EFFICACY OF COGNITIVE BEHAVIOUR AND RELAXATION TECHNIQUES ONINSOMNIA AMONG STUDENTS OF AHMADU BELLO UNIVERSITY,

ZARIA, KADUNA STATE, NIGERIA

BY

Moses VICTOR

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY AND COUNSELLING

FACULTY OF EDUCATION

AHMADU BELLO UNIVERSITY,

ZARIA, NIGERIA

JANUARY, 2017

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ZARIA, KADUNA STATE, NIGERIA

BY

Moses VICTOR

N.C.E. Soc. Stud. (FCE, Katsina, 2004)

B.Ed. Soc. Stud. (ABU, 2008)

M.Ed. Educ. Psych. (ABU, 2013)

P13EDPC9009

A THESIS SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES, AHMADU BELLO UNIVERSITY, ZARIA, NIGERIA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF DOCTOR OF PHILOSOPHY IN EDUCATIONAL PSYCHOLOGY,

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY AND COUNSELLING, FACULTY OF EDUCATION, AHMADU BELLO UNIVERSITY, ZARIA

JANUARY, 2017

DECLARATION

I declare that this thesis entitled; "**Efficacy of Cognitive Behaviour and Relaxation Techniques on Insomnia Among Students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria"** is the result of my research effort and to the best of my knowledge it has not been produced and presented for the award of any degree or diploma at this or any other Institution. All quotations and citations made in this work have been fully acknowledged in the reference pages.

_____ _____

Moses VICTOR Date

CERTIFICATION

This thesis entitled;**"Efficacy of Cognitive Behaviour and Relaxation Techniques on Insomnia among Students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,"** meets the requirements governing the award of degree of Doctor of Philosophy in Educational Psychology of the Ahmadu Bello University, Zaria and is approved for its contribution to knowledge and literary presentation.

_____ _____

Prof. M. Balarabe Date
Chairman, supervisory committee

_____ _____

Dr. A.I. Mohammed Date Member,
supervisory committee

_____ _____

Dr. U. Yunusa Date
Member, supervisory committee

_____ _____

Dr. A.I. Mohammed Date
Head of Department (HOD)

_____ _____

Prof. K. Bala Date Dean,
School of Postgraduate Studies

DEDICATION

This thesis is dedicated to my beloved parents,I. K. Victorand Ngozi Victor for their love and supportand to my beloved wife, Blessing Victor Moses, for her encouragement and for being patient with me throughout the period of this study.

ACKNOWLEDGEMENTS

First, the researchergives God the glory for his mercies and grace which help to see him through this research work.While reflecting on the number of people and scholars who have played key roles in helping to organize this work, the researcher acknowledges the contributions of hissupervisors;Prof. M. Balarabe, Dr. A.I. Mohammed and Dr. U. Yunusa.Yourmany useful pieces of adviceand corrections provided the direction for this work. The researcher appreciates thefact that you have always been available to assist to make this work better. No doubt, you have provided the researcher with the solid foundation from which to build a career and isforever grateful.

To the internal examiners, Prof. R.M. Bello, Rev. Fr. Prof. D.A. Oliagba, and Prof. E.A. Gunen, the researcher appreciates the priceless time and expertise you have volunteered to review this thesis. Your meticulous scrutiny and guided observations have made this work what it is. Thank you so much for your effort.

The researcher also understand that this work could not have been better without the untiring efforts and pivotal role played by Prof. E.F. Adeniyi, Prof. K.H. Mahmoud, Prof. S. Sambo, Prof. J.A. Gwani, Dr. M.I. Abdullahi, Dr. J.O. Bawa, Dr. U. Abdulwahid, Dr. H.A. Tukur, and Mr. L.K. Maude. Your useful scholarly contributions have helped immensely in enriching this research. The researcher is conscious that this work may not be where it is if it were not for your support.

The research assistant, Miss. Helen Opuana, is appreciated for giving up several Saturdays in order to assist. You provided the much needed research support, visiting, calling and encouraging the female subjects in the hostel. Thank you, Helen.

In addition, the researcher appreciatesMalam A. Mua'zu, Mr. M. Markus and Malam S. Abubakar for their contribution to the progress and success of this work. Lastly, the researcher appreciates friends and relatives that have contributed in one way or the other in making this programme a success. Worthy of mention but a few, isthe researcher's uncle, SP. Bernard David, who on several occasions assisted the researcher financially. In addition, the researcher appreciatesDr. Gateh, a Ghanaian from University of Cape coast, who upon graduation from faculty of Agriculture, ABU, Zaria, gave the researcher his HP Laser Jet

printer free of charge. The printer helped immensely in minimizing the cost of doing the research.

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OPERATIONAL DEFINITION OF TERMS

Cognitive Behaviour Techniqueis an interventiontechnique that help studentswith insomnia change their thinking habit at bedtime and behaviour to healthy ones to enhance good sleep quality.

Insomniais a sleep condition characterized by difficulty falling or staying asleep long enough to feel refreshed the next morning with attendant daytime side effects

Relaxation Techniqueis an interventiontechnique that help studentswith insomnia to relax their mind and be calm while on bed to achieve good sleep quality

ABSTRACT

The study investigated the efficacy of cognitive behaviour and relaxation techniqueson insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria. Five objectives, five research questions and five null hypotheses guided the study.Quasi-experimental design involvingpretest posttest experimental/control group was used. A total sample of thirty-sixstudentsof Ahmadu Bello University, Zaria with amean age of24.5,who were identified withprimary insomnia condition wereused for the study and purposively divided into three groups of twelve each.One group was exposed toCognitive Behaviour Technique for insomniawhich include; stimulus control instruction, sleep restriction, sleep hygiene education, and cognitive technique while the other was exposed toRelaxation Techniquewhich include;deep breathing exercise, progressive muscle relaxation,guided visualization meditation, and mindfulness technique. The thirdgroup was used as a control group. The intervention lasted for a period of six weeks.The instrument used for data collection was Insomnia Severity Index,which wasadministered at pre andpost intervention stages to assess treatment outcome.The Statistical Package for the Social Sciences (SPSS version 24) was used to analyse the data collectedusing the one-way between groups analysis of covariance method.After adjusting for the covariate,findings suggesta significant effect of cognitive behaviour technique in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,$F(1,21) = 22.416$, $p = .000$.Relaxation technique is also efficacious in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria, $F(1,21)= 12.630$, $p =.002$. The study also found a significant differential effect of cognitive behaviour technique when compared torelaxation techniquein reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,$F(1,21)= 12.206$, $p =.002$, with cognitive behaviour technique producing higher remission rate of about 65.6% compared to 45.6% for relaxation technique.No significant differential effect of cognitive behaviour technique,$F(1,9) = .106$, $p = .752$, and relaxation technique, $F(1,9)= .369$, $p = .559$,in reducing insomnia of male compared to female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria was found.Based on the findings of the study, it was recommended thatschool psychologists and counsellors should be encouraged to use cognitive behaviour and relaxation technique asa more viable interventionto re-address insomnia condition among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria.

CHAPTER ONE
INTRODUCTION

1.1 Background to the Study

Higher institutions of learning like the universitiesare dominated in number by young adult students,transitingto a world of relative freedom, new experiences and choices, and based on the choices they made, their campus and off-campus experiences can contribute to healthor disorderpromoting lifestyles and ultimately relate to their quality of lives and school activities.

National Sleep Foundation (2005), reported that young adults are at greater risk than other age groups for developing first onset psychopathology (mental/behaviour disorder), which may lead to lower social and academic performance, lethargy and worse long-term outcomes such as school dropout and possibly poor productivity at work or school. The foundation also noted that majority of students due to new environment; life experiences and choices at schoolreceive significantly less sleep. A survey in 26 of 36 States in Nigeria on the prevalence of insomnia showed that 25.68% of people aged 18 – 30 years had insomnia (Gureje, Oladeji, Abiona, Makanjuola &Esan, 2011). A national survey in US byHicks and Pellegrini (2001), showed that 68.3% of College students reported sleep problems, against 26.7% found in 1982.This suggests an upward trend. In 2009, Lund, Reider, Whiting and Prichard,found 60% of College students reported poor sleep quality. Approximately 35% of students' population with sleep problems have reported experience of insomnia during the course of a year in school (Walsh, Benca & Bonnet, 1999), 11.4% reported that their symptoms started when they were between 21 and 30 years old (Hardison, Neimeyer, & Lichstein, 2005).

A study in Europe using randomly selected young adults ages 21-30 years found that 16.6% reported experiencing insomnia. About 45% of these young adults with insomnia continued to report sleep problems at a 3.5-year follow-up (Breslau, Roth, Rosenthal,& Andreski, 1996). Out of over 1,000 College students surveyed by Taylor and Bramoweth (2010), 6.8% of the students with insomnia used medication (either prescription or over the counter) to help them sleep and 11.4% used alcohol as a sleep aid.What precipitates and perpetuates insomnia among students' population may vary from medical to non-medical reasons.However, studies by Tagaya, Uchiyama, Ohida, Kamei, and Shibuti (2004), reported that pressure from academic workload was one of the reasons for sleep disturbance among students. Aside academic pressure, social activities, social pressures, and college cultures contributed to the problem.

Harvey (2000), stated that approximately 80% of individuals with insomnia reported an inability to "empty their mind," and "unwind their thinking" at bedtime. Harvey stressed that the thought content of people with insomnia is full of worries, emotionally arousing, and focused on daily and future concerns particularly, their consequences. In addition, Gellis and Lichstein (2009), reported that insomniac possess a number of behaviors that has to do with the frequency of "worry, planning, or thinking about important matters at bedtime". This arousing pre-sleep cognitive activity is believed to be one of the causesof insomnia among students. When the condition persists, it taxes its sufferer mentally and physically, and can cause a lot of stress and distress for the individual(Vgontzas, Liao, Bixler, Chrousos, & Vela-Bueno, 2009; Byles,Mishra, Harris& Nair, 2003). However, despite the existence of several psychological treatment options for insomnia, people who experience sleep difficulties usually start with passive strategies. When surveyed, many insomnia patients

reported that they read, watch television, or listen to the radio when they are unable to sleep. Many people with insomnia say that they "do nothing" except lie in bed, tossing and turning, waiting for sleep to come. The first level of active treatment among sufferers is self-medication with alcohol, over-the-counter sleep aids, or natural/herbal dietary supplements. If people with insomnia eventually seek medical help for their condition, they are generally prescribed hypnotic drugs as a second level of active treatment (National Sleep Foundation, 2001).

Literature have shown that psychological approaches like cognitive behavioral techniques produced sustained benefits in management of insomnia without the risk for tolerance or adverse effects associated with pharmacologic approaches. Cognitive behaviour technique for insomnia (CBT) is a brief, sleep-focused, multimodal intervention that includes psychological and behavioural intervention such as stimulus control, sleep restriction, cognitive therapy, and education about sleep hygiene, and sometimes combined with relaxation technique (Morin & Benca 2012). Relaxation technique,which improvescalmness,may include deep breathing exercise, progressive muscle relaxation, guided visualization among others (Ethan, 2014, 2008, NSF 2015). A combined approach is usually preferred because several dimensions of insomnia can be addressed(Morin & Benca, 2012). The techniques, when combined have shown high potency and are now commonly recommended as first-line psychological treatment for insomnia due to its success rateof 70 to 90% (Morin, 2004).

1.2 Statement of the Problem

Insomnia, a sleep condition that is characterized by persistence difficulty in falling or staying asleep long enough to feel refreshed the next morning, with daytime side effects among young adult students studying in higher institutions of learning is taking a worrisome proportion particularly in this computer age and demanding 21st century academic environment.It was reported that insomnia affects about a quarter of young people in Nigeria (Gureje, et al. 2011). As a common sleep, complain that makes restful and restorative sleep often a painstaking and exasperating experience, this condition if not mitigated could give rise to the development of other comorbidconditions.It has been reported that students who experience persistence insomnia might suffer from fatigue, reduced quality of life, and are likely to experience depression and engage in substance abuse to enable them sleep. Lack of sleep may also weaken students' daytime performance, leading to falling asleep in the class, lateness to lecture, lacking energy to perform academic tasks, poor concentration, inattentiveness, indolence, and absenteeism and can escalate tendency for memory deterioration, which might consequently affect negatively on students' school outcomes. Despite theseeffects however, the condition is still an under-recognized and under studied problem in this clime. Even empirical studies examining remediation among young adult students of Ahmadu Bello University,Zaria, Kaduna State, Nigeria are lacking.

Although, studies (Edinger & Sampson, 2003;Fornal–Pawłowska & Szelenerger, 2013), have supported the potency ofcognitive behaviour technique in reducing insomniabut locally,studies investigating its relative effectiveness with relaxation technique among young adults in the university domiciling in the hotels are rare.Therefore, in view of the

foregoing, and realizing the importance of good sleep quality, particularly to students'memory, and learning outcome and other school activities, the need to address insomnia in a psychologicalwaybecomes very essential.

In the light of the above, this study finds out the efficacy ofcognitive behavior and relaxation techniques on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria.

1.3 Objectives of the Study

The objectives of the study were to:

i. Determine the effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria
ii. Determine the effect of relaxation techniqueon insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria
iii. Determine therelative effects of cognitive behaviour and relaxation techniqueson insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria
iv. Examine thedifferential effects of cognitive behaviour technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria
v. Examine the differentialeffects of relaxation techniqueon insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

1.4 Research Questions

The study was tailored towards answering the following research questions;

i. What is the effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?
ii. What is the effect of relaxation technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?
iii. What is therelativeeffects of cognitive behaviour and relaxation techniques on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?
iv. What is the differentialeffect of cognitive behaviour technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?
v. What is the differentialeffect of relaxation technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?

1.5 Research Hypotheses

Based on the research questions, the following non-directional hypotheses wereused to guide the study;

H_{01} There is no significant effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

H_{02} There is no significant effect of relaxation techniqueon insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

H_{03} There is no significant differential effect of cognitive behaviour and relaxation techniques on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

H_{04} There is no significant differential effect of cognitive behaviour technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

H_{05} There is no significant differentialeffect of relaxation technique on insomnia condition ofmaleandfemale students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

1.6 Significance of the Study

The findings will help students suffering from sleep initiation/maintenance difficulty by providing them with tested techniques they can use to resolve such problem without resorting to sleeping pills or substance abuse, which can lead to dependence and addiction consequently. Using these techniques will help students have a good sleep quality and enhanced daytime performance.

The study will also benefit school psychologists andcounsellors by providing them with tested cognitive and behavioural techniques that can be used to help students who are suffering from insomnia or sleeplessness.It is hoped that when students sleep needs are address, attention and concentration in class will likely improve and teachers will need less effortin term of behavior modification or management in the class most especially among students who are fond of sleeping in the class while lecture is ongoing.

The findings of this study will add to the existing body of literature and theories concerning behavioural and psychological interventions formanagement of Insomnia particularly among young adult students' population in Ahmadu Bello University, and the community as a whole.

It will help create awareness to the public in general and provide government and non-governmental organizations with data about the existence and efficacy of psychological and behavioural techniques in managing insomnia. This information can help to improve public and mental health of students and the community in general.

When published, this study will provide future researchers, and readers with valuable information and findings regarding the effectiveness of using various psychological techniques and methods such as cognitive behaviour technique and relaxation techniquein reducing insomnia.

The findings from this study can lead to new methods or strategies for dealing with insomnia and can shed more light on new findings, suggestions for improvements and useful recommendations.

1.7 Basic Assumptions of the Study

Based on available literature regarding the problem under investigation, the studyassumes that;

i. Cognitive behaviour technique might be effective inreducing insomnia amongstudents of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

ii. Relaxationtechnique mightbe effective inreducing insomnia amongstudents of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

iii. Cognitive behaviour technique might be more effective when compared to Relaxation technique in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

iv. The effect of cognitive behavior technique on insomnia between male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeriamight not differ

v. The effect of relaxation technique in reducing insomnia between male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria might not differ

1.8 Scope and Delimitation of the Study

The scope of the study was all students ofAhmadu Bello University, Zaria, Kaduna State, Nigeria who show symptoms ofprimary insomnia. The study was delimitedto assessing the efficacy of CBT (stimulus control instruction, sleep restriction, sleep hygiene education, and cognitive technique) and Relaxation technique(deep breathing exercise, progressive muscle relaxation, guided visualization meditation, and mindfulness technique). It was delimitedbecause of prevalence of insomnia,which is caused by cognitive and behaviour problems among the target group. Another reason is the dearth of research in this clime targeting young adults in the universities,and toexaminewhethercognitive behaviour and relaxationtechniques can be applied effectivelyin managing insomnia among young adults' university students of different demography domiciling mostly on universityfacilities.

CHAPTER TWO
REVIEW OF RELATED LITERATURE

2.1 Introduction

The section presents review of materialsrelated to the study, and this was done under different subheadings;

2.2.1 Concept of Insomnia

2.2.2 Concept of cognitive behaviour techniques

2.2.3 Concept of relaxation technique

2.3 Theoretical Framework

2.3.1 The 3Ps theory of insomnia

2.3.2 Cognitive theory of insomnia

2.3.3 Hyper-arousal theory of insomnia

2.3.4 Selective attention theory of insomnia

2.3.5 Sleep-wake regulation theory of insomnia

2.3.6 Model of insomnia

2.4 Review of Empirical Studies

2.5 Summary

2.2.1 Conceptof Insomnia

The term insomnia is derived from the Latin word *in-, meaning "not" or "without," and, somnus, meaning, "sleep."*Insomnia is a sleep complain that is characterized by difficulty falling or staying asleep long enough to feel refreshed the next morning, even when there is enough time to sleep. Occasional experience ofinsomnia, especially during times of stress, are not abnormal, but persistent insomnia characterized by recurrent difficulty getting to sleep or remaining asleep is an abnormal behavior pattern (Harvey & Tang, 2012). About 6% to 10% of U.S. adults (Bootzin & Epstein, 2011; Smith & Perlis, 2006), about 12% of students in Nigeria (Gureje, Makanjuola, & Kola, 2007), and 25.68% of young people aged 18 – 30 years in Nigeria also (Gureje et al. 2011) suffer from the most commonly occurring sleep-wake disorder - primary insomnia.

Insomnia as a sleep disorder is distinguished from sleep problem by it persistence and effects on an individuals' daily functioning. Sleep problems tend to be random occurrences that peak during stressful times, and are accentuated by worry and rumination, but usually goes away after the stressful situation passes, sleep disorders on the other hand persist during times of no stress and are themselves a cause of stress for most insomnia sufferers. Unlike occasional sleep disturbances, sleep disorders significantly affect the individual's

physiological and psychological health, and the individual's productivity as a whole. In particular, insomnia is commonly cited to be the primary sleep disorder found in those with chronic sleep problems. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition – (DSM-V-TR) has a specific chapter devoted to Sleep Disorders (American Psychiatric Association, 2013). The Sleep Disorders are organized into four sections according to presumed etiology: Primary Sleep Disorders (which is further subdivided into Dysomnia and Parasomnia), Sleep Disorder related to another Mental Disorder, Sleep Disorder due to a General Medical Condition, and Substance Induced Sleep Disorder (APA, 2013). Primary Insomnia – a type of Dysomnia is characterized by abnormalities in the amount, quality, or timing of sleep, and includes sleep disorders such as Primary Insomnia, Primary Hypersomnia, Narcolepsy, Breathing-Related Sleep Disorder, and Circadian Rhythm Disorder (APA, 2013). In particular, Primary Insomnia is characterized by difficulty falling asleep, difficulty maintaining sleep, or a feeling of non-restorative sleep not due to any other identifiable causes such as a mental disorder or physical illness (APA, 2013).

According to the DSM-V-TR, Primary Insomnia is defined by the following diagnostic criteria: 1) difficulty initiating or maintaining sleep, or non-restorative sleep being the predominant complaints; 2) sleep disturbance or associated daytime fatigue causes significant distress or impairment in important areas of functioning (e.g., academic, social, occupational, among others.); 3) sleep disturbance does not occur exclusively during the course of other Primary Sleep Disorders (e.g., Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, and others.); 4) sleep disturbance does not occur exclusively during the course of a psychiatric disorder (e.g., major depression or

anxiety disorders); and 5) sleep disturbance is not due to direct effects of a substance (e.g., drug abuse, medication) or a general medical condition (APA, 2013).

However, it can be difficult to differentiate Primary Insomnia from secondary insomnia and the vast number of sleep disorders mirroring Primary Insomnia's symptoms. For example, cases of secondary insomnia (when insomnia is not the primary cause of the sleep disorder) can be observed in Sleep Disorders due to a General Medical Condition, Mental Disorder, and/or are substance-induced, but insomnia in these cases is secondary to a condition that is severe enough to warrant independent attention in precedence to the insomnia (APA, 2013). Other Sleep Disorders such as Circadian Rhythm Sleep Disorder, Narcolepsy, and Parasomnias may also have symptoms similar to Primary Insomnia but have different etiologies (APA, 2013). There are additional challenges to assessing insomnia. As insomnia has been operationally defined in multiple ways with varying standards and criteria.Most academic authors acknowledge the lack of consistency found in the concept construction of insomnia. Some researchers have used the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM) to operationalize insomnia, while other researchers have focused on specific symptoms of insomnia (e.g., quality of sleep, duration of sleep, level of difficulty initiating and maintaining sleep, among others). This is particularly common in large scale, epidemiological surveys where the criteria used to define insomnia varies between surveys, and there is a lack of comparability between the population studied, time frames used, and the time between follow up periods. Therefore, although attempts have been made, no standard definition of insomnia has prevailed as the accepted definition used for research purposes. Insomnia precise definition accounts the difficulties in sleep initiation or maintenance and how that amounts to unproductiveness in many areas of an

individual's situations. According to World Health Organization (WHO) 1997 report on international classification of sleep disorders, Insomnia can be defined as the ineffectiveness in achieving quality and or sufficient sleep. Insomnia has been rated as the most common sleep disorder (Grewal & Doghramji 2010). It is commonly distinguished by complains on sleep duration and quality (Morin, 2012).

Insomnia isalso defined as a condition that is related to significant distress or impairments in an individual's functioning (e.g., physical, academic, mental, occupational, among others) due to difficulty initiating or maintaining sleep (Maxmen, Ward, & Kilgus, 2009). The purpose of this study is not to examine the prevalence rate of Insomnia but to examine the effects of psychological techniques on insomnia among young adult students in universities.

It should be noted that there is a price to be paid for sleep deprivation associated with insomnia. Research evidence shows that the sleep-deprived brain is less able to concentrate, pay attention, respond quickly, solve problems, and remember recently acquired information (Florian, Vecsey, Halassa, Haydon & Abel, 2011; Lim & Dinges, 2010). Chronic sleep deprivation – regularly getting too little sleep – is linked to a range of serious physical health problems, including poorer immune system functioning (Carpenter, 2013). The immune system protects the body against disease, so it is not surprising that researchers report that people who sleep less than 7 hours a night had a threefold higher risk of developing the common cold after exposure to cold viruses than those who sleep 8 or more hours nightly (Cohen et al., 2009; Reinberg, 2009). If we miss a few hours of sleep, we may feel a little groggy the next day, but we will probably be able to muddle through. Nevertheless, over time, continued sleep deprivation takes a toll on our ability to function at our best, leading to daytime fatigue and creating difficulties performing our usual social, occupational, student,

or other roles. Not surprisingly, people with the disorder often have other psychological problems as well, especially anxiety and depression. Psychological factors contribute to primary insomnia. People troubled by insomnia tend to bring their anxieties and worries to bed with them, which raises their bodily arousal to a level that can prevent natural sleep. Another source of anxiety comes in the form of performance anxiety, or pressure felt from thinking one must get a full night's sleep to be able to function the next day (Sánchez-Ortuño & Edinger, 2010). People who are struggling with insomnia may try to force themselves to sleep, which typically backfires by creating more anxiety and tension, thus making sleep even less likely to occur.

