Cognitive Behavioral Therapy for Insomnia in Breast Cancer Survivors: A
Randomized Controlled Crossover Study

A dissertation submitted in partial satisfaction of the requirements for the degree

Doctor of Philosophy

in

Clinical Psychology

by

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Chair

University of California, San Diego

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2008
DEDICATION

To my Family:

Mamma,
Papa’ & Paola,

Dary,

Marco

&

Eythan

With

LOVE
A human being is a part of a whole, called by us the “universe,” a part limited in time and space. He experiences himself, his thoughts and feelings, as something separated from the rest - a kind of optical delusion of his consciousness. This delusion is a kind of prison for us, restricting us to our personal desires and to affection for a few persons nearest us. Our task must be to free ourselves from this prison by widening our circles of compassion to embrace all living creatures and the whole of nature in its beauty.

Albert Einstein
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LIST OF ABBREVIATIONS

1. CBT-I: Cognitive behavioral therapy for insomnia
2. BzRA: Benzodiazepine receptor agonist
3. PMR: Progressive Muscle Relaxation
4. TST: Total sleep time
5. TIB: Total time in bed
6. WASO: Wake time after sleep onset
7. SOL: Sleep onset latency
8. SE: Sleep efficiency
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ACKNOWLEDGEMENTS

I would like to acknowledge my graduate advisor, Sonia Ancoli-Israel, PhD, for her support during graduate school and as chair of my dissertation committee. Her guidance in the development and implementation of this study, patience in editing the drafts and unyielding optimism in the recruitment phase have been instrumental. Above all, her support, respect and interest in my academic career, as well as her contribution to my intellectual and personal growth have been truly invaluable.

I would like to thank the many people who have helped make this study possible, including: John McQuaid, PhD, for his insightful clinical guidance as my therapy supervisor; Lianqi Liu, MD, for the timely scoring of all the actigraphic data; Sue Lawton, MA, for data management; Monique Cornejo, BA, for data entry; Barbara Parker, MD, for referring patients to the study; Matthew Marler, PhD, for his statistical guidance during the proposal of the study; Loki Natarajan, PhD, and Feng He, MS, for their statistical guidance and consultations during the study; and Lee Cohen, BA, for his priceless help in the bureaucratic administration of the grant.

I would also like to thank the wonderful women who have participated in the study. Their personal strength and dedication have been a great source of inspiration to me. These women have made my work worthwhile.

Finally, I would like to acknowledge the California Breast Cancer Research Program for funding this study (CBCRP 11GB-0049).

This manuscript is currently being prepared for publication.
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ABSTRACT OF THE DISSERTATION

Cognitive Behavioral Therapy for Insomnia in Breast Cancer Survivors: A Randomized Controlled Crossover Study

by

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Doctor of Philosophy in Clinical Psychology

University of San Diego California, 2008

San Diego State University, 2008

Professor Sonia Ancoli-Israel, Chair

Insomnia is characterized by complaints of difficulty initiating or maintaining sleep, or non-restorative sleep which last for at least one month and which cause clinically significant distress or impairment in functioning. Estimates of insomnia in women with breast cancer range from approximately 20% to 70%, with reports of poor sleep lasting for years after completion of the cancer treatment. Insomnia in breast cancer patients is often associated with depression, anxiety, fatigue, and low quality of life.

This study aimed at understanding whether an individual cognitive behavioral treatment for insomnia (CBT-I) would result in improvements in sleep as well as
improvements in fatigue, depression, anxiety and quality of life (QOL) in breast cancer survivors. Fourteen breast cancer survivors (age $M=61$, $SD=11.6$, range $=45-85$) were randomly assigned to either 6 weeks of CBT-I followed by 6 weeks of follow up, or 6 weeks of treatment as usual (TAU) followed by 6 weeks of CBT-I.

The hypotheses tested were that subjective and objective measures of sleep would improve during CBT-I compared to during TAU, that the QOL, fatigue, depression and anxiety would improve during CBT-I compared to during TAU, and that the effects of CBT-I on sleep and other symptoms would be maintained at 6 weeks. The results revealed that the participants assigned to receiving the CBT-I in the first six weeks had improved self-rated insomnia after treatment compared to the participants assigned to TAU (Insomnia severity index. Post-CBT-I: $M=12.20$, $SD=6.57$, range=2-19; Post-TAU: $M=20.71$, $SD=3.99$, range=16-26, $p=0.03$).

The pooled analyses of pre and post CBT-I treatment for all 14 participants revealed significant improvements in self-rated insomnia and sleep quality as well as improvements in objective measures of sleep. The analyses of the group that received CBT-I followed by 6 weeks of follow-up revealed that the sleep benefits gained during treatment were maintained at follow-up. In addition, QOL significantly improved at follow-up. No significant effects were found for psychological or fatigue variables.

The results are comparable to the sleep findings in previous studies that looked at group CBT-I therapies in breast cancer survivors, but differ in the effects found in psychological, fatigue and QOL measures. These results show that individual CBT-I is
efficacious in improving sleep in breast cancer survivors. Further studies with
greater sample size will help better understand the relationship between treating
insomnia and psychological, fatigue and QOL variables in breast cancer survivors.
INTRODUCTION

Poor sleep is a common complaint in cancer populations, yet this has been a neglected problem (Savard & Morin, 2001). As described by Vena, Parker, Cunningham, Clark, and McMillan (2004) and Clark, Cunningham, McMillan, Vena, and Parker (2004), poor sleep is common in all types of cancer and can often have serious consequences. Most of the studies on sleep disturbance in cancer, however, have been conducted in women with breast cancer. Estimates of insomnia and poor sleep quality in breast cancer patients range from 19% (Savard, Simard, Blanchet, Ivers, & Morin, 2001) to 73% (Carpenter et al., 2004).

As reviewed by Fiorentino and Ancoli-Israel (2006), poor sleep in women with breast cancer has been quantified both objectively and subjectively. Women with breast cancer may be prone to insomnia for various reasons, including a general increase in psychological distress after the cancer diagnosis and disruption of sleep due to increased frequency and severity of hot flashes caused by menopause, which often is induced by the breast cancer treatment (Kryger, 2004).

Most studies examining distress and psychological disorders in breast cancer patients have suggested that the most common diagnoses are insomnia, major depressive disorder, dysthymic disorder, adjustment disorder with depressive mood or mixed depressive and anxious mood, and anxiety disorders, including generalized anxiety disorder, post-traumatic stress syndrome, and adjustment disorder with anxious mood (Kissane, Grabsch, Love, Clarke, Bloch, & Smith, 2004; Miller, Jones,
While rates of each disorder vary from study to study, results of all studies agree that insomnia, depression, and anxiety are the most common. This symptom cluster is debilitating and decreases quality of life in women with breast cancer.

INSOMNIA

Insomnia is characterized by complaints of difficulty initiating or maintaining sleep, or non-restorative sleep which last for at least one month and which cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (APA, 2000). Insomnia is a heterogeneous disorder that has been classified by the International Classification of Sleep Disorders into subtypes, primary and comorbid insomnias, each with different characteristics (AASM, 2005). Primary insomnia includes any insomnia which has no other cause, such as psychophysiological insomnia (i.e., heightened arousal and learned sleep-preventing associations), physiological insomnia (i.e., the subjective reports of disturbed sleep are corroborated by objective polysomnography) and idiopathic insomnia (e.g., childhood onset chronic inability to obtain adequate sleep). Comorbid insomnia includes insomnia associated with mental disorders, medical disorders, medications (both prescription and over-the-counter), alcohol or drug dependency, environmental factors, sleep induced respiratory impairment, movement disorders, disorders of the sleep wake schedule, and /or parasomnias (e.g., somnambulism, REM sleep behavior disorder) (NIH, 2005).
Both primary and comorbid insomnia are characterized by reduced sleep duration and intensity. The underlying symptoms can be initial or sleep onset insomnia (i.e., difficulty falling asleep), middle or sleep maintenance insomnia (i.e., difficulty staying asleep), terminal or late insomnia (i.e., early morning awakening), and/or non-restorative sleep. Each of these can be transient, transient recurring, or chronic. In addition, many patients suffer from a combination of insomnia types.

Insomnia is a very common sleep disorder, with reports suggesting that 20% of the adult population and 30-50% of the elderly population complain of difficulty with sleep (Hublin & Partinen, 2002). Other estimates of insomnia prevalence in the general adult population have ranged from 9% to 17% (Ancoli-Israel & Roth, 1999; Ford & Kamerow, 1989; Ishigooka et al., 1999; Ohayon & Roth, 2001; Simon & VonKorff, 1997).

Chronic insomnia is defined by the duration of the insomnia episode ranging from 30 days or six months depending on the study (NIH, 2005). Chronic insomnia has a host of debilitating consequences that include tiredness, negative mood, the inability to enjoy family and social relationships, difficulty concentrating, memory problems, decreased quality of life, increased absenteeism, decreased job performance, increased severity of pain and poor health, and increased risk of falls (Roth & Ancoli-Israel, 1999). Chronic insomnia has also been associated with greater functional impairment, loss of productivity (Roth & Ancoli-Israel, 1999), excess health care utilization (Simon & VonKorff, 1997), and high psychiatric comorbidity, particularly depression and anxiety (Ford & Kamerow, 1989), as well as increased suicide risk.
(Agargun, Kara, & Solmaz, 1997; Fawcett et al., 1990). In addition, chronic insomnia is very often comorbid with medical disorders, including irritable bowel syndrome (Elsenbruch, Thompson, Hamish, Exton, & Orr, 2002), fibromyalgia (Jenum, Drewes, Andreasen, & Nielsen, 1993), asthma (Jenum et al., 1993) and gastroesophageal reflux disease (Raiha, Impivaara, Seppala, Knuts, & Sourander, 1993).

Not much is known on how to prevent insomnia; however, there is some evidence that leading an active life style and having a satisfying social life are correlates of healthy sleep, and might represent protective factors against insomnia (Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001). Factors that make people more vulnerable to insomnia, particularly complaints of initiating and maintaining sleep, include previous complaints of insomnia (odds ratio 3.5), female gender (odds ratio, 1.5), advancing age (odds ratio, 1.3), snoring (odds ratio, 1.3), and multiple concomitant health problems (odds ratios, 1.1 to 1.7) (Klink, Quan, Kaltenborn, & Lebowitz, 1992). Women with breast cancer have at least two of these main vulnerability factors for insomnia (i.e., being female and having cancer and its related health disturbances). In addition, many women with breast cancer are older, which may put them at even greater risk of developing insomnia. While most of the literature on aging and sleep shows that the prevalence of insomnia increases with age (e.g., Foley et al., 1995), a study by Davidson MacLean, Brundage, and Schulze (2002) showed that in cancer patients this relationship might be inverted with younger patients having more insomnia than older patients.
As mentioned above, insomnia is often comorbid with other psychological disorders. The 2005 NIH State-of-the-Science conference on insomnia emphasized the necessity to explore the relationship between characteristics of insomnia and other psychiatric and medical comorbid disorders (NIH, 2005). Additionally, the NIH panel encouraged randomized controlled trials that would validate treatment options in different populations suffering from insomnia. These types of studies are scarce in cancer patients.

INSOMNIA IN BREAST CANCER PATIENTS: OBJECTIVE MEASUREMENT STUDIES

There have been few objective studies of sleep in breast cancer patients and the results have been contradictory. Silberfarb and colleagues (Silberfarb, Hauri, Oxman, & Schnurr, 1993) compared polysomnographically recorded sleep of 32 cancer patients (15 with breast cancer) with age- and sex-matched healthy volunteers and 32 otherwise healthy insomnia patients and found that the breast cancer patients had sleep architecture similar to the normal sleepers. On the contrary, polysomnographic studies in women with breast cancer who had completed chemotherapy showed that they had lighter sleep (i.e., increased stages 1 and 2), less deep sleep (i.e., decreased stages 3 and 4), less REM sleep, and lower sleep efficiency compared to normative data (Fiorentino et al., 2005).

