, herring, meat, milk, eggs, whole grains, peas, beans soybean curd, raisins, dried figs and apricots. The RDA is 12 milligrams per day.

5. **Iron and copper:** Recent studies reported that a deficiency in either copper or iron has an effect on sleep patterns. Women who received insufficient amounts of copper or iron, reported that they found themselves sleeping longer than usual and also waking more frequently during the night. They also reported that they would awaken tired and not refreshed. There is no RDA for copper. However, it can be found in whole-grain cereals and breads, shellfish, nuts, organ meats, eggs, poultry, dried beans and peas and
leafy dark-green vegetables. The RDA for iron is 15 milligrams a day. It is found in organ meats and dark-green leafy vegetables, as well as in beef, sardines, oysters, prunes and other dried fruits, peas and lima beans.

**Physical Fitness:**

Trying to keep fit is important for several reasons. Many jobs do not provide the opportunity for the human system to maintain heart, lungs and muscles in good working order. The human body has evolved under a physically active life and stays healthier when there is some regular physical activity. Doing at least twenty minutes of exercise, three times a week is said to improve mood and increase resistance to stress. Shift workers are in a difficult position because of their hours, in that, their working hours can interfere with normal participation in regular team sports and group physical activities.

The following are some rules that can help with physical fitness:

1. **Keep your stamina up.** Try to take part in some physical activity. Aiming to raise your heart beat is a good start. This can be done by training for 20 minutes a day, 3 times a week. The purpose is to raise your hear-beat from its normal 72 beats per minute towards a training-rate: a minimum of 180 minus your age, i.e. 140 for a 40 year old. Using large muscle groups, like in your legs is quite effective. Such exercises include, swimming, cycling or jogging. Brisk walking is also effective.

2. **Make good use of your days.** When working nights, you are usually free during the day while most people are at work. This means you can take advantage of avoiding crowds for leisure activities like golf, swimming-pools, etc.

3. **Don’t over do it.** Do not exercise too strenuously before starting your night shift. You do not want to exhaust yourself before work.
4. **Visit your doctor for an annual check.** You should have a regular medical check-up, at least once a year. Many problems can be treated if detected early.

5. **Check yourself out.** Be aware of any gradual developments of problems that may result from shiftwork. Such as, weight loss or gain, gastric or digestive problems, excessive fatigue or nervous disorders. These are early signs of ill-effects resulting from night work.

6. **Take care of your life-style.** Working different hours can lead to health related habits, like smoking, drinking caffeine or alcohol and dependency on sleeping pills. Keeping a diary of your life-style, may help to warn you of any growing problem areas.
APPENDIX D

PSYCHOMETRIC BATTERY

1. Sleep Diary: Center for Sleep and Chronobiology- University of Toronto

2. The Insomnia Severity Index (ISI)

3. The Beck Depression Inventory-II (BDI-II)

4. The Beck Anxiety Inventory (BAI)
1. Sleep Diary
**SLEEP DISORDERS CLINIC**

Center for Sleep and Chronobiology
University of Toronto

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Name ____________________________________________

Date Started ____________________ Day of Week ____________

Instructions:
- Please leave diary near your bedside.
- It is important that you fill out this chart each morning.
- Mark your diary in the following way:

<table>
<thead>
<tr>
<th>ACTIVITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>A - each alcoholic drink</td>
</tr>
<tr>
<td>C - each caffeinated drink includes coffee, tea, chocolate, cola</td>
</tr>
<tr>
<td>P - every time you take a sleeping pill or tranquilizer</td>
</tr>
<tr>
<td>M - meals</td>
</tr>
<tr>
<td>S - snacks</td>
</tr>
<tr>
<td>X - exercise</td>
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<tr>
<td>T - use of toilet during sleep-time</td>
</tr>
<tr>
<td>B - noises that disturb your sleep</td>
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<tr>
<td>W - time of wake-up alarm (if any)</td>
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**SLEEP TIME (including naps):**

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**Example:**

**ACTIVITIES**

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**SLEEP TIME**

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<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>A</td>
<td>S</td>
<td>T15</td>
<td>W</td>
<td>S</td>
<td>M</td>
<td>M</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Lights Out** 12:30 am
**Total Sleep Time** 5 hrs
<table>
<thead>
<tr>
<th>Week 1</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>7</td>
</tr>
</tbody>
</table>
2. Insomnia Severity Index (ISI)
(Morris, 1993)