2.2.1.1 Types of Insomnia

Insomnia can be grouped into *primary and secondary, or comorbid, insomnia* (Buysse, 2008; Erman, 2007). Primary insomnia is a sleep difficulty or insomnia not attributable to a medicalor psychiatric disorder (World Health Organization, 2007). It is described as a complaint of prolonged sleep onset latency, disturbance of sleep maintenance, or the experience of no refreshing sleep (Riemann & Ulrich, 2002). A complete diagnosis will differentiate between primary insomnia, insomnia as secondary to another condition, or co-morbid with one or more conditions.

American Academy of Sleep Medicine (2005) international classification of insomnia include the following;

A. One or more of the following symptoms:

- Difficulty initiating sleep

- Difficulty maintaining sleep
- Waking up too early
- Non-restorative sleep

B. Sleep difficulty occurs despite adequate opportunity for sleep

C. At least one of the following daytime symptoms related to the nighttime sleep difficulty reported:

- Fatigue or malaise
- Attention, concentration, or memory impairment
- Social or vocational dysfunction or poor school performance
- Mood disturbance or irritability
- Daytime sleepiness
- Motivation or energy or initiative reduction
- Proneness for errors or accidents
- Tension headaches or gastrointestinal symptoms, or both
- Concerns or worries about sleep

The DSM-5 updated criteria for insomnia include the following (Diagnostic and Statistical Manual of Mental Disorders, 2013) predominant complaint of dissatisfaction with sleep quantity or quality, associated with one (or more) of the following symptoms:

Difficulty initiating sleep. Sleep onset insomnia is difficulty falling asleep at the beginning of the night, often a symptom of anxiety disorders. Delayed sleep phase disorder can be

misdiagnosed as insomnia, as sleep onset is delayed to much later than normal while awakening spills over into daylight hours (Kertesz& Cote, 2011).

Difficulty maintaining sleep,(maintenance insomnia) characterized by frequent awakenings or problems returning to sleep after awakenings.

Early morning awakening with inability to return to sleep.

In addition,

The sleep disturbance causes clinically significant distress or impairment in social, occupational, educational, behavioral, or other important areas of functioning.

The sleep difficulty is present for more than one month for chronic and less for acute.

The sleep difficulty occurs despite adequate opportunity for sleep.

The insomnia is not better explained by and does not occur exclusively during the course of another sleep-wake disorder (e.g., narcolepsy, a breathing related sleep disorder, a circadian rhythm sleep-wake disorder, a parasomnia).

The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication). Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia.

Insomnia can be classified as transient, acute, or chronic.

i. Transient insomnia lasts for less than a week. It can be caused by another disorder, by changes in the sleep environment, by the timing of sleep, severe depression, or by

stress. Its consequences – sleepiness and impaired psychomotor performance – are similar to those of sleep deprivation (Roth& Roehrs, 2003).

ii. Acute insomnia is the inability to consistently sleep well for a period of less than a month. Insomnia is present when there is difficulty initiating or maintaining sleep or when the sleep that is obtained is non-refreshing or of poor quality. These problems occur despite adequate opportunity and circumstances for sleep and they must result in problems with daytime function. Acute insomnia is also known as short-term insomnia or stress related insomnia that last less than 30 days.

iii. Chronic insomnia lasts for longer than a month. It can be caused by another disorder, or it can be a primary disorder. People with high levels of stress hormones or shifts in the levels of cytokines (a protein secreted by lymph cells that affects cellular activity and controls inflammation) are more likely than others to have chronic insomnia. Its effects can vary according to its causes. They might include muscular fatigue, hallucinations, and/or mental fatigue. Chronic insomnia can cause double vision (Roth, & Roehrs, 2003).

Insomnia affects people of all age groups but people in the following groups have a higher chance of acquiring insomnia.

i. Individuals of any age but especially those older than 60
ii. History of mental health disorder including depression, etc.
iii. Emotional stress
iv. Working late night shifts
v. Travelling through different time zones (Golub, 2012).

Young people with insomnia disorder usually complain that it takes too long to get to sleep. Older people are more likely to complain of waking frequently during the night or of waking too early in the morning. Interestingly, many insomnia patients underestimate how much sleep they actually get – thinking they were lying awake when they actually had nodded off (Harvey & Tang, 2012).

2.2.1.2 Causes of Insomnia among students

There are a many common factors associated with the development and maintenance of insomnia such as age, physiology, circadian rhythms, and environment.

Age

Aging is one of the most significant factors associated with changes in sleep across the lifetime (Carskadon & Dement, 2011). Sleep consists of two physiologically distinct states: rapid eye movement (REM) and non-rapid eye movements (NREM) sleep (Carskadon & Dement, 2011). NREM sleep is associated with minimal mental activity and is divided into three stages, with increasing depth of sleep achieved from stages N1 through N3. REM sleep consists of electro-encephalography (EEG) activation, muscle atonia, and rapid eye movements. For most adult sleepers, sleep onset occurs through NREM sleep with REM sleep occurring at least 80 minutes afterwards and NREM and REM sleep typically alternating throughout the rest of the sleeping period. REM cycles become longer over time, with a reduction of stage 3 and 4 sleep across the sleep period. One of the biggest changes in the structure of sleep (also known as 'sleep architecture') is seen in newborn infants. During the first year of life, infants transition from wake to sleep through REM sleep, as opposed to the NREM to REM progression seen in older ages (Carskadon & Dement, 2011). Infants

also begin to develop consolidated nocturnal sleep during their first year of life; with slow wave sleep (also known as deep sleep'), occurring with the greatest frequency in young children and then decreasing with age (Carskadon & Dement, 2011). Other age-related changes in sleep (sometimes beginning as early as young adulthood) include an increasing amount of time spent in the lighter stages (i.e., N1 and N2) of sleep, more time spent awake, and an advancing of the circadian rhythm (i.e., becoming sleepier earlier in the cycle) (Carskadon & Dement, 2011). Sleep architecture changes across the lifespan (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Although there are age-related changes in sleep architecture, the majority of changes in sleep are not due to age but rather are a result of various medical and psychiatric comorbidities that become increasingly prevalent with age (Carskadon & Dement, 2011).

In addition to factors predisposing adults, most especially older adults to poor sleep, there are a number of factors that can precipitate poor sleep such as the onset of an illness, loss of physical functioning, or another primary sleep disorder (e.g., obstructive sleep apnea) (Spielman, Caruso, & Glovinsky, 1987; Vaz Fragoso, & Gill, 2007). Furthermore, once an older adult develops insomnia, there are a number of factors that can perpetuate poor sleep such as social isolation, caregiving, or bereavement. Understanding the changes in sleep that occur with age is important because older adults may have lowered expectations for their sleep and assume that poor sleep is a natural consequence of aging. As a result, it may be up to the clinician and the psychologist to inquire about sleep complaints from older patients. Poor sleep is an important complaint to assess in older adults as it is associated with poorer overall physical and mental health status (Reid, Martinovich, & Finkel, 2006).

Specifically, poor sleep has been implicated in an increased risk for falls, impaired physical functioning, cognitive decline, and memory problems in older adults (St George, Delbaere, Williams, & Lord, 2008; Foley, Ancoli-Israel, Britz, Walsh, 2004). Another consideration for a clinician treating insomnia in older adults is the use of medication. Many medications (e.g., anti-hypertensive, inhaled steroids) used in conditions commonly seen in advanced age such as hypertension and chronic obstructive pulmonary disease are known to cause sleep difficulties in older adults. Furthermore, many sedating medications (e.g., long acting benzodiazepines and muscle relaxants) can cause daytime napping that, in turn, could impair nocturnal sleep. Older adults can work with their physician to adjust the dosage or timing of their mediation use to avoid impaired sleep. Additionally, older adults may sometimes employ alcohol as a sleep aid due to its sedating effects. Although initial consumption of alcohol can have a relaxing effect, it can result in a 'rebound' of insomnia, causing the older adult to wake during the night. Accordingly, greater alcohol consumption has been linked to poor sleep in older adults (Dufour, Archer, &Gordis, 1992; Yao, Yu, Cheng, &Chen, 2008).

Environmental factors

Several environmental factors can contribute to the development and maintenance of insomnia, including noise, light, temperature, and presence of electronic and communication devices in the bedroom. Individuals with insomnia are often more susceptible to sleep interference and disruption related to external environmental stimuli, such as noise and temperature, than patients without insomnia (Jones, Macphee, Broomfield, Jones, & Espie, 2005). Noise and light levels that may not bother other people can have a profound disruptive impact on the sleep of patients with insomnia. For example, external noise from

traffic or insects, as well as indoor noises (e.g., television, radio, a bed partner's snoring, other people in the household, even a squeaky door hinge) can contribute to a restless and sleepless night for patients with insomnia. Interestingly, white noise or other repetitive noise (e.g., sound of a fan) can have a soothing effect and can be conducive to promoting sleep for some patients with insomnia (Richards, Nagel, Markie, Elwell, Barone, 2003, Stanchina, Abu-Hijleh, Chaudhry, Carlisle, & Millman, 2005).

It has been demonstrated that aspects of the bedroom environment, such as the use (or even just the presence) of an electronic device (e.g., television, phone) or a desk where finances and/or other paperwork are done, can lead to the bedroom becoming associated with arousing, non-sleep conducive behaviors (Robertson, Broomfield, & Espie, 2007; Brunborg, Mentzoni, & Molde, 2011). This may be due to classical conditioning, a theory, which helps to explain the importance of 'cues' (i.e., discriminative stimuli) in either promoting or disrupting sleep. For patients with normal sleeping patterns, the bed, bedroom, and bedtime are usually strong cues for sleep; inversely, they become strong cues for wakefulness in patients with insomnia, as they adopt and perpetuate arousal-inducing behaviors (e.g., worrying, watching TV, surfing the internet paying bills) while in the bed or bedroom. Another reason for limiting the use of electronic devices in the bedroom is the light from these devices also contributes to arousal. Another important environmental consideration is how comfortable a patient finds his/her mattress and pillow (e.g., size, firmness). For example, patients with insomnia who have comorbid pain and other medical conditions can be greatly impacted by their mattress and pillow, as both affect sleeping position and

aggravate pain, which can negatively affect sleep (Colbert, Markov, Banerji, & Pilla, 1999; Young, & Jewell, 2002).

Physiology

Insomnia is generally considered a disorder of hyper arousal and the manifestations of this excessive arousal are varied (Riemann, Spiegelhalder, &Feige, 2010). Initially, investigations focused on somatic hyperarousal in patients with insomnia compared to good sleepers. In these investigations, patients with insomnia were found to have elevations in heart rate, body temperature, galvanic skin conductance, and completely body metabolic rate, all suggestive of elevated activity of the sympathetic nervous system (Bonnet, & Arand, 1998; Bonnet, & Arand, 2003). These effects are paralleled by findings that there is elevated activation of the hypothalamic-pituitary-adrenal (HPA) axis in terms of higher levels of cortisol in the blood (Vgontzas, Bixler, & Lin, 2001). These differences seem to be strongest at night and it is as if the body is in a state of 'fight-or-flight,' instead of minimizing arousal in preparation for sleep. For a number of years, an enigma in the sleep research field was the finding that patients with insomnia often did not have evidence of disturbed sleep during overnight sleep studies when compared to good sleepers, despite feeling that they had slept poorly. One theory about this discrepancy between subjective perceptions and the objective evidence was that there is an inherent limitation in the traditional method of conducting sleep studies (Carskadon, Dement, Mitler, Guilleminault, Zarcone, & Spiegel, 1976). This is because sleep study records are visually scored and so determinations of wake and the different sleep stages rely on direct observation.

An alternative approach is to use computer-based spectral analysis measures that decompose the EEG signal into different frequency bands. When this approach was applied to sleep studies of patients with insomnia, it was found that they often display elevated activity in the beta frequency range during sleep (Buysse, Germain, & Hall, 2008; Perlis, Merica, Smith, & Giles, 2001). Beta EEG activity is usually seen while awake and actively engaged in mental processing. It is believed that patients with insomnia can experience a state that is a mixture of waking and sleeping features. They appear to be asleep, but at the cortical level they are continuing to process information; this type of 'sleep' is thus, perceived as wakefulness. This phenomenon has been referred to as cortical hyper-arousal. Another line of research has been the use of neuroimaging methods to study hyper-arousal in insomnia. These studies have been difficult to apply to the study of insomnia, in part due to the loudness of functional magnetic resonance imaging (fMRI) scanners, which often does not allow the patient to undergo scanning while asleep.

A few studies have utilized positron emission tomography (PET) imaging in which infusion of the radioisotope can occur during sleep and then the patient can be awoken for scanning (Nofzinger, Nissen, & Germain, 2006; Smith, Perlis, Chengazi, Soeffing, & McCann, 2005). These studies have found that, for patients with insomnia and without insomnia, there is a decrease in whole brain arousal in the transition from wakefulness to sleep, as is logical. However, for those with insomnia there is less of a reduction compared to those without. Certain brain regions remain more active during sleep and are another indicator of hyper-arousal. One region prone to remaining more active is the reticular activating system, the brainstem region in which most of the major neurotransmitter pathways (e.g. serotonin,

norepinephrine, acetylcholine, histamine, dopamine, neuropeptide A and B) originate. Given that, these neurotransmitters modulate levels of brain arousal; this implies that these systems are maintaining a higher level of arousal in patients with insomnia. It is noteworthy that most sleep medications used to treat insomnia act by way of γ-aminobutyric acid (GABA) mechanisms, as GABA inhibits the activity of the neurotransmitter systems that originate in the reticular activation system. Thus, efficacy of medications used to treat insomnia appears to be due to the ability to reduce brain hyper arousal.

Insomnia, socioeconomic status, and ethnicity

Among adults, the evidence suggesting that income and education pose, as independent risk factors for insomnia are not well supported (Ohayon, 2002). Although the prevalence of insomnia has been found to be higher in individuals with lower incomes, lower socioeconomic status, lower education, among the unemployed, and in individuals working nights or shift work (Katz & McHorney, 1998; Leblanc et al., 2007; Ohayon, 2002), these findings have been inconsistent. Similarly, generalizations also cannot be made for individuals who do not work – although they have been found to be more likely to report having insomnia symptoms, compared to their working counterparts – as this risk appears to be higher among retirees and homemakers (Ohayon, 2002), and retirees and homemakers are variables that are closely related to factors such as age and sex.

Among adolescents, most studies have found no family income effect on the prevalence of DSM-IV-TR insomnia or on the incidence of insomnia over a 12 months' period (Johnson et

al., 2006; Patten et al., 2000; Roberts et al., 2008a). For example, Patten et al. (2000) found that family income did not predict insomnia chronicity over a 4-year period, meaning that trouble sleeping among adolescents suggested little, if any, association with socioeconomic status. Johnson et al. (2006) and Roberts et al. (2008a), however, did find that adolescents whose parents were less educated had higher lifetime prevalence rates of insomnia compared to their peers who had parents that were better educated, and Roberts et al. (2008a) also found lower family income to be a univariate risk factor for insomnia. Although inconclusive, there appears to be a strong reciprocal relationship between psychosocial risk factors and insomnia (Roberts et al., 2008b); this meant that negative psychosocial factors tend to increase an adolescent's risk of having insomnia and, in turn, insomnia, particularly chronic insomnia, is a good predictor of negative psychosocial outcomes. There has also been little research on how ethnicity influences insomnia prevalence. Roberts et al. (2008a) found no ethnic status effects on incidence of insomnia over a 12 month in adolescents, and no significant differences were found in regards to race/ethnicity, parental marital status, or household location between adolescents with insomnia and those with insomnia and a co-morbid psychiatric disorder as well in Johnson et al.'s (2006) study. Patten et al. (2000), however, found large racial/ethnic differences between ethnic groups' reports of sleep problems, with Asians reporting the highest rates of sleep problems and frequent sleep problems and "Blacks" reporting the lowest rate of sleep problems and frequent sleep problems.

Similarly, Katz and McHorney's analysis suggest that "nonwhite" participants were more likely to report insomnia than their "white" participants were. Unfortunately, similar to the evidence on education and income, the influence of race/ethnicity on adolescent insomnia is

inconsistent, and, therefore, inconclusive, which is usually further compounded by the fact that the role of income, socioeconomic status, race/ethnicity, and education in adolescents' insomnia has been little explored.

Circadian rhythms

Circadian rhythms refer to patterning of biological rhythms, including sleep and wake periods, that occur across a 24-hour cycle (Reid, & Zee, 2011). Circadian rhythms work with sleep homeostasis to maintain discrete periods of alertness and sleepiness. Sleep homeostasis refers to the pressure to sleep that accumulates as the duration of wakefulness increases. For example, the homeostatic drive to sleep decreases as the sleep need is met.

Circadian regulation of sleep is not influenced by the amount of preceding sleep, but rather is controlled by an endogenous biological clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus (Glickman, 2010; Berson, Dunn, & Takao, 2002). These rhythms typically run longer than 24 hours but they become synchronized to the 24-hour clock via environmental cues (e.g., the light–dark cycle). Sunlight has the strongest influence, on the timing of sleep and wake. For example, sunlight impacts sleep and wakefulness through its impact on the secretion of melatonin, a hormone, which promotes sleep (Guardiola-Lemaitre, & Quera-Salva, 2011). This is due to sunlight transmitted through the retina along the optic nerve to the SCN in the hypothalamus, which regulates melatonin, body temperature, and other functions that contribute to sleep. Specifically, the pineal gland of the SCN produces melatonin at night (or in darkness), which promotes sleepiness. Thus, melatonin production is indirectly inhibited by sunlight and stimulated by darkness. Circadian rhythms complement the homeostatic process by maintaining alertness

during day and facilitating sleepiness at night. Circadian rhythms are implicated in the development of insomnia when there is a mismatch between an individual's internal circadian rhythm and the timing of their sleep-wake cycle. For example, on Sunday night, after staying up late on Friday and Saturday nights, an individual may not experience the onset of sleepiness until midnight. However, because they have to be at work early on Monday, they may attempt to sleep earlier in the evening (e.g., 10 pm), and as a result, experience sleep-onset insomnia. In some cases, the patient may have a circadian rhythm sleep disorder resulting from a significant misalignment between the internal circadian clock and the physical social environment of the individual that is presenting as insomnia (Reid, & Zee, 2011). This could be caused by a change in the environment outside of the patient's control, such as shift work or jet lag, or simply that the patient's internal circadian clock does not match that of the 'societal norms,' as seen with advanced sleep phase disorder (i.e., circadian rhythm causes significantly earlier sleep and wake times [e.g., 8 pm and 2 am, respectively]) or delayed sleep phase disorder (i.e., delayed sleep onset and wake times [e.g., 2 am and 11 am, respectively]).

Use of psychoactive drugs (such as stimulants), including certain medications, herbs, caffeine, nicotine, cocaine, amphetamines, methylphenidate, aripiprazole, MDMA, modafinil, or excessive alcohol intake and withdrawal from anti-anxiety drugs such as benzodiazepines or pain relievers such as opioids can lead to insomnia (Morin, 2003). Insomnia may also result from heart disease and deviated nasal septum and nocturnal breathing disorders, restless legs syndrome, which can cause sleep onset insomnia due to the discomforting sensations felt and the need to move the legs or other body parts to relieve

these sensations. Pain, (Ramakrishnan & Scheid, 2007) an injury or condition that causes pain can preclude an individual from finding a comfortable position in which to fall asleep, and can in addition cause awakening. Hormone shifts such as those that precede menstruation and those during menopause and life events such as fear, stress, anxiety, emotional or mental tension, work problems, financial stress, birth of a child and bereavement (Mayo Clinic, 2013).Mental disorders such as bipolar disorder, clinical depression, generalized anxiety disorder, posttraumatic stress disorder, schizophrenia, obsessive compulsive disorder, dementia, (Gelder, Mayou & Geddes, 2005) and ADHD (Bendz & Scates, 2009).

Certain neurological disorders, brain lesions, or a history of traumatic brain injury. Medical conditions such as hyperthyroidism and rheumatoid arthritis (Mendelson, 2008). Abuse of over the counter or prescription sleep aids (sedative or depressant drugs) can produce rebound insomnia and poor sleep hygiene, e.g., noise or over-consumption of caffeine.

Sleep studies using polysomnography have suggested that people who have sleep disruption have elevated nighttime levels of circulating cortisol and adrenocorticotropic hormone They also have an elevated metabolic rate, which does not occur in people who do not have insomnia but whose sleep is intentionally disrupted during a sleep study. Studies of brain metabolism using positron emission tomography (PET) scans indicate that people with insomnia have higher metabolic rates by night and by day. The question remains whether these changes are the causes or consequences of long-term insomnia (Mendelson, 2008).

Studies have been conducted with steroid hormones and insomnia. Changes in levels of cortisol, progesterone in the female cycle, or estrogen during menopause are correlated with

increased occurrences of insomnia. Those with differing levels of cortisol often have long-term insomnia, where estrogen is onset insomnia catalyzed by menopause, and progesterone is temporary insomnia within the monthly female cycle. Cortisol is typically the stress hormone in humans, but it is also the awakening hormone. Analyzing saliva samples taken in the morning has shown that patients with insomnia wake up with significantly lower cortisol levels when compared to a control group with regular sleeping patterns. Further studies have revealed that those with lower levels of cortisol upon awakening also have poorer memory consolidation in comparison to those with normal levels of cortisol (Backhaus, Junghanns, Born, Hohaus, Faash, & Hohagen, 2006). Studies support that larger amounts of cortisol released in the evening occurs in primary insomnia. In this case, drugs related to calming mood disorders or anxiety, such as antidepressants, would regulate the cortisol levels and help prevent insomnia.

2.2.1.3 Prevalence of Insomnia among students of higher institution

Although older age groups most frequently report complaints of insomnia, a significant percentage of young people experience insomnia (Lichstein, 2004; Ohayon, 2002). A longitudinal study of over 1,000 randomly selected young adults ages 21-30 found 16.6% reported experiencing insomnia (Breslau et al., 1996). Forty-five percent of these young adults with insomnia continued to report sleep problems at a 3.5-year follow-up. Of the young adults without insomnia at baseline, 13.1% developed insomnia during the course of the study. Studies conducted in Europe have found 4%-14% of young adults between the ages of 19-24 experience insomnia (Ohayon & Roberts, 2001; Ohayon, Roberts, Zulley, Smirne, & Priest, 2000).

A study of American college students found 15% reported having poor sleep quality and 12%-13% reported experiencing difficulty falling asleep, waking during the night, or waking too early in the morning at least three times per week (Buboltz et al., 2001). Studies conducted at the University of North Texas (UNT) found 28.4% of students' report experiencing insomnia at least once a week (Taylor et al., 2007). Out of over 1,000 college students surveyed, 6.8% of students use medication (either prescription or over the counter) to help them sleep and 11.4% use alcohol as a sleep aid (Taylor & Bramoweth, 2010).