There is evidence of objective sleep being disrupted by hot flashes in women with breast cancer. Carpenter et al. (2004) found that 67% of survivors of breast cancer experienced nighttime hot flashes compared to only 37% of healthy women.
matched, among other variables, on age and menopausal status. Savard et al. (2004) reported that nights with hot flashes were associated with more percentage wake time, lower percentage stage two, and less efficient sleep compared to nights with no hot flashes. Therefore, hot flashes may be one of the causes of complaints of poor sleep in women with breast cancer. Hence, whenever possible, recording and measuring hot flashes in future studies investigating sleep in women with breast cancer is desirable.

INSOMNIA IN BREAST CANCER PATIENTS: SUBJECTIVE MEASUREMENT STUDIES

Studies that examined subjective sleep reports in cancer patients found significant complaints of difficulty sleeping (Ancoli-Israel, Kryger, Roth, & Dement, 2005) with the severity of the complaints being comparable to the insomnia complaints in other medical conditions (Fortner, Stepanski, Wang, Kasprowicz, & Durrence, 2002). In particular, women with breast cancer have severe complaints of poor sleep, insomnia, and fatigue (Savard et al., 2001; Silberfarb et al., 1993). Savard (2001) studied insomnia prevalence in 300 non-metastatic breast cancer women and found that 19% met diagnostic criteria for an insomnia syndrome with 95% of the cases being chronic insomnia. They also reported that the onset of insomnia followed the breast cancer diagnosis in only 33% of the cases and that 58% of the patients reported that cancer either caused or aggravated their sleep problems. Ancoli-Israel et al. (2006) found that women with breast cancer complained of poor sleep after they were diagnosed even before they began their chemotherapy. Carpenter et al. (2004) compared breast cancer survivors’ sleep and fatigue levels to age, race and
menopausal status matched healthy women and found that 73% of breast cancer survivors and 67% of healthy women had poor sleep quality and high sleep disturbance. Women with breast cancer also experienced shorter sleep duration and higher rates of nighttime flashes (67%) compared to healthy women (37%). Results were unlikely to be confounded by other psychological stressors given that the researchers found no group differences between women with breast cancer and healthy women in levels of fatigue and depression. However, other studies report that fatigue is one of the primary complaints in cancer patients (see Ancoli-Israel, Moore, & Jones, 2001 for a more complete review).

Koopman et al. (2002) looked at sleep disturbances in women with metastatic breast cancer. The results showed that 63% reported sleep disturbance, and that 37% reported using sleeping pills in the previous 30 days. This study also found interesting relationships between types of insomnia and correlates of breast cancer experience; all types of insomnia correlated with increased depression. In addition, problems falling asleep (i.e., initial insomnia) were associated with greater pain. Problems with awakenings during the night (i.e., middle insomnia) were related to less education, and finally, problems with early awakenings (i.e., terminal insomnia) were associated with less social support. Although insomnia in the context of metastatic cancer may be different than that of early stage cancer, these results still increase the overall understanding of sleep in breast cancer. The relationship between insomnia and other psychiatric disorders such as depression and anxiety in the breast cancer population still needs to be established. Some studies show a decrease in depression (Quesnel,
Savard, Simard, Ivers, & Morin, 2003; Savard, Simard, Ivers, & Morin, 2005a, 2005b) and anxiety (Savard et al., 2005a) in women with breast cancer, following successful treatment of insomnia with cognitive behavioral therapy for insomnia.

In summary, many women with breast cancer have complaints of poor sleep with prevalence estimates that range from approximately 20% to 70%. Most but not all breast cancer patients report having poor sleep prior to the breast cancer diagnosis, but others also report that the breast cancer exacerbated their sleep problems.

TREATMENT OF INSOMNIA

The severity and frequency of insomnia in the breast cancer population warrants a closer look at what efficacious treatments are available for insomnia and, in particular, which treatments are more adaptable to the specific needs of this population.

PHARMACOTHERAPY

Although pharmacotherapy is the most prescribed therapy for cancer patients with insomnia (Davidson, MacLean, Brundage, & Schulze, 2002; Derogatis, Feldstein, Morrow, & et al., 1979; Stiefel, Kornblith, & Holland, 1990), to our knowledge, there have been no studies examining the effect of pharmacotherapy on insomnia in patients with breast cancer. The medications most often used to treat insomnia in the general population are the benzodiazepines, benzodiazepine receptor agonists (BzRAs), anti-depressants, anti-histamines, and most recently, a melatonin receptor agonist. A meta-analysis of randomized controlled trials of benzodiazepine efficacy versus placebo for insomnia in otherwise healthy
individuals, showed that benzodiazepines were superior to placebo in shortening sleep latency (mean of 14.3 minutes, and 95% confidence intervals of 10.6 and 18.0) (Holbrook, Crowther, Lotter, Cheng, & King, 2000). Studies on BzRAs show that they are also efficacious in improving various aspects of sleep including self-reported sleep latency, total sleep time, and wake time after sleep onset (Walsh et al., 1998).

The NIH State-of-the-Science conference on insomnia (NIH, 2005), concluded that the BzRAs are efficacious in the short-term management of insomnia and that the frequency and severity of adverse effects associated with BzRAs are much lower than those seen with the older, longer acting benzodiazepines or with other sedating drugs. They also concluded that all antidepressants have potentially significant adverse effects raising concerns about the risk–benefit ratio, that barbiturates and antipsychotic drugs have significant risks and thus their use in the treatment of chronic insomnia cannot be recommended, and that there is no systematic evidence for efficacy of the antihistamines (H1 receptor antagonists) yet there is significant concern about risks associated with these drugs.

In summary, insomnia in the cancer population is usually treated with sedating drugs (Davidson et al., 2002; Derogatis et al., 1979; Stiefel et al., 1990). The literature on these drugs in general insomnia populations shows that benzodiazepines, BzRAs (zaleplon, zolpidem and zolpidem CR, eszopiclone) and the melatonin receptor agonist (ramelteon) are generally effective in reducing sleep latency, augmenting total sleep time and/or improving sleep continuity, and the BzRAs have been shown to be safe. However, there are drawbacks to the use of
some hypnotic drugs, including the non-curative nature of the treatment, the various side effects, as well as possible psychological and sometimes physiological dependence. As described below, in cancer patients, these disadvantages are often complicated by other factors.

BEHAVIORAL AND COGNITIVE BEHAVIORAL TREATMENTS

In the last seven years a growing literature on effectiveness of behavioral treatments for insomnia fostered a shift in what sleep medicine experts consider the gold-standard treatment for chronic insomnia. Cognitive behavioral therapy for insomnia (CBT-I) combines behavioral therapies with cognitive restructuring and educational sleep hygiene. Cognitive behavioral therapy’s efficacy in treating insomnia and improving sleep has been shown on both subjective and objective measures of sleep (Cervena et al., 2004). CBT-I is a safe and effective treatment for insomnia (Morin, 2004).

In 1999, Edinger and Wohlgemuth (1999) reviewed the literature on the management of persistent primary insomnia and reported the inadequacies of hypnotics in treating this disorder. They talked favorably about the promising behavioral and cognitive behavioral, patient specific approaches to insomnia treatment. More recently, randomized controlled trials have shown some clear advantages of cognitive behavioral techniques in comparison to pharmacotherapies. A randomized controlled trial on young and middle aged adults with chronic sleep onset insomnia found CBT-I more effective than pharmacotherapy in decreasing sleep latency and increasing sleep efficiency, and in promoting the largest number of normal
sleepers at the end of treatment (Jacobs, Pace-Schott, Stickgold, & Otto, 2004). This study found no advantages of combined CBT-I and pharmacotherapy treatments versus CBT-I alone.

Another randomized controlled study on older adults suffering from chronic, primary insomnia found that initially CBT-I and pharmacotherapy were similarly efficacious in bettering sleep and that there was a trend for the combined CBT-I and pharmacotherapy approach to decrease wake time after sleep onset to a greater degree than each treatment modality separately (Morin, Colecchi, Stone, Sood, & Brink, 1999). However, at one-year and two-year follow-up, only the CBT-I group still showed continued improvements in sleep. An earlier study by McClusky and colleagues (1991) comparing pharmacotherapy to behavioral therapy for sleep onset insomnia found that pharmacotherapy was efficacious immediately in reducing sleep latency, while behavioral therapies started having effects by the second week but then maintained their effects at follow-up to a larger degree than the pharmacological treatment. Finally, a meta-analysis by Smith and colleagues (2002) comparing pharmacotherapy and cognitive behavior therapies (e.g., sleep restriction, stimulus control, cognitive restructuring) for chronic insomnia showed that both treatment approaches ameliorate sleep, but that cognitive behavior therapies result in greater reduction of sleep latency compared to pharmacotherapy. The average number of CBT sessions for the studies included in this meta-analysis was 4.9 over an average period of time of 5.3 weeks. The average length of the pharmacotherapy was 2.0 weeks.
There are also randomized controlled trials of CBT-I versus placebo and comparisons of different modalities of CBT-I. A randomized controlled trial of CBT-I versus relaxation therapy and a placebo (i.e., quasi-desensitization treatment) in adults with sleep maintenance insomnia showed that CBT-I was more efficacious in improving measures of sleep fragmentation than either the relaxation training or the placebo (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). A randomized controlled trial of different modalities of CBT-I (i.e., individual therapy, group therapy, and telephone consultations) applied to the treatment of primary sleep onset and sleep maintenance insomnia showed that all modalities were efficacious in treating insomnia, that improvements in sleep were maintained at six months follow-up, and most interestingly that there were no significant differences between modalities (Bastien, Morin, Ouellet, Blais, & Bouchard, 2004). Cervena et al. (2004) found that CBT-I for insomnia was associated with improved sleep architecture, specifically an increased amount of stage 2, REM and slow wave, and reduced beta and sigma activity.

In summary, several studies have shown that behavior therapies and CBT-I in particular are as effective and longer lasting than pharamcotherapies for treating insomnia. The NIH State-of-the-Science conference on insomnia also concluded that CBT-I is as effective as prescription medications for brief treatment of chronic insomnia and that there are indications that CBT-I’s beneficial effects, in contrast to those produced by medications, may last well beyond termination of treatment. There is some evidence that pharmacotherapy is faster acting in acute stages of insomnia,
and that cognitive behavioral treatments have better long-term outcomes. The better long-term effects of cognitive behavioral therapy compared to pharmacotherapy may be due to the fact that with this therapy the individual has learned coping skills (e.g., behavioral and cognitive) to tackle acute insomnia as well as to prevent or mitigate the severity of future insomnia episodes.

Within the cancer and sleep literature, other promising behavioral approaches to aid sleep have been reported. Berger et al. (2002; 2003) studied the feasibility and the effects of an individualized sleep promotion plan composed of sleep hygiene, relaxation therapy, stimulus control and sleep restriction on women undergoing chemotherapy for breast cancer. The authors reported that the intervention was feasible with adherence rates improving over time. In addition, with the exception of nighttime awakenings which were higher than desired (>30 Min. per night), most of the sleep measures remained consistent with normal values. Cannici, Malcolm, and Peek (1983) reported a reduction in sleep onset latency in cancer patients with insomnia after a muscle relaxation training. These results were sustained at the three month follow-up.

Allison et al. (2004) used a psycho-educational intervention that emphasized skills building (i.e., problem solving techniques, relaxation, cognitive coping, goal setting, communication, social support and lifestyle factors) in head and neck cancer patients and found that the intervention was feasible and was related to increased self reported sleep quality. Simeit, Deck, & Conta-Marx (2004) compared the subjective sleep of cancer patients with insomnia after either progressive muscle relaxation or
autogenic training in addition to a standard rehabilitation program. Both interventions reduced sleep disturbance compared to a standard rehabilitation program, with no significant differences between the two interventions. Finally, Coleman et al. (2003) reported an increase in total nighttime sleep minutes after an individualized exercise program in patients treated with aggressive chemotherapy for multiple myeloma.

Interventions based on eastern medicine and holistic practices have also recently been studied as possible treatments for poor sleep in cancer patients, with promising results. Carlson, Speca, Patel, and Goodey (2003) reported that mindfulness-based stress reduction techniques may help improve sleep in breast and prostate cancer. Tibetan yoga practices have been reported to increase self-perceived quality of sleep in lymphoma patients (Cohen, Warneke, Fouladi, Rodriguez, & Chaoul-Reich, 2004). Gentle touch healing may also have a positive effect on sleep in cancer patients (Weze, Leathard, Grange, Tiplady, & Stevens, 2004).