1. Please rate the current severity of your insomnia problem(s):

<table>
<thead>
<tr>
<th>Difficulty failing asleep:</th>
<th>None</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Difficulty staying asleep:</th>
<th>None</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Problems waking up too early:</th>
<th>None</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

2. How satisfied/dissatisfied are you with your current sleep pattern?

<table>
<thead>
<tr>
<th>Very satisfied</th>
<th>Moderately satisfied</th>
<th>Very dissatisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Not at all</th>
<th>A little</th>
<th>Somewhat</th>
<th>Much</th>
<th>Very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

4. How noticeable to others do you think your sleeping problem is in terms of impairing the quality of your life?

<table>
<thead>
<tr>
<th>Not at all</th>
<th>A little</th>
<th>Somewhat</th>
<th>Much</th>
<th>Very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

5. How worried/distressed are you about your current sleep problem?

<table>
<thead>
<tr>
<th>Not at all</th>
<th>A little</th>
<th>Somewhat</th>
<th>Much</th>
<th>Very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks, including today. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

<table>
<thead>
<tr>
<th>1. Sadness</th>
<th>6. Punishment Feelings</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: I do not feel sad.</td>
<td>0: I don’t feel I am being punished.</td>
</tr>
<tr>
<td>1: I feel sad much of the time.</td>
<td>1: I feel I may be punished.</td>
</tr>
<tr>
<td>2: I am sad all the time.</td>
<td>2: I expect to be punished.</td>
</tr>
<tr>
<td>3: I am so sad or unhappy that I can’t stand it</td>
<td>3: I feel I am being punished.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2. Pessimism</th>
<th>7. Self-Dislike</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: I am not discouraged about my future.</td>
<td>0: I feel the same about myself as ever.</td>
</tr>
<tr>
<td>1: I feel more discouraged about my future than I used to.</td>
<td>1: I have lost confidence in myself.</td>
</tr>
<tr>
<td>2: I do not expect things to work out for me.</td>
<td>2: I am disappointed in myself.</td>
</tr>
<tr>
<td>3: I feel my future is hopeless and will only get worse.</td>
<td>3: I dislike myself.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. Past Failure</th>
<th>8. Self-Criticalness</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: I do not feel like a failure.</td>
<td>0: I don’t criticize or blame myself more than usual.</td>
</tr>
<tr>
<td>1: I have failed more than I should have.</td>
<td>1: I am more critical of myself than I used to.</td>
</tr>
<tr>
<td>2: As I look back, I see a lot of failures.</td>
<td>2: I criticize myself for all of my faults.</td>
</tr>
<tr>
<td>3: I feel I am a total failure as a person.</td>
<td>3: I blame myself for everything bad that happens.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4. Loss of Pleasure</th>
<th>9. Suicidal Thoughts or Wishes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: I get as much pleasure as I ever did from the things I enjoy.</td>
<td>0: I don’t have any thoughts of killing myself.</td>
</tr>
<tr>
<td>1: I don’t enjoy things as much as I used to.</td>
<td>1: I have thoughts of killing myself, but I would not carry them out.</td>
</tr>
<tr>
<td>2: I get very little pleasure from the things I used to enjoy.</td>
<td>2: I would like to kill myself.</td>
</tr>
<tr>
<td>3: I can’t get any pleasure from the things I used to enjoy.</td>
<td>3: I would kill myself if I had the chance.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>5. Guilty Feelings</th>
<th>10. Crying</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: I don’t feel particularly guilty.</td>
<td>0: I don’t cry anymore than I used to.</td>
</tr>
<tr>
<td>1: I feel guilty over many things I have done or should have done.</td>
<td>1: I cry more than I used to.</td>
</tr>
<tr>
<td>2: I feel quite guilty most of the time.</td>
<td>2: I cry over every little thing.</td>
</tr>
<tr>
<td>3: I feel guilty all of the time.</td>
<td>3: I feel like crying, but I can’t.</td>
</tr>
</tbody>
</table>
4. BAI
Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

1. Numbness or tingling.
2. Feeling hot.
3. Wobbliness in legs.
4. Unable to relax.
5. Fear of the worst happening.
6. Dizzy or lightheaded.
7. Heart pounding or racing.
8. Unsteady.
11. Feelings of choking.
14. Fear of losing control.
15. Difficulty breathing.
17. Scared.
18. Indigestion or discomfort in abdomen.
19. Faint.
20. Face flushed.
21. Sweating (not due to heat).
APPENDIX E