Insomnia is particularly problematic in the college student population. When compared to normal sleepers, college students who have trouble sleeping have slower reaction times, lower GPAs, higher levels of daytime sleepiness, fatigue, worry, and a higher risk for traffic accidents (Lindsay, Hanks, Hurley, & Dane, 1999; Means et al., 2000; Trockel, Barnes, & Egget, 2000). Nearly one-third (32%) of college students report having a sleep-related incident while driving, 81.4% of those students momentarily dozed off and 18.6% actually fell asleep at the wheel (Lindsay et al.). As many as 72% of car crashes in which college students died may be attributed to fatigue or sleepiness while driving (Lindsay et al.). The negative impact of poor sleep on driving may be worse in college students than the general population. This age group tends to have a low perception of personal susceptibility to injuries and may be subjected to peer influence or social demand to continue driving despite feeling tired (Lindsay et al. 1999). The combination of sleepiness, feeling invincible, and peer pressure may be dangerous in sober students and the risk of fatal accidents is higher if alcohol or drugs are also involved.

It is important therefore to study the effectiveness of CBT and Relaxation techniquein a university student population because these young adults are different from the general adult population. Since most university students are developmentally transitioning between adolescence and adulthood, they may respond to interventions differently than other adults. During early adulthood, students normally undergo changes in their circadian rhythms, which affect the mechanisms that regulate sleep and wake cycles (Crowley et al., 2007). As students get to higher institution, their drive for sleep decreases (Taylor, Jenni, Acebo, & Carskadon, 2005). These changes in their circadian rhythms result in students having a natural tendency to delay their sleep onset and stay awake later. This phase delay tendency usually starting from adolescence may carry over into the university years as students' transit into adulthood (Valdez, Ramirez, & Garcia, 1996). During adulthood, individuals generally fall asleep earlier as they age (Bliwise, 2005). Most CBT-I studies have been conducted with individuals who have grown out of their early adulthood sleep patterns. The mean ages of participants for most insomnia studies in one large meta-analyses of CBT-I range from 41.7 to 47.2 years (Morin, Culbert,& Schwartz, 1994; Smith,Perlis, Park,& Buysse, 2002).

College students are also a unique subgroup because they tend to be sleepier than the general adult population. Comparisons between students and healthy adults (ages 30-80) indicate students score higher on subjective measures of sleepiness (Johns, 1992) and demonstrate shorter SOL on objective measures of sleepiness (Levine, Roehrs, Zorick,& Roth, 1988). The level of daytime sleepiness observed in college students is greater than the level observed in similarly aged young adults who are not in school (Fukuda & Ishihara, 2001). The high level of sleepiness in this population is worse in college students with insomnia,

who score higher on measures of daytime sleepiness than students without insomnia (Means,Lichstein, Epperson, & Johnson 2000). This finding is unique to college students because in the general population adults with insomnia often report feeling sleepy during the day; however, their scores on objective and subjective measures of sleepiness are not significantly different from normal sleepers (Seidel et al, 1984).

Additionally, many college students have academic, work, and social demands that can result in irregular sleep patterns. In order to meet these demands, many students restrict the amount of time they sleep during the week, then try to make up for this sleep deprivation by sleeping longer on weekends (Breslau et al., 1997; Tsai & Li, 2004). Students who work as well asgo to school tend to get the smallest amounts of sleep during the week and students with early morning classes tend to have the greatest variability between their weekday and weekend sleep schedules (Machado,Varella,& Andrade, 1998). Even though students may be,attempting to make up for lost sleep on weekends, they often delay their weekend bedtimes (Valdez, Ramirez, & Garcia, 1996), possibly due to social activities and the carryover effects of the pubertal circadian shift. Social pressures and college cultures may reinforce these irregular sleep patterns. For instance, many social activities for these young adults start when many older adults are getting ready for bed. It is commonplace for students to sleep late on days in, which they do not have early morning classes. In addition to the variability between weekdays and weekends, college students' sleep patterns may also fluctuate throughout the year as their workloads, study needs increase during midterm, and final exams then drastically change over school vacations period.

CBT-I procedures with the general adult population emphasize the importance of maintaining regular sleep schedules with similar bed and wake times on weekdays and

weekends (Morin &Espie, 2003; Perlis, Jungquist, Smith,& Posner, 2005), and studies have suggested that failure to adhere to the prescribed sleep schedule may interfere with treatment outcomes (Riedel & Lichstein, 2001). It is uncertain what would happen when the intervention is provided to students who are unable to maintain a regular schedule seven days a week for six weeks of therapy and after therapy ends.

Frequently, college students live in dormitories and other group living arrangements, such as community housing or small apartments. Often these types of housing may not provide students with living areas that are separate from their sleeping areas. Thus, their beds become the all-purpose piece of furniture in their room where they study, watch TV, socialize, use their laptop computers, and sleep. According to stimulus control theory, when beds are used for activities other than sleep, the bed loses its ability to be a discriminative stimulus for sleep (Bootzin, 1972; Morin, 2005). This loss of association may be especially prominent in dormitory residents since they could be in their bedrooms throughout their waking hours because their living and study space is their bedroom.

To counter this problem, a primary intervention employed in CBTi is to restrict the use of the bed and bedroom to sleep related activities only. As part of this intervention, participants will be instructed to get out of bed and leave the bedroom if they do not fall asleep quickly (Bootzin, Epstein, & Wood, 1991). College students may have difficulty following this instruction if they do not have another room to go to outside of their bedroom. Although Zwart and Lisman (1979) found insomniacs who read in bed during the night when they had trouble sleeping achieved similar improvements in SOL as participants who got out of bed, it is uncertain what would happen when students who live in dormitories are given stimulus control instructions.

2.2.1.4 Effects of Insomnia on students

Insomnia generally, in adults, refers to sleep difficulty leading to shorter duration than the average basal need of 7 to 8 hours per night. The main symptom of sleep loss is excessive daytime sleepiness, but other symptoms include depressed mood and poor memory or concentration (Dinges, Rogers & Baynard, 2005). Chronic sleep loss, while neither a formal syndrome nor a disorder, has serious consequences for health, performance, and safety. Insomnia is a highly prevalent problem that continues to worsen in frequency, as individuals grow older. Recent studies find that at least 18 percent of adults' report receiving insufficient sleep (Liu et al., 2000; Kapur, Redline, Nieto, Young, Newman, Henderson, 2002; Strine & Chapman, 2005). Historically, there have been a limited number of nationally representative surveys that provide reliable data on sleep patterns in the population.

The National Health Interview Survey (NHIS), run by the Centers for Disease Control and Prevention (CDC) included the following question in the 1977, 1985, 1990 cycles: "On average how many hours of sleep do you get a night (24hour period)?" The same question was added to the core NHIS questionnaire in 2004. Based on these data, it has been estimated that the percentage of men and women who sleep less than 6 hours has increased significantly over the last 20 years as well as the number of people facing difficulty to initiate and sustain sleep (CDC, 2005). Adolescents also frequently report receiving insufficient sleep.

Contrary to public perceptions, adolescents need much sleep as preteens. A large survey of over 3,000 adolescents in Rhode Island found that only 15 percent reported sleeping 8.5 or

more hours on school nights, and 26 percent reported sleeping 6.5 hours or less (Wolfson & Carskadon, 1998). The optimal sleep duration for adolescents, about 9 hours per night, is based on research about alertness, sleep-wake cycles, hormones, and circadian rhythms (Carskadon, 2004). Among adolescents, extensive television viewing and growing social, recreational, and academic demands contribute to sleep loss or sleep problems (Wolfson & Carskadon, 1998). In the past decades or more years, research has overturned the dogma that insomnia has no health effects, apart from daytime sleepiness. Studies suggest that having sleep less than 7 hours per night may have wide-ranging effects on the cardiovascular, endocrine, immune, and nervous systems, including the following:

• Obesity in adults and children

• Diabetes and impaired glucose tolerance

• Cardiovascular disease and hypertension

• Anxiety symptoms

• Depressed mood

• Alcohol use

Many of the studies find graded associations, insofar as the greater the degree of sleep deprivation, the greater the apparent adverse effect.

A. Health consequences

i. Insomnia and Obesity

When a person sleeps less than 7 hours a night there is a dose-response relationship between sleep loss and obesity: the shorter the sleep, the greater the obesity, as typically measured by body mass index (BMI)—weight in kilograms divided by height in meters squared. Although most studies were cross-sectional, one prospective study was a 13-year cohort study of nearly 500 adults. By age 27, individuals with short sleep duration (less than 6 hours) were 7.5 times more likely to have a higher body mass index, after controlling for confounding factors such as family history, levels of physical activity, and demographic factors (Hasler, Buysse, Gamma, Ajdacic, Eich, Rossler & Angst, 2005). Another study, a large population-based study of more than 1,000 adults, found a U-shaped relationship between sleep duration, measured by polysomnography, and BMI. Adults who slept 7.7 hours had the lowest BMI; those with shorter and longer sleep duration had progressively higher BMI. The U-shaped association also applies to other health outcomes, such as heart attacks. The impact of sleep loss diminishes with age. The study also sought to investigate physiological mechanisms behind the relationship between sleep duration and BMI. Measuring two appetite-related hormones, the study found that sleep insufficiency increased appetite. Sleep insufficiency was associated with lower levels of leptin, a hormone produced by an adipose tissue hormone that suppresses appetite, and higher levels of ghrelin, a peptide that stimulates appetite (Taheri Lin, Austin, Young & Mignot, 2004). Another study a small randomized, crossover clinical trial also found that sleep restriction was associated with lower leptin and higher ghrelin levels (Spiegel, Tasali, Penev, Van & Cauter, 2004). The findings suggest that a hormonally mediated increase in appetite may help to explain why short sleep is related to obesity. Several mediating mechanisms have been proposed, including effects of sleep deprivation on the sympathetic nervous system and/or

hypothalamic hormones (Spiegel et al., 2004), which also influence appetite. Taken as a whole, the body of evidence suggests that the serious public health problem of obesity may continue to grow as sleep loss trends continue to worsen. It also suggests that addressing obesity will likely benefit sleep disorders, and treating sleep deprivation and sleep disorders may benefit individuals with obesity (Taheri et al., 2004).

ii. Insomnia and diabetes

Two large epidemiological studies and one experimental study found an association between sleep loss and diabetes, or impaired glucose tolerance. Impaired glucose tolerance, which is a precursor to diabetes, is manifested by glucose levels rising higher than normal and for a longer period after an intravenous dose of glucose. In the Sleep Heart Health Study, which is a community-based cohort, adults (middle-aged and older) who reported 5 hours of sleep or less were 2.5 times more likely to have diabetes, compared with those who slept 7 to 8 hours per night (Gottlieb et al., 2005). Those reporting 6 hours per night were about 1.7 times more likely to have diabetes. Both groups were also more likely to display impaired glucose tolerance. Adults with sleep times of 9 hours or more also showed these effects, a finding consistent with the Nurses' Health Study. Adjustment for waist girth, a measure of obesity, did not alter the significance of the findings, suggesting that the diabetes effect was independent of obesity. The relationship between shorter sleep times and impaired glucose tolerance is also supported by an experimental study in which 11 healthy male volunteers were restricted to 4 hours of sleep forsix nights (Spiegel, Leproult, Van & Cauter, 1999). Even after this relatively short period, the study found that sleep loss, compared with a fully rested state, led to impaired glucose tolerance. The effect resolved after restoring sleep to

normal. Glucose clearance was 40 percent slower with sleep loss than with sleep recovery. Further, mice that have a mutation in a gene that regulates circadian rhythms have metabolic disorders (Turek et al., 2005).

iii. Insomnia and Cardiovascular Morbidity

Sleep loss and sleep complaints are associated with heart attacks (myocardial infarction) and perhaps stroke, according to several large epidemiological studies (Eaker, Pinsky, & Castelli, 1992; Qureshi, Giles, Croft, & Bliwise, 1997; Schwartz et al., 1998; Newman et al., 2000; Ayas, Pittman, MacDonald, & White, 2003; Yaggi et al., 2005; Bradley et al., 2005; Caples, Gami, & Somers, 2005) and one case-control study (Liu et al., 2002). One of these studies, of incident cases of heart attacks in the Nurses' Health Study, was discussed earlier because it also found increased incidence of diabetes (Ayas et al., 2003). The cohort had no coronary heart disease at baseline. Ten years later, in 1996, the likelihood of nonfatal and fatal heart attack was modestly increased for both short and long sleep duration. Five hours of sleep or less was associated with a 45 percent increase in risk (odds ratio [OR] = 1.45, 95% confidence interval [CI], 1.10–1.92), after adjusting for age, BMI, smoking, and snoring. Similarly, elevated risks were also found for sleeping 9 hours or more. The effects were independent of a history of hypertension or diabetes because additional adjustment for these conditions yielded slightly lower, but still significantly elevated, relative risks. Several potential mechanisms could explain the link between sleep loss and cardiovascular events, including blood pressure increases, sympathetic hyperactivity, or impaired glucose tolerance. Experimental data, showing that acute sleep loss (3.6 hours sleep) for one-night results in increased blood pressure in healthy young males, may provide a biological

mechanism for the observed associations between sleep loss and cardiovascular disease (Tochikubo et al., 1996; Meier-Ewert et al., 2004).

iv. Insomnia and Mood, Anxiety, and Alcohol Use

Insomnia is associated with adverse effects on mood and behavior. Adults with chronic sleep loss report excess mental distress, depressive symptoms, anxiety, and alcohol use (Baldwin & Daugherty, 2004; Strine & Chapman, 2005; Hasler et al., 2005). A meta-analysis of 19 original articles found that partial sleep deprivation alters mood largely that it does cognitive or motor functions (Pilcher & Huffcutt, 1996). Several studies of adolescents, including one with more than 3,000 high school students, found that inadequate sleep is associated with higher levels of depressed mood, anxiety, behavior problems, alcohol use (Carskadon, 1990; Morrison et al., 1992; Wolfson & Carskadon, 1998), and attempted suicide (Liu, 2004). Nevertheless, it is not clear from cross-sectional studies whether sleep influences mood or anxiety level, or vice versa. On the other hand, a large, 3-year longitudinal study of more than 2,200 middle school students (ages 11 to 14) found that self-reported sleep loss was associated with more depressive symptoms and lower self-esteem over time (Fredriksen et al., 2004). The study measured sleep loss using a single question about sleep duration on school nights and measured depressive symptoms and self-esteem by the Children's Depressive Inventory and the Self-Esteem Questionnaire, respectively. Therefore, although this study suggests an association, the evidence is still limited.

v. Insomnia and Disease Mortality

Sleep loss is also associated with increased age specific mortality, according to three large, population-based, prospective studies (Kripke et al., 2002; Tamakoshi et al., 2004; Patel et

al., 2004). The studies were of large cohorts, ranging from 83,000 to 1.1 million people. In three studies, respondents were surveyed about their sleep duration, and then they were followed for periods ranging from 6 to 14 years. Deaths in short or long sleepers were compared with those who slept 7 hours (the reference group), after adjusting for numerous health and demographic factors. Sleeping 5 hours or less increased mortality risk, from all causes, by roughly 15 percent. The largest American study found a U-shaped curve, showing that progressively shorter or longer sleep duration is associated with greater mortality. Other epidemiological studies suggest that sleep-loss-related mortality is largely from acute heart attacks (Ayas et al., 2003).

B. Functional consequences of insomnia

The public health consequences of insomnia and other sleep disorders are far from benign. Some of the most devastating human and environmental health disasters have been partially attributed to sleep loss due to insomnia and night shift work-related performance failures, including the tragedy at the Bhopal, India, chemical plant; the nuclear reactor meltdowns at Three Mile Island and Chernobyl; as well as the grounding of the Star Princess cruise ship and the Exxon Valdez oil tanker (NCSDS, 1994; NTSB, 1997; Moss & Sills, 1981; United States Senate Committee on Energy and National Resources, 1986; USNRC, 1987; Dinges, Graeber, Carskadon, Czeisler, & Dement, 1989). Each of these incidents not only cost millions of dollars to clean up, but also had a significant impact on the environment and the health of local communities. Less visible consequences of sleep conditions take a toll on nearly every key indicator of public health: mortality, morbidity, performance, accidents and injuries, functioning and quality of life, family well-being, and health care utilization. The

following is an overview of the consequences of sleep loss and sleep disorders on an individual's performance, safety, and quality of life. Nearly all types of sleep problems are associated with performance deficits in occupational, educational, and other settings. The deficits include attention, vigilance, and other measures of cognition, including memory and complex decision making.

i. Insomnia and Cognitive Performance

Sleep loss had been largely dismissed as the cause of poor cognitive performance by early, yet poorly designed, research. The prevailing view until the 1990s was that people adapted to chronic sleep loss without adverse cognitive effects (Dinges et al., 2005). Research that is more recent has revealed sleep loss-induced neurobehavioral effects, which often go unrecognized by the affected individuals. The neurobehavioral impact extends from simple measures of cognition (i.e., attention and reaction time) to far more complex errors in judgment and decision making, such as medical errors. Performance effects of sleep loss include the following:

• Involuntary micro sleeps occur.

• Attention to intensive performance is unstable, with increased errors of omission and commission.

• Cognitive slowing occurs in subject-paced tasks, while time pressure increases cognitive errors.

• Response time slows.

• Performance declines in short-term recall of working memory.

• Performance requiring divergent thinking deteriorates.

• Learning (acquisition) of cognitive tasks is reduced.

• An increase in response suppression errors in tasks requiring normal primarily prefrontal cortex function.

• The likelihood of response preservation on ineffective solutions is increased.

• Compensatory efforts to remain behaviorally effective are increased.

• Although tasks may be done well, performance deteriorates as tasks duration increases (Durmer & Dinges, 2005).

Attention and reaction time are altered by experimental sleep loss, which leads to cumulative dose-dependent, deterioration of attention and reaction time. Deterioration is measured in part using the psychomotor vigilance task (PVT), a test that requires continuous attention to detect randomly occurring stimuli and that is impervious to aptitude and learning effects. In one study, 48 healthy subjects were randomized to 4, 6, or 8 hours' time in bed for 14 days (Van Dongen et al., 2003). Investigators found a dose-dependent effect, which increased over time. Performance deficits in individuals who slept 6 hours or less per night were similar to those observed in individuals after two nights of total sleep deprivation. Most striking was that study subjects remained largely unaware of their performance deficits, as measured by subjective sleepiness ratings. A second study (Belenky et al., 2003) showed a similar dose-dependent, cumulative effect over 7 days of sleep loss in 66 healthy volunteers. Subjects were followed for 3 days after the period of sleep restriction, during

which time they recovered, but not enough to return to their baseline levels. Imaging studies have demonstrated a physiological basis for cognitive impairments with sleep loss that has been linked with metabolic declines in the frontal lobe of the brain (Thomas et al., 2000). Although there is not a large body of evidence, associations are also likely between sleep loss and increased risk taking (Roehrs, Greenwald, & Roth, 2004).

ii. Insomnia and Academic Performance

Sleep loss due to insomnia in adolescence is common and grows progressively worse over the course of adolescence, according to studies from numerous countries (Wolfson & Carskadon, 2003; Howell et al., 2004). Average sleep duration diminishes by 40 to 50 minutes from ages 13 to 19. Despite the physiological need for about 9 hours of sleep, sleep duration, across this age span, averages around 7 hours and about a quarter of high school and college students are sleep deprived (Wolfson & Carskadon, 1998). Research indicates that patterns of shortened sleep occur in the preadolescent period, and may be most marked in African American boys, compared to white children or African American girls (Spilsbury et al., 2004). The decline in adolescent sleep duration is attributed to psychological and social changes, including growing desire for autonomy, increased academic demands, and growing social and recreational opportunities, all of which take place in spite of no change in rise time for school (Wolfson & Carskadon, 1998). Furthermore, the need to earn income adds to the burden. Students who worked 20 or more hours weekly, compared with those who worked less than 20 hours, were found to go to bed later, sleep fewer hours, oversleep, and fall asleep more in class (Millman, 2005). Sleep loss affects alertness, attention, and other cognitive functions in adolescents (Randazzo,Muehlbach, Schweitzer,& Walsh, 1998),

but demonstrating a causal relationship between sleep loss and academic performance has been difficult. Most studies attempting to link the two are cross-sectional in design, based on self-reporting of grades and sleep times, and lack a control for potential confounders (Wolfson & Carskadon, 2003).

An association between short sleep duration and lower academic performance has been demonstrated (Wolfson & Carskadon, 1998; Drake, Roehrs& Roth, 2003; Shin, Kim, Lee, Ahn, & Joo, 2003), but the question of causality has not been resolved by longitudinal studies. A 3-year study of 2,200 middle school students did not find that sleep loss resulted in lower academic performance. It only found a cross-sectional association at the beginning of the study. However, by the end of the study, as sleep time worsened, grades did not proportionately decrease (Fredriksen et al., 2004). A study of the Minneapolis School District, which delayed start times for its high schools by almost 1.5 hours (from 7:15 a.m. to 8:40 a.m.), found significant improvements in sleep time, attendance, and fewer symptoms of depressed mood (Wahlstrom et al., 2001). Further, there was a trend toward better grades, but not of statistical significance. The study compared grades over the 3 years prior to the change with grades 3 years afterwards. Much of the difficulty in studying sleep loss and its relation to academic performance stems from multiple, often unmeasured, environmental factors that affect sleep (such as school demands, student employment after school, family influences, TV viewing, and Internet access). These are set against the rapid developmental and physiological changes occurring in adolescence. Another difficulty is the challenge of objectively assessing school performance (Wolfson & Carskadon, 2003).

iii. Insomnia and motor vehicle crashes

Sleepiness is a significant, and possibly growing, contributor to serious motor vehicle injuries. Almost 20 percent of all serious car crash injuries in the general population are associated with driver sleepiness, independent of alcohol effects (Connor et al., 2002). Driver sleepiness is most frequently a manifestation of sleep loss, as discussed below, but other sleep disorders, which have lower prevalence, contribute to the problem, including sleep disordered breathing, restless legs syndrome, and narcolepsy. The 20 percent figure, cited above, is the population-attributable risk, which is a key public health measure indicating what percentage of car crash injuries, including fatal injuries of passengers, could be avoided by eliminating driver sleepiness. The finding was based on a population-based case-control study in a region of New Zealand in which 571 car drivers and a matched control sample were asked detailed questions about measures of acute sleepiness while driving (Connor et al., 2002). The study adjusted for potential confounding factors, including alcohol. Crashes examined in this study involved a hospitalization or death. The greatest risk factor for the crashes was sleep loss and time of day (driving between 2:00 a.m. to 5:00 a.m.), but sleep apnea symptoms were not risk factors. Indications are that the public health burden of sleepiness-related injuries is likely increasing, given recent trends in drowsy driving. The National Sleep Foundation found that self-reported drowsy driving has increased significantly over the past years, from 51 percent of respondents in 2001 to 60 percent in 2005 (NSF, 2005). Similarly striking was that more than 10 percent of the entire sample reported nodding off or falling asleep while driving at least 1 to 2 days per month. The impact of driver sleepiness is similar in magnitude to that of alcohol consumption. A study of all crashes between 1990 to 1992 reported to North Carolina's uniform reporting system found that fall-asleep crashes (ones in which a law officer determines the driver to be

asleep or fatigued) and alcohol-related crashes were similar in terms of serious injuries (13.5 and 17.8 percent of crashes from all causes, respectively) and fatalities (1.4 and 2.1 percent of all fatalities, respectively) (Pack et al., 1995).