INTEGRATING CBT-I IN BREAST CANCER PATIENTS

There are several published CBT-I manuals. Two of these manuals can be used in group or individual therapy settings (Morin, 1993; Morin & Epsie, 2003) and the third is geared specifically for individual settings (Perlis, Jungquist, Smith, & Posner, 2005). Briefly, the components of these CBT-I treatment manuals are cognitive (e.g., maladaptive thoughts restructuring), behavioral (e.g., sleep restriction, stimulus control), and educational (e.g., sleep stages, sleep hygiene). Both manuals spell out the agenda components for each session.
In research settings however, there is flexibility in terms of what components to apply and emphasize depending on the type of population being treated. With breast cancer patients, Quesnel and colleagues (2003) and Savard et al. (2005a) used additional strategies to encourage coping with fatigue by encouraging physical activity, and by educating participants on fatigue and the differentiation between fatigue and somnolence.

The theoretical framework on which the manuals are based is Spielman’s three factor (3-P) model of insomnia (Spielman, Caruso, & Glovinsky, 1987). This model postulates three factors which are necessary for the development of chronic insomnia: predisposing factors that make the person prone or vulnerable towards insomnia, precipitating factors that trigger the insomnia, and perpetuating factors that maintain the insomnia. The 3-P model postulates that predisposing, precipitating, and perpetuating factors may have different weights or importance at different times in the course of the insomnia. The cognitive behavioral technique for combating insomnia is to target the perpetuating factors, such as maladaptive thoughts about sleep (e.g., I have to sleep for at least eight hours or I will not function during the day), and/or maladaptive behaviors (e.g., staying in bed when one cannot fall asleep). Behavioral perpetuating factors are often maladaptive coping attempts to counteract the effects of the insomnia (e.g., napping for sleepiness).

The predisposing factor most often hypothesized for insomnia is arousability or hyperarousal. Arousability is a relatively stable tendency to be easily aroused (Coren, 1988) and can be expressed through physiological, cognitive and/or emotional
hyperarousal (Morin, 1993). According to Morin (1993), arousability is a central mediating feature of insomnia. He suggests that people with insomnia are generally in a state of greater arousal both during the day and during the night compared to people without insomnia.

The precipitating factors of insomnia have been reported in the literature as events having mostly, but not necessarily, negative valance, and being related to experiences with family, health, or work-school events (Bastien, Vallieres, & Morin, 2004).

An example of an application of Spielman’s model to an insomnia patient with breast cancer can be seen in Appendix 1. It is reasonable to assume that in many women with breast cancer the precipitating event is the diagnosis of breast cancer, which is a life threatening and life disrupting trauma. In the present study one of the participants recalled refusing to sleep when she first got diagnosed with breast cancer because “I thought I was going to die, and felt I had more important things to do than sleep.” The same participant reported that, as time went on and she didn’t die, she had the desire to get back to a regular sleep/wake cycle, but that at this point she had “forgotten how to sleep” (see Appendix 2 for additional patients’ comments).

Kryger (2004) reported that women with breast cancer are prone to insomnia and sleep disturbances for various reasons. As mentioned above, the treatment for breast cancer may cause menopause, thus increasing the frequency and severity of hot flashes which are known to disrupt sleep. Furthermore, being diagnosed with breast cancer and having to face treatment may create or increase symptoms of anxiety
and/or depression that also disrupt sleep. It is important to note that at times the
insomnia is a pre-breast cancer condition, and the cancer diagnosis most likely
aggravates the severity of the insomnia. Once the insomnia is triggered, many factors
including comorbid conditions such as depression and anxiety may work as
perpetuating factors. In addition, medical and psychiatric illnesses, such as cancer or
depression, may play a role in predisposing, precipitating and perpetuating the
insomnia.

Recognizing the predisposing and precipitating factors of insomnia is
important for the clinical formulation of the case (e.g., theoretical background) and to
help the patients understand the multi-factorial nature of their symptoms and hence
‘make sense’ of their insomnia. However, the primary focus of the CBT-I
intervention is the factors that can be changed (i.e., the perpetuating factors).
Therefore, the CBT-I protocol delivery will not change based on the etiology of the
insomnia or whether a breast cancer patient developed insomnia before the breast
cancer diagnosis or as a result of the breast cancer.

STUDIES USING CBT-I IN BREAST CANCER PATIENTS

The efficacy of a multimodal CBT intervention for insomnia in 10 breast
cancer survivors was tested with an A-B multiple baseline non-randomized design
study looking at the effects of the treatment on insomnia, depression, anxiety, fatigue,
and quality of life (Quesnel et al., 2003). Eight weekly group sessions of
approximately 90 minutes and one optional booster session one month after the end of
treatment were administered. The therapy was based on the clinical procedures
explained by Morin (1993) with a focus on specific needs of the breast cancer population, such as managing fatigue. Therapy was led by a single therapist and began with education about the conceptual model of insomnia and continued with sleep restriction techniques (where the amount of time in bed prescribed is set at the amount of actual sleep the person is getting based on the sleep diary entries), stimulus control (where the aim is to associate the bed with sleep), and cognitive restructuring of the dysfunctional thoughts and attitudes regarding sleep. Sleep hygiene principles and relapse prevention strategies were also taught to the participants. Objective sleep data were collected with overnight polysomnograms (PSG) and subjective sleep measures were collected with sleep diaries and questionnaires.

The participants that completed the treatment showed improvements in both subjective and objective sleep data, particularly in sleep efficiency, (SE = total sleep time/time in bed X 100, and total wake time). The objective sleep data continued to show improvements on SE at follow-up of 6-months with all participants being above the 85% cutoff for insomnia. Four out of 8 participants showed improvements in SE (above the 85% cutoff for insomnia) on sleep diary data at the end of treatment and 5 out of 7 that completed the 6-month follow-up were above 71%. Six out of the 8 patients that completed the study significantly improved over pre-treatment baseline scoring below the cut off of 8 on the insomnia questionnaire (both patient and clinician rated). These gains were maintained at follow up. In addition, the CBT-I intervention was significantly associated with amelioration of depressive symptoms, general and physical fatigue, global quality of life, and cognitive aspects of quality of
life. Although the small sample size and the non-randomized design reduce the
generalizability and the attribution of causality of the results of the study, the
treatment offered was clearly associated with various sleep and psychological benefits
(Quesnel et al., 2003).

Another study by the same group of researchers looked at the effects of CBT-I on sleep, psychological and immunological effects in breast cancer survivors (Savard et al., 2005a, 2005b). Fifty-seven women were randomly assigned to either group CBT-I or waitlist control. The therapy consisted of eight weekly group sessions with 5 or 6 participants and followed the same protocol described above (Quesnel et al., 2003). Objective and subjective sleep measures were recorded, as well as hypnotic medication use, psychological distress, quality of life, and immune measures that included enumeration of blood cell counts and cytokine production. Results showed clear improvements in the subjective sleep data. Significant group-time interactions (two groups= treatment or waitlist control, five times= pre-treatment, post-treatment, and three, six and twelve month follow ups) with a priori contrast revealed improvements in all variables except total sleep time. The analysis of subjective sleep data pooled from all participants showed significant improvements after treatment in all sleep variables including sleep onset latency, wake time and sleep efficiency. The improvements were maintained for all variables at the three, six and twelve month follow up, and continued to improve further for total sleep time and the insomnia questionnaire filled out by a significant other at the 12 month follow up.
More ambivalent results came from the objective PSG data, which showed no significant group by time interactions for any variable, but showed significant positive changes from pre-treatment to post-treatment for all variables except total sleep time. The changes were maintained at the six month follow-up. A significant decrease in hypnotic use was also revealed by the participants after being treated and improvements in psychological variables (i.e., anxiety, depression, global quality of life, fatigue) were maintained at the follow-up assessments (Savard et al., 2005a). The analysis of immunological measures revealed that at post-treatment participants had higher cytokine production (i.e., interleukin-1-beta, interferon gamma) and lower increases of lymphocytes, which then increased at follow-up, as did the white blood cell counts. The mediation analysis revealed that some of these changes were partially mediated by improvement in psychological and insomnia related sleep variables. These results are preliminary, but promising and provide some support to the hypothesis that there is a causal relationship between insomnia and immunological functioning, and that treating insomnia might benefit the immune system (Savard et al., 2005b).

However, both these studies have been conducted by researchers from the same lab hence, according to the criteria set forth by Chambless & Ollendick (2001), replications of the findings from independent investigators are needed before considering CBT-I an empirically validated treatment for insomnia in breast cancer survivors.
Other cognitive behavioral interventions have been implemented to treat different mental illnesses and psychological distress, to alleviate or cope with pain, and to improve quality of life in women with breast cancer. The efficacy of these interventions varies from study to study (Fiorentino & Ancoli-Israel, in preparation). Studies have used a variety of different modalities (e.g., group, individual, phone-therapy) with varying degrees of emphasis on either the cognitive or the behavioral techniques. Some studies have also looked at the effects of cognitive behavioral interventions on physiological variables and immune functioning. Although more research is needed, in general, cognitive behavioral interventions in breast cancer patients seem both feasible and efficacious practices for breast cancer patients’ symptom reduction.

THE PRESENT STUDY

This study aimed at understanding the relationship between insomnia, fatigue, depression and anxiety in breast cancer by examining the effects of CBT-I on this symptom cluster and on quality of life in survivors of breast cancer.

HYPOTHESES AND AIMS

Aim 1. To examine the effect of CBT-I on the sleep of women with breast cancer.

Hypothesis 1: Subjective (as measured by the Insomnia Severity Index and the Pittsburg Sleep Quality Index) and objective (as measured by actigraphy) measures of sleep would improve during CBT-I compared to during the treatment-as-usual period.
Aim 2. To examine the effect of CBT-I on the quality of life, fatigue, depression and anxiety.

Hypothesis 2. Quality of life (as measured by the Medical Outcomes Study Short Form Health Survey, and the Functional Outcomes of Sleep Quality), and fatigue (as measured by the Multidimensional Fatigue Symptom Inventory), depression and anxiety (as measured by the Center of Epidemiological Studies Depression scale, and the Brief Symptom inventory) would all improve as sleep improved during CBT-I compared to during the treatment-as-usual period.

Aim 3. To examine whether the effects of CBT-I on sleep and other symptoms were maintained 6 weeks post-treatment.

Hypothesis 3. The effects of CBT-I on sleep and other symptoms would be maintained at 6 weeks.
METHOD

RECRUITMENT

Forty-four women were referred to the study from various sources including word of mouth, oncologists working in the San Diego community and the Rebecca and John Moores UCSD Cancer Center. Brochures and fliers promoting the study were distributed in wig and coffee shops in the San Diego community. An advertisement promoting the study appeared in the Union Tribune, and information on the study was posted on the internet on Craig’s List and the Cancer Navigator websites. As shown in Figure 1, of the 44 women referred, 23 women did not participate (for various reasons, see Figure 1), and 21 were consented. Of these 21, 5 were ineligible either because they did not have insomnia, they had other current psychological disorders, or they had active cancer. Two women dropped during the first week of the study (one reported that the study “was too much”, and another realized in therapy that the root of her insomnia would better be addressed with a more comprehensive psychotherapy addressing psychological events and traumas experienced in childhood). In the end, 14 cancer survivors who had finished their cancer treatment were recruited and all 14 completed the study protocol.

PARTICIPANTS’ DEMOGRAPHIC CHARACTERISTICS

The mean age of the 14 participants was 61 years (SD=11.6; range = 45-85 years). Twelve participants self-identified their race as Caucasian, one as Asian, and one reported more than one race. One participant self-identified her ethnicity as Hispanic and 13 as not-Hispanic. At study enrollment one participant was single, 10
were married, 2 were divorced, and one widowed. One participant’s husband died during the study. All participants completed high school, two completed some college (Associate Degree), and 11 participants completed college (Bachelor Degree) or higher education (Master’s Degree or Ph.D.). Five participants reported a professional occupation, three reported being a homemaker, one reported other occupation, and five participants were retired. The average yearly income reported was between $50,000 and $100,000 with 2 participants reporting less than $15,000 and 4 participants reporting greater than $100,000. One participant refused to answer the income question.