DEMOGRAPHICS QUESTIONNAIRE, GROUP INTERVENTION EVALUATION, BELIEFS AND ATTITUDE ABOUT SLEEP SCALE, AND COGNITIVE VIGNETTES RELATED TO INSOMNIA

1. Demographics Questionnaire

2. "Group Intervention" Evaluation

3. Beliefs and Attitude about Sleep Scale

4. Cognitive Vignettes Related to Insomnia
1. DEMOGRAPHICS

1) Today's Date: __________

2) Age: __________

3) Sex: Male  Female

4) Current Status: a) Single  b) Married  c) Living with a partner  d) Separated  e) Divorced  f) Widowed
   (Please circle)

5) How long have you been suffering from insomnia? ________________

6) Do you have difficulty with: (please circle)
   a) falling asleep
   b) staying asleep
   c) waking up early
   d) all of the above
   e) both "a" and "b"
   f) both "a" and "c"
   g) both "b" and "c"

7) Are you currently taking medication for your insomnia?
   a) No  (if no, please go to question #8)
   b) Yes  (if yes, please specify the type and amount) ________________

8) Have you ever taken medication in the past to treat your insomnia?
   a) No
   b) Yes (if yes, please answer question #9)

9)  i) Type of medication and amount:

    ii) How long did you take the medication for: __________

    iii) Was it helpful (please circle): No  Yes

    iv) Why did you stop (use space below):

    ______________________________________________________________________

10) Have you ever sought treatment for your insomnia before?
   a) No
   b) Yes (if yes, answer question #11)

11) What type of treatment did you receive?

    ______________________________________________________________________
2. EVALUATION OF THE GROUP INTERVENTION

Did you find the following interventions helpful in dealing with your insomnia?

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Somewhat</th>
<th>Very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) guided imagery</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>b) progressive muscle relaxation</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>c) Breathing exercises</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>d) autogenic training</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>e) Sleep Restriction</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>f) Stimulus control</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>g) Sleep Hygiene</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>h) Cognitive restructuring (changing beliefs &amp; attitudes regarding your sleep and insomnia)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>i) Knowledge regarding Sleep physiology</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

Please indicate whether the factors listed below were helpful to you as a group member, in dealing with your insomnia.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Not at all</th>
<th>Minimally</th>
<th>Moderately</th>
<th>Intensely</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) A feeling of belonging</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>b) Instilling hope</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>c) Expressing my concerns</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>d) Self Understanding</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>e) Giving me a chance to help others</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>f) Understanding that my problems, feelings and fears were not unique to me</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>g) Getting advice or suggestions from other group members.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>h) Learning from watching others</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Several statements reflecting people’s beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement, place a mark (✓) along the line wherever your personal rating falls. Try to use the whole scale, rather than placing marks at one end of the line.

1. I need 8 hours of sleep to feel refreshed and function well during the day.

2. When I don’t get a proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer.

3. Because I am getting older, I need less sleep.

4. I am worried that if I go for one or two nights without sleep, I may have a nervous breakdown.

5. I am concerned that chronic insomnia may have serious consequences for my physical health.

6. By spending more time in bed, I usually get more sleep and feel better the next day.

7. When I have trouble getting to sleep, I should stay in bed and try harder.

8. I am worried that I may lose control over my abilities to sleep.

9. Because I am getting older, I should go to bed earlier in the evening.
10. After a poor night's sleep, I know that it will interfere with my daily activities on the next day.

Strongly disagree  Strongly agree

11. In order to be alert and function well during the day, I am better off taking a sleeping pill rather than having a poor night's sleep.

Strongly disagree  Strongly agree

12. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before.

Strongly disagree  Strongly agree

13. Because my bed partner falls asleep as soon as his or her head hits the pillow and stays asleep thorough the night, I should be able to do so too.

Strongly disagree  Strongly agree

14. I feel that insomnia is basically the result of aging, and there isn't much that can be done about this problem.

Strongly disagree  Strongly agree

15. I am sometimes afraid of dying in my sleep.

Strongly disagree  Strongly agree

16. When I have a good night's sleep, I know that I will have to pay for it on the following night.

Strongly disagree  Strongly agree

17. When I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week.

Strongly disagree  Strongly agree

18. Without an adequate night's sleep, I can hardly function the next day.

Strongly disagree  Strongly agree

19. I can't ever predict whether I'll have a good or poor night's sleep.

Strongly disagree  Strongly agree

20. I have little ability to manage the negative consequences of disturbed sleep.
When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before.