In actual driving performance on a closed course, sleep-deprived adults performed as poorly as did alcohol-challenged adults. After a night of total sleep deprivation, impairments in lane-keeping ability were similar to those found with blood alcohol content of 0.07 percent (Fairclough & Graham, 1999). Fall-asleep crashes have distinct patterns by type, age, and time of day. According to the North Carolina study, fall-asleep crashes are largely off the road and at higher speeds (in excess of 50 mph) (Pack et al., 1995). Adolescents and young adults between the ages of 16 and 29 are the most likely to be involved in crashes caused by the driver falling asleep (Horne and Reyner, 1995; Pack et al., 1995). They account for about 50 percent of all crashes (Horne and Reyner, 1995; Pack et al., 1995). Fall-asleep crashes occur at two periods of day that coincide with circadian variation in sleepiness, in the early morning (2:00 a.m. to 8:00 a.m.) (Pack et al., 1995; Connor et al., 2002) and during the midafternoon (Horne &Reyner, 1995; Pack et al., 1995; Carskadon, 2004). The most common reasons behind fall-asleep crashes are working multiple jobs, night shift work, and sleep duration of less than 5 hours (Connor et al., 2002; Stutts, Wilkins, Scott & Vaughn, 2003). Sleep apnea accounts for a small, but measurable percentage of motor vehicle crashes, primarily in drivers above the age of 25 (Sassani et al., 2004). Individuals with sleep apnea are at twice the risk of having a traffic accident as unaffected individuals (Teran-Santos, Jimenez-Gomez, & Cordero-Guevara, 1999), the higher the apnea-hypopnea index, the higher the risk (Young &Jewell, 2002).

Sleepy drivers tend to display reduced vigilance, slow reaction times, and loss of steering control. Steering impairment in sleep deprivation, and alcohol intoxication was compared in a controlled clinical trial. Untreated OSA and sleep deprivation were similar in producing progressive steering deterioration throughout the drive, whereas alcohol-impaired individuals steered equally throughout the drive (Hack et al., 2001). Occupational groups at high risk of sleep-related crashes are night shift workers (Horne and Reyner, 1995; Ohayon et al., 2002; Drake, Roehrs, & Roth 2004), medical residents and house staff (Marcus & Loughlin, 1996; Barger et al., 2005), and commercial truck drivers (Walsh et al., 2005). Commercial truck drivers have attracted the most study because of the prevalence, severity, and public health impact of crashes involving commercial trucks. There are an estimated 110,000 injuries and 5,000 fatalities each year in motor vehicle accidents involving commercial trucks (CNTS, 1996). The National Transportation Safety Board (NTSB) determined that fatigue (including sleepiness) was the probable cause of 57 percent of crashes leading to a truck driver's death (NTSB, 1990). Although this figure is not universally accepted, the definition of fatigue by the NTSB is equivalent to the term sleepiness or sleep-related fatigue used by sleep experts (i.e., fatigue that results in human performance failure) (Walsh et al., 2005). For each truck driver fatality, another three to four people are killed (NHTSA, 1994).

iv. Insomnia and other injuries

Sleep related fatigue is an independent risk factor in work related injuries and fatalities, according to two large and well-designed studies (Akerstedt,Fredlund, Gillberg, & Jansson, 2002; Swaen et al., 2003). Swaen and coworkers prospectively studied a cohort of more than

7,000 workers in numerous industries in the Netherlands over a 1-year period before studying the occurrence of occupational accidents. During the year, they collected information about sleep patterns and other potential risk factors for work-related injuries. The 108 employees who reported being injured during the next year could be assessed for risk factors without recall bias affecting the results. The study found a dose-response relationship between two sleep-related fatigue measures and injuries. For example, highly fatigued workers were 70 percent more likely to be involved in accidents than were workers reporting low fatigue levels, after adjustment for other risk factors. Workers with chronic insomnia were also far more likely than those who were good sleepers to report industrial accidents or injuries (Leger,Guilleminault, Bader, Levy & Paillard, 2002). Finally, disturbed sleep plays a role in occupational fatalities. In a large 20-year prospective study in Sweden of nearly 50,000 individuals, those reporting disturbed sleep were nearly twice as likely to die in a work-related accident (OR = 1.89, 95% CI 1.22–2.94) (Akerstedt et al., 2002). Similarly, workers who report snoring and excessive daytime sleepiness, indications of sleep apnea, are twice as likely to be involved in workplace accidents, as verified by registry data and after adjusting for all potential confounders (Lindberg, Carter, Gislason, & Janson, 2001).

C. Insomnia and quality of life

Sleep problems, difficulty initiating and maintaining sleep, non-restorative sleep, and excessive daytime sleepiness are associated with adverse effects on well-being, functioning, and quality of life, according to numerous studies covering the general population (Baldwin et al., 2001, 2004; Hasler et al., 2005; Strine & Chapman, 2005), working people

(Kuppermann et al., 1995), and clinical populations (Simon & VonKorff, 1997), including pediatric samples (Rosen, Palermo, Larkin,& Redline, 2002). Studies have used various measures of quality of life and functional status, the most common of which is a validated questionnaire known as the SF-36, a 36-item measure that asks about eight domains: (1) physical functioning; (2) role limitation due to physical health problems (role physical); (3) bodily pain; (4) general health perceptions; (5) vitality; (6) social functioning; (7) role limitations due to emotional health problems (role emotional); and (8) mental health. A similar measure is the health-related quality of life survey, which asks fewer questions. Individuals who suffer from primarily sleep apnea, narcolepsy, restless legs, primary parasomnias, and insomnia constantly report poorer quality of life compared to population norms (Reimer & Flemons, 2003).

Using health-related measures of quality of life, the functional impact of sleep loss was assessed by a large and nationally representative survey, the United States Behavioral Risk Factor Surveillance System (Strine & Chapman, 2005). The study focused on nocturnal sleep time in nearly 80,000 respondents. About 26 percent of the respondents reported obtaining insufficient sleep on a frequent basis (not enough sleep on 14 days or more over the past 30 days). This group was significantly more likely than those without frequent sleep insufficiency to report poorer functioning and quality of life on each of the eight items of the health-related quality of life. Several studies have dealt with insomnia and its adverse impact on quality of life (Zammit, Weiner, Damato, Sillup,& McMillan, 1999; Leger et al., 2002; Katz & McHorney, 2002).

People with severe insomnia reported lower quality of life on all eight domains of the SF-36 (Leger et al., 2002). Their low quality of life ratings was similar to ratings by patients with congestive heart failure and depression, according to a study of nearly 3,500 primary care patients (Katz and McHorney, 2002). About 16 percent of the sample had severe insomnia, and the study adjusted for numerous factors including health habits, obesity, other chronic conditions, and severity of disease. A study of a large health maintenance organization population (n = 2,000) found that insomnia (versus no current insomnia) was associated with significantly greater impairment, as measured by the self-rated Social Disability Schedule and the interviewer rated Brief Disability Questionnaire. Individuals with insomnia also had more days of restricted activity due to illness and more days spent in bed (Simon & VonKorff, 1997). One study revealed a dose-response relationship, with higher levels of insomnia being associated with greater impairments in the ability to accomplish daily tasks and decreased enjoyment of interpersonal relationships (Roth & AncoliIsrael, 1999).

Approximately a quarter of children and adolescents report difficulty with sleep (Stein,Mendelsohn, Obermeyer, Amromin, & Benca, 2001; Archbold, Pituch, Panahi, & Chervin, 2002). However, very few studies have assessed the association between sleep loss and sleep disorders and health-related quality of life in children. Analysis of a widely used parent report measure of children is physical, emotional, and social functional status and wellbeing the CHQ-PF50 found the quality of life of their children deteriorated with the severity of OSA (Rosen et al., 2002). This is consistent with a negative association between sleep difficulties and health related quality of life that was observed a similar analysis of 80 parents of children referred to a pediatric sleep disorders clinic (Hart et al., 2005). Thus,

sleep difficulties may broadly affect a child's development through its impact on children's social, emotional, and physical functioning.

D. Economic impact of insomnia

Although problems falling asleep or daytime sleepiness affect 35 to 40 percent of the population (Hossain & Shapiro, 2002), the full economic impact of sleep loss and sleep disorders on individuals and society is not known. There are limited data on the economic impact of insomnia, sleep disordered breathing, and narcolepsy; the economic impact of other sleep disorders has not been analyzed. Individuals suffering from insomnia place an increased burden on the health care system (Ohayon & Roth, 2003). Their activity is more limited (Simon and VonKorff, 1997), and they are significantly more likely to access medical and psychiatric care than are individuals that do not have a sleep or psychiatric disorder (Weissman et al., 1997). Individuals with insomnia who also have an associated psychiatric disorder are more likely to seek treatment for emotional problems (14.9 percent versus 8 percent) (Weissman et al., 1997), have a greater number of physician visits, and be admitted to a hospital twice as often (Leger et al., 2002). The burden insomnia place on the health care system is long-term—the majority of individuals with either mild (59 percent) or severe (83 percent) insomnia continue to suffer symptoms of insomnia 2 years after initial diagnosis (Katz & McHorney, 1998).

Consequently, individuals suffering from insomnia place a significant economic burden on society resulting in increased health care costs. Billions of dollars are spent each year in the United States on the direct costs of sleep loss and sleep disorders. These medical costs include expenses associated with doctor visits, hospital services, prescriptions and over the

counter medications. In 1995, the direct cost of insomnia in the United States was estimated to be $13.9 billion (Walsh & Engelhardt, 1999). Further, based on the costs associated with a laboratory-based polysomnogram, it would cost over $17.5 billion to test and $3 billion to treat every person in the United States who has sleep apnea (Sassani et al., 2004). A 1988 study estimated that productivity loss resulting from insomnia cost $41.1 billion (Stoller, 1994). Absenteeism cost more than $57 billion (Walsh, 2004). Therefore, once the costs of industrial and motor vehicle collisions and related morbidities are included, the indirect cost of insomnia could top $100 billion. Two factors contribute to these higher costs associated with insomnia.

First, the general population is typically reluctant to consult doctors about their sleep problems, and second, inadequate physician training prevents proper recognition, diagnosis, and treatment of patients with insomnia (Walsh & Engelhardt, 1999; Benca, 2005). Although it is predicted that the advent of more effective portable monitoring devices will decrease the costs associated with testing and diagnosis of sleep disorders, the total direct costs will still remain high and be a burden. The indirect costs associated with sleep loss and sleep disorders also result in billions of dollars of annual expenditures, including costs associated with illness related morbidity and mortality, absenteeism, disability, reduction or loss of productivity, industrial and motor vehicle accidents, hospitalization, and increased alcohol consumption (Hossain & Shapiro, 2002). As is the case with direct costs, for each of these categories further analysis is required to determine the complete indirect costs of sleep loss and sleep disorders. The annual economic impact of sleep problems relating to workers'

inability to adjust to late shifts are estimated to be at minimum over $60 billion (Moore-Ede, 1993).

In addition, it has been estimated that sleep-related fatigue costs businesses $150 billion a year in absenteeism, workplace accidents, and other lost productivity (Sleep Disorders Create Growing Opportunities for Hospitals, 2001). A 1994 analysis of automobile accidents estimated the cost of accidents attributed to sleepiness to be $29.2 to $37.9 billion (Leger, et al.2002). Over 50 percent of automobile crashes involving a truck, where a fatality occurred, were caused by sleep-related fatigue, costing approximately $2.7 million and 4,800 lives (NTSB, 1990; Mitler et al., 2000). However, there is no standardized mechanism to record fatigue- and sleep related accidents; therefore, these figures are likely underestimates of the total cost of automobile accidents. Although the complete economic impact of sleep disorders and sleep loss is limited, the available data demonstrates the high burden that inadequate sleep has on the economy. With the average age of the population rising, incidence of sleep disorders is likely to rise, leading to increased costs (Phillips, 2005). Two factors contribute to these higher costs associated with insomnia. First, the general population is typically reluctant to consult doctors about their sleep problems, and second, inadequate physician training prevents proper recognition, diagnosis, and treatment of patients with insomnia (Walsh & Engelhardt, 1999; Benca, 2005; NIH, 2005).

2.2.2 Concept of cognitive behaviour technique for insomnia

Cognitive-behavioral therapy for Insomnia (CBT-I) is a safe 4 –8 weeks' effective treatment for insomnia that may be used to augment hypnotic drugs or as a monotherapy. Cognitive-behavioral management of insomnia includes 3 components behavioral, cognitive, and

educational modules— and is usually presented in a group or individual therapy setting. Traditionally, CBT-I and/or CBT for other sleep disorders have been developed, and provided by psychologists (Perlis et al., 2005). The evidence supporting this behavioral approach shows that CBT is effective for 70% to 90% of patients and that it can significantly reduce several measures of insomnia, including sleep-onset latency and wake-after-sleep onset. Aside from the clinically measurable changes, this therapy system enables many patients to regain a feeling of control over their sleep, thereby reducing the emotional distress that sleep disturbances cause (Morin, 2004). The most common cognitive behavioral therapies for insomnia are stimulus control, sleep restriction, sleep hygiene, relaxation training, and cognitive therapy. Typically, therapy includes three or more of the above components (Perlis et al., 2005).

Stimulus Control Technique (SCT)

This treatment modality is recommended for both sleep initiation and maintenance problems (Bootzin 1972; Bootzin, Epstein, & Ward, 1991). The therapy is considered, according to the American Academy of Sleep Medicine (Chesson, Anderson & Littner, 1999), to be thefirst-line behavioral treatment for chronic insomnia. This is the case because this intervention has been assessed extensively as a monotherapy and has been found to reliably produce good clinical results. Proposed by Bootzin (1972, 1977), stimulus control therapy (SCT) was the first nonpharmacological treatment developed specifically for insomnia. SCT has become the gold standard against which new interventions are tested. SCT is based on an operant paradigm within which the bed and bedroom no longer function as discriminative stimuli for sleep (Blampied & Bootzin 2011, Bootzin, Smith, Franzen, & Shapiro 2010). For the person with insomnia, the bed and bedroom become associated with behaviors that are

incompatible with sleep such as watching television, eating, reviewing the day's events, planning, worrying, lying awake, and becoming anxious and frustrated from trying to fall asleep or fall back to sleep. There is also a Pavlovian conditioning component in the development of insomnia in that the bed and bedroom become conditioned stimuli for stress and frustration associated with being unable to fall asleep. The aims of SCT are to strengthen the bed and bedroom as cues for sleep, to weaken them as cues for behaviors that are incompatible with sleep, and to develop a consistent sleep-wake pattern (Bootzin 1972, 1977).

The bed should be strictly reserve for sleepand this will help to establish new sleep habits. Staying out of the bed and bedroom when unable to sleep decreases sleep anticipatory anxiety, dysfunctional sleep-related cognitions, and arousal (Morin & Epsie 2003). Getting out of bed during the night if unable to fall asleep is likely to increase the patient's sleep debt and homeostatic sleep drive. Following stimulus control instructionsmakes it more likely that patients will fall asleep quickly and stay asleep, thereby strengthening the bed and bedroom as discriminative stimuli for sleep. Getting up at the same time each day affects the circadian clock by helping to develop a regular sleep-wake schedule. Detailed rationales for each of the stimulus control instructions are provided to patients (Bootzin & Epstein, 2000, Bootzin et al. 2010). Meta-analyses and systematic reviews (Morin et al. 2006) indicate that SCT is one of the most effective, if not the most effective, single-component intervention for insomnia. Although SCT has received less testing as a single intervention in the past 10 years (Pallesen, Nordhus, Kvale, Nielsen, & Havik2003, Reidel, Lichstein, Peterson, Means, & Epperson 1998), its inclusion in multicomponent interventions has increased (Lichstein,

Wilson, & Johnson, 2000; Morin et al. 2004; Rybarczyk, Lopez, Benson, Alsten, & Stepanski, 2002).

Sleep Restriction Technique

Sleep restriction therapy (SRT) is a behavioral treatment for insomnia developed by Spielman, Caruso and Glovinsky (1987). The theoretical underpinnings of SRT emphasize that individuals with insomnia spend too much time in bed attempting to sleep, which leads to increased wakefulness, fragmented sleep, and variability in the timing of sleep and wake. The aims of SRT are the consolidation of sleep and the establishment of a consistent sleep-wake schedule by limiting time spent in bed. An individualized sleep-wake schedule is developed to limit the patient's amount of time in bed to the estimated mean time spent asleep. The nightly mean total sleep time is typically determined through weeks of data collected through sleep diaries. The therapist and the patient agree upon a wake time, which is followed throughout treatment. A bedtime is then established that gives the patient an amount of time in bed equivalent to the baseline mean total sleep time. For instance, if the mean total sleep time is 5.5 hours, and the patient agrees to awaken each day at 5:30 AM, then a bedtime is set for about 11:30 or midnight for the first week of treatment (Spielman, Caruso & Glovinsky, 1987).During each week of treatment, the bedtime is advanced about 15 to 30 minutes based on the previous week's sleep efficiencycalculated as TST/TIB x 100.

Table 2.1 Prescribing adjustments to time in bed based on sleep efficiency

Sleep efficiency	Adjustment to bed time
<85%	15 minutes later
85%–89%	No change
90%–94%	15 minutes earlier
=95%	30 minutes earlier

After the patient has restricted his/her time in bed to his/her initial sleep window for 1 week, the bedtime is adjusted based on the sleep efficiency attained. Source: (Perlis, Jungquist, Smith, & Posner 2005).

The prescribed time in bed is probably less than is actually needed since persons with insomnia (Wohlgemuth & Edinger 2000) often underestimate total time slept. As a result, some partial sleep deprivation is induced, the homeostatic sleep drive is increased, and sleep consolidation occurs on subsequent nights. Patients develop a regular sleep-wake rhythm through consistent scheduling. Patients are not prescribed less than five hours of time in bed. Length of treatment is six weeks to obtain improvement, although eight weeks have also been recommended (Spielman & Glovinsky 1991). Further details on the use of SRT are provided in Epstein & Bootzin (2002), Spielman and colleagues (2010), and Wohlgemuth and Edinger (2000). SRT has been used as a single treatment (Friedman et al. 2000) and included in multicomponent interventions (Edinger et al. 2001, Espie et al. 2001). Sleep compression is a modification of SRT that gradually reduces time in bed over the treatment period rather than the immediate reduction approach used in SRT. This gentler variation has been used successfully with older adults (Lichstein et al. 2001, Reidel et al. 1995).

Sleep Hygiene and Education

Sleep hygiene and education (SHE) is composed of both information about sleep and a group of lifestyle recommendations under the label of sleep hygiene. Sleep education consists of knowledge about sleep processes and function, developmental changes in sleep, sleep homeostasis, circadian rhythms, and individual sleep needs (Bootzin et al. 1996).

Sleep hygiene consists of rules to improve sleep, for example, put the bedroom clock where you cannot see it and avoid coffee, nicotine, and alcohol (Hauri 1991). There is a lack of agreement among experts regarding a definition of sleep hygiene (Stepanski & Wyatt 2003), and the term is often used incorrectly by healthcare providers to referto SCT (Edinger, Wohlgemuth, Radtke, Coffman, & Carney, 2007).Stepanski and Wyatt were unable to findany studies that used the same sleep hygiene instructions. Although a core set of instructions was identified, the specific recommendations differed across studies. SHE, particularly the education component, is a fundamental part of insomnia intervention (Morin et al. 1999) that can lay the basis for patients' understanding of theory-based interventions such as SCT and SRT. As such, it is usually delivered at the beginning of the CBT intervention period. Although SHE has limited efficacy as a single-component intervention (Morin et al. 1999), it is included in the recent surge of multicomponent intervention studies (Edinger & Sampson 2003, Espie et al. 2007, Morin, et al. 2004, Reidel 2000).

Cognitive technique (CT) for maladaptive belief/intrusive thoughts

While maladaptive behaviors perpetuate insomnia, faulty beliefs and unrealistic expectations about sleep and insomnia bolster these behaviors. Cognitive therapy targets these erroneous beliefs and attempts to alter them (Morin, Blais, & Savard 2002). First, the dysfunctional cognition is identified, which reveals the faulty underlying belief. Then, the clinician offers alternative interpretations, and the patient can begin to think about his or her insomnia in a different way. There are 5 main targets of cognitive therapy.

1.Misconceptions about causes of insomnia. Many patients attribute their insomnia to specific causes or precipitating factors, such as pain, allergies, age, or depression. The patient then believes that these conditions must be resolved before the insomnia will abate.

Although such factors are often involved in sleep disturbances, attribution of insomnia to those factors alone is self-defeating because the patient may indeed have little control over them. Cognitive therapy shows the patient that chronic insomnia always involves behavioral and psychological factors over which the patient can exercise some control. By understanding that insomnia is not caused solely by external causes, the patient may then learn how to overcome or adapt to it.

2. Misattribution and amplification of consequences. While some patients may experience real daytime consequences of insomnia, the fear of these consequences and concerns about the health effects of sleep loss magnify the subjective report of these consequences and amplify the calamitous nature of insomnia. Tiredness, irritability, and inefficiency may be subjectively attributed to a poor night's sleep(Zammit 1988) but objective measurements of daytime sleepiness and lack of vigilance in patients with insomnia are quite limited (Lichstein, Wilson, & Noe 1994, Stepanski, Zorick & Roehrs 1988). Reports of adverse effects are higher among worry-prone individuals (Coyle & Watts 1991). In fact, people with subjective insomnia exhibit more daytime sequelaeof adverse effects than those with psychophysiological insomnia, who have objective evidence of disturbed sleep (Stepanski, Zorick & Roehrs 1988).The worry-prone personality of many patients with subjective insomnia may be the cause of this phenomenon, or it may confirm the tendency of psychophysiologicalinsomnia patients toward physiologic hyperarousal, during both the night and the day.

A rational look at the objective consequences of sleep loss can help patients reattribute the presumed consequences of sleep loss and see that worry about insomnia may be more detrimental to their health than the actual sleep loss.

3. Unrealistic sleep expectations. Everyone sleeps differently. People with insomnia may become overly focused on achieving some "normal" sleep duration, onset, or quality by comparing their sleep patterns with those of friends or bed partners. While 8 hours may be the average sleep time for most adults, the self-imposed pressure of reaching this sleep duration may worsen sleep difficulties or even cause people without definable sleep disturbances to believe they have insomnia. In these cases, baseline clinical assessment of sleep parameters will (Stepanski, Zorick & Roehrs 1988)reveal the severity and significance of the sleep disturbance and determine whether it is pathologic (sleep-onset latency or wake time after sleep onset > 30 minutes; total sleep time < 6.5 hours) (Morin 1993, Morin & Espie 2003)**,** or within normative ranges. For those patients with unrealistic expectations, cognitive therapy helps them change their expectations about sleep and reappraise the clinical importance of their sleep difficulties.

4. Performance anxiety and learned helplessness. For many people, insomnia constitutes a lack of control. The variability of sleep patterns from night to night only magnifies the representation of sleep as unpredictable and out of control. As the feeling of powerlessness increases, patients place more pressure on themselves to fall asleep, creating a sense of performance anxiety that can make matters worse. Cognitive strategies for this situation may include paradoxical recommendations (e.g., "Try not to fall asleep"), which increase the patient's perception of control. When insomnia is unpredictable and seemingly, without cause, patients may fall into a state of learned helplessness, believing that hypnotics are the only source of predictability or that there will never be any control or predictability. Then, patients begin to position themselves as victims and catastrophize the power, effect, and duration of insomnia.