PARTICIPANTS’ BREAST CANCER DIAGNOSIS INFORMATION

All participants reported a history of breast cancer and had completed the treatment for breast cancer (some participants were continuing hormonal therapy during the study). The mean number of years since the completion of treatment was six years (range of 5 months prior to enrolling in the study to 24 years).

Four participants reported having been diagnosed with an invasive lobular carcinoma, three participants reported an invasive ductal carcinoma, two participants reported a mixed ductal and lobular carcinoma, three reported other pathology (of which one specified having had inflammatory breast cancer), and two could not remember their diagnosis and could not find their medical reports. Seven participants reported stage 1 cancer, three reported stage 2 cancer, two reported stage 3 cancer, and two reported advanced stage 3 cancer.
PARTICIPANTS’ BREAST CANCER TREATMENT INFORMATION

All participants except one underwent surgery. Eight participants had a lumpectomy, 3 had a mastectomy, and 2 had a double mastectomy. Seven participants received chemotherapy, 11 received radiation, and 8 received hormonal therapy. Most participants received a combination of chemotherapy, radiation therapy and hormonal therapy: 3 participants received chemotherapy, radiation therapy and hormonal therapy, 3 participants received chemotherapy and radiation therapy, 3 participants received radiation therapy and hormonal therapy, and 1 participant received chemotherapy and hormonal therapy.

STUDY PROTOCOL

During an initial brief telephone screening and study information phone call, information was gathered on the completion of the cancer treatment and nature, frequency, and duration of sleep complaints. The questions asked were:

a. Have you finished your cancer treatment?

b. Can you describe your sleep problem/s?

c. How many times per week would you say you have this/ese problem/s?

d. How long have you had this (these) problem(s)?

Those who finished cancer treatment and had insomnia symptoms occurring at least 3 times per week for at least a month duration were invited to meet with the study coordinator for formal IRB consent to participate in the study, a more thorough screening of other sleep disorders, and a semi-structured clinical inventory for DSM-IV-TR for axis I psychiatric screening (Spitzer, Williams, Gibbon, & First, 1992).
The inclusion criteria for the study included meeting the DSM IV criteria for insomnia (i.e., difficulty falling or staying asleep 3 or more times per week for at least one month, with daytime consequences/complaints), and being breast cancer survivors who had finished their breast cancer treatment (e.g., radiation therapy, chemotherapy, surgery). Women who were still on hormonal therapy were allowed in the study. The exclusion criteria included having other current medical or psychiatric conditions that would interfere with the research.

All participants were asked not to start any new insomnia treatments during the study. If participants were already having their insomnia treated, they were allowed to continue with their regular treatment (e.g., hypnotic drugs, herbs, etc.) but were encouraged to stop or decrease dosages during the treatment phase of the study. Medication intake was monitored throughout the study.

Screening for inclusion and exclusion criteria was conducted at the first face-to-face visit via interview and questionnaires. The Sleep Disorders Symptom Checklist (Perlis, Jungquist, Smith & Posner, 2005) was used to rule out sleep disorders other than insomnia. While not a validated measure, the SDSC is used in clinical settings. Patients rate their symptoms as happening never, seldom, sometimes often or frequently in response to questions aimed at ruling out sleep disorders, including sleep apnea, narcolepsy, delayed sleep phase disorder, advanced sleep phase disorder, and nightmares. If symptoms for a specific condition are endorsed, further investigation of the history of the patient in regards to the condition is warranted.
During this meeting, the SCID-I for DSM-IV-TR (Spitzer, Williams, Gibbon, & First, 1992) was administered by a trained therapist (LF) as a screening for psychological disorders. The SCID is a semi-structured diagnostic interview designed to assist clinicians, researchers, and trainees in making reliable DSM-IV psychiatric diagnoses. The SCID-I is a reliable and validated interview. Several studies have used the SCID as the "gold standard" in determining the accuracy of clinical diagnoses (e.g., Shear et al., 2000; Steiner, Tebes, Sledge, & Walker, 1995). If participants were eligible for the study they were randomly assigned through a computer generated random list to either 6 weeks of CBT-I followed by 6 weeks of follow-up (condition called CBT-I) or 6 weeks of treatment as usual followed by 6 weeks of CBT-I (condition called TAU).

CBT-I TREATMENT

The treatment consisted of six sessions of individual CBT-I. The sessions were approximately one hour each and were conducted in therapy rooms either at the Moores UCSD Cancer Center in La Jolla, or at the SDSU/UCSD Joint Doctoral Program Psychology Clinic in San Diego, depending on the patients’ preferences. All sessions were conducted by a graduate level therapist trained in CBT-I, and supervised by a licensed clinical psychologist who specializes in cognitive behavioral interventions. The therapy sessions were audiotaped for supervision purposes.

The treatment was based on the manual by Perlis and colleagues (Perlis, Jungquist, Smith & Posner, 2005), and combined educational information on sleep, and behavioral and cognitive strategies to improve sleep. The educational component
was introduced during the first session and consisted of information on sleep stages, processes regulating sleep (i.e., circadian and homeostatic), sleep throughout life, sleep in cancer patients, Spielman et al.’s (1987) insomnia 3-P model and cognitive behavioral therapy basics (i.e. the interconnectedness of thoughts, feelings and behaviors).

The behavioral component was introduced during the second session and entailed sleep restriction, stimulus control, adhering to the sleep hygiene rules (see Appendix 3), and training in progressive muscle relaxation techniques (Bernstein, Borkovec, & Hazlett-Stevens, 2000). In order to implement the sleep restriction the participants kept a baseline sleep diary for the first week of therapy. During the second session of CBT-I they were prescribed to stay in bed the amount of hours that they had on average slept during the first week of therapy. For this study no one was ever sleep restricted below 4 ½ hours for safety reasons. As the patients’ sleep diary sleep efficiency [SE=(total sleep time/total time in bed) x 100] increased to close to 90% the patients were prescribed 15 to 30 minutes increases in their time spent in bed by going to bed 15 to 30 minutes earlier.

The stimulus control consisted of advising the patient to only use the bed for sleep, and getting out of the bed if awake for more than 15 min or if they felt frustrated or alert. The progressive muscle relaxation was administered during the sixth session with the purpose of teaching the patients a technique to relax in bed as they were waiting to fall asleep. The patients were encouraged to recognize the tension in isolated muscles by purposefully tensing and activating the muscles and then relaxing
them and focusing on the self-infused relaxed state and the difference between the sensations of tension and relaxation.

The cognitive component was introduced during the fourth session of the therapy and entailed challenging the dysfunctional beliefs about sleep and any other belief or thought that kept the patient awake at night, and restructuring the maladaptive thoughts into more realistic, functional, and less anxiety provoking thoughts. Examples of maladaptive thoughts encountered were: “If I don’t sleep 8 hours per night my cancer will come back”, “I deal with lack of sleep worse than anyone I know”, “Sleep is like dying and I want to live”, “I won’t be able to have fun playing bridge tomorrow if I don’t get a good night sleep”, “I won’t be able to function well at work and will loose my job if I don’t sleep”, “I need to sleep when my husband sleeps”, and “I should always be happy because I got a second chance in life, and I should not feel badly about looking older.”

CBT-I also entails homework assignments including sleep diaries (see Appendix 4), scheduling worry time, and use of thought restructuring techniques. The worry time is a 15-30 minute time scheduled during the day, preferably at the same time each day, during which the patient was advised to allow herself to worry about anything that was concerning her. Once the worry time was over, anytime that the patient found herself worrying she could then say to herself “I have my worry time scheduled I don’t need to worry now.” This technique was adopted to allow a time-limited opportunity for patients to express their concerns and consequently reduce the likelihood of bringing their problems to bed.
The 3Cs thought restructuring technique (Catch, Check, and Change the thought) was taught to the patients. This technique consists of recognizing maladaptive and anxiety provoking thoughts, checking them against reality, and changing them to more realistic and adaptive thoughts that lower anxiety in the patient. Patients were also encouraged to recognize thinking errors (e.g., all or none thoughts, overgeneralizing, catastrophizing) as they came up in therapy. Patients were encouraged to reformulate the cognitions containing thinking errors into more realistic thoughts that improved patients overall mood and lessened anxiety.

The overview of each session is summarized in Appendix 5

DESIGN

This study was a cross-over design with participants being randomly assigned to one of two conditions CBT-I or TAU. In the first condition the participants received 6 weekly CBT-I sessions and were followed for six additional weeks. In the second condition, participants were first followed for six weeks in a treatment-as-usual protocol and then received the 6 weekly sessions of CBT-I (see Figure 2). The study was approved by the UCSD and the SDSU Committees on Human Research.

MEASURES

OBJECTIVE MEASURE OF SLEEP

Sleep/wake activity was recorded with actigraphy. Actigraphs are small devices worn on the wrist which measure wrist activity. The Actillume II actigraph (Ambulatory Monitoring, Inc) was used in this study. It is approximately 1" diameter x 0.35" height, weighing under ½ oz. It has non-volatile 32K memory, 16 Hz. sample
rate and two modes of operation (zero crossing or low and high sensitivity proportional integrating measure). Data were averaged and stored in 1 minute fixed epoch lengths to yield up to 22 days of recording time per initialization.

Our laboratory has extensive experience with these devices and has collected similar data in middle-aged adults (Kripke et al., 1997), in older schizophrenic patients (Martin et al., 2001), in community dwelling elderly (Ancoli-Israel et al., 1991), in demented nursing home patients, (Ancoli-Israel, Clopton, Klauber, Fell, & Mason, 1997; Ancoli-Israel, Klauber et al., 1997) and in breast cancer patients (Ancoli-Israel, Cohen-Zion, Gehrman, Jones, & Johnson, 2002; Cohen-Zion et al., 2002; Jones, Cohen-Zion, Johnson, & Ancoli-Israel, 2002; Liu et al., 2003; Moore et al., 2001). The output from the actigraph supplies information about day and night sleep and wake variables, and circadian rhythm variables. This study focused on the night variables. Only results for total sleep time (TST), Wake After Sleep Onset (WASO), number of awakenings per night, and percent sleep (percent of time asleep at night out of the time in bed) are reported.

SUBJECTIVE MEASURES OF SLEEP

Subjective sleep assessments were obtained by self-report using the Insomnia Severity Index (ISI) (Bastien, Vallieres, & Morin, 2001), and the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989).

ISI: The ISI is a 7-item insomnia severity questionnaire that assesses 7 aspects of insomnia: difficulty falling asleep, nighttime awakenings, early morning awakenings, impairment of daytime functioning, noticeability of impairments, distress
and worry about sleep, and current dissatisfaction with sleep. Each item is rated on a Likert scale ranging from 0 (not at all) to 4 (extremely). The ISI total score is obtained by adding the scores on the 7 items (range 0-28). The ISI internal consistency is high (i.e., 0.78). Any score below 8 is considered not clinically significant insomnia.

PSQI: The Pittsburgh Sleep Quality Index (PSQI) (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Buysse et al., 1989) measures reported sleep patterns and sleep problems, including sleep quality, sleep latency, sleep efficiency and napping behavior. The PSQI is a 19-item questionnaire that has been demonstrated to have high internal consistency (0.83), test-retest reliability (0.85) and diagnostic validity. A global sleep quality score derived from the PSQI can be used to index overall quality of sleep over the prior one-week period. Global sleep quality scores are continuous (range 0-21) with high scores reflecting poor sleep quality. A score below 5 is considered good sleep.

Sleep Diaries (see Appendix 4): A daily sleep diary was completed by each participant during the first 5 weeks of the CBT-I. The variables extracted from the sleep diaries were time in bed (TIB), TST, WASO, sleep onset latency (SOL, the time it takes to fall asleep), sleep efficiency (SE= (TST/TIB) x 100), and number of wakes. During each session (an exception being the first session during which the sleep diaries were introduced as homework assignments) each variable was calculated for each day and averaged for the week.
FATIGUE

Fatigue assessments were obtained by self-report using the short form of the Multidimensional Fatigue Symptom Inventory (MFSI-SF) (Stein, Martin, Hann, & Jacobsen, 1998). The original MFSI was an 83-item self-report questionnaire designed to assess the principle manifestations of fatigue in cancer patients. It has been reported as a valid and reliable tool to assess the full spectrum of fatigue symptoms for both clinical and research applications. The symptoms are classified into 5 rationally derived subscales: global, somatic, affective, cognitive, and behavioral. Based on factor analysis, a 30-item short form (MFSI-SF) was empirically derived from the 83 items of MFSI, which were collapsed into five subscales: General, Physical, Emotional, Mental, and Vigor. Each subscale includes 6 items and each item is rated on a 5-point scale indicating how true the statement was during the last week (0=not at all, 4=extremely). The sum of General, Physical, Emotional, and Mental subscale scores minus the Vigor subscale score generates a total score. Range of possible score for each subscale is 0 to 24, and the range for total score is -24 to 96, with higher score indicating more severe fatigue, except for the Vigor subscale, where a higher score indicates less fatigue. A score greater than 8 is symptomatic of clinically significant fatigue. Only total score results are presented.