I get overwhelmed by my thoughts at night and often feel I have no control over my racing mind.

I feel I can still lead a satisfactory life despite sleep difficulties.

I believe insomnia is essentially the result of a chemical imbalance.

I feel insomnia is ruining my ability to enjoy life and prevents me from doing what I want.

I avoid or cancel obligations (social, family, occupational) after a poor night’s sleep.

A “nightcap” before bedtime is a good solution to sleep problems.

Medication is probably the only solution to sleeplessness.

My sleep is getting worse all the time, and I don’t believe anyone can help.

It usually shows in my physical appearance when I haven’t slept well.
4. COGNITIVE VIGNETTES RELATED TO INSOMNIA

1) Relationships between Sleep Cognitions and Affect

Situation One:

Eating breakfast in the morning

Cognitions/thoughts:
"How am I gonna get through the day after such a miserable night?"

Feelings:
Depressed, helpless

Situation Two:

Poor functioning at work

Cognitions/thoughts:
"I just can’t do my work after a bad night’s sleep”.

Feelings:
Angry, irritable

Situation Three:

Watching TV in the evening

Cognitions/thoughts:
"I must have some sleep tonight”.

Feelings:
Anxious or apprehensive

Situation Four:

Getting ready for bed

Cognitions/thoughts:
“What’s the use of going to bed tonight when I know I won’t be able to go to sleep?”

Feelings:
Helpless, out of control
2) Altering Beliefs and Attitudes about Sleep

Five Typical Dysfunctional Sleep Cognitions:

1) Misconceptions of the causes of insomnia
2) Misattributions or amplifications of its consequences
3) Unrealistic sleep expectations
4) Diminished perceptions of control and predictability of sleep
5) Faulty beliefs about sleep promoting practices

Vignette 1: Misconceptions of Insomnia Causes

Dysfunctional Cognition: "I feel my insomnia is basically the result of some biochemical imbalance or pain."

Underlying Belief: "Unless these underlying problems are corrected, there is nothing I can do to improve my sleep."

Cognitive Errors: Misattribution, faulty evidence, absolute thinking

Interventions/Alternative Interpretations:
1) Exclusive attribution of insomnia to these external causes is self-defeating because you may indeed have little control over them.

2) Regardless of the initial precipitating causes, psychological and behavioural variables are almost always involved in chronic insomnia.

3) Because you have some control in changing these variables, you can also improve your sleep patterns.

Vignette 2: Other Misconceptions of Insomnia Causes

Dysfunctional Cognition: "I feel my sleep problem is essentially the result of aging and I can't do anything about it."

Underlying Belief: "Disturbed sleep is an inevitable consequence of aging."

Cognitive Errors: Misattribution, faulty evidence, absolute thinking

Interventions/Alternative Interpretations:
1) Do all older people you know have sleep disturbances?
2) Beyond some normal age related changes in sleep patterns, not all older people suffer from insomnia; therefore, other factors must be involved.

3) Lifestyle changes accompanying retirement may alter your sleep patterns; thus, you can make some adjustments in these areas to improve sleep.

Vignette 3: Magnifications/Misattributions of Insomnia Consequences

Dysfunctional Cognitions: “After a poor night’s sleep, I know I won’t be able to function the next day.”

“When I feel irritable, depressed, or anxious during the day, it is because I haven’t slept well the night before.”

Underlying Belief: “Insomnia is necessarily detrimental to daytime functioning.”

Cognitive Errors: Misattributions, magnification, overgeneralization.

Interventions/Alternative Interpretations:
1) Do you always experience daytime impairments after a poor night’s sleep?
2) Are these daytime sequelae always experienced with the same intensity?
3) Is it possible that other factors might also be causing these problems?

Vignette 4: Consequences of Insomnia

Dysfunctional Cognitions: “I am concerned that chronic insomnia may have serious consequences for my physical health.”

Underlying Belief: “Insomnia is necessarily detrimental to health.”

Cognitive Errors: Faulty evidence, overgeneralization, catastrophizing

Interventions/Alternative Interpretations:
1) There is no evidence that anyone has ever died from lack of sleep alone.
2) Excessive worrying about insomnia may be more detrimental to health than sleep loss itself.
Vignette 5: Sleep Requirements Expectations

Dysfunctional Cognitions:  "I must get 8 hours of sleep every night."

Underlying Belief:  "It is essential to sleep 8 hours to feel refreshed and function well during the day."