5. Faulty beliefs about sleep-promoting practices. Preconceived ideas about solutions for insomnia may only worsen the case. The counterproductive tactics most commonly practiced by people who are unable to fall asleep are to stay in bed and try harder or to sleep in later in the morning to compensate for poor sleep. Most erroneous practices are altered through the behavioral component of therapy, but patients also need to cognitively restructure the way they think about sleep promotion. In general, cognitive therapy for insomnia helps to change the underlying ideas that perpetuate insomnia. People with insomnia should learn 6 basic cognitive strategies:

(1) keep realistic expectations; (2) do not blame insomnia for all impairments; (3) never try to sleep; (4) do not give too much importance to sleep; (5) do not catastrophize after a poor night's sleep; and (6) develop tolerance to the effects of insomnia.

2.2.3 Concept of relaxation technique

Relaxation technique (also known as relaxation training) is any method, process, procedure, or activity that helps a person to relax, to attain a state of increased calmness, or otherwise reduce levels of pain, anxiety, stress or anger. Relaxation in psychology is the emotional state of a living being, of low tension in, which there is an absence of arousal that could come from sources such as anger, anxiety, or fear. Relaxation techniques are often employed as one element of a wider stress management program and can decrease muscle tension, lower the blood pressure and slow heart and breathe rates, among other health benefits (Goleman, 1986).

People respond to stress in different ways, namely, by becoming overwhelmed, depressed or both. Some relaxation may include *deep breathing,* which tend to calm people who are

overwhelmed by stress, while rhythmic exercise improves the mental and physical health of those who are depressed. People who encounter both symptoms simultaneously, feeling depressed in some ways and overexcited in others, may do best by walking or performing yoga techniques that are focused on strength (Robinson, Segal, Segal, & Smith, 2011). Research has indicated that removing stress helps to increase a person's health (Smith, 2007). People use relaxation techniques for the following reasons, among others:Anger management, Anxiety attack, Immune system support, Pain management, Depression, Insomnia, Cardiac health, Addiction treatment, Childbirth, Headache, and Nightmare disorder among others (Smith, 2007).

People to improve their state of relaxation use different techniques. Some of the methods are performed alone; some require the help of another person. Some involve movement, some focus on stillness, while other methods involve different elements. Certain relaxation techniques known as "formal and passive relaxation exercises" are generally performed while sitting or lying quietly, with minimal movement and involve "a degree of withdrawal" (Smith, 2007). These include autogenic training, Biofeedback, Deep breathing, Mindfulness Meditation, Progressive muscle relaxation, Visual imagery, and Hypnosis among others.

Relaxation training has a long history as a treatment for insomnia. If hyperarousal were a primary determinant of insomnia, it would be expected that methods to reduce arousal would be among the treatments to produce the most improvement. There are a number of different methods for reducing arousal, including *progressive muscle relaxation, diaphragmatic breathing, autogenic training, electromyography biofeedback, meditation, yoga, Visual imagery and hypnosis* (Bootzin & Rider 1997). Progressive muscle relaxation

(Bernstein & Borkovec 1973) is the most widely researched single psychological and behavioral treatment for insomnia (Lichstein & Riedel 2001). Relaxation has been designated as an effective treatment in practice guidelines (Morgenthaler et al. 2006). Nevertheless, relaxation has not been found to be as effective in producing improved sleep as a single treatment as other treatments combined (Morin et al. 2006). Multicomponent treatment that includes relaxation training has been found to be effective (Jacobs et al. 2004). *Mindfulnessmeditation* is a recently added component in multicomponent treatments for insomnia. Mindfulness is defined as the awareness that arises out of intentionally attending in an open, accepting, and discerning way (Shapiro & Carlson 2009), and it involves formal meditation practices as well as principles for applying this awareness to one's moment-to-moment experience. It promises to be particularly effective at reducing mind-racing and ruminative worrying (Bootzin et al. 2010).

Bootzin and Stevens (2005) developed a manualized, small group treatment to improve sleep, daytime sleepiness, and emotional distress in teens with substance-abuse treatment histories. The multicomponent treatment consisted of six 90-minute weekly small-group sessions, with the first session dedicated to sleep education and sessions 2–6 divided equally into segments for the cognitive-behavioral sleep intervention and a modified mindfulness-based stress reduction (MBSR) program. The multicomponent cognitive-behavioral sleep treatment consisted of SCT, emphasis on regularizing sleep-wake schedules across school and weekend days, the use of bright light therapy to advance circadian sleep-wake schedules, and cognitive therapy. The multicomponent treatment program was successful in improving the sleep of adolescents who attended four or more of the six treatment sessions.

The frequency of mindfulness meditation practice, but not duration of practice, was significantly related to improvement in total sleep time and to improvement in self-efficacy about sleep problems (Britton et al. 2010). Mindfulness principles and practices have also been integrated with CBTi for adults with insomnia (Ong et al. 2008, Ong & Sholtes 2010).

In a treatment development study, 27 adults with insomnia completed a multicomponent group treatment that consisted of SCT, SRT, sleep education, sleep hygiene, and MBSR in an integrative framework. There were significant changes in both sleep measures and measures of arousal (Ong et al. 2008). Similar to the results from the study of substance-abusing adolescents, frequency, but not duration of meditation practice, was significantly related to reductions in arousal. Both studies suggest that emphasis should be placed more on the frequency than the duration of mindfulness meditation practice. Furthermore, both studies indicate that mindfulness meditation may contribute to improvement of sleep problems and reduction in arousal in multicomponent treatment studies for insomnia (Bootzin et al. 2010).

2.3 Theoretical Framework

There are different theoretical viewpoints on insomnia. “While the generic [CBT] model is applicable to all disorders, each of the disorders has its own model derived from the generic model” by other scholars (Beck, 2012 p.2). Insomnia theory that were used in this work include;the 3Ps theory of insomnia, cognitive theory, hyper-arousal theory of insomnia, selective attention theory of insomnia, and sleep-wake regulation theory of insomnia view.

2.3.1 The 3Ps theory of insomnia (Spielman, Caruso & Glovinsky1987)

Since the late 1980s, insomnia has largely been conceptualized from within a behavioral framework. Spielman, Caruso, and Glovinsky (1987) proposed the original model 3Ps theoryand it continues to be the leading theory for both Sleep Medicine and the subspecialty area of Behavioral Sleep Medicine.

The theory derived its strength from Skinner (1938) operant conditioning, which states that a behavior is more likely to recur if its consequence (1) generates pleasure (positive reinforcement) or (2) removes an aversive state (negative reinforcement). In contrast, a behavior is less likely to recur if it leads to (i) undesirable consequences or (ii) removes a pleasant state (punishment). On this basis, it was emphasized that behaviour problem develops because of maladaptive transactions between the individual and the environment. In other words, when the environment becomes less rewarding and more punishing, the individual develops mental/behaviour problems.

This behavioral model therefore posits that insomnia occurs acutely in relation to both predisposing (trait) and precipitating (state) factors and occurs chronically in relation to perpetuating or maintaining factors. Thus, an individual may be prone to insomnia due to trait characteristics, experience acute episodes because of precipitating events, and have chronic insomnia owing to a variety of perpetuating factors. With respect to trait factors, personality characteristics (Stepanski 2000; Perlis, Giles, Mendelson, Bootzin, Wyatt 1997), physiologic arousal (Stepanski 2000, Perlis, et al. 1997), and genetic predisposition (Bastien, & Morin 2000), are each thought to contribute to predispose the individual to acute episodes of insomnia. Typical precipitating events (which represent stressors within the larger stress diathesis model of disease) include situational stress (Hall, Buysse, Reynolds, Kupfer,

&Baum 1996), acute injury or pain, bereavement, etc. Perpetuating factors, as the term implies, maintain the chronic form of the disorder even after the precipitating events have either been stabilized or resolved. Perpetuating factors are any of a variety of compensatory strategies in which the patient engages in an attempt to cope with insomnia symptoms. Typical examples of such factors include: excessive daytime napping, extending sleep opportunity, keeping variable sleep–wake schedules, using alcohol as a hypnotic, spending excessive time awake in bed, diminishing daily activity level due to fatigue, etc.

Central to the behavioral model of insomnia is the role of classical conditioning as the primary maintaining factor. It is hypothesized that over time, insomnia becomes a conditioned response to the bed and bedroom environment. This process presumably occurs via traditional principles of classical conditioning, due to repeated parings of the bed and bedroom (conditioned stimuli) with states of psychophysiologic hyperarousal (unconditioned stimuli) that are thought to interfere with the normal biologic processes of sleep initiation and maintenance.

2.3.2 Cognitive theory (Beck, 1964)

Concisely, Beck's cognitive model (1964) proposes that dysfunctional thinking (which influences the patient's mood and behavior) is common to all psychological disturbances. Thinking negative about oneself or situation affect the mood and in turn affect sleep, therefore, when people learn to evaluate their thinking in a more realistic and adaptive way, they experience improvement in their emotional state and in their behavior (Beck, 2011). A number of authors have stressed the importance of cognitive factors in primary insomnia (Morin, 1993; Harvey 2002). Given their emphasis on the role of cognition, they and others have developed interventions, which provide for the cognitive component of the more

broad-based cognitive behavioral approach. Within this perspective, two related types of cognitions are thought to be operational: one set is related to the patient's beliefs about their disorder; the other set is related to cognitive processes like intrusive thoughts and worry. Morin et al., for example, have found that patients with primary insomnia have a number of maladaptive beliefs about sleep, including unrealistic views about what constitutes adequate sleep and catastrophic beliefs about the consequences of insomnia. Such beliefs presumably contribute to insomnia (Spielman, Caruso, & Glovinsky 1987) via increasing sleep-related performance anxiety and (Stepanski, 2000) by prompting and promoting maladaptive compensatory behaviors.

Support for the role of such factors derives from data showing that successful cognitive-behavioral treatment of insomnia is associated with a reduction in negative beliefs and attitudes about sleep (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Morin, Blais, & Savard, 2002). While this is suggestive, more work is needed to demonstrate the "insomnogenic" potential of such cognitions. This is so, because one can easily imagine that successful therapy may change one's thoughts and beliefs, but also that such changes may not be responsible for the treatment gains. Hall et al. (1996) and Harvey and Payne have focused more on cognitive process (vs. content) issues. Central to this area is that patients with insomnia often complain that they are unable to sleep because of intrusive thoughts or excessive worry.

These thoughts and images are characterized as being "intrusive" and may occur in isolation or as unwanted perseverative-type problem solving (worry). The content of the "thoughts and worry" may be centered on the kind of dysfunctional attitudes and beliefs described above, but they are often more general in content. The ideation and imagery that occurs as

intrusive thoughts is often related to mundane daily activities and/or work or relationship issues. As with dysfunctional attitudes and beliefs, intrusive thoughts and perseverative thinking (from within the radical cognitive perspective) are thought to be responsible for the occurrence and severity of insomnia. The more moderate view is that these phenomena are, along with behavioral and conditioning factors, contributory.

Support for the cognitive perspective comes from a variety of studies, which have found that patients with primary insomnia complain of higher levels of presleep rumination compared with normal controls (Lichstein, & Rosentha 1980; Nicassio, Mendlowitz, Fussell, & Petras, 1985). Investigations of pre-sleep thought content have found that the pre-sleep cognitions of patients with primary insomnia tends to be more negatively toned, and that patients report increased general problem solving and thoughts pertaining to environmental stimuli at or around sleep onset (Van Egeren, Haynes, Franzen, & Hamilton, 1983; Watts, Coyle, & East, 1994).

2.3.3 Hyper-arousal theory of insomnia (Perlis, Giles, Bootzin & Wyatt 1997)

In sharp contrast to the cognitive model, the neurocognitive perspective all but suggests that dysfunctional beliefs and worry are epiphenomena. It is posited that cognitive factors are likely to mediate the occurrence and severity of insomnia when the disorder is acute. When, however, the disorder is chronic, cognition occurs secondary to conditioned arousal. Put differently, patients with chronic insomnia are not awake because they are given to rumination and worry, but rather ruminate because they are awake.

The neurocognitive perspective (Perlis, et al. 1997) is an extension of the traditional behavioral model. As laid out by Spielman et al. (1987), the behavioral model allows for a compelling conceptualization regarding how maladaptive behaviors lead to conditioned

arousal and chronic insomnia. The Spielman model does nothowever spells out what the conditioned arousal is, or why and how "arousal" interferes with sleep initiation and/or maintenance and/or the perception of sleep. These latter issues are precisely the province of the neurocognitive model, which defines "arousal" as conditioned cortical arousal. This form of arousal may be observed in patients with primary insomnia as high-frequency EEG activity (14–45 Hz) at or around sleep onset and during NREM sleep (Perlis, Smith, Orff, Andrews, & Giles, 2001). High frequency EEG activity, it is hypothesized, allows for abnormal levels of sensory and information processing and long-term memory formation. Increased sensory processing is thought to interfere with the ability to initiate sleep (as measured by traditional PSG measures). Increased information processing during PSG-defined sleep is thought to interfere with the subject's ability to perceive PSG sleep as "sleep." Increased long-term memory formation (attenuation of the normal mesograde amnesia of sleep) is thought to interfere with the patient's morning judgments about sleep quality and quantity.

Support for the neurocognitive perspective (Perlis, Merica, Smith, & Giles, 2001) comes from a variety of studies which have found Beta EEG (14–45 Hz) to (1) be elevated in patients with insomnia (Perlis et al. 2001; Merica, Blois, & Gaillard, 1998; Lamarche, & Ogilvie, 1997), (2) be positively associated with patient perceptions of sleep quality (Perlis et al. 1997; Hall, Buysse, Nowell, Nofzinger, Houck, Reynolds, & Kupfer, 2000), (3) be positively associated with sleep state misperception (the degree of discrepancy between subjective and objective measures of sleep) (Perlis, et al. 2001; Krystal, Edinger, Wohlgemuth, & Michaels, 2002), and (4) vary with successful CBT treatment for insomnia (Krystal, Edinger, Wohlgemuth, & Marsh, 2001). There is also preliminary evidence that the

occurrence of elevated NREM Beta activity is sensitive and specific to primary insomnia (vs. insomnia secondary to major depressive disorder) (Perils et al. 2001) and data which suggest the long-term memory function at peri-sleep-onset intervals is altered in patients with chronic insomnia Perils et al. 2001).

2.3.4 Selective attention theory of insomnia(Espie, Broomfield, MacMahon, Macphee, &Taylor 2006).

This theory addresses the role of selective attention in the development and maintenance of insomnia. Psychological and/or physiological stress is posited to lead to selective attention toward stressors, and inhibition of the "de-arousal" that normally accompanies sleep. Inappropriate arousal may then lead to selective attention to sleep-related cues (implicit or explicit) and increased explicit intention and effort to sleep, which further inhibit normal sleep-related de-arousal (Espie, et al. 2006). The psychobiological inhibition model has good face validity and leads to specific, testable hypotheses regarding selective attention to sleep-related cues, which have received some support from empirical studies (Jones, Macphee, Broomfield, Jones, & Espie 2005; Spiegelhalder, Espie, Nissen & Riemann, 2008). Limitations include the difficulty of measuring intention and effort in a comparative sense, and the lack of specification of neural substrates.

2.3.5 Sleep-wake regulation theory of insomnia (Buysse, Germain, Hall, Monk & Nofzinger, 2011)

This neurobiological theory of insomnia draws on previous psychological-behavioral models as well as the central and local neurobiological models of sleep regulation. The theory hypothesize that insomnia is a disorder of sleep-wake regulation characterized by persistent

wake-like activity in neural structures during NREM sleep, resulting in simultaneous and regionally-specific waking and sleeping neuronal activity patterns ((Buysse, Germain, Hall, Monk & Nofzinger, 2011). During NREM sleep defined by central and frontal EEG patterns, the theory hypothesize that regional neuronal activity and metabolism is greater in limbic and parietal cortices, thalamus, and hypothalamic-brainstem arousal centers in individuals with insomnia, as compared to individuals with good sleep. The simultaneous occurrence of sleep-like and wake-like neural activity may help to explain clinical phenomenology and treatment effects in insomnia. For instance, individuals with insomnia may perceive wakefulness or persistent awareness of the environment despite the occurrence of cortical EEG patterns consistent with sleep. Relatively small increases in sleep time or decreases in wakefulness with treatment could be associated with large subjective improvements if treatment led to reduced local waking neural activity (Buysse, Germain, Hall, Monk & Nofzinger, 2011).

The model suggests that insomnia results from persistent activity in wake-active brain regions and circuits during NREM sleep, including cortical regions (prefrontal, parietal, and precuneus), paralimbic cortex (anterior cingulate, mesial temporal), thalamus, and hypothalamic/brainstem arousal centers. Persistent activity in the precuneus during EEG-defined NREM sleep may be particularly relevant to the experience of insomnia: Such activity may contribute to the subjective experience of self-awareness. Increased activation of components of the default-mode network during slow wave activity has been suggested to facilitate neuronal interactions and information processing (Maquet, 2010).

Neurobiological model of insomnia differs from the most widely cited models of insomnia primarily based on level of inquiry. Most previous models have been developed from psychological and behavioral constructs, which are measured by self-report or behavioral response patterns, or global EEG characteristics. The neurobiological model has been developed from clinical and basic neuroscience observations, which are measured in the activity patterns of more specific brain regions or circuits. Ultimately, psychological and behavioral constructs must have neurobiological determinants. Conversely, the activity of brain regions and circuits is ultimately expressed in the behavior of the organism (Buysse, Germain, Hall, Monk & Nofzinger, 2011).

2.3.6 Model of insomnia

The following is an overview of theintegrative model of insomnia:

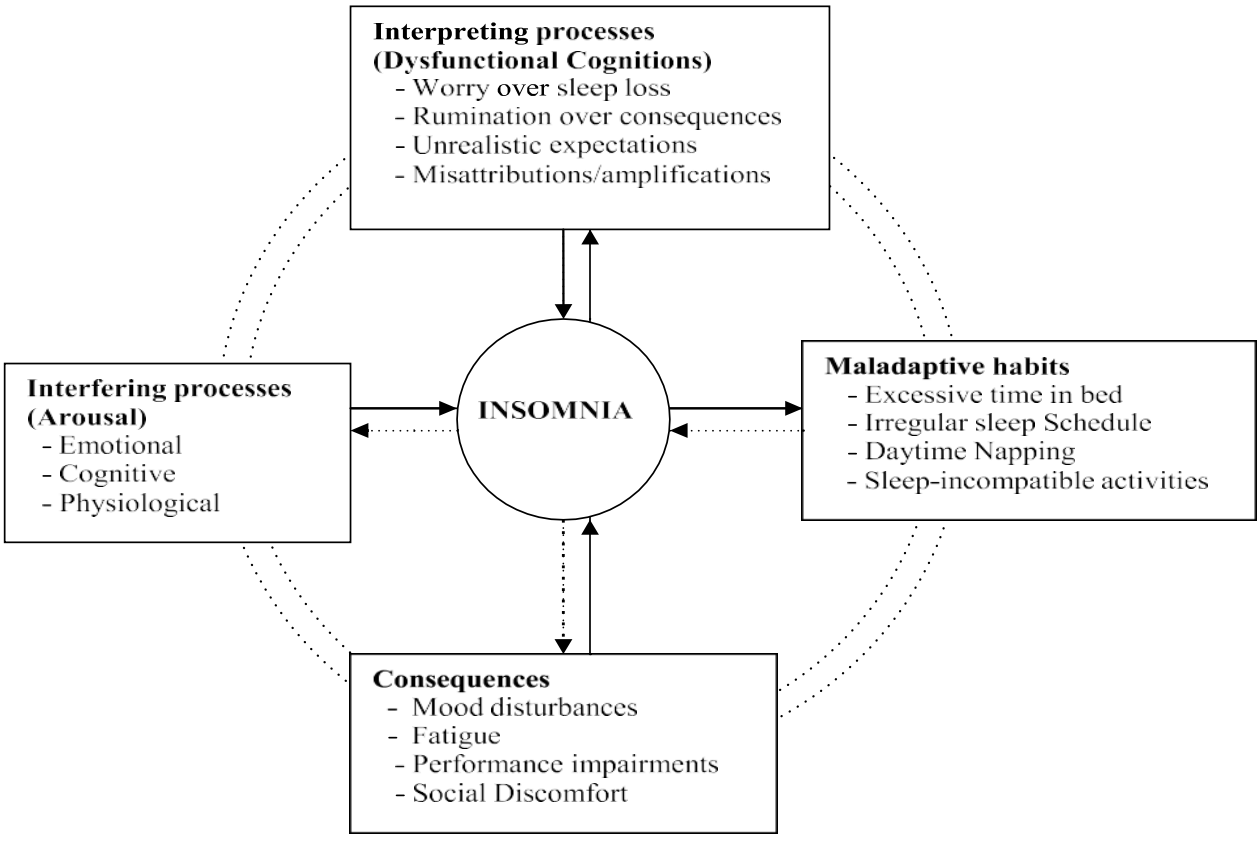


Figure 1: An Integrative Model of Insomnia. Source: (SpielmanCaruso & Glovinsky 1987).

The model posits that insomnia occurs acutely in relation to both predisposing (trait) and precipitating (state) factors and occurs chronically in relation to perpetuating or maintaining factors. Typical precipitating events include situational stress, acute injury or pain, bereavement, among others while, perpetuating factors, as the term implies, maintain the form of the disorder even after the precipitating events have either been stabilized or resolved. Typical examples of such factors include: excessive daytime napping, extending sleep opportunity, keeping variable sleep–wake schedules, using alcohol as a hypnotic, spending excessive time awake in bed, diminishing daily activity level due to fatigue. The model also gave emphasis on the role of cognition. Cognitive processes like intrusive thoughts and worry, maladaptive beliefs about sleep, including unrealistic views about what constitutes adequate sleep and catastrophic beliefs about the consequences of insomnia. Such beliefs presumably contribute to insomnia via increasing sleep-related performance anxiety and by prompting and promoting maladaptive compensatory behaviors. Therefore, since insomnia is because of these factors, it is believed that using cognitive and behavioural method can as well provide help in resolving the condition.

2.4 Review of Empirical Studies

Several empirical studies have been conducted on cognitive and behavioural techniques for treating insomnia. For instance, Vitiello, Rybarczyk, Von Korff, and Stepanski (2009) investigated whether Cognitive Behavioral Therapy for Insomnia Improves Sleep and Decreases Pain in Older Adults with Co-Morbid Insomnia and Osteoarthritis. Twenty-three patients (mean age 69.2 years) were randomly assigned to CBT-I and 28 patients (mean age 66.5 years) to an attention control. Participants were recruited by placement of brochures,

memos, and flyers in places where medical patients who qualified for the study might see them. Potential volunteers who contacted the investigators about the study were initially screened by telephone. Sleep and pain were assessed by self-report at baseline, after treatment, and (for CBT-I only) at 1-year follow-up. Result revealed CBT-I significantly improved sleep and significantly reduced pain after treatment. Control subjects reported no significant improvements. One-year follow-up found maintenance of improved sleep and reduced pain for both the CBT-I group alone and among subjects who crossed over from control to CBT-I.The study concluded that CBT-I but not an attention control, without directly addressing pain control, improved both immediate and long-term self-reported sleep and pain in older patients with osteoarthritis and comorbid insomnia.