QUALITY OF LIFE

Quality of life assessments were obtained by self-report using the Medical Outcomes Study Short Form 36 item Health Survey (SF-36) (Ware & Sherbourne,
1992) and the Functional Outcomes of Sleep Questionnaire (FOSQ) (Weaver et al., 1997).

SF-36: The SF-36 is the most widely used short form of the Medical Outcomes Study (MOS) Functioning and Well-being Profile (Stewart & Ware, 1992), and measures aspects of health relevant to functional status and well-being that are not age, disease, or treatment specific (Stewart & Ware, 1992). It has been used for assessment and evaluation of various populations (Ganz, Day, Ware, Redmond, & Fisher, 1995; Stewart & Ware, 1992; Wyatt et al., 1998), including women with breast cancer (Ganz et al., 1995; Goodwin, Black, Bordeleau, & Ganz, 2003). Responses are summarized into four mental (mental health index, vitality, role limitations due to emotional problems, social functioning) and four physical (physical functioning, general health perceptions, bodily pain, role limitations due to physical health problems) subscales. These dimensions can be consolidated into separate mental and physical health related quality of life summary scales (Ware, Snow, Kosinski, & Gandek, 2000). Subscale scores range from 0 to 100, with higher scores indicating better health. Only general health perception score results are presented.

FOSQ: The Functional Outcomes of Sleep Questionnaire (FOSQ) (Weaver et al., 1997), was used to measure daytime functional consequences of sleep problems. The questionnaire is designed to measure functional status in situations that produce sleepiness. The measure has content validity based on 100% agreement by a panel of experts composed of individuals with expertise in sleep disorders, geriatrics and instrument design. There are six subscales, including vigilance, intimacy and sexual
relationships, general productivity, activity level, and social outcome and a total score. Test-retest reliability for the scale was .91 when administered a week apart. The range of scores of possible scores is 0-20, with lower scores considered poorer functioning. Only total score results are presented.

DEPRESSION

Subjective depression level assessments were obtained by self-report using the Center of Epidemiological Studies-Depression (CES-D) scale (Beeber, Shea, & McCorkle, 1998). The CES-D is a 20-item scale of depressive symptoms. Answers to the items on the scale are based on the degree to which symptoms were experienced during the last week. This scale has been shown to have high reliability and validity in the assessment of depressive symptoms (Radloff, 1977), yet it is brief and easy to administer which makes it appropriate for use in this population of patients in the context of the current study. Since the CES-D reflects cognitive and affective symptoms rather than somatic symptoms of depression, it is highly recommended for use with patients with medical problems. Beeber et al.(Beeber et al., 1998) used the CES-D to assess symptoms of depression in a group of newly diagnosed cancer patients. Their data supported the use of the CES-D as a valid and reliable tool for assessing depressive symptoms in this population. Scores of 16 or higher are symptomatic of significant depression.

MOOD AND ANXIETY

Subjective psychological and somatic symptoms assessments were obtained by self-report using the Brief Symptom Inventory (BSI-18) (Zabora et al., 2001). The
BSI-18 has six questions for each type of symptoms assessed: Depression, anxiety, and somatic symptoms. The BSI is written at a sixth-grade reading level. Each item is rated on a 5-point Likert scale from 0 (not at all) to 4 (always). The patient is asked to respond to each item in terms of "how they have been feeling during the past 7 days". Each participant receives a score on the Global Severity Index, as well as the 3-symptom specific subscales. The BSI-18 has been tested in 1,543 cancer patients and has an internal consistency reliability of 0.89 (Cronbach alpha). Scores of 63 or higher are clinically significant. Only global score results are presented.

MENOPAUSAL SYMPTOMS

Subjective menopausal symptoms assessments were obtained by self-report using the Greene Climacteric Scale (GCS) (Greene, 1998). The GCS is a self-rated questionnaire that provides a comprehensive measure of wide-ranging symptoms experienced by menopausal women. It consists of 21 items with 5 separate subscales which generate one total score, and sub-scores of psychological (anxiety, depression), physical (e.g. muscle and joint pains, headaches), and vasomotor symptoms (hot flashes, night sweats). Its predictive and construct validity have been established in several studies (Pearce et al., 1997; Wu et al., 2001). Higher scores reflect a severe menopause problem. The range of possible scores is 0-63, with normative means differing by menopausal status: premenopausal $M=10.56$, perimenopausal $M=15.84$, postmenopausal $M=15.48$, and post-hysterectomy=15.28. Most of the participants in this study were either post-menopausal or post hysterectomy. Only total score results are presented.
ASSESSMENT OVERVIEW

Demographic and cancer diagnosis information were collected one time at baseline. The assessments for medication intake, insomnia (ISI), quality of life (SF-36, FOSQ), depression (CESD), anxiety and mood (BSI), and menopausal symptoms (Greene), were collected at baseline, at six weeks and at twelve weeks. In addition, weekly sleep quality (PSQI) assessments were collected for the 13 weeks (baseline + 12 weeks), and daily sleep diary data were collected during the treatment phase for 5 weeks. Actillumes were worn for 72 consecutive hours at each phase (baseline, six weeks and twelve weeks) of the study.

DATA ANALYSIS

All questionnaires and sleep diary data were entered and double checked by research assistants and the study PI. The actigraphy data were scored by a trained scorer blind to the study condition (CBT-I or TAU) of the participants. All analyses were performed using the statistical software for the social sciences SPSS version 15 (SPSS, 2006), except the repeated measures analysis on the PSQI data which were done using the statistical package R (R Development Team, 2005).

Differences between groups at study entry in the major outcome variables and in the demographic and cancer diagnosis and treatment variables were tested using non-parametric independent sample tests (Mann-Whitney U). The differences between the CBT-I group and the TAU group after the first six weeks of the study were analyzed with non-parametric independent sample tests (Mann-Whitney U) on
change scores (calculated by subtracting the post-six weeks scores from the baseline scores).

To examine differences before and after treatment, data from the entire sample (N=14) were analyzed using non-parametric Wilcoxon Signed Rank Tests for paired analyses. The non-parametric Wilcoxon Signed Rank Test was also used to examine differences between Pre-CBT-I, Post-CBT-I and Follow-Up in the CBT-I group.

A repeated measures analysis of variance was used to examine differences in the 13 assessments of the PSQI between the groups. Two models were used for this analysis: One used the PSQI data from weeks 0-6 and the other the PSQI data from weeks 7-12. Each model had the repeated PSQI values as outcome, group as the between subject factor, and time as the within subject factor, and groupXtime (interaction) as effects.
RESULTS

The results of the non-parametric independent sample tests (Mann-Whitney $U$) showed no statistically significant baseline differences between the two groups on demographic variables (age, income, education) or cancer treatment variables (type of treatment, stage).

SUBJECTIVE SLEEP VARIABLES (see Table 1)

ISI

BASELINE GROUP DIFFERENCES

Although both groups scored in the range suggesting clinically relevant insomnia at Phase 0, participants in the CBT-I group reported significantly less insomnia compared to participants in the TAU group (CBT-I: $M=16.80$, $SD=3.83$, range= 13-22; TAU: $M=21.57$, $SD=2.70$, range= 18-26, $U=8.50$, $p=0.04$).

CBT-I COMPARED TO TAU

Participants reported significantly less insomnia Post-CBT-I compared to Post TAU (CBT-I, $M=12.20$, $SD=6.57$, range=2-19; TAU $M=20.71$, $SD=3.99$, range=16-26, $U=7.50$; $p=0.03$) (see Figure 3).

PRE-POST CBT-I DIFFERENCES

Participants reported significantly less insomnia post-CBT-I compared to pre-CBT-I (Pre-CBT-I: $M=18.36$, $SD=4.40$, range=12-26; Post-CBT-I: $M=10.93$, $SD=7.28$, range=2-26, $z=-3.194$, $p=0.001$).
PHASE DIFFERENCES IN CBT-I GROUP

Participants in the CBT-I group reported significantly less insomnia post-CBT-I compared to Pre-CBT-I (Pre-CBT-I: $M=16.80$, $SD=3.83$, range=13-22; Post-CBT-I: $M=12.20$, $SD=6.57$, range=2-19, $z=-2.21$, $p=0.03$). In addition, participants reported significantly less insomnia at the 6-week follow-up compared to immediately after the 6 sessions of CBT-I (Follow-Up: $M=10.20$, $SD=6.22$, range=0-15, $z=-2.23$, $p=0.03$) and significantly less insomnia at the 6-week follow-up compared to at Pre-CBT-I ($z=-2.20$, $p=0.03$).

PSQI

BASELINE GROUP DIFFERENCES

Although both groups scored in the range suggesting clinically relevant sleep disruption at Phase 0, participants in the CBT-I group reported significantly better sleep quality compared to participants in TAU group (CBT-I: $M=11.00$, $SD=1.41$, range=9-13; TAU: $M=14.43$, $SD=2.63$, range=11-19, $U=4.00$, $p=0.008$).

CBT-I COMPARED TO TAU

The analyses of differences between groups revealed no statistically significant differences in sleep quality between the groups. However, the direction of the findings suggest better sleep quality Post-CBT-I compared to Post-TAU (see Table 1).

PRE-POST CBT-I DIFFERENCES

Participants reported significantly better sleep quality Post-CBT-I compared to Pre-CBT-I (Pre-CBT-I: $M=12.14$, $SD=3.21$, range=9-19; Post-CBT-I: $M=7.57$, $SD=4.22$, range=3-16, $z=-3.125$, $p=0.002$).
PHASE DIFFERENCES IN CBT-I GROUP

Participants in the CBT-I group reported significantly better sleep quality at Follow-Up compared to Pre-CBT-I (Pre-CBT-I: $M=11.00$, $SD=1.41$, range=9-13; Follow-Up: $M=7.00$, $SD=4.53$, range=1-12, $z=-1.99$, $p=0.046$). The difference between the sleep quality at Pre-CBT-I and Post-CBT-I, and the difference between Post-CBT-I and Follow-Up were not statistically significant.

REPEATED MEASURES ANALYSIS OF VARIANCES

The mixed models repeated measures analysis of variance of the PSQI data revealed that, over time, the two groups had different slopes. The CBT-I group’s sleep quality improved from baseline through the first six assessments while the TAU group’s sleep quality stayed consistent during this same time period. In the first model analysis of the first 7 assessments (Time 0 to Time 6) there was a significant effect of the within subject factor Time ($t_{(12,81)}=-3.91$, $p=0.0002$), suggesting that the sleep quality within the two groups changed over time. There was also a borderline significant Group by Time interaction ($t_{(12,81)}=1.95$, $p=0.054$) suggesting that ratings of sleep quality within the groups changed over time at different rates with the slope for the CBT-I group being -0.68 (sleep quality improving) and the slope for the TAU group being -0.23 (little or no change in sleep quality) (see figure 4).

Inversely, during the second six assessments, the CBT-I group’s sleep quality stayed the same (i.e., maintained the gains from the treatment phase) while the TAU
group’s sleep quality improved. In the second model analysis of the last 6 assessments (Week 7 to Week 12) there was a significant effect for the between subject factor group \((t_{(12,68)}=3.80, p=0.002)\) meaning that the groups differed in mean PSQI scores, and a significant group by time interaction \((t_{(12, 68)}=-5.36, p<0.0001)\) meaning that the groups changed over time in different directions. The slope for the CBT-I group was 0.09 (improvement in sleep quality was maintained) and the slope of the TAU group was -1.21 (showing that sleep quality improved over time) (see Figure 4).