Cognitive Errors:  Unrealistic expectations, absolute thinking

Interventions/Alternative Interpretations:
1) Sleep need vary widely among individuals, and there is no "gold standard" that everyone should aim for.

2) Avoid placing undue pressure on yourself to achieve such a standard, as it may increase your anxiety and perpetuate insomnia.

3) Remember also, that too much sleep may be a waste of time; some very productive people are short sleepers.

Vignette 6: Unrealistic Expectations of self

Dysfunctional Cognitions:  "Because my spouse (significant other) falls asleep in minutes, I should be able to do the same."

Underlying Belief:  "Everyone must sleep alike."

Cognitive Errors:  Overgeneralization; absolute thinking.

Interventions/Alternative Interpretations:
1) Do all people you know have the same height and weight?

2) Beyond some normative range, there is wide variability among individuals in terms of how fast they fall asleep, how often they wake up, and the overall quality and duration of sleep.

3) It is best to avoid social comparisons, as there will always be someone who is wealthier, is taller, and sleeps better than you.
Vignette 7: Diminished Control over Sleep and Performance Anxiety

Dysfunctional Cognition: “I am afraid of losing control over my sleep abilities.”
“T have lost control of my sleep.”

Underlying Belief: “It is essential to be in full control of all aspects of one’s life.”

Cognitive Errors: Catastrophizing; irrational belief

Interventions/Alternative Interpretations:
1) What is the worst thing that could happen if you never got to sleep tonight? It is not catastrophic to go without sleep.

2) The harder you try to control sleep, the less control you will achieve; it is much easier to force wakefulness than to fall asleep at will.

3) Sleep will come more easily if you do not try so hard to control it.

Vignette 8: Unpredictability of Sleep and Learned Helplessness

Dysfunctional Cognitions: “I usually can’t predict whether I’ll have a good or poor night’s sleep.”
“I must rely on a sleeping aid to make my sleep more predictable.”

Underlying Belief: “No matter what I do, it has no effect on sleep. Insomnia is mostly a result of external factors that I have little control over. I am a victim.”

Cognitive Errors: Overgeneralization, faulty evidence, circular reasoning

Interventions/Alternative Interpretations:
1) When you use a sleeping pill and experience some temporary relief, it only reinforces your conviction that you have little control over sleep.

2) Nighttime sleep is not independent of daytime activities, thoughts, and feelings; therefore, you must carefully examine these relationships in order to make your sleep more predictable and develop more self-control.

3) Remember also that the most predictable consequences of sleeplessness is that it will eventually lead to sleepiness.
Vignette 9: Excessive Emphasis on Sleep and Control

Dysfunctional Cognitions:  
"I feel insomnia is destroying my entire life."
"I have little control in managing the negative consequences of disturbed sleep."

Underlying Belief:  
"I am helpless/hopeless; unless sleep improves, my life will remain miserable."

Cognitive Errors:  
Magnification; overgeneralization; catastrophizing.

Interventions/Alternative Interpretations:
1) Since sleep is supposed to occupy only a third of your life, aren't you giving it more importance than it deserves?

2) Is it possible the next time you have a bad night's sleep to ignore it and go about your daily routines just pretending you slept well?

Vignette 10: Misconceptions about Good Sleep Practices

Dysfunctional Cognitions:  
"When I don't get an adequate amount of sleep, I need to catch up by sleeping late the next morning or by napping the next day."

Underlying Belief:  
"It is essential to make up for all sleep loss."

Cognitive Errors:  
Faulty evidence; absolute thinking.

Intervention/Alternative Interpretations:
1) Sleeping too late in the morning or taking daytime naps is likely to delay sleep onset the next night.

2) Sleep deprivation experiments have shown that people only need to make up for about one-third of previous sleep loss.
Vignette 11: Other Misconceptions about Good Sleep Practices

Dysfunctional Cognitions: "When I have trouble sleeping, I should just stay in bed and try harder."

Underlying Beliefs: "If I get out of bed, I will wake up even more and surely won't be able to go back to sleep."

Cognitive Errors: Faulty assumption/evidence

Interventions/Alternative Interpretations:
1) The harder you try to induce sleep at will, the less likely you are to succeed. Have you ever noticed that you fall asleep unintentionally when you are not even trying (e.g., reading, watching TV)?

2) When you stay in bed awake for too long, it only strengthens the association between your sleep surrounding and tension/frustration.

Vignette 12: Further Misconceptions about Good Sleep Practices

Dysfunctional Cognitions: "If I just spend more time in bed, I will eventually get all the sleep/rest I need."