Kaldo, Jernelöv, Blom, Ljótsson, Brodin, Jörgensen, Kraepelien, Rück, and Lindefors (2015) in a randomized trial researched on Guided internet cognitive behavioral therapy for insomnia compared to a control treatment. The basic aim was to evaluate if internet-delivered Cognitive Behavioral Therapy for insomnia (ICBT-i) with brief therapist support outperforms an active control treatment. Adults volunteers diagnosed with insomnia were recruited via media (n = 148) and randomized to either eight weeks of ICBT-I or an active internet-based control treatment. Primary outcome was the insomnia severity index (ISI) assessed before and after treatment, with follow-ups after 6 and 12 months. Secondary outcomes were use of sleep medication, sleep parameters (sleep diary), perceived stress, and a screening of negative treatment effects. Hierarchical Linear Mixed Models were used for intent-to-treat analyses and handling of missing data. Result shows that ICBT-I was significantly more effective than the control treatment in reducing ISI (Cohen's d = 0.85),

sleep medication, sleep efficiency, sleep latency, and sleep quality at post-treatment. The positive effects were sustained. However, after 12 months the difference was no longer significant due to a continuous decrease in ISI among controls, possibly due to their significantly higher utilization of insomnia relevant care after treatment. Forty-six negative effects were reported but did not differ between interventions. Kaldo et al. (2015) concluded that supported ICBT-I is more effective than an active control treatment in reducing insomnia severity and treatment gains remain stable one year after treatment.

Using a randomized controlled trial, other studies tried to examine whether a video-based cognitive behavioral therapy for insomnia is as efficacious as a professionally administered treatment in breast cancer (Savard, Ivers, Savard, & Morin 2014). Two hundred and forty-two women with breast cancer who had received radiation therapy in the past 18 months and who had insomnia symptoms or were using hypnotic medications were randomized to: (1) professionally administered CBT-I (PCBT-I; n = 81); (2) video-based CBT-I (VCBT-I; n = 80); and (3) no treatment (CTL; n = 81). PCBT-I composed of six weekly, individual sessions of approximately 50 min; VCBT-I composed of a 60-min animated video + six booklets. Insomnia Severity Index (ISI) total score and sleep parameters were derived from a daily sleep diary and actigraphy, collected at pretreatment and post treatment. Results revealed that PCBT-I and VCBT-I were associated with significantly greater sleep improvements, assessed subjectively, as compared to CTL. However, relative to VCBT-I, PCBT-I was associated with significantly greater improvements of insomnia severity, early morning awakenings, depression, fatigue, and dysfunctional beliefs about sleep. The remission rates of insomnia (ISI < 8) were significantly greater in PCBT-I as compared to

VCBT-I (71.3% versus 44.3%, P < 0.005). Savard et al (2014) concluded that a self-administered cognitive behavioral therapy for insomnia (CBT-I) using a video format appears to be a valuable treatment option, but face-to-face sessions remain the optimal format for administering CBT-I efficaciously in patients with breast cancer. Self-help interventions for insomnia may constitute an appropriate entry level as part of a stepped care model.

Ahmed and Younis (2014) studied Effect of Relaxation Techniques on Quality of Sleep for Patients with End-Stage Renal Failure Undergoing Hemodialysis. The researcher used a convenient sample of 20 adult patients undergoing hemodialysis, with one group, before and after intervention. Two tools were used for data collection, which includes socio-demographic data assessment andPittsburgh Sleep Quality Index that measures the sleepquality. Patients were exposed to fourweeks' treatment sessions using progressive muscle relaxation, deep breathing exercise and guided visualization. The main result showed that the relaxation technique improved the total score of sleep quality and itsdimensions in hemodialysis patients. It showed that the mean score of total Pittsburgh Sleep Quality Index decreased significantly after demonstration of relaxation techniques compared with the mean score of total Pittsburgh Sleep Quality Index one month before application of relaxation techniques. Ahmed and Younis (2014) recommended that periodic clinical assessment of sleep complaintsshould become routine for dialysis patients. In addition, non-pharmacological methods such as relaxation techniquesshould be used for the treatment of sleep problems in hemodialysis patients.

Sivertsen, Omvik, Pallesen, Bjorvatn, Havik, Kvale, Nielsen, and Nordhus, (2006) examined Cognitive Behavioral Therapy vs Zopiclone for Treatment of Chronic Primary Insomnia in Older Adults: A Randomized Controlled Trial FREE. The main Objective was to examine short- and long-term clinical efficacy of cognitive behavioral therapy (CBT) and pharmacological treatment in older adults experiencing chronic primary insomnia. The study used A randomized, double-blinded, placebo-controlled trial designinvolving 46 adults who respondent to advertisement made by the researcher (mean age, 60.8 y; 22 women) with chronic primary insomnia reported Norwegian university-based outpatient clinic for adults and elderly patients. The Intervention CBT (sleep hygiene, sleep restriction, stimulus control, cognitive therapy, and relaxation; n = 18), sleep medication (7.5-mg zopiclone each night; n = 16), or placebo medication (n = 12). All treatment duration was 6 weeks, and the 2 active treatments were followed up at 6 months. Main Outcome Measures used in the study were Ambulant clinical polysomnographic data and sleep diaries to determine total wake time, total sleep time, sleep efficiency, and slow-wave sleep (only assessed using polysomnography) on all 3 assessment points. Results according to Sivertsen et al. (2006) showed CBT improved short- and long-term outcomes compared with zopiclone on 3 out of 4 outcome measures. For most outcomes, zopiclone did not differ from placebo. Participants receiving CBT improved their sleep efficiency from 81.4% at pretreatment to 90.1% at 6-month follow-up compared with a decrease from 82.3% to 81.9% in the zopiclone group. Participants in the CBT group spent much more time in slow-wave sleep (stages 3 and 4) compared with those in other groups, and spent less time awake during the night. Total sleep time was similar in all 3 groups; At 6 months, patients receiving CBT had better sleep efficiency using polysomnography than those taking zopiclone. The study concluded that

interventions based on CBT are superior to zopiclone treatment both in short- and long-term management of insomnia in older adults.

Fiorentino (2008), investigates Cognitive behavioral therapy for insomnia in breast cancer survivors using a randomized controlled crossover study. 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.

Morgan, Dixon, Mathers, Thompson & Tomeny, (2004), assessed psychological treatment for insomnia in the regulation of long-term hypnotic drug use. The objectives were to evaluate the clinical and cost impact of providing, in routine general practice settings, a cognitive behaviour therapy (CBT) package for insomnia to long-term hypnotic drug users with chronic sleep difficulties; and to identify factors associated with variations in clinical outcomes. Design: A pragmatic cluster randomised controlled trial with two treatment arms (a CBT-treated 'sleep clinic' group, and a 'no additional treatment' control group), with post-treatment assessments starting at 3, 6 and 12 months. Setting: Twenty-three general practices in Sheffield, UK. Participants: In total, 209 patients (aged 31–92 years) with chronic sleep problems who had been receiving repeat hypnotic drug prescriptions for at least 1 month (mean = 13.4 years) were recruited into the trial. Interventions: The intervention consisted of six 50-minute sessions as follows: introduction and sleep assessment, basic sleep hygiene, stimulus control and sleep restriction procedures, progressive relaxation, cognitive treatments, and review and discharge. Main outcome measures: These included global sleep quality [as measured by the Pittsburgh Sleep Quality Index (PSQI)], frequency of hypnotic drug use, mean dose of hypnotics consumed, health-

related quality of life [as measured by the Short-Form 36 (SF-36)], NHS service costs and overall cost utility. Results: At 3- and 6-month follow-ups, patients treated with CBT showed improved global PSQI scores as well as improvements in the SF-36 dimensions of vitality at 3 months, physical functioning, and mental health at 6 months. CBT-treated patients also reported reductions in the frequency of hypnotic drug use compared with the control group, with many CBT treated patients reporting zero drug use at the follow-up assessments. Clinical improvements were maintained within the CBT group at the 12-month follow-up, with PSQI scores and the frequency of hypnotic drug use continuing to show significant reductions relative to the control group. Concluded that in routine general practice settings, psychological treatment for insomnia can improve sleep quality, reduce hypnotic drug use, and improve health- related quality of life at a favourable cost among long-term hypnotic users with chronic sleep difficulties. These positive outcomes appear robust over time, persisting for at least 1 year among the more treatment-adherent patients.

Gałuszko-Węgielnik,Jakuszkowiak-Wojten, Wiglusz,Cubałaand Landowski (2012) investigated the efficacy of cognitive-behavioural therapy (CBT) as related to sleep quality and hyperarousal level in the treatment of primary insomnia. The aim of the study was to determine the efficacy of a CBT-protocol in the treatment of PI by means of sleep onset latency and the number of awakenings during night parameters along with sleep quality and the level of psychophysiological hyper-arousal. 26 individuals from a tertiary reference sleep disorders outpatients' clinic (22 women; mean age 41.4; 4 men; mean age 42.5) with primary insomnia (DSM-IV-TR) were used in the study. The participants were scored with Ford Insomnia Response to Stress Test (FIRST) at the beginning of the study. The Athens

Insomnia Scale (AIS), Hyperarousal Scale, Leeds Sleep Questionnaire (LSEQ) were also applied at the beginning, at the end and three months after the end of the study. The participants during the course of the treatment patients completed a Sleep Diary (SD). The CBT program employed was based on the Perlis protocol. Standard individual sessions of 50 minutes were provided on a weekly basis for 8-10 weeks by the therapist. After 3 months, a follow-up session was scheduled. Results as reported by Gałuszko-Węgielnik et al. (2012) showedsignificant improvement as related to the CBT treatment was present in the measures of sleep onset latency, numbers of awakenings during night, sleep efficiency, quality of falling asleep, quality of sleep, and quality of morning awakening. The improvement reached the significance level in the measure of psychophysiological arousal (52.3 vs. 42.4; $p<0.000$) and AIS (15.7vs. 6.8; $p<0.000$). Gałuszko-Węgielnik et al. (2012) concluded thatCBT is an effective treatment in resolving insomnia.

Trauer, Qian, Doyle, Rajaratnam, and Cunnington, (2015). Investigated Cognitive Behavioral Therapy for Chronic Insomnia using A Systematic Review and Meta-analysis. The objective was to determine the efficacy of CBT-i on diary measures of overnight sleep in adults with chronic insomnia. Searches of MEDLINE, EMBASE, PsycINFO, CINAHL, the Cochrane Library, and PubMed Clinical Queries from inception to 31 March 2015, supplemented with manual screening. Study Selection: Randomized, controlled trials assessing the efficacy of face-to-face, multimodal CBT-i compared with inactive comparators on overnight sleep in adults with chronic insomnia, with studies of insomnia comorbid with medical, sleep, or psychiatric disorders excluded. Among 292 citations and 91 full-text articles reviewed, 20 studies (1162 participants [64% female; mean age, 56

years]) were included. Approaches to CBT-i incorporated at least 3 of the following: cognitive therapy, stimulus control, sleep restriction, sleep hygiene, and relaxation. At the posttreatment time point, SOL improved by 19.03 (95% CI, 14.12 to 23.93) minutes, WASO improved by 26.00 (CI, 15.48 to 36.52) minutes, TST improved by 7.61 (CI, 0.51 to 15.74) minutes, and SE% improved by 9.91% (CI, 8.09% to 11.73%), and changes seemed to be sustained at later time points. No adverse outcomes were reported. CBT-i is an effective treatment for adults with chronic insomnia, with clinically meaningful effect sizes.

Edinger, Olsen, Stechuchak, Means, Lineberger, Kirby and Carney (2009) in a randomized clinical trial, investigated the effects of Cognitive behavioral therapy for patients with primary insomnia or insomnia associated predominantly with mixed psychiatric disorders: The objective of the study was to evaluate the efficacy of cognitive behavioral therapy (CBT) against a sleep hygiene education control therapy in patients with primary or comorbid insomnia. A randomized, parallel-group design was used, and clinical trial conducted at a single Veterans Affairs medical center. Eighty-one adults (n = 11 women; mean age, 54.2 years) with chronic primary (n = 40) or comorbid insomnia associated predominantly with mixed psychiatric disorders (n = 41). Patients, screened via structured interviews and diagnostic polysomnography, were randomly assigned to receive CBT (sleep education, stimulus control, and time-in-bed restrictions; 20 patients with primary and 21 with comorbid insomnia), or sleep hygiene (SH: education about aspects of lifestyle and the bedroom environment that affect sleep; 20 patients with primary and 20 with comorbid insomnia). Outpatient treatment included 4 biweekly sessions with a post treatment assessment and a follow-up conducted at 6 months. Participants completed actigraphy and

sleep diaries for 2 weeks prior to therapy, during a 2-week post treatment assessment, and during 2 weeks at follow-up. They also completed questionnaires measuring global insomnia symptoms, general sleep quality, and sleep-disruptive beliefs before treatment, immediately following treatment, and at the follow-up time point. Edinger et al (2009) reported that consistent with previous studies, CBT outperformed sleep hygiene across several study outcome measures for the sample as a whole. Statistical analyses showed no significant 3-way interaction of treatment group, time, and insomnia type for any of the sleep or questionnaire measures, suggesting the benefits of CBT over sleep hygiene were comparable for patients with primary insomnia and comorbid insomnia. Moreover, only 1 of several indexes of clinically notable improvement suggested a significantly better response to CBT by patients with primary insomnia, as compared with those with comorbid insomnia. The study concluded that a fixed 4-session "dose" of CBT produced similar benefits for patients with primary and those with comorbid insomnia across most measures examined. Thus, CBT appears to be a viable psychological insomnia therapy both for those with primary insomnia and for groups composed mainly of patients with insomnia and nonpsychotic psychiatric conditions.

In another study, Jernelöv, Lekander, Blom, Rydh, Ljótsson, Axelsson, and Kaldo (2012) studied the Efficacy of a behavioral self-help treatment with or without therapist guidance for co-morbid and primary insomnia, using a randomized controlled trial. The researcher hypothesized that a cognitive behaviorally based self-help book is effective to treat insomnia in individuals, also with co-morbid problems, and that the effect is enhanced by adding brief therapist telephone support. One hundred and thirty three volunteers were recruited from all

over Sweden, through media and websites. Interested individuals were directed to a web site with a description of the study, informed consent and screening forms. All assessments were conducted on the Internet or telephone. No monetary compensation was given for participation, but the participants did not pay for the treatment. Parallel randomized design was used with (block-randomization, $n \geq 21$) controlled "open label" trial; three groups-bibliotherapy with ($n = 44$) and without ($n = 45$) therapist support, and waiting list control ($n = 44$). Assessments before and after treatment, and at three-month follow-up was carried out. Intervention was six weeks of bibliotherapeutic self-help, with established cognitive behavioral methods including sleep restriction, stimulus control, and cognitive restructuring. Therapist support was a 15-minute structured telephone call scheduled weekly. Main outcome measures were sleep diary data, and the Insomnia Severity Index. Analyses of data showed significant improvements in both self-help groups from pre to post treatment compared to waiting list. For example, treatment with and without support gave shorter sleep onset latency (improvement minutes [95% Confidence Interval], 35.4 [24.2 to 46.6], and 20.6 [10.6 to 30.6] respectively), and support gave a higher remission rate (defined as ISI score below 8; 61.4%), than bibliotherapy alone (24.4%, $p < .001$). Improvements were not seen in the control group (sleep onset latency 4.6 minutes shorter [-1.5 to 10.7], and remission rate 2.3%). Self-help groups maintained gains at three-month follow-up. The study concluded that participants receiving self-help for insomnia benefited markedly. Self-help, especially if therapist-supported, has considerable potential to be as effective as individual treatment at lower cost, also for individuals with co-morbid problems.

In addition, Espie, Kyle, Williams, Ong, Douglas, Hames and Brown (2012) used randomized, placebo-controlled trial to examine the efficacy of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. Three arms: CBT, imagery relief therapy (IRT: placebo), treatment as usual (TAU) comprising one hundred sixty-four adults (120 F: [mean age 49y (18-78y)] meeting proposed DSM-5 criteria for Insomnia Disorder, randomly assigned to CBT (n = 55; 40 F), IRT placebo (n = 55; 42 F) or TAU (n = 54; 38 F). CBT and IRT each comprised 6 online sessions delivered by an animated personal therapist, with automated web and email support. Participants also had access to a video library/back catalogue of session content and Wikipedia style articles. Online CBT users had access to a moderated social network/community of users. TAU comprised no restrictions on usual care and access to an online sleep diary. Major assessments at baseline, post-treatment, and at follow-up 8-weeks post-treatment; outcomes were appraised by online sleep diaries and clinical status. Result showed on the primary endpoint of sleep efficiency (SE; total time asleep expressed as a percentage of the total time spent in bed), online CBT was associated with sustained improvement at post-treatment (+20%) relative to both TAU (+6%; d = 0.95) and IRT (+6%: d = 1.06), and at 8 weeks (+20%) relative to IRT (+7%: d = 1.00) and TAU (+9%: d = 0.69). These findings were mirrored across a range of sleep diary measures. Clinical benefits of CBT were evidenced by modest superiority over placebo on daytime outcomes (d = 0.23-0.37) and by substantial improved sleep-wake functioning on the Sleep Condition Indicator (range of d = 0.77-1.20). Three-quarters of CBT participants (76% [CBT] vs. 29% [IRT] and 18% [TAU]) completed treatment with SE > 80%, more than half (55% [CBT] vs. 17% [IRT] and 8% [TAU]) with SE > 85%, and over one-third (38% [CBT] vs. 6% [IRT]

and 0% [TAU]) with SE > 90%; these improvements were largely maintained during follow-up. Espie et al. (2012) concluded that CBT delivered using a media-rich web application with automated support and a community forum is effective in improving the sleep and associated daytime functioning of adults with insomnia disorder.

Edinger,William, Wohlgemuth, Radtke, Marsh, andQuillian, (2001) studied Cognitive Behavioral Therapy for Treatment of Chronic Primary Insomnia using a Randomized, double blind, placebo-controlled clinical trial. The major objective of the study was to test the efficacy of a hybrid cognitive behavioral therapy (CBT) compared with both a first-generation behavioral treatment and a placebo therapy for treating primary sleep-maintenance insomnia. Seventy-five adults (n=35 women; mean age, 55.3 years) with chronic primary sleep-maintenance insomnia (mean duration of symptoms, 13.6 years) were randomly assigned to receive CBT (sleep education, stimulus control, and time-in-bed restrictions; n=25), progressive muscle relaxation training (RT; n=25). Treatment lasted 6 weeks, with follow-up conducted at 6 months. Instruments used were13-item Insomnia Symptom Questionnaire (ISQ), a 9-item Self Efficacy Scale (SES), and the Beck Depression Inventory (BDI) at baseline, mid-treatment (i.e., end of third treatment week), post treatment, and the 6-month follow-up time points. Result as reported by Edinger et al. (2001) showed that Cognitive behavioral therapy produced larger improvements across the majority of outcome measures than did RT or placebo treatment. For example, sleep logs showed that CBT-treated patients achieved an average 54% reduction in their WASO whereas RT-treated and placebo-treated patients, respectively, achieved only 16% and 12% reductions in this measure. Recipients of CBT also showed a greater normalization of sleep

and subjective symptoms than did the other groups with an average sleep time of more than 6 hours, middle WASO of 26.6 minutes, and sleep efficiency of 85.1%. In contrast, RT-treated patients continued to report a middle WASO of 43.3 minutes and sleep efficiency of 78.8%. Edinger et al. (2001) concluded that CBT represents a viable intervention for primary sleep-maintenance insomnia. This treatment leads to clinically significant sleep improvements within 6 weeks and these improvements appear to endure through 6 months of follow-up.

Jungquist, O'Brien, Matteson-Rusby, Smith, Xia, Lu, Perlis, and Pigeon (2010) assessed the efficacy of cognitive-behavioral therapy for insomnia in patients with chronic pain. Twenty-eight subjects with chronic neck and back pain were stratified according to gender, age, and ethnicity, then assigned to one of the two treatment groups: CBT-I, or a contact control condition. Eight weeks of CBT-I including sleep restriction, stimulus control, sleep hygiene, and one session of cognitive therapy devoted to catastrophic thoughts about the consequences of insomnia. Outcomes included sleep diary assessments of sleep continuity, pre–post measures of insomnia severity Index (ISI), pain (Multidimensional Pain Inventory), and mood (BDI and POMS). Result revealed that subjects receiving CBT-I (n = 19), as compared to control subjects (n = 9), exhibited significant decreases in sleep latency, wake after sleep onset, number of awakenings, and significant increase in sleep efficiency. The diary findings were paralleled by significant changes in the ISI ($p = 0.05$). Jungquist et al. concluded that CBT-I was successfully applied to patients experiencing chronic pain. Significant improvements were found in sleep as well as in the extent to which pain interfered with daily functioning.

Sánchez, Díaz-Piedra, Miró, Gálvez, and Buela-Casal (2012) also investigated the effects of cognitive-behavioral therapy for insomnia on polysomnographic parameters in fibromyalgia patients. The study aimed to evaluate the efficacy of cognitive-behavioral therapy for insomnia (CBT-I) on polysomnographic parameters in patients with fibromyalgia (FM). Twenty-six women with FM (M = 46.79 years of age, SD = 5.15) participated in the study and were assigned to a CBT-I group (n = 13; M_{age}= 44.83, SD = 5.30) or a sleep hygiene (SH) condition (n = 13; M_{age} = 48.75, SD = 4.37). Simple randomization (1:1) was implemented by a computerized number generator designed by an investigator with no clinical involvement in the trial. The evaluation consisted in two interview sessions and domiciliary polysomnography study before and after treatment. The results show that time-in-bed and wake percentage diminish after CBT-I. Improvements were also observed in sleep efficiency, which was close to normal levels. The percentage of NREM stage 1 sleep decreased and NREM stages 3 sleep and 4 increased. Similarly, light sleep (stages 1 and 2) diminished and deep sleep increased (stages 3 and 4) after CBT-I. No improvements were observed in any of these parameters in the individuals undergoing SH therapy. Sánchez et al. (2012) concluded that the randomized controlled trial provides new evidence that the use of CBT-I in FM patients can significantly improve objective sleep parameters.

Edingerand Sampson (2003) studied A Primary Care "Friendly" Cognitive Behavioral Insomnia Therapy. The study objective was to test the effectiveness of an abbreviated cognitive-behavioral insomnia therapy (ACBT) with primary care patients. A single-blind, randomized group design was used in which study patients were randomized to either a brief, 2-session ACBT or a similarly brief intervention (SHC) that included only generic sleep hygiene recommendations.18 men and 2 women veteran patients (M_{age} = 51.0 yrs., SD

= 13.7 years) who met criteria for chronic primary insomnia were used in the study.Participants completed sleep logs for 2 weeks and questionnaires to measures insomnia symptoms, sleep-related self-efficacy, and dysfunctional beliefs about sleep before treatment, during a 2-week post treatment assessment, and again at a 3-month post treatment follow-up. Statistical analyses showed that ACBT produced significantly larger improvements across a majority of outcome measures than did SHC. Case-by-case analyses showed that only the ACBT produced consistent positive effects across study patients, and a sizeable proportion of these patients receiving this treatment achieved clinically significant improvements by their study endpoints. Approximately 52% of those receiving the ACBT reported at least a 50% reduction in their wake time after sleep onset, and 55.6% of ACBT-treated patients who entered the study with pathologic scores on an Insomnia Symptom Questionnaire (ISQ), achieved normal ISQ scores by their outcome assessment. Edinger and Sampson (2003) therefore concluded that ACBT is effective for reducing subjective sleep disturbance and insomnia symptoms in primary care patients.