SLEEP MEDICATION COUNT

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in sleep medication count.

CBT-I COMPARED TO TAU

There were no statistically significant differences in sleep medication count found between groups. However the direction of the findings suggests lower sleep medication use after CBT-I compared to after TAU (see Table 1).

PRE-POST CBT-I DIFFERENCES

Participants reported a significant decrease in sleep medication use Post-CBT-I compared to Pre-CBT-I (Pre-CBT-I: \(M=1.00, \text{Median}=1, SD=1.46, \text{range}=0-5\); Post-CBT-I: \(M=0.07, \text{Median}=0, SD=0.27, \text{range}=0-1, z=-2.264, p=0.02)\).
PHASE DIFFERENCES IN CBT-I GROUP

There were no statistically significant differences for sleep medication count between the different phases.

SLEEP DIARIES (see Table 2)

TIME IN BED (TIB)

DIFFERENCES BETWEEN WEEKS 1-5

Participants spent significantly less time in bed during the night during week 5 compared to week 1 (TIB in minutes. Week 1: $M=474, SD=67$, range=372-610; Week 5: $M=416, SD=52$, range=333-506, $z=-2.60$, $p=0.009$).

SLEEP EFFICIENCY (SE)

DIFFERENCES BETWEEN WEEKS 1-5

Participants had a significantly higher SE at week 5 compared to week 1 (SE percentage. Week 1: $M=73, SD=15$, range=35-92; Week 5: $M=83, SD=11$, range=57-99, $z=-2.54$, $p=0.01$) (see figure 5).

WASO

DIFFERENCES BETWEEN WEEKS 1-5

Participants had significantly lower WASO during week 5 compared to week 1 (WASO in minutes. Week 1: $M=53, SD=43$, range=3-164; Week 5: $M=25, SD=26$, range=0-96, $z=-3.04$, $p=0.002$) (see figure 6).

NUMBER OF WAKES

DIFFERENCES BETWEEN WEEKS 1-5
Participant woke up significantly less during week 5 compared to week 1
(Week 1: \( M=3, SD=1, \) range=1-6; Week 5: \( M=2, SD=2, \) range=0-7, \( z=-2.42, p=0.016 \)).

TST, SOL, AND SLEEP AIDS COUNT

There were no statistically significant findings for TST, and SOL. The analysis for Sleep Aids Count revealed a tendency towards statistical significance with less
Sleep Aids used at week 5 compared to Week 1 (Week 1: \( M=1.3, SD=2.4, \) range=0-7;
Week 5: \( M=0.50, SD=1.3, \) range=0-5, \( z=-1.83, p=0.068 \)).

OBJECTIVE SLEEP VARIABLES (see Table 3)

ACTIGRAPHIC VARIABLES

TST

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in TST

CBT-I COMPARED TO TAU

There were no statistically significant differences between the groups in the
TST change scores from Phase 0 to Phase 1.

PRE-POST CBT-I DIFFERENCES

Participants slept significantly less at Post-CBT-I compared to Pre-CBT-I
(TST in hours. Pre-CBT-I: \( M=7.26, SD=0.76, \) range=6.2-8.5; Post-CBT-I: \( M=6.64, SD=1.41, \) range=4.77-9.68, \( z=-2.133, p=0.033 \)).

PHASE DIFFERENCES IN CBT-I GROUP

There were no significant differences in TST between Phase 0, Phase 1, and
Phase 2 in the CBT-I group.
WASO

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in WASO.

CBT-I COMPARED TO TAU

There were no statistically significant differences between the groups in the WASO change scores from Phase 0 to Phase 1.

PRE-POST CBT-I DIFFERENCES

Participants had significantly less WASO Post-CBT-I compared to Pre-CBT-I (WASO in hours. Pre-CBT-I: $M=1.63, SD=0.66$, range=0.69-2.54; Post-CBT-I: $M=1.04, SD=0.31$, range=0.56-1.61, $z=-2.621, p=0.009$) (see Table 7).

PHASE DIFFERENCES IN CBT-I GROUP

Participants in the CBT-I group had significantly less WASO at Follow-Up compared to Pre-CBT-I (Pre-CBT-I: $M=1.41, SD=0.73$, range=0.69-2.23; Follow-Up: $M=1.03, SD=0.52$, range=0.4-1.7, $z=-2.023, p=0.043$). The difference between Pre-CBT-I and Post-CBT-I, and Post-CBT-I and Follow-Up were not significant.

NUMBER OF WAKES

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in Number of Wakes.

CBT-I COMPARED TO TAU

There were no statistically significant differences between the groups in the Number of Wakes change scores from Phase 0 to Phase 1.
PRE-POST CBT-I DIFFERENCES

Participants woke up significantly less at Post-CBT-I compared to Pre-CBT-I (Pre-CBT-I: $M=26.18$, $SD=7.80$, range=13.67-36.33; Post-CBT-I: $M=23.41$, $SD=6.35$, range=14.67-33.00, $z=-1.992$, $p=0.046$).

PHASE DIFFERENCES IN CBT-I GROUP

Participants in the CBT-I group woke up significantly less at Follow-Up compared to Pre-CBT-I (Pre-CBT-I: $M=23$, $SD=8.88$, range=14-36; Follow-Up: $M=20$, $SD=8.44$, range=9-31 $z=-2.023$, $p=0.043$). The differences between Pre-CBT-I and Post-CBT-I, and Post-CBT-I and Follow-Up in Number of Wakes were not significant.

SLEEP PERCENT

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in Sleep Percent.

CBT-I COMPARED TO TAU

There were no statistically significant differences between the groups in Sleep Percent change scores from Phase 0 to Phase 1.

PRE-POST CBT-I DIFFERENCES

Participants slept a greater percentage of their time in bed at Post-CBT-I compared to Pre-CBT-I (Pre-CBT-I: $M=0.82$, $SD=0.06$, range=0.74-0.92; Post-CBT-I: $M=0.86$, $SD=0.03$, range=0.79-0.92, $z=-2.268$, $p=0.023$).
PHASE DIFFERENCES IN CBT-I GROUP

There were no differences between Phase 0, Phase 1 and Phase 2 in Sleep Percent in the CBT-I group.

QUALITY OF LIFE (Table 4)

MOS-SF 36 (General Health Subscale)

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in Quality of Life.

CBT-I COMPARED TO TAU

There were no statistically significant differences between the groups in Quality of Life change scores from Phase 0 to Phase 1.

PRE-POST CBT-I DIFFERENCES

There were no statistical significant differences found in general health before and after CBT-I.

PHASE DIFFERENCES IN CBT-I GROUP

Participants in the CBT-I group reported significantly worse health Post-CBT-I compared to Pre-CBT-I (Pre-CBT-I: $M=66.00$, $SD=8.94$, range=55-75; Post-CBT-I: $M=59.00$, $SD=14.31$, range=40-75, $z=-2.04$, $p=0.04$). Participants reported significantly better health at Follow-Up compared to Post-CBT-I (Follow-Up: $M=71.00$, $SD=14.32$, range=55-90, $z=-2.23$, $p=0.03$). The difference in general health between Pre-CBT-I and the Follow-Up was not significant, although the means are in the correct direction (i.e., higher at Follow-Up, see Table 4).
FOSQ

BASELINE GROUP DIFFERENCES

There were no differences between the groups at Phase 0 in daytime functioning.

CBT-I COMPARED TO TAU

There were no statistically significant differences between the groups in daytime functioning change scores from Phase 0 to Phase 1.

PRE-POST CBT-I DIFFERENCES

There were no statistically significant findings in daytime functioning before and after CBT-I.

PHASE DIFFERENCES IN CBT-I GROUP

Participants in the CBT-I group reported significantly better daytime functioning at Follow-Up compared to Post-CBT-I (Post-CBT-I: $M=16.50$, $SD=2.19$, range=13-19; Follow-Up: $M=17.45$, $SD=1.85$, range=15-20, $z=-2.02$, $p=0.043$). The difference between daytime functioning Pre-CBT-I and Post-CBT-I, and the difference between Pre-CBT-I and Follow-Up were not statistically significant.

OTHER VARIABLES

The analysis of the Depression (CESD), Mood and Anxiety Symptoms (BSI), Fatigue (MFSI), and Menopausal (Greene) variables revealed no statistically significant findings. However, the descriptive statistics show that all of the results are in the direction of less distressful symptoms after CBT-I compared to after TAU, and that, for the CBT-I group, the benefits are either maintained (depression), improved
(menopausal symptoms), or slightly decreased (fatigue and mood and anxiety) at follow up (see Tables 5-8).

QUALITATIVE DATA

In addition to the quantitative data reported above, the participants spontaneously commented on the efficacy of the CBT-I treatment. The comments are summarized in Appendix 2.
DISCUSSION

The results of this study suggest that survivors of breast cancer with insomnia benefit from CBT-I treatment. The CBT-I group reported significantly less insomnia (ISI) after CBT-I compared to the TAU group after TAU. The pooled analyses with all 14 participants confirmed that insomnia was significantly decreased after treatment compared to before treatment. While the subjective sleep quality (PSQI) change scores were not significantly different between the CBT-I group and the TAU group, the pooled analysis revealed better sleep quality after treatment compared to before treatment in the whole sample. The findings from the self-reported questionnaires were corroborated by the pooled objective actigraphic data, which showed better sleep quality (higher sleep percent) and decreased sleep disruption (decreased WASO and Number of Wakes) after treatment.

In addition, these findings were confirmed by the sleep diary data during treatment which, similar to the actigraphic data, showed better sleep quality (higher SE) and decreased sleep disruption (decreased WASO and Number of Wakes) at week 5 compared to week 1 of treatment. Furthermore, consistent with the participants’ sleeping better after treatment, participants also significantly decreased the use of sleep medication after treatment compared to before treatment. This finding was evident in both the self reported questionnaire assessments at Pre-CBT-I and Post-CBT-I, and in the sleep diaries during treatment.

The first hypothesis of this study, that the subjective and objective measures of sleep would improve during CBT-I compared to during the treatment-as-usual period
was therefore confirmed for the subjective insomnia (ISI), but rejected for the subjective and objective sleep quality (PSQI and actigraphy).

Results of this study also showed that the effects of the CBT-I on sleep and other symptoms were maintained at six weeks post-treatment follow-up. The sleep variable results suggest that the treatment benefits described above were in all cases maintained and sometimes increased over the six weeks of follow up. Specifically, self reported insomnia levels continued to improve, self reported sleep quality continued to improve, objective sleep quality maintained, objective WASO maintained, and objective number of wakes continued to improve. The decrease in sleep medication use was maintained at follow up as well. Therefore, the third hypothesis that the effects of CBT-I on sleep would be maintained at 6 weeks was confirmed for both subjective and objective sleep.

These results are consistent with the research findings from the CBT-I studies in breast cancer conducted by the Morin group (Quesnel et al., 2003; Savard et al., 2005a), that showed significant improvements after treatment in subjective insomnia (ISI), sleep diary variables, medication use, and polysomnographic variables (Savard et al., 2005a) and improvements in insomnia (ISI), and sleep efficiency and total wake time (from both sleep diaries and polysomnography) (Quesnel et al., 2003). The significant decrease in actigraphic TST after CBT-I compared to before CBT-I revealed from the pooled actigraphy analysis of the present study do not contradict the findings from the Morin group studies who report no significant improvements in polysomnographic TST after CBT-I treatment compared to before treatment (Savard
et al., 2005a; Quesnel et al., 2003). The fact that participants slept less after treatment compared to before treatment may seem paradoxical for an insomnia treatment study, however this finding is not uncommon and is usually the result of the prescribing sleep restriction and stimulus control, which decrease the opportunity for sleep, with the intent of improving sleep consolidation (decreasing SOL, WASO and number of wakes, and increasing sleep efficiency). In fact, WASO and the number of wakes did decrease supporting the notion that the patients’ sleep was consolidated. On a side note, participants in the present study spontaneously reported that as the quality of their sleep improved they felt as though they needed fewer hours of sleep to wake up feeling refreshed. Hence the decrease in TST post CBT-I found in this study may in some cases reflect a spontaneous morning awakening resulting from the homeostatic pressure to wake up after a well consolidated and restful sleep.