Underlying Belief: "Lying down in bed, even if I am awake, provides some very needed rest."


Interventions/Alternative Interpretations:
1) Have you ever paid close attention to the way you feel after spending 12 hours in bed? Do you really feel good and refreshed upon arising?

2) It is best to spend less time in bed and sleep more efficiently.
APPENDIX F

FREQUENCY AND PERCENTAGE TABLES OF CONTROL GROUPS, PEARSON CORRELATION TABLES & SLEEP PHYSIOLOGY BOOKLET

1. Frequency and Percentage Tables of Control groups that did not meet clinical significance for sleep latency, sleep efficiency, sleep duration, insomnia severity, depression and anxiety.

2. Pearson Correlation Tables for performance, sleep, and depression and anxiety measures.

3. Sleep Physiology Booklet
1. FREQUENCY AND PERCENTAGE TABLES OF CONTROL GROUPS

**Summary of Frequency and Percentages of Control groups that met Clinical Significance for Sleep Diary Latency, Sleep Diary Efficiency and Sleep Diary Duration Improvements**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Clinical Status</th>
<th>10 weeks</th>
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<tr>
<td></td>
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<td></td>
<td>3 months</td>
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<tr>
<td>Sleep Diary Latency</td>
<td>Normal range</td>
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<td>2 (5.7)</td>
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<tr>
<td></td>
<td>Clinical range</td>
<td>34 (97.1)</td>
<td>33 (94.3)</td>
</tr>
<tr>
<td></td>
<td>Normal range</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Sleep Diary Efficiency</td>
<td>Clinical range</td>
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</tr>
<tr>
<td></td>
<td>Normal range</td>
<td>2 (5.7)</td>
<td>2 (5.7)</td>
</tr>
<tr>
<td>Sleep Diary Duration</td>
<td>Clinical range</td>
<td>13 (94.3)</td>
<td>33 (94.3)</td>
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</table>

Note. Numbers in parentheses represent percentages of the Control groups.

**Summary of Frequency and Percentages of Control Groups Clinical Status regarding the Insomnia Severity Index (ISI)**

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<tr>
<td></td>
<td></td>
<td></td>
<td>10 weeks</td>
</tr>
<tr>
<td>Insomnia Severity Index</td>
<td>Severe</td>
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<td>35 (100)</td>
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Note. Numbers in parentheses represent percentages of the control groups.

**Summary of Frequency and Percentages of Control Groups Clinical Status regarding Depression (BDI-II) and Anxiety (BAI)**

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<tr>
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<th>BDI-II</th>
<th>BDI-II</th>
<th>BDI-II</th>
<th>BDI-II</th>
<th>BAI</th>
<th>BAI</th>
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<td>Mild</td>
<td>2 (5.7)</td>
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<td>1 (2.9)</td>
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<td></td>
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</tr>
<tr>
<td>Moderate</td>
<td>25 (71.4)</td>
<td>24 (68.6)</td>
<td>23 (65.7)</td>
<td>24 (68.6)</td>
<td>24 (68.6)</td>
<td>21 (60.0)</td>
<td>22 (62.9)</td>
<td>21 (60.0)</td>
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<tr>
<td>Severe</td>
<td>8 (22.9)</td>
<td>10 (28.6)</td>
<td>11 (31.4)</td>
<td>11 (31.4)</td>
<td>11 (31.4)</td>
<td>14 (40.0)</td>
<td>13 (37.1)</td>
<td>14 (40.0)</td>
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</table>

Note. Numbers in parentheses represent percentages of the control groups.
2. PEARSON CORRELATION TABLES

Correlations Between Measures of Performance, Sleep, and Depression and Anxiety at Baseline (n=70)

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<tr>
<th>Variable</th>
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<td>3. Manikin</td>
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<tr>
<td>5. Sleep Efficiency</td>
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<td>0.57**</td>
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<td>-</td>
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*p < .05. **p < .01.

Correlations Between Measures of Performance, Sleep, and Depression and Anxiety at 2 Weeks Follow-Up (n=70)

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<td>0.71**</td>
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Note: n = 70
* p < .05. ** p < .01.
### Correlations Between Measures of Performance, Sleep, and Depression and Anxiety at 3 Months Follow-Up (n=79)

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*p < .05. ** p < .01.