Fornal–Pawłowska and Szelenerger (2013) evaluate Cognitive behavioral therapy for chronic insomnia. The major objective was to evaluate the efficacy of cognitive behavioral therapy (CBT-I) for chronic insomnia treatment. 236 patients with nonorganic insomnia were assigned to group CBT-I (6 sessions, 6-10 patients). From this pool, 72 participants with no history of other psychiatric or sleep disorders conditions were selected. Eventually, 51 patients (40f, mean age: 54.6±13.9y, mean insomnia duration: 7±6.3y) and 51 matched healthy controls (mean age: 55.4±14.3y) completed the study. Outcomes in the insomnia group at baseline and post-treatment were compared to control group. Subjects underwent sleep diary, the Athens Insomnia Scale (AIS), the Beck Depression Inventory (BDI), the

Ford Insomnia Response to Stress Test (FIRST), the SF-36 questionnaire and the State-Trait Anxiety Inventory (STAI). Result revealed that at baseline, groups differed significantly in most dependent variables. At post treatment, a substantial improvement in all sleep parameters was observed in insomnia group: sleep latency, number of awakenings, wake time after sleep onset, sleep time, sleep efficiency, sleep quality and frequency of hypnotic use. Lower AIS and FIRST scores, reductions of depression and anxiety symptoms, and improved energy and social functioning ratings, accompanied these outcomes. All changes were maintained during the 3-month follow-up. Only 10/51 patients had no clinically meaningful improvement at any post-treatment time points. After the therapy, patients did not differ significantly from good sleepers in number of awakenings, sleep quality, feeling in the morning, depression and anxiety symptoms, and quality of life related to mental health. Fornal–Pawłowska andSzelenerger (2013) concluded that The CBT-I produced a sustained, clinically meaningful improvement in nocturnal sleep and daytime functioning.

In another study on CBT-I, Matthews, Berger, Schmiege, Cook, McCarthy, Moore, and Aloia (2014) examine the effect of cognitive-behavioral therapy for insomnia (CBTI) on sleep improvement, daytime symptoms, and quality of life (QOL) in breast cancer survivors (BCSs) after cancer treatment. A prospective, longitudinal, randomized, controlled trial design was used. 56 middle-aged BCSs (breast cancer survivors) with chronic insomnia. Women were randomly assigned to CBTI or behavioral placebo treatment (BPT) and completed measures of sleep, QOL, functioning, fatigue, and mood at baseline, post intervention, and at three- and six-month follow-ups. The major research variables were sleep outcomes (e.g., sleep efficiency, sleep latency, total sleep time, wake after sleep onset, and number of nightly awakenings); secondary variables included sleep medication use,

insomnia severity, QOL, physical function, cognitive function, fatigue, depression, anxiety, and sleep attitudes or knowledge. Result shows that sleep efficiency and latency improved more in the CBTI group than the BPT group; this difference was maintained during follow-up. Women in the CBTI group had less subjective insomnia, greater improvements in physical and cognitive functioning, positive sleep attitudes, and increased sleep hygiene knowledge. No group differences in improvement were noted relative to QOL, fatigue, or mood. Matthews et al. (2014) concluded that CBTI appears to be beneficial for BCSs' sleep latency/efficiency, insomnia severity, functioning, sleep knowledge, and attitudes more than active placebo, with sustained benefit over time.

Trauer, Qian, Doyle, Rajaratnam, & Cunnington (2015) carried out a Systematic Review and Meta-analysis of Cognitive Behavioral Therapy for Chronic Insomnia. To determine the efficacy of CBT-I on diary measures of overnight sleep in adults with chronic insomnia. Data were collected from Searches of MEDLINE, EMBASE, PsycINFO, CINAHL, the Cochrane Library, and PubMed Clinical Queries from inception to 31 March 2015, supplemented with manual screening. Among 292 citations and 91 full-text articles reviewed, 20 studies (1162 participants [64% female; mean age, 56 years]) were included. Approaches to CBT-I incorporated at least 3 of the following: cognitive therapy, stimulus control, sleep restriction, sleep hygiene, and relaxation. The output revealed that at the post treatment time point, SOL improved by 19.03 minutes, WASO improved by 26.00 minutes, TST improved by 7.61 minutes, and SE% improved by 9.91% and changes seemed to be sustained at later time points. No adverse outcomes were reported. Trauer et al. (2015)

concluded that CBT-I is an effective treatment for adults with chronic insomnia, with clinically meaningful effect sizes.

Deora and Anthony (2013) evaluated the Effectiveness of Relaxation Training and Sleep Hygiene Education for Insomnia of Depressed Patients. The basic study objective was to evaluate whether relaxation training and sleep hygiene education intervention can be effective for female patients who experience major depressive disorder and insomnia. Therefore, a convenience sample of 10 female participants were recruited from an outpatient psychiatric private practice. Participants attended four consecutive, weekly outpatient sessions lasting approximately 1 hr. The initial session consisted of conducting a psychiatric evaluation and administration of the Pittsburgh Sleep Quality Index to measure rating of sleep quantity and quality. Principles of sleep hygiene and relaxation exercises were introduced during the initial session. Subsequent sessions focused on reinforcement of the principles of sleep hygiene and relaxation training. Results showed that Sleep hygiene education and relaxation training were effective in treating insomnia of depressed patients. Deora and Anthony (2013) concluded that Implementation of sleep hygiene education and relaxation training would provide nurses with evidence-based treatment alternatives or complements to pharmacotherapy in depressed patients.

In another study, Talbot, Maguen, Metzler, Schmitz, McCaslin, Richards, Perlis, Posner, Weiss, Ruoff, Varbel, and Neylan (2014) researched on Cognitive Behavioral Therapy for Insomnia in Posttraumatic Stress Disorder, with a major objective to examine whether cognitive behavioral therapy for insomnia (CBT-I) improves sleep in posttraumatic stress disorder (PTSD) as well as nightmares, nonsleep PTSD symptoms, depression symptoms,

and psychosocial functioning. Randomized controlled trial design with two arms: CBT-I, and monitor-only waitlist control was used. Forty-five adults (31 females: [mean age 37 y (22-59 y)] with PTSD meeting research diagnostic criteria for insomnia, randomly assigned to CBT-I (n = 29; 22 females) or monitor-only waitlist control (n = 16; 9 females). Eight-session weekly individual CBT-I delivered and measures included continuous monitoring of sleep with diary and actigraphy; pre-polysomnography and post-polysomnography and Clinician-Administered PTSD Scale (CAPS); and pre, mid, and post self-report questionnaires, with follow-up of CBT-I participants 6 months later. Result shows that CBT-I was superior to the waitlist control condition in all sleep diary outcomes and in polysomnography-measured total sleep time. Compared to waitlist participants, CBT-I participants reported improved subjective sleep (41% full remission versus 0%), disruptive nocturnal behaviors (based on the Pittsburgh Sleep Quality Index-Addendum), and overall work and interpersonal functioning. These effects were maintained at 6-month follow-up. Both CBT-I, and waitlist control participants reported reductions in PTSD symptoms and CAPS-measured nightmares. The study concluded that Cognitive behavioral therapy for insomnia (CBT-I) improved sleep in individuals with posttraumatic stress disorder, with durable gains at 6 months. Overall psychosocial functioning improved following CBT-I. The initial evidence regarding CBT-I, and nightmares is promising but further research is needed. Results suggest that a comprehensive approach to treatment of posttraumatic stress disorder should include behavioral sleep medicine.

To examine gender responsiveness to CBTi, Lami, Martínez, Sánchez, Miró, Diener, Prados, and Guzmán (2016), conducted a study titled “Gender Differences in Patients with

Fibromyalgia Undergoing Cognitive-Behavioral Therapy for Insomnia: Preliminary Data." The aim of this study was to assess the efficacy of cognitive-behavioral therapy for insomnia (CBT-I) in men and women with FM and compare sleep and clinical features between both genders.Fifteen women and 13 men were selected to participate in nine weekly CBT-I sessions that involved completing several self-reported questionnaires at pretreatment, post-treatment, and follow-up. Result reveals that both groups showed significant clinical and statistical improvements in sleep quality. Differential treatment responsiveness between sexes was observed. Male group exhibited significant changes at post-treatment in sleep disturbances compared to female the female.

2.5 Summary

This chapter reviewed literature related to the study of efficacy of Cognitive Behaviour and Relaxation Techniquesinreducing Insomnia. It conceived cognitive behavior technique as a process thathelp people change unhelpful thinking habits and behaviour to healthy ones so as to enhance good sleep quality while, relaxation technique was viewed as a technique which include activities that help a person to relax, and to attain a state of increased calmness by reducing levels of feelings and consequently improve sleep quality. Theoretically, speaking, behavioral model posits that insomnia occurs acutely in relation to both predisposing (trait) and precipitating (state) factors and persist in relation to perpetuating or maintaining factors. Thus, an individual may be prone to insomnia due to trait characteristics, experience acute episodes because of precipitating events, and have chronic insomnia owing to a variety of perpetuating factors. Central to cognitive view is that patients with insomnia often complain that they are unable to sleep because of intrusive

thoughts or excessive worry, but put differently, neurocognitive perspective assert that patients with chronic insomnia are not awake because they are given to rumination and worry, but rather ruminate because they are awake. The theory views maladaptive behaviors as one, which lead to conditioned arousal at bedtime and thus lead to insomnia.

This study is unique fromprevious empirical studies reviewed because it can be observed that most interventions were carried out using patients from general population with or without general medical condition.This study does not use subjectsfrom general population with medical condition or those with ailments but involved only students withoutself-reported ailment. The study comprised of young adult studentsnot >38 years of age unlike most reviewed studies which used older adults. The study also used students who in most cases live on campus dormitories, and compare sex difference in responsiveness to treatment, a gap many empirical studies reviewed on insomniafailed to address. It was also realized that published empirical research work used relaxation technique (progressive muscle relaxation only) for control, but this study employed full package of relaxation technique as an active treatment option to compare its efficacy to CBT.

CHAPTER THREE
METHODOLOGY

3.1 Introduction

The methodology of the study waspresented under the following subheadings;

Research design

Population of the Study

Sample and sampling technique

Instrumentation

Validity of Research Instruments

Reliability of Research Instruments

Procedure for Data Collection

Procedure forData Analysis

3.2 Research design

The study used a quasi-experimental design involving pretest-posttest experimental and control group. This design was helpful as it enables the researcher to purposively, select

sample with the condition of interest for the study. Other reason for using this type of design wasthatit ensuresparticipants are measured on an outcome variable both before and after the intervention of interest hoping that if the intervention is effective, outcome scores should improve compared to control or baseline outcomewhile, scores will hold steady if the intervention has no effect(Mark & Gamble 2009). Max and Lynn(2003), also states that the design is appropriate if the problem being studied has a likely chance of progression during the time in which the study takes place before subsiding due to intervention. The conceptual model of the design is graphically presented as follows:

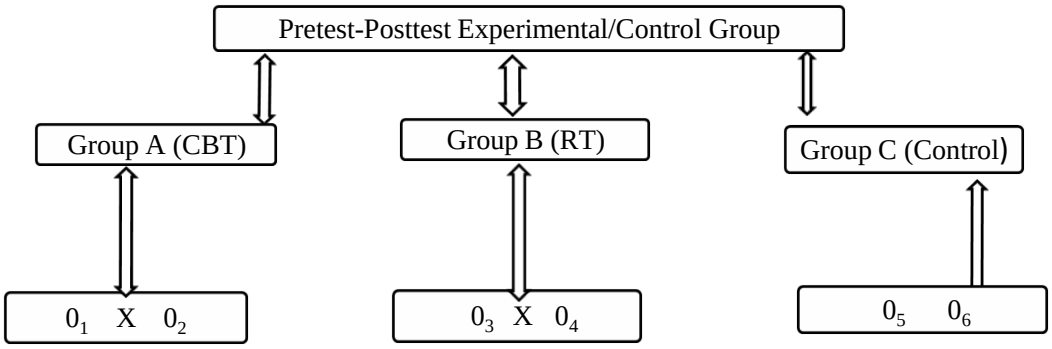


Figure 2: A flowchart of proposed research design for the study

Interpretation

0_1 - Pretreatment

X - Treatment

0_2 - Post treatment

3.3 Population of the study

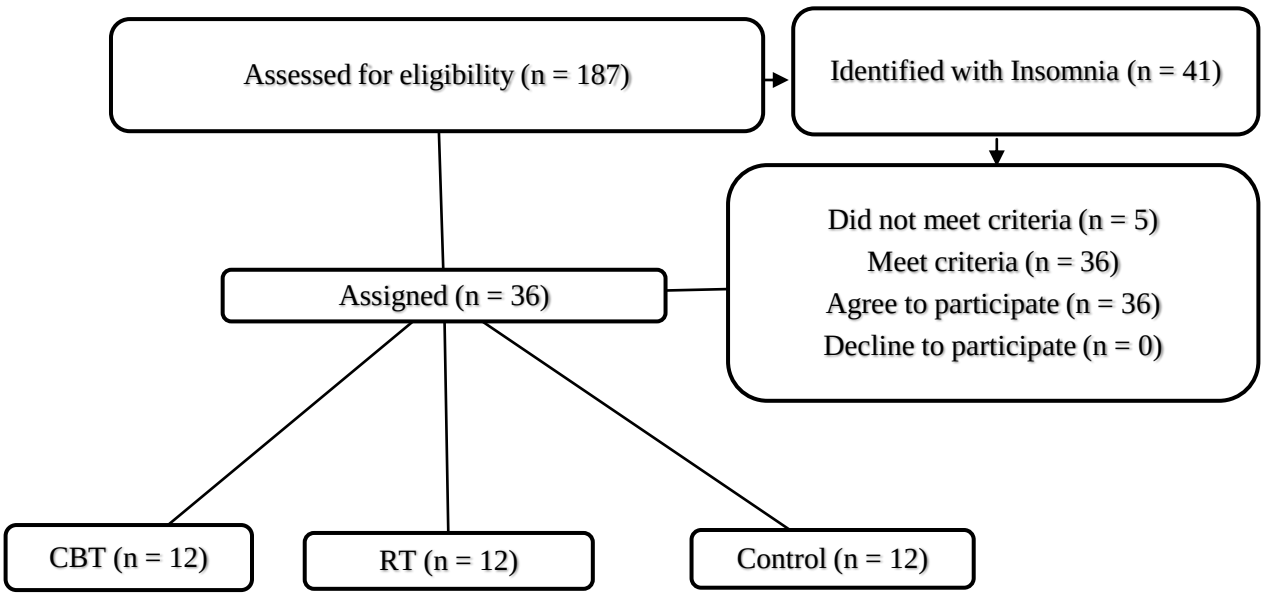
The population of the study includes all young adults'male and female students of Ahmadu Bello University Zaria, whoshowsymptoms of primary insomnia.However, the total number of students identified with the insomnia condition using Insomnia Severity Index were forty-one out of one hundred and eighty-seven contact made. From this figure, five were excluded

from the study due to medical condition that requires independent attention. Ahmadu Bello University, Zaria is adjudged the largest university in Sub-Saharan Africa and the second largest in the continent of Africa. The university boast of an estimated forty-five thousand students comprising undergraduate, and postgraduate undergoing various programs across the thirteen faculties(https://abu.edu.ng/history.html&http://spgs.abu.edu.ng/, 2017). There are students also undergoing various programs at its different institutes. The population of students in this university cut across gender, various ethnic, tribal, religious, cultural and social background and it is true representation of the Nigerian population.

3.4 Sample and Sampling Technique

Atotal sample size of thirty-six (36) participantswith (12) per group were used for the study. VanVoohis and Morgan (2007), and De Winter (2013),state that, depending on the nature, risk, cost,and prevalence of the researched condition, a sample size of 3 – 40 can be used in an experiment.Moreover, that, having a small sample size can give the researcher adequate control over the subjects and eliminate chances of attrition.Purposive sampling technique was used because only studentswho the meet study criteria (those identified with primary insomnia condition) were assigned into experimental and control group. That is, participants were required to (a) score ≥ 8 on the Insomnia Severity Index (ISI), (Bastien, Vallieres, & Morin, 2001); (b) their insomnia must not be because of comorbid condition such as General Medical Condition like; diseases, illnesses, and injuries, Psychiatric or other sleep disorders. These criteria are consistent with elements of a Diagnostic and Statistics Manual of Mental Disorders (DSM-IV) diagnosis of insomnia and quantitative criteria for primary insomnia (Lichstein, Durrence, Taylor, & Riedel, 2003).

The thirty-six students were volunteers willing to participate in the study in order to resolve their insomnia problem. They were gotten via notices placed around hostels, sharing handbills, and through one-on-one contact on campus. Similar strategy for recruiting volunteers for insomnia researchwas also used by Division of Sleep Medicine, Harvard Medical School (2015), Carney, Harris, Moss and Edinger (2010), Adamu (2012), Vitiello et al. (2009), and it is recommended by Agency for Healthcare Research and Quality (2012). The following flowchart shows an overview of the sample:



Source: (the researcher, 2016)

3.5 Instrumentation

Subjective standardized instrumentused and tested in several insomnia research (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin 2006) wasadopted andused to assess the participants during pretreatment and post treatment.The instrument wasdivided into section A = demographic data (sex, age, marital status, personal income, cost of insomnia, and

perceived academic outcome). Section B; consist of standardizedreliable instrument that wasused as outcome measures.

3.5.1 Insomnia Severity Index (ISI)

The Insomnia Severity Indexdeveloped by Bastien, Vallieres and Morin(2001) was used to assess students' insomnia condition. The Insomnia Severity Index (ISI) was designed to be both an*identificationtool* of insomnia and an *outcome measure* for use in insomnia intervention research (Bastien, et al. 2001). It is designed with a five response of none = 0, mild = 1, moderate = 2, severe = 3, very severe = 4. The ISI measured subjective insomnia severity during the previous weeks and the measures is relevant for the entire age range (Edinger, Buysse, Deriy, Germain, Lewin, Ong, & Morgenthaler, 2015). The measure included seven questions bothering on sleep onset, sleep maintenance, and consequences. Thescores ranged from 0 to 28, andtotal scores are categorized into 'absence of insomnia (0-7), 'sub threshold insomnia' (8-14), 'moderately insomnia' (15-21) and 'severe insomnia' (22-28). Consistent with Bastien et al. recommendation, a cutoff of ≥ 8 (Gellis, Arigo & Elliott, 2013)serves as threshold for participation in the study.The ISI is a recommendedtool for assessment of insomniaand is sensitive to intervention (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006).

3.6. Validity of Research Instrument

A group of insomnia research experts produced the Insomnia Severity Index (ISI) that was used in the study, and its content have been validated and approved for use by research supervisors and internal examiners in the Department of Educational Psychology and Counselling, Ahmadu Bello University, Zaria, Kaduna State, Nigeria.Experts in behavioural

and psychological sleep medicine also recommend the instrumentas preferred self-report measure of insomnia condition for studies that evaluate sleep difficulty as an outcome (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin 2006).

3.7 Reliability of Research Instrument

The instrumentused for data collection in the study was anadopted instrument, which yielded an acceptable reliability index and consistency as revealed by the developer.Morin, Belleville, Bélanger and Ivers (2011), reported a Crombach's alpha of 0.90 for Insomnia Severity Index.The ISI has been found to be a reliable and valid instrument to measure perceived insomnia severity (Morin et al., 2011). ISIhas demonstrated 80% sensitivity and specificity (Bastien et al., 2001).The reliability indexwas adequate and consistence with Creswell (2002) recommendation of .70 and above.

3.8 Procedure for Data Collection

The data collection procedure was discussed in three phases; pretreatment phase, treatment phase, and post-treatmentphase. Control of extraneous variables were also discussed.

Pretreatment Phase (week 1)

Beforethe placement of notices, and administration of instrument, a letter of introduction was collected from the Department of Educational Psychology and Counselling, which the researcher used to introduce himself to the research participants. Time was taken to socialize and create a good rapport with the students. After that, the students were briefed on the essence of the study.In addition, consent was obtained from the participants after which they filledquestionnaires that were used toidentifythem for insomnia, and insomnia associated

with other medical, psychiatric/other sleep conditions.Data collected at this point served as baseline or pretest data, and bases for inclusion/exclusion. Participants were instructed on how to fill the instruments, after which arrangements were made regarding further meetings.

Treatment Phase (week 2-5)

Twodifferent intervention procedures(see appendix II page 161 and 166 for the treatment manual) weregiving tothe two treatment groups at varied time of the day. The CBT for insomnia intervention used was based on two treatment manuals developed by Morin and Espie (2003), and Perlis et al (2005), while the Relaxation Technique used was based on Ethan (2014), and National Sleep Foundation (2015), recommendations for reducing insomnia respectively. Other relaxation recommendations used were duly acknowledged. The interventions are those that have been shown efficacious in prior researches and have been recommended by the American Academy of Sleep Medicine for the treatment of insomnia (Morgenthaler et al., 2006, NFS, 2015). However, the interventions in brief, starting with activities from week two – six are as follows;

Weeks	Cognitive Behaviour Technique	Brief
Wk 2	Stimulus control	Instructions designed to associate the bed/bedroom with sleep and to reestablish a consistent sleep-wake schedule
Wk 3	Sleep restriction	A method for strict schedule of bed times and wake times with no midday naps which results in more consolidated and more efficient sleep
Wk 4	Sleep hygiene	General guidelines about health practices and education, environmental factors that may affect sleep
Wk 5	Cognitive technique	It is a psychological method aimed at counteringnegative intrusive thoughts at bedtime and belief about sleep
Wk 6	Posttest	Revision and re-administration of the instrument
	Relaxation Technique	
Wk 2	Deep breathing exercise	It is encouraged so that individual can have a controlled breath that can release tension, worries and control racing heart rate at bed time
Wk 3	Progressive muscle relaxation techniques	Exercise aimed at inducing a state of deep muscular relaxation as well as helping one to learn the difference between tension

		and relaxation
Wk 4	Guided visualization meditation	GVM was instructed by encouraging positive feelings of peacefulness. The idea in this exercise is to focus attention on a pleasant image or story, so that the mind can let go of worries or thoughts that keepsone awake.
Wk 5	Mindfulness technique	It is instructed so that individual can stop thinking too much once they've gone to bed, and it will help them to learn to calm their mind and do away with racing thoughts and worries.
Wk 6	Posttest	Revision and re-administration of the instrument

The treatment sessionstook place on Saturdays with those receiving cognitive behaviour and relaxation techniques undergoingsix-week intervention sessions, which took place during first semester, 2016.After each week of intervention, participants were given a handout containing treatment recommendations, to serve as a guide or reminder during practice. All treatments instructions weredelivered around the participants' hostel at an agreed time and sometimes at a serene location for convenience. The average intervention time was 48 minutes with a minimum of 30 minutes to maximum of 1 hour. All these were carried out with the help of a female research assistant who was taught on the study protocol.Although,eachsessionconducted with the two treatment groups differsin specific content, all sessions center onthe following main activities: (i) finding out about progress(ii) ascertaining problems in home practice (iii) encouraging levelof adherenceand (iv)introducingnew intervention.

Post-treatment Phase (week 6)

During this phase, post intervention test using Insomnia Severity Index was administered. This was used to compare with pretest data to examine changes that have occur over the course of the intervention. Before termination, the researcher informed the participants on what to do when there is a relapse (fall back to the manual). For ethical reason, control group were given the intervention manual for self-help. Finally, the participants were made

to understand that longitudinal data suggests that treatment gains are extremely durable with time. They are most often maintained or improved over time, and that relapse rates will be small provided they continue goodhabits (abstain from things that precipitate and perpetuate the condition in the first place).