Another goal of this study was to examine the effect of CBT-I on quality of life, fatigue, depression and anxiety. Contrary to the effects found by Morin and colleagues (Quesnel et al., 2003; Savard et al., 2005a), which showed significant improvements in mood, fatigue, and quality of life, the present study failed to reveal significant improvements in depression, mood or fatigue variables, although the results were in the direction of less distress after treatment. While the pooled analysis before and after treatment for quality of life were not significant, there was a decrease in quality of life (MOS-SF 36) immediately after treatment in the CBT-I group, followed by a significant improvement in quality of life (MOS-SF 36 and FOSQ) at the six week follow-up, suggesting that the benefits of better sleep took time before
showing up in the participants’ overall quality of life. Hence, the second hypothesis of the study that quality of life, fatigue, depression and psychological symptoms would all improve during CBT-I compared to during the treatment as usual period was rejected.

The differences in the psychological variables outcomes noted between the present study and the Morin and colleagues studies (Quesnel et al., 2003; Savard et al., 2005a) are puzzling, particularly in light of the similar sleep improvement findings. There are several differences between CBT-I implemented in this study and the Morin and colleagues studies (Quesnel et al., 2003; Savard et al., 2005a) that may account for the different findings. First, the present study delivered an individual treatment as opposed to a group treatment. It is possible that the group environment with its inherent peer support, group feedback, and social setting facilitated the faster quality of life improvements and fostered the greater change in mood, depression, and fatigue found in the other studies. Second, this study’s treatment was delivered in 6 sessions of approximately one hour each (with the exception of the last session which included the PMR and lasted approximately 1 ½ hours), while the Morin and colleagues studies were delivered in 8 sessions lasting 90 minutes each. The difference in length and number of sessions might also have accounted for the differences in psychological and fatigue variables outcomes between the studies.

Furthermore, as opposed to the Morin and colleagues studies (Quesnel et al., 2003; Savard et al., 2005a) and, to our knowledge, to all the CBT-I studies implemented in the general population, this study’s cognitive behavioral sessions
tackled not only the maladaptive thoughts related to sleep, but also the maladaptive thoughts not regarding sleep that were keeping the participant awake, and preventing them from relaxing at night. Many of the thoughts dealt with in therapy were related to worries regarding cancer recurrence, physical effects of the cancer treatment, family, work, body images, and existential dilemmas. While the participants clearly described benefits related to this approach, spontaneously reporting in therapy lowered anxiety and appreciation for the realizations regarding the effects of their thought patterns on their anxiety and sleep, it is possible that focusing on these matters created an awareness of problems that could not be appropriately dealt with in the 6 session of CBT-I offered. Notably, the participants whose cognitive behavioral sessions mostly focused on the non-sleep thoughts were also the ones that asked about the possibility and expressed the desire to continue working with the therapist on non-insomnia issues at the end of the protocol.

Another factor that might have contributed to the non-significant effects of the CBT-I treatment on the psychological variables is that while the improvement in insomnia levels before and after treatment was statistically significant, the means at post treatment were not below the clinical cut-off of for insomnia. Not having the insomnia completely resolved might have contributed to the non significant improvements found in the psychological and fatigue measures in the present study. The ISI means at post treatment in the Morin group studies were either closer to the cut-off (Savard et al., 2005a) or below the cut-off (Quesnel et al., 2003) which may have contributed to the participants overall feeling better.
A floor effect could also explain the non significant findings in the psychological variables in the present study. Given that the mean of the present study’s participant scores at baseline was below the cut-offs for the psychological measures (CESD and BSI), there might simply have not been any chance to detect or any need for improvement in those symptoms. This was different in one of the Morin studies (Savard et al., 2005a) for example in which anxiety levels at baseline were above the clinical cut off of 7 on the Hospital Anxiety and Depression Scale-Anxiety. This explanation however does not apply to the fatigue findings, because the participants in the study scored within the levels suggesting fatigue.

The small sample size of 14 participants and consequential lowered power to detect and effect of the treatment, is another important possible explanation for the lack of some findings in this study. Low power is also most likely responsible for the non significant between group change scores differences between the CBT-I group and the TAU group. As mentioned above the only between group analysis that produced a significant effect was for the insomnia measure (ISI), where the strong effect size of the treatment was well captured by the main insomnia measure.

The general aim of study was to understanding the relationship between insomnia, fatigue, depression and anxiety in breast cancer by examining the effects of CBT-I on the symptom cluster typically observed in breast cancer patients. Because of the small sample size and low power, this aim was only partially achieved. Many of the non-significant findings could be due to the low power instead of a real absence of a relationship. Therefore, reliable conclusions about the effects of improving insomnia
in breast cancer survivors on the symptom cluster of psychological distress, and fatigue are not possible from this study. Nevertheless, this study has several merits and is an important contribution to the scientific literature on non-pharmacological treatments for sleep disturbance in breast cancer patients. First, it is the first study outside of the Morin lab and has in part replicated that research group’s findings on the efficacy of CBT-I in breast cancer survivors. Replication of findings from independent investigators is a necessary benchmark toward the validation of a treatment (Chambless & Ollendick, 2001). Second, it is the first study in breast cancer that has used individual instead of group therapy settings, and hence informs the literature on the feasibility, benefits and possible drawbacks of this therapeutic form for this population. Third, it is the only study in breast cancer that has used 6 sessions instead of 8 sessions of treatment with results showing statistically significant improvement in sleep quality and sleep disruption after only 5 sessions (sleep diary data) and noticeable improvements in sleep quality and sleep disruption after the second session (see figures 4-6). This finding will be helpful in those clinical settings where there may not be time for longer treatments.

LIMITATIONS AND FUTURE DIRECTIONS

Along with the study’s strengths there are also some limitations. The small sample size is a strong limitation of the study, not only for the low power to detect effects, previously discussed, but also for limitations in reliability and generalizability of the findings. Another limitation of the present study was that it was not possible to examine the relationship between the subjective and objective data, as these data were
collected at different time points. However, correlations between subjective and objective sleep have historically not been very high, and they are not diagnostically necessary to assess good or poor sleep.

Other inadequacies of the study design included missing sleep diary baseline and post PMR assessments. A pre-CBT-I, post-CBT-I difference analysis would have possibly yielded significant effects for variables such as sleep onset latency (SOL) and sleep aid use which were in the expected direction (with shorter SOL and less sleep aid use at week 5 compared to week 1) but not statistically significant (although sleep aids was close to significance). Furthermore, this study has limited external generalizability to culturally and racially diverse populations. With a few exceptions, most of the sample was non-Hispanic and Caucasian, hence limiting the generalizability of the findings to these ethnic and racial backgrounds. On the other hand, the present study sample encompassed a wide range of age, income, years of education and practiced occupations, which increased the generalizability of the findings.

Future studies with greater sample sizes and greater power are necessary to understand the relationships between insomnia, CBT-I treatment and the psychological, cognitive, life style and physical symptoms that are known as the symptom cluster in breast cancer survivors. In particular, studies exploring the effect of CBT-I on the cognitive functioning on women with breast cancer would be particularly illuminating. The effects of the condition known as chemobrain, which affects many women with breast cancer who have undergone chemotherapy with
varying degrees of memory and cognitive problems, may be lessened by a successful treatment of sleep disturbance and insomnia (also known to affect cognitive abilities). In the present study two of the patients that were complaining of chemobrain-like symptoms (particularly memory problems and confusion) spontaneously reported that their memory improved after the CBT-I treatment.

The small sample size of the present study did not allow for exploration of the relationships between different types of breast cancer diagnoses, different cancer treatments (e.g., chemotherapy, radiation, surgeries) and CBT-I outcomes. Future studies with larger sample size should investigate these relationships. Knowing the degree of effectiveness of CBT-I with specific types of breast cancer patients would be an important step in tailoring the treatment for sleep disturbance offered to these patients.

Other research is also needed to develop the optimal CBT-I protocol to treat women with breast cancer. The findings from this study regarding the positive effects found after fewer sessions of CBT-I compared to earlier studies will inform future research about the number of necessary sessions to produce improvements in sleep, and the sufficient sessions to produce good sleepers (scoring below the clinically significant cut-offs as well as reporting sleep satisfaction) and reduce the distress of the breast cancer symptom cluster. Future studies should also explore the effects of re-arranging the components of the therapy according to the type of insomnia and the specific needs of the breast cancer survivors. In the present study, during the CBT-I treatment it became evident that different participants responded positively to different
components of the therapy; participants who experienced higher anxiety and worries seemed to benefit mostly from the last 3 sessions (cognitive component and PMR) VS. participants’ whose main problems related to learned maladaptive behaviors and habits perpetuating their insomnia benefited to a greater degree from the first 3 session (education, stimulus control, sleep restriction and sleep hygiene). Identifying the different types of sleep disturbances and delivering a CBT-I treatment tailored to the specific needs of a specific breast cancer survivor would facilitate the delivery of efficient as well as effective CBT-I treatment.

Future studies should have multiple, longer follow up assessments. This study followed its participants weekly for 6 weeks after the CBT-I treatment and found the CBT-I benefits maintaining, improving and sometimes manifesting for the first time (i.e., quality of life) at the 6 week follow-up assessment. Future studies with additional follow-ups (e.g., 12 weeks, 6 months, 1 year) would be helpful to capture and explain the full effects of, and durability of the CBT-I treatment.

A final recommendation for the future of this field of research is to test the efficacy of the CBT-I during breast cancer treatment. Because insomnia and its consequences are often experienced during cancer treatment and may interact and exacerbate the side effects of the cancer treatment, as well as affect the treatment adherence and outcome, an investigation of the effects of CBT-I during cancer treatment would be meaningful.

In summary, the results of this study suggest that CBT-I improves sleep in women survivors of breast cancer. Although more research is needed to better
understand the effects of CBT-I on the psychological and physical symptom cluster presented in breast cancer patients, and to find the optimal treatment protocol that maximizes the gains this population can reap from the treatment, the present research study in combination with the previous studies, shows that CBT-I in breast cancer patients is both a feasible and efficacious treatment in reducing sleep disturbance.
Table 1. *Means, Standard Deviations and Ranges of Subjective Insomnia by Phase and Group*

**Insomnia (ISI)**

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>16.80</td>
<td>3.83</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>12.20</td>
<td>6.57</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>10.20</td>
<td>6.22</td>
</tr>
</tbody>
</table>
Table 2. *Means, Standard Deviations and Ranges of Subjective Sleep Quality by Phase and Group*

Sleep Quality (PSQI)

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>11.00</td>
<td>1.41</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>8.60</td>
<td>4.10</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>7.00</td>
<td>4.53</td>
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</table>
Table 3. *Means, Standard Deviations and Ranges of Subjective Sleep Medication Count by Phase and Group*

Sleep Medication Count

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th></th>
<th>TAU Group</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>Range</td>
<td>M</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>0.80</td>
<td>1.30</td>
<td>0-3</td>
<td>0 TAU</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>0</td>
<td>0</td>
<td>0-0</td>
<td>1 Pre-Tx</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>0.20</td>
<td>0.45</td>
<td>0-1</td>
<td>2 Post-Tx</td>
</tr>
<tr>
<td>Variable</td>
<td>Session 1</td>
<td>Session 5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>------------</td>
<td>------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>Range</td>
<td>$M$</td>
</tr>
<tr>
<td>TIB (Min.)</td>
<td>474.22</td>
<td>66.5</td>
<td>372–610</td>
<td>416.18</td>
</tr>
<tr>
<td>SE (%)</td>
<td>73.09</td>
<td>15.0</td>
<td>35–92</td>
<td>83.25</td>
</tr>
<tr>
<td>WASO (Min.)</td>
<td>53.24</td>
<td>42.9</td>
<td>3–164</td>
<td>25.20</td>
</tr>
<tr>
<td># of Wakes</td>
<td>3.15</td>
<td>1.36</td>
<td>1–6</td>
<td>2.16</td>
</tr>
<tr>
<td>TST (Min.)</td>
<td>344.82</td>
<td>78.3</td>
<td>178–504</td>
<td>347.49</td>
</tr>
<tr>
<td>SOL (Min.)</td>
<td>44.19</td>
<td>41.5</td>
<td>5–143</td>
<td>24.77</td>
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<tr>
<td># Sleep Aids</td>
<td>1.29</td>
<td>2.37</td>
<td>0–7</td>
<td>0.50</td>
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</table>
Table 5.