### Correlations Between Measures of Performance, Sleep, and Depression and Anxiety at 6 Months Follow-Up (n=79)

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<td>3. Manikin</td>
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<td>0.75**</td>
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<tr>
<td>6. Sleep Duration</td>
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<td>0.73**</td>
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<td>8. BDI-II</td>
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*p < .05. ** p < .01.
SLEEP PHYSIOLOGY

Treating Chronic Insomnia: A Cognitive-Behavioural Group Therapy Approach
Why Do We Sleep?

Restorative Theory of Sleep

Many studies have shown that one of the main reasons we sleep is for restorative purposes. The restorative nature of sleep happens in slow wave sleep, stages 3 and 4. The following is a summary of studies that supports the above theory of Restorative Sleep:

*Exercise & Sleep: Some studies have shown that when exercise has been performed there is: (1) a decrease in the time it takes to fall asleep (2) an increase in slow wave sleep (3) a suppression of REM sleep (4) an increase in total sleep time. Some studies of marathon runners have shown that Slow wave sleep increases dramatically after running a marathon.

*Pregnancy: Increased metabolism and growth was present during pregnancy which coincided with increased slow wave sleep. Other studies have shown that when subjects had their sleep disrupted or deprived, subsequent sleep had a greater proportion of slow wave sleep.
*Cell Division: Studies have shown that the highest peak of cell division (mitoses) occurs during sleep. This supports the theory that sleep is a period of growth and restoration.

*Growing at night: Some studies have shown that there is a massive increase in growth hormone release shortly after sleep onset. This also tends to coincide with slow wave sleep. Approximately 80% of the daily release of growth hormones are released in the first two slow wave sleep cycles at night. Other studies have shown that after two sleep studies, one of normal sleep and the other after depriving subjects of sleep for 40 hours, the second study found that there was a great increase in the nocturnal growth hormone on the second sleep.

*Energy Consumption during Sleep: After an intense-energy consuming day, the body tends to conserve energy during the night while asleep. Oxygen consumption is lowest during slow wave sleep, thereby serving a restorative function. Usually after a good night of sleep we awake refreshed and alert.
Stages of Sleep

**Stage 1**
- alpha waves turn into theta waves, slower
- it makes up about 5% of sleep
- transition from wakefulness to sleep
- can be awoken very easily

**Stage 2**
- very abundant, makes up 50% of our sleep
- its the real first stage of true sleep
- its marked by K-complexes and spindles

**Stage 3 & 4**
- known as slow wave sleep
- it occupies about 20% of our sleep
- these are recuperative stages of sleep
- if awoken in this stage, person will be in a state of confusion
- made up of delta waves

**Rapid Eye Movement (REM)**
- makes up about 20% of our nights
- it looks very close to wake or stage 1, but the muscle tone is much lower
- dreams are very abundant in this stage and are usually more emotional than when we dream in other stages
Normal Sleep Cycle

- 4 to 6 alternating cycles of NREM (Stages 1-4) and REM sleep, each cycle lasts in approximately 90 minute intervals.

- Subject usually falls asleep in NREM sleep

- Stages of sleep descend into deeper sleep, passing from Stage 1 to 2 to 3 to 4.

- From stages 1-4, the waves become slower and more abundant

- There is then a brief return to Stage 2, after which the first REM episode begins

- REM is usually interrupted with a body movement, and the Subject enters Stage 2 sleep again or is awoken and begins the entire cycle again

- In the beginning of the sleep, Slow wave sleep is abundant, but becomes less frequent as the night progresses

- Inversely, REM sleep is less in the beginning of the night, but progressively increases in the latter part of the night sleep

- The amount of SWS depends on how long the subject has been awake

- REM sleep on the other hand, is based more on a Circadian Cycle, where it seems to take place during the night when the Subject’s body temperature rises.
Figure 1 Changes in sleep stages with Aging.

- **Newborn**: 25% REM, 24% light, 51% deep, 1% wake.
- **Young Adult**: 27.1% REM, 19.8% light, 51.0% deep, 2.1% wake.
- **Middle Aged**: 26.6% REM, 11.7% light, 59.3% deep, 3.2% wake.
- **Elderly**: 19.8% REM, 5.5% light, 15.4% deep, 58.5% wake.

Legend:
- Deep-stages 3&4
- REM
- Light- stages 1&2
- Wake
Figure 2 Graph showing changes with age in total amounts of NREM and REM sleep. There is a sharp diminution of REM sleep in the early years. REM sleep falls from 8 hours at birth to less than 1 hour in old age. The amount of NREM sleep remains more constant, falling from 8 hours to 5 hours.
Circadian Rhythm
Your Biological Clock

Circadian Rhythms: Human are a rhythmic species, we experience cycles with a period of about a day, 24 hours. Chronobiology is the study of these circadian rhythm cycles in humans.

In determining a person's circadian rhythm, a particular variable must be measured repeatedly at different points of the day and from this you can see systematic changes from one time of the day to another, these changes are consistent over a 24 hour period. For example, when body temperature is taken throughout a 24 hour period, in a normal sleeper, body temperature usually goes down during the night and early morning when one is sleeping. This cycle usually repeats every day.
We have a biological clock inside of us and it's usually resistant to abrupt change. The purpose of our internal clock is to prepare the body and brain for sleep at certain times of the day and active wakefulness at others.

This clock lowers body temperature, heart rate and blood pressure, suppresses the excretion of hormones like melatonin, which helps the individual feel drowsy. The biological clock also protects our sleep by suppressing renal and bowel functions and hunger so we get a long and uninterrupted period of sleep.

The fact that the circadian rhythm is resistant to abrupt changes, creates problems for shiftworkers. Many of the internal functions of the circadian rhythm, like body temperature, cortisol and melatonin excretion and REM sleep, all have rhythms that influence are sleep. Some of these rhythms can take several days to adjust to any disruption or desynchronization that may result from shiftwork. Heart rate can adjust rather quickly, but other factors like body temperature or melatonin can take longer.
Figure 3 Plasma cortisol and Melatonin levels from the same group of nine healthy young subjects. From "Making Shiftwork Tolerable" by T. Monk and S. Foildard, 1992, pp. 12. Note that the Cortisol levels are lower in the night, just before going to bed. However, the levels increase throughout the night and morning, until awakening. Notice that the opposite is true of the Melatonin. Melatonin provokes sleep and increases around bedtime and the early morning, but decreases just before awakening.
Body Temperature and Alertness

Figure 4 Body temperature and alertness from a group of 15 subjects experiencing 72 hours of constant wakefulness. Note that as the temperature rises so does alertness. Temperature is usually higher during periods of awake and goes down during periods of sleep. (after Froberg, 1977).
Psychophysiological Insomnia

- a form of insomnia caused by tensions and learned associations that prevent sleep and result in a complaint of insomnia and decreased functioning during wakefulness

- **Mild Insomnia**: occurs almost nightly
  - patient receives an insufficient amount of sleep or doesn’t feel rested
  - this really doesn’t affect one’s social or occupational functioning
  - usually feel irritable, mild anxiety and daytime fatigue

- **Moderate Insomnia**: occurs nightly
  - patient receives insufficient sleep and doesn’t feel rested
  - there’s moderate impairment to social and occupational functioning
  - there’s feelings of fatigue, tiredness, irritability and mild anxiety

- **Severe Insomnia**: occurs nightly
  - patient receives insufficient sleep and doesn’t feel rested
  - there’s severe impairment to social and occupational functioning
  - feelings always associated with feelings of anxiety, daytime fatigue, tiredness, restlessness and irritability
Jet-lag Syndrome:
The jet-lag syndrome is a common disorder of the sleep/wake schedule. It results from airplane trips that may span over 4-5 times zones when going east or west bound. This tends to dissociate passengers endogenous rhythms (which includes cortisol, prolactin and potassium secretion) and external synchronizers or stimuli like sunrise, sunset and social activities (Nahon & Hedouin). Jet-lag leads to decreased alertness and insomnia. Research has shown that it takes anywhere from 1-6 days to resynchronize after a west-bound trip and 3-11 days after an east-bound trip (Nahon & Hedouin).

Shiftwork Sleep Disorder:
Night shift work and alternating schedules can also lead to disorders of the sleep/wake schedule. Night shift work affects approximately 20% of the active population in Canada and the U.S. About one million Canadians work shiftwork. This kind of work however, inevitably affects the sleep/wake rhythm in that even if one constantly works nights shift, conventional time-schedules will be resumed in order to avoid social isolation. Because the human body does not have a predisposition to sleeping during the day and because of environmental factors like noise and light, recuperative sleep for night shift workers is very poor. The result is what’s called “blue-collar jet lag”. This can lead to problems that can range from feeling bodily aches and pains, to lack of concentration, to even personality problems (Nahon & Hedouin). Three factors contribute to the diminished alertness and performance of night-shift workers:
(1) Misalignment of circadian phase
(2) Chronic and acute sleep deprivation
(3) Increased duration of waking hours before beginning night work.