*Means, Standard Deviations and Ranges of Actigraphic TST by Phase and Group*

**TST in Hours**

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>7.21</td>
<td>1.02</td>
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<tr>
<td>1 Post-Tx</td>
<td>6.66</td>
<td>1.76</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>6.58</td>
<td>1.15</td>
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Table 6.

*Means, Standard Deviations and Ranges of Actigraphic WASO by Phase and Group*

<table>
<thead>
<tr>
<th>WASO in Hours</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>1.41</td>
<td>0.73</td>
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<tr>
<td>1 Post-Tx</td>
<td>1.04</td>
<td>0.27</td>
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<tr>
<td>2 Follow-Up</td>
<td>1.03</td>
<td>0.52</td>
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Table 7.

**Means, Standard Deviations and Ranges of Actigraphic Number of Wakes by Phase and Group**

<table>
<thead>
<tr>
<th>Number of Wakes</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>0 Pre-Tx</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>1 Post-Tx</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>2 Follow-Up</td>
<td>20</td>
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Table 8.

Means, Standard Deviations and Ranges of Actigraphic Sleep Percent by Phase and Group

Sleep Percent

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>0.83</td>
<td>0.07</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>0.86</td>
<td>0.04</td>
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<tr>
<td>2 Follow-Up</td>
<td>0.86</td>
<td>0.07</td>
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Table 9.

Means, Standard Deviations and Ranges of Subjective Quality of Life Questionnaires

by Phase and Group

<table>
<thead>
<tr>
<th>Quality of Life (MOS-SF 36)</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>66.00</td>
<td>8.94</td>
</tr>
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<td>1 Post-Tx</td>
<td>59.00</td>
<td>14.31</td>
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<tr>
<td>2 Follow-Up</td>
<td>71.00</td>
<td>14.32</td>
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</table>

<table>
<thead>
<tr>
<th>Quality of Life (FOSQ)</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>16.07</td>
<td>2.36</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>16.50</td>
<td>2.19</td>
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<tr>
<td>2 Follow-Up</td>
<td>17.45</td>
<td>1.85</td>
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Table 10.

*Means, Standard Deviations and Ranges of Subjective Depression by Phase and Group*

<table>
<thead>
<tr>
<th>Depression (CESD)</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase</td>
<td>M</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td></td>
<td>13.20</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td></td>
<td>9.60</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td></td>
<td>9.00</td>
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Table 11.

*Means, Standard Deviations and Ranges of Subjective Psychological Symptoms by Phase and Group*

**Psych Symptoms (BSI)**

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>6.40</td>
<td>3.05</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>5.20</td>
<td>2.59</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>6.20</td>
<td>5.21</td>
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Table 12.

*Means, Standard Deviations and Ranges of Subjective Fatigue by Phase and Group*

**Fatigue (MFSI)**

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th>TAU Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>18.80</td>
<td>15.74</td>
</tr>
<tr>
<td>1 Post-Tx</td>
<td>15.20</td>
<td>19.63</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>16.20</td>
<td>18.47</td>
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Table 13.

Means, Standard Deviations and Ranges of Subjective Menopausal Symptoms by Phase and Group

Menopausal Symptoms (Greene)

<table>
<thead>
<tr>
<th>Phase</th>
<th>CBT-I Group</th>
<th></th>
<th></th>
<th>TAU Group</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>Range</td>
<td>M</td>
<td>SD</td>
<td>Range</td>
</tr>
<tr>
<td>0 Pre-Tx</td>
<td>19.00</td>
<td>6.28</td>
<td>10-27</td>
<td>0 TAU</td>
<td>18.71</td>
<td>7.91</td>
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<tr>
<td>1 Post-Tx</td>
<td>17.60</td>
<td>5.22</td>
<td>12-26</td>
<td>1 Pre-Tx</td>
<td>21.57</td>
<td>5.41</td>
</tr>
<tr>
<td>2 Follow-Up</td>
<td>15.00</td>
<td>7.38</td>
<td>8-27</td>
<td>2 Post-Tx</td>
<td>18.71</td>
<td>6.82</td>
</tr>
</tbody>
</table>
Graph 1. Participant Flowchart

Assessed for eligibility (N=44)

Did not participate (n=23)
  Reasons:
  Refused to participate (n=15)
  Satisfied taking medications for sleep (n=1)
  Study too cumbersome (n=2)
  Unknown (n=12)
Ineligible (n=8)
  No insomnia (n=5)
  Other psychological disorders (n=2)
  Not finished with cancer treatment (n=1)

Enrollment
n=21

Allocated to CBT-I, Follow-Up
  (n=11)
  Completed allocated intervention (n=6)
  Did not receive allocation intervention (n=5)
  Reasons:
  Dropped (n=2)
    To address other psychological issues (n=1)
    Study too troublesome (n=1)
  Ineligible (n=3)
    Active cancer (n=1)
    Major depressive disorder (n=2)

Allocation

Allocated to TAU, CBT-I
  (n= 10)
  Completed allocated intervention (n= 8)
  Did not receive allocation intervention (n=2)
  Reasons:
  Ineligible (n=2)
    No insomnia (n=1)
    Active cancer (n=1)
Graph 2.

Study Design

<table>
<thead>
<tr>
<th>CBT-I Group</th>
<th>CBT-I</th>
<th>FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAU Group</td>
<td>TAU</td>
<td>CBT-I</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Phase 0</th>
<th>Phase 1</th>
<th>Phase 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 0</td>
<td>Week 6</td>
<td>Week 12</td>
</tr>
</tbody>
</table>
Graph 3.

Mean ISI by group. The solid black represents the baseline assessment, the checkered pattern represents the post CBT-I assessment, the blank white pattern represents the post TAU assessment (difference between CBT-I and TAU, p=0.03), and the dotted pattern represents the post Follow-Up assessment (Pre CBT-I - Follow-up difference: p=0.03; post-CBT-I - Follow-up difference: p=0.03).
Graph 4.

PSQI by group and week of study. Solid lines represent the treatment phase, dotted lines represent a non-treatment phase (TAU for the TAU group and Follow-Up for the CBT-I group) (first 7 weeks time factor: p=0.0002, second 6 weeks group factor: p=0.002, groupXtime: p<0.0001).
Graph 5.

Sleep diary sleep efficiency for all 14 participants by treatment week (p=0.01).
Graph 6.

Mean minutes of WASO as recorded on sleep diaries for all 14 participants by treatment week (p=0.002).
Graph 7.

Mean Actigraphy WASO in hours by group. The solid black represents the baseline assessment, the checkered pattern represents the post CBT-I assessment, the blank white pattern represents the post TAU assessment (pooled pre CBT-I - Post CBT-I difference: $p=0.009$), and the dotted pattern represents the post Follow-Up assessment (Pre CBT-I - Follow-Up difference: $p=0.04$).
Appendix 1. An example of Spielman’s three factor model of insomnia in a patient with breast cancer.

1. Predisposing factors:
   a. Arousability
   b. Decreased homeostatic sleep drive

2. Precipitating factors:
   a. Acute stress related to breast cancer diagnosis
   b. Breast cancer treatment side effects
   c. Surgical pain

3. Perpetuating factors:
   a. Poor bed-sleep stimulus control
   b. Poor sleep hygiene
   c. Maladaptive thoughts, e.g., “I will not be able to sleep tonight, which will make me miserable throughout the whole day tomorrow”
   d. Psychological conditioning
   e. Depression
   f. Anxiety
Appendix 2. Patients’ Comments.

Comments about insomnia and breast cancer:

1. “The breast cancer took away the only thing I was ever good at: Sleep.”

2. “When I got diagnosed with breast cancer, I thought I was going to die. I never wanted to go to bed and sleep because for me sleep was like not living and I wanted to live as much as possible…. Then when I was cured, I realized that I should sleep, but I had forgotten how to do it.”

3. “It was being off of the hormonal replacement therapy that made me not sleep well, and then it just got worse with the cancer treatment.”

4. “I use to be a great sleeper. Then my insomnia started with the breast cancer and I haven’t slept since.”

Comments about the CBT-I:

1. “I remembered my dreams for the first time in years.”

2. “I feel like I have control over my sleep now.”

3. “This therapy has changed my life.”

4. “I feel like I am regaining my life back.”

5. “Even my husband told me he thinks I am sleeping better.”

6. “I have more time and energy for family and fun now.”

7. “I haven’t slept this well in 10 years.”

8. “I slept better after the educational session… because it helped me to know that even if I don’t sleep the whole night I can go in all the stages of sleep. This makes me
less anxious, because I know that my body will get at least a little bit of what it
needs.”

9. “I am not sure if I sleep better or not, but the therapy has helped me because I don’t
stress about sleeping or not anymore, and I have learned good patterns and behaviors.”

Sleep Restriction

- The participant fills out a baseline sleep diary for one week
- During the second week participants were prescribed to stay in bed the amount of hours that they had on average slept during the first week of therapy
- As the patients’ sleep efficiency \[SE=(\text{Total sleep time/total time in bed}) \times 100\] increased to close to 90% the patients were prescribed 15 to 30 minutes increases in their time spent in bed by going to bed 15 to 30 minutes earlier

Stimulus Control

- Participants were told to use their bed for sleep and sex only
- And to get out of the bed if awake for more than 15 minutes or if frustrated or alert

Sleep Hygiene Rules

The following sleep hygiene rules were discussed in session and also given to the patient as a handout to bring home:

- Sleep only as much as you need to feel refreshed during the following day
- Get up at the same time every day, 7 days a week
- Exercise regularly
- Make sure your bedroom is comfortable and free from noise and light
- Make sure that your bedroom temperature is comfortable during the night
- Eat regular meals and do not go to bed hungry
- Avoid excessive fluids in the evening
• Cut down on all caffeine products
• Avoid alcohol, especially in the evening
• Avoid smoking, especially during the evening
• Don’t take your problems to bed
• Don’t try to fall asleep
• Put the clock under the bed or turn it so you cannot see it
• Avoid naps
Appendix 4. Sleep Diary

Name:____________ Date:____________

My Daily Sleep Diary - MONDAY

Please fill out during the morning:

1. Last night my bed time was: ________ am/pm.

2. It took me ___________ minutes/hours to fall asleep.

3. I took ________ mg of ______________ and/or ________ oz of alcohol to help
   me sleep.

4. Once I fell asleep I woke up _______ times. The reason(s) I think I woke up
   for are: _________________________________________________________
   ______________________________________________________________ 

5. Each time I woke up I stayed awake (please use back of paper if necessary):
   1. ________ min/hours
   2. ________ min/hours
   3. ________ min/hours
   4. ________ min/hours
   5. ________ min/hours

6. I woke up for the last time in the morning at ______________am/pm.

7. I got out of bed at ________________ am/pm.

8. My sleep was:_________ (1=very restless, 5= very sound)

9. This morning I feel ________ (1=exhausted, 5=refreshed)
Please fill out at the end of your day:

1. Today I took __________ naps for a total of _________ min/hours.

2. Today I exercised _____ times for a total of ___________ min/hours.

3. From 1 (minimum) to five 5 (maximum) I felt:
   
   1. Alert __________
   
   2. Concentrated____
   
   3. Happy __________
   
   4. Tired __________
   
   5. Anxious __________
Appendix 5. Overview of the focus of each Session:

First session:
- Overview of treatment program
- Goal setting
- Education about sleep, sleep stages, sleep throughout life
- Spielman’s 3-P insomnia model
- Homework explanation (sleep diaries)

Second session:
- Sleep diary variables explanation and calculation
- Sleep restriction
- Stimulus control

Third session:
- Review of sleep diary and discussion
- Sleep hygiene rules

Fourth session:
- Review of sleep diary and discussion
- Introduction to the CBT model
  - Cognitive errors, cognitive restructuring, worry-time

Fifth session:
- Review of sleep diary and discussion
- Continuation of cognitive restructuring
Sixth session:

- Review of sleep diary and discussion
- Review and integration of therapy components
- Relapse prevention
- Identification of high-risk insomnia situations
- Progressive muscle relaxation
- Feedback on therapy
REFERENCES