Control of extraneous variables

Some of the measures that were taken include;

i. Only students who scored ≥ 8 on the Insomnia Severity Index (ISI) were used.In addition, Insomnia Screening Questionnaire(Alberta Health and Wellness, 2007) was used to assess forabsence or presence of a medical/psychiatric condition, substance abuse disorder, and other sleep disorder. These criteria are consistent withDiagnostic and Statistics Manual of Mental Disorders (DSM-5) algorithm for diagnosis of primary insomnia (DSM, 2013; Lichstein, Durrence, Taylor, & Riedel, 2003; Morin & Espie, 2003; and American Academy of Sleep Medicine, 2005).
ii. More often, participants were encouraged to ensure compliance and practice as homework, all the RT and the CBT instructions received.
iii. In order to prevent or reduce interaction effect, different locations were usedat different time of the day (Saturday) for administering the intervention.

3.9 Procedure for Data Analysis

Data collected weresorted and subjected to statistical analysis using Statistical Package for the Social Sciences (IBM SPSS version 24). The five research questions andfive null hypotheses wereanswered and tested with varied tools. Frequency and percentage wasused to analyze subject's demographic data while, mean and standard deviation was used to

answer research questions. All the null hypothesesstated for the study were tested usinga one-way between groupanalyses of covariance with pretest scores of both groups used as the covariate. The justification for using ANCOVA is that, it is appropriate when a researcher has two or more groups subjected to pre-test/post-test (for example; comparing the effects of two or more different interventions, taking before and after measures for each group). The scores on the pre-test are treated as a covariate to 'control' for pre-existing differences between the groups(Pallant, 2005, 2011).Decisionto reject or retain theoutcome ofhypotheses testing was made at the .05 significance level.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Introduction

The major goal of this study was to assess the efficacy of cognitive behaviour and relaxation techniques on insomnia among students of Ahmadu Bello University, Zaria. The data collated at pre and post intervention stages were sortedand analysed statistically with the help of Statistical Package for the Social Sciences (SPSS version 24). Descriptive statistics of frequency and percentage was used to analyse the demographic characteristics of the participants while mean and standard deviation was used for the research questions. One-way analysis of covariance was used to test the stated null hypotheses at the 0.05 significance level.

4.2 Demographic Characteristics of the Participants

The demographic characteristics of the participants used in this study are analysed and presented in table 4.1

Table 4.1 Descriptive Statistics of theDemographic Characteristics of Participants

Variable	***Option***	***Frequency***	***Percent***	***Cumulative Percent***

Age range	18-22	15	41.7	41.7
	23-27	13	36.1	77.8
	28-32	5	13.9	91.7
	33-38	3	8.3	100.0
	Total	36	100.0	
Marital Status	Married	4	11.1	
	Single	32	88.9	
	Total	36	100.0	

Table 4.1 reveals that 15 participants representing 41.7% of the total number of subjects that participated in the study were <22 years of age. Those within the age range of 23 – 27 (n = 13), constitute 36.1% of the research participants. This suggests that out of every ten (10) participants that participated in the study, almost eight (8) of them were <27 years old, and cumulatively formed 77.8% of the research participants. Only 13.9% (n = 5) and 8.3% (n = 3) were within the age range of 28 – 32 and 33 – 38 respectively. Cumulatively, they (n = 8) constitute 22.2% of the total number of participants used in the study.

The table also reveals the marital status of the research participants. It shows that 88.9% (n = 32) of the participants were single while 11.1% (n = 4) were married.

4.3 Hypotheses Testing

In testing the five null hypotheses stated for the study, a one-way analysis of covariance was used to control for pre-intervention differences in participant's score on the insomnia severity index. In the following subsections, the research questions were answered with their corresponding hypotheses.

Research Question 1: what is the effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?

To answer research question one, themean and standard deviation of the participant's pretest and posttest scoreswerepresented in table 4.3.

Table 4.2 Mean and Standard Deviation of Pre Post Insomnia Condition of CBTi and CTL

Condition	***Intervention***	***N***	***Mean***	***Std. Deviation***
Pretest Insomnia	CBT	12	14.75	4.18
	Control group	12	10.92	2.02
	Total	24	12.83	3.76
Posttest Insomnia	CBT	12	5.08	.79
	Control group	12	9.92	2.84
	Total	24	7.50	3.20

Table 4.2 shows the pretest and posttest mean scores and standard deviation of participants in the intervention group and control group. It reveals that at pretest, students assigned to receive cognitive behaviour technique had a higher mean score (14.75±4.18) when compared to students in the control group (10.92±2.02). However, after treatment, students in the intervention group had a mean score lower (5.08±.79) than control group (9.92±2.84). This outcome suggests that the intervention may have an effect with intervention group having a better within group average remission rate of 65.6%when compared to 9.2% for the control group.

Null Hypothesis 1: There is no significant effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

To test the null hypothesis one, aone-way analysis of covariance was carried out and pretest scores of students who received CBT and that of CTL were used as the covariate to control for initial differences. The result is presented in table 4.3.

Table 4.3 One-way Analysis of Covariance on Efficacy of CBTagainsta Control Group

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	140.167[a]	2	70.083	15.357	.000
Intercept	76.233	1	76.233	16.705	.001
Pretest Insomnia	.000	1	.000	.000	.996
Group	102.295	1	102.295	22.416	.000
Error	95.833	21	4.563		
Total	1586.000	24			
Corrected Total	236.000	23			

a. R Squared = .594 (Adjusted R Squared = .555)

Table 4.3shows a one-way analysis of covariance,which was conducted to compare the effectiveness of cognitive behaviour technique interventions designed to reduce participants' insomnia condition against an untreated control group. Pretest scores of participantsonInsomnia Severity Index were used as the covariate in this analysis. After adjusting for the covariate, there was a statistically significant effect of cognitive behaviour technique in reducing insomnia when compared to control group on post-intervention scores, $F(1,21)= 22.416$, $p = 0.000$. The R Squared value suggeststhat 59.4% of variance in the post intervention scores can be explained by the intervention. It can therefore, be said that to a large extend;CBT was effective in reducing insomnia condition. Therefore, the null hypothesis which states that there is no significant effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria is rejected.

Research Question 2: What is the effect of relaxation techniqueon insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?

To answer research question two, themean and standard deviation of the participant's pretest and posttest scores wereused as presented in table 4.4.

Table 4.4 Means and Standard Deviation of Pretest and Posttest Insomnia Condition of Students Exposed to Relaxation Technique and the Control Group

Condition	***Intervention***	***N***	***Mean***	***Std. Deviation***
Pretest Insomnia	Relaxation Technique	12	12.42	2.64
	Control group	12	10.92	2.02
	Total	24	11.67	2.43
Posttest Insomnia	Relaxation Technique	12	6.75	1.36
	Control group	12	9.92	2.84
	Total	24	8.33	2.71

Table 4.4 shows the pretest and posttest mean scores of participants in the intervention group and control group. It reveals that at pretest, students assigned to receive relaxation techniquefor insomnia had a higher mean score (12.42±2.64) when compared to students in the control group (10.92±2.02). However, after treatment, students in the intervention group had a mean score lower (6.75±1.36) than control group (9.92±2.84). This outcome suggests that the intervention may have an effect with intervention group having a better within group average remission rate of 45.6%when compared to 9.2% for the control group.

Null Hypothesis 2: There is no significant effect of relaxationtechnique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

To test the null hypothesis two, aone-way between-group analysis of covariance was carried out and the pretest mean scores of students who were exposed to relaxation technique, and that of control group were used as the covariate to control for initial differences. The result is presented in table 4.5.

Table 4.5 One-way Analysis of Covariance on the Effect of Relaxation Technique against Control group

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	63.844[a]	2	31.922	6.355	.007
Intercept	34.319	1	34.319	6.832	.016
Pretest Insomnia	3.677	1	3.677	.732	.402
Group	63.447	1	63.447	12.630	.002
Error	105.489	21	5.023		
Total	1836.000	24			
Corrected Total	169.333	23			

a. R Squared = .377 (Adjusted R Squared = .318)

Table 4.5shows a one-way analysis of covariance (ANCOVA) which was used to compare the effectiveness of relaxation technique designed to reduce participants' insomnia condition against a control group. The test was carried out with participants' pretest scores on Insomnia Severity Index used as the covariate. After adjusting for the covariate, the analysis found a statistically significant effect of relaxation technique in reducing insomnia when compared to the participants in the control group, $F(1,21) = 12.630$, $p = 0.002$. The R Squared value indicates that 37.7% of the variance in post intervention scores as measured by the insomnia severity index can be explained by the intervention.Thus, relaxationtechnique was effective in reducing insomnia condition. Therefore, the null hypothesis which states that there is no significant effect of relaxationtechnique oninsomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeriais rejected.

Research Question 3: What is the differential effects of cognitive behaviour and relaxation techniqueon insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?

To answer research question three, the mean and standard deviation of the participant's pretest and posttest scores werepresented in table 4.6.

Table 4.6 Means and Standard Deviation of Pretest and Posttest Insomnia of Students Exposed to Cognitive Behaviour technique and those Exposed to Relaxation Technique

Condition	***Intervention***	***N***	***Mean***	***Std. Deviation***
Pretest Insomnia	Cognitive Behaviour Technique	12	14.75	4.18
	Relaxation Technique	12	12.42	2.64
	Total	24	13.58	3.62
Posttest Insomnia	Cognitive Behaviour Technique	12	5.08	.79
	Relaxation Technique	12	6.75	1.37
	Total	24	5.93	1.38

Table 4.6 shows the pre-intervention and post intervention mean scores of participants exposed to cognitive behaviour technique and those exposed to relaxation technique. It reveals that at pretreatment stage, students assigned to receive cognitive behaviour technique for insomnia had a higher mean score (14.75±4.18) when compared to students in the relaxation technique group (12.42±2.64). However, after treatment, students exposed to cognitive behaviour technique had a mean score lower (5.08±.79) than those exposed to relaxation technique (6.75±1.37). This outcome suggests different treatment effect with a better average remission rate of 65.6% for cognitive behaviour technique group compared to 45.6% for those exposed to relaxation technique.

Null Hypothesis 3: there is no significant differential effect of cognitive behaviour and relaxation techniqueson insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

To test null hypothesis three, aone-way analysis of covariance was carried out and the pretest mean scores of participantsin both interventions were used as the covariate to control for initial differences. The result ispresented in table 4.7.

Table 4.7 One-way Analysis of Covariance on the Differential Effect of Cognitive Behaviour and Relaxation Technique on Insomnia

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	16.779[a]	2	8.390	6.512	.006
Intercept	43.732	1	43.732	33.946	.000
Pretest Insomnia	.112	1	.112	.087	.771
Group	15.725	1	15.725	12.206	.002
Error	27.054	21	1.288		
Total	884.000	24			
Corrected Total	43.833	23			

a. R Squared = .383 (Adjusted R Squared = .324)

Table 4.7shows a one-way analysis of covariance (ANCOVA) which was used to test null hypothesis three and the essence was to compare the differential effectiveness of cognitive behaviour technique against a relaxation techniquedesigned to reduce participants' insomnia condition. The test was conducted using participants' scores on the pre-intervention administration of the Insomnia Severity Index as the covariate. After adjusting for the covariate, the study finds a statistically significant differentialeffect of cognitive behaviour technique in reducing insomnia when compared to relaxation technique, $F(1,21) = 12.206$, p=0.002. The R Squaredvalue indicates that 38.3% of the variance in post intervention scores as measured by the insomnia severity index can be explained by the intervention after adjusting for covariate. Therefore, the null hypothesis which states that there is no significant differential effect of cognitive behaviour and relaxation techniques on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeriais rejected.

Research Question 4: what is the differentialeffect of cognitive behaviour technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?

To answer research question four, themean and standard deviation ofpretest and posttest scores of male and female participants arepresented in table 4.8.

Table 4.8 Means and standard deviation of Pretest and Posttest Insomnia of Male and Female Subjects Exposed to Cognitive Behaviour Technique

Condition	***Sex***	***N***	***Mean***	***Std. Deviation***
Pretest Insomnia	Male	6	14.67	2.42
	Female	6	14.83	5.71
	Total	12	14.75	4.18
Posttest Insomnia	Male	6	5.17	.41
	Female	6	5.00	1.09
	Total	12	5.08	.79

Table 4.8 shows the pre-intervention and post intervention mean scores of male and female participants exposed to cognitive behaviour technique for insomnia. It reveals that at pretreatment stage, male subjects assigned to receive cognitive behaviour technique for insomnia had a lower mean score (14.67±2.42) when compared to female subjects (14.83±5.71). However, after treatment, male subjects exposed to cognitive behaviour technique had a mean score higher (5.17±.41) than the female counterpart (5.00±1.09). This outcome may suggest a better average remission rate of 66.3% for the female subjects when compared to 64.7% for the male subjects.

Null Hypothesis 4: there is no significant differential effect of cognitive behaviour technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

To test null hypothesis four, aone-way analysis of covariance was carried out and the pretest mean scores of male and female participants were used as the covariate. The result is presented in table 4.9.

Table 4.9 One-way Analysis of Covariance on differential Effect of Cognitive Behaviour Technique on Insomnia of Male and Female Students of Ahmadu Bello University, Zaria

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	.389[a]	2	.195	.268	.771
Intercept	26.466	1	26.466	36.491	.000
Pretest Insomnia	.306	1	.306	.422	.532
Gender	.077	1	.077	.106	.752
Error	6.527	9	.725		
Total	317.000	12			
Corrected Total	6.917	11			

a. R Squared = .056 (Adjusted R Squared = -.153)

Table 4.9shows a one-way analysis of covariance (ANCOVA) which was used to compare the responsiveness between male and female participants exposed to cognitive behaviour technique. Participants' scores on the pre-intervention administration of the Insomnia Severity Index was used as the covariate. After adjusting for the covariate, the result of the analysis suggests there was no statistically significant variation in responsiveness between male and female students who received cognitive behaviour technique for insomnia, $F(1,9) = .106$, $p = 0.752$.The R Squared value of .056 indicates that only 5.6% of the variance in post intervention scores can be explained by the gender after adjusting for covariate. The non-significant differential effect outcomesuggests that the intervention (CBT) was just as effective for the male as it was for the female as both group show good level of improvements after intervention. Therefore, the null hypothesis which states that there is no significant differential effect of cognitive behaviour technique on insomnia condition of

male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria is retained.

Research Question 5: what is the differentialeffect of relaxation technique on insomnia of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria?

To answer research question five, themean and standard deviation of the male participant's pretest and posttest scores as measured by insomnia severity index was conducted using descriptive statistics and the result is presented in table 4.10.

Table 4.10 Means, Standard Deviation of Pretest, Posttest Insomnia Condition of Male, and Female Subjects Exposed to Relaxation technique for Insomnia

Condition	***Sex***	***N***	***Mean***	***Std. Deviation***
Pretest Insomnia	Male	6	14.00	2.19
	Female	6	10.83	2.14
	Total	12	12.42	2.64
Posttest Insomnia	Male	6	6.83	1.47
	Female	6	6.67	1.37
	Total	12	6.75	1.36

Table 4.10 shows the pre-intervention and post intervention mean scores of male and female participants exposed to relaxation techniquefor insomnia. It reveals that at pretreatment stage, male subjects assigned to receive relaxation technique for insomnia had a higher mean score (14.00±2.19) when compared to female subjects (10.83±2.14). However, after treatment, male subjects exposed to relaxation technique for insomnia had a mean score higher (6.83±1.47) than their female counterpart (6.67±1.37). This outcome suggests a better

average remission rate of 51.2% for the male subjects when compared to 38.4% for female subjects.

Null Hypothesis 5: There is no significant differential effect of relaxation technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria

To test null hypothesis five, aone-way analysis of covariance was carried out and pretest mean scores of male and female participants were used as the covariate. The result is presented in table 4.11.

Table 4.11 One-way Analysis of Covariance on the Effect of Relaxation Techniqueon Insomnia condition of Male and Female Students of Ahmadu Bello University, Zaria

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	2.990[a]	2	1.495	.779	.487
Intercept	3.962	1	3.962	2.066	.184
Pretest Insomnia	2.906	1	2.906	1.515	.250
Gender	.707	1	.707	.369	.559
Error	17.260	9	1.918		
Total	567.000	12			
Corrected Total	20.250	11			

a. R Squared = .148 (Adjusted R Squared = -.042)

Table 4.11show a one-way between-group analysis of covariance test of null hypothesis five, to compare the responsiveness between male and female participants exposed to relaxation techniquefor insomnia. After adjusting for the covariate, the result of the analysis suggests there was no statistically significant variation between male and female students' responsiveness to relaxation technique intervention for insomnia, $F(1,9) = .369$, p =

0.559.The R Squared value of .148 indicates that only 14.8% of the variance in post intervention scores can be explained by gender after adjusting for covariate. The non-significant between group outcomessuggests that male and female responsiveness to relaxation technique for insomnia wasthe same as both group show good level of improvements after intervention. Therefore, the null hypothesis which states that there is no significant differential effect of relaxation technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria is retained.

4.4 Summary of Major Findings

The findings of the study established that:

i. Cognitive behaviour technique significantly reduces insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,$F(1,21) = 22.416$, p= 0.000.When compared to control group, subjects exposed to cognitive behaviour technique for insomnia achieved a significant remission of averagely 65.6% as measured by Insomnia Severity Index.

ii. Relaxation technique significantly reduces insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,$F(1,21) = 12.630$, p =0.002.This result also suggests that when compared to control group, subjects exposed to relaxation technique for insomnia achieved significant remission of averagely 45.6% in their scores on ISI.

iii. Both cognitive behaviour technique and relaxation techniquewere effective in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,$F(1,21) = 12.206$, $p = 0.002$. However, students treated with cognitive

behaviour technique achieved anaverage65.6% remission when compared to 45.6% for relaxation technique.

iv. Male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria do not significantlydiffer in their response to cognitive behaviour technique forinsomnia,$F(1,9) = .106$, $p = 0.752$.

v. No significant differentialeffect of relaxation technique on insomnia condition of maleandfemale students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,$F(1,9) = .369$,$p = 0.559$.

4.5 Discussion

The findings of this studyreveals that students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria who were exposed to cognitive behaviour technique had reduced insomnia.This suggests that when compared to control group, subjects exposed to six weeks CBT show significant improvement with a higher between and within group remission rate of about 65.6%. This result corroborates with previous finding of Edinger et al (2009),who reported that CBT is effective in treating insomnia condition. In addition, CBT outperformed sleep hygiene (control) across several study outcome measures for the sample as a whole. Edinger et al.(2009) conclude that CBT is a viable psychological insomnia therapy both for those with primary insomnia and for groups composed mainly of patients with insomnia and nonpsychotic psychiatric conditions. It also corroborates earlier finding byFornal–Pawłowska and Szelenerger (2013), who reported that after the therapy, patients did not differ significantly from good sleepers in number of awakenings, sleep quality, and feeling in the morning. Fornal–Pawłowska and Szelenerger (2013), concluded that the CBT

produced a sustained, clinically meaningful improvement in nocturnal sleep and daytime functioning. In addition, Kaldo et al. (2015), result also shows that CBT was significantly more effective than the control treatment in reducing Insomnia Severity.Similar finding by Morgan et al. (2004), also showed that at 3- and 6-month follow-ups, patients treated with CBT showed improved global PSQI scores as well as improvements in the SF-36 dimensions of vitality at 3 months and physical functioning and mental health at 6 months. CBT-treated patients also reported reductions in the frequency of hypnotic drug use compared with the control group, with many CBT treated patients reporting zero drug use at the follow-up assessments. Clinical improvements were maintained within the CBT group at the 12-month follow-up, with PSQI scores and the frequency of hypnotic drug use continuing to show significant reductions relative to the control group. Concluded that in routine general practice settings, psychological treatment for insomnia can improve sleep quality, reduce hypnotic drug use, and improve health- related quality of life at a favourable cost among long-term hypnotic users with chronic sleep difficulties. These positive outcomes appear robust over time, persisting for at least 1 year among the more treatment-adherent patients.

Findings also shows that students exposed to relaxation technique had reduced insomnia.When compared to control group, this result suggests that subjects exposed to relaxation technique show significant improvement with a higher between and within group remission rate. This outcome corroborates previous findings byAhmed and Younis (2014), who reported that relaxation technique improved the total score of sleep quality. It showed that the mean score of total Pittsburgh Sleep Quality Index decreased significantly after

demonstration of relaxation techniques compared with the mean scores of total Pittsburgh Sleep Quality Index one month before application of relaxation techniques. It also corroborates results presented by Deora and Anthony (2013), which showed that Sleep hygiene education (SHE) and relaxation training were effective in treating insomnia of depressed patients. Therefore, Deora and Anthony (2013), concluded that Implementation of sleep hygiene education and relaxation training will provide nurses with evidence-based treatment alternatives or complements to pharmacotherapy in depressed patients. Cooke (2015) also reported that progressive Muscle Relaxation (PMR) reduces feelings of tension, to lower perceived stress, and to induce relaxation. That PMR decreases the arousal of the autonomic and central nervous system and to increase parasympathetic activity. Reports of patients who participated in PMR training following cancer treatment indicate that they experienced reduced state anxiety, pain, and symptoms of depression, as well as improvements in sleep parameters and overall quality of life. The evidence is however insufficient due to the limited number of trials per outcome and methodological limitations of published studies. PMR is considered to have few adverse effects, although some concern has been raised about the use of relaxation therapy interventions among individuals who have a history of psychiatric disorders.

The study also reveals differential effect of cognitive behaviour and relaxation technique in reducing insomnia among students of Ahmadu Bello University, Zaria. It suggests that when compared to relaxation group who achieved 45.6% remission after intervention, subjects exposed to cognitive behaviour technique for insomnia show significant improvement of about 65.6% in their post intervention score as measured by insomnia severity index. The

reason for the significant differential effect in the intervention, which favoured cognitive behaviour techniquegroup, could be the fact that they were introduced to a combined intervention of both behavioural and cognitive techniques while, relaxationtechnique group were exposed to a standalone technique. This findingcorroborates initial result by Edinger et al. (2001), which showed that Cognitive behavioral therapy produced larger improvements across the majority of outcome measures than did Relaxation Training. For example, sleep logs showed that CBT-treated patients achieved an average 54% reduction in their WASO whereas RT-treated and placebo-treated patients, respectively, achieved only 16% and 12% reductions in this measure. Recipients of CBT also showed a greater normalization of sleep and subjective symptoms than did the other groups with an average sleep time of more than 6 hours, middle WASO of 26.6 minutes, and sleep efficiency of 85.1%. In contrast, RT-treated patients continued to report a middle WASO of 43.3 minutes and sleep efficiency of 78.8%. Edinger et al. (2001) concluded that CBT represents a viable intervention for primary sleep-maintenance insomnia because it leads to clinically significant sleep improvements within 6 weeks and these improvements appear to endure through 6 months of follow-up.Trauer, Qian, Doyle, Rajaratnam, and Cunnington, (2015) in their investigation on Cognitive Behavioral Therapy for Chronic Insomnia using A Systematic Review and Meta-analysis incorporated at least 3 CBT-i approaches: cognitive therapy, stimulus control, sleep restriction, sleep hygiene, and relaxation. At the posttreatment time point, SOL improved by 19.03 (95% CI, 14.12 to 23.93) minutes, WASO improved by 26.00 (CI, 15.48 to 36.52) minutes, TST improved by 7.61 (CI, 0.51 to 15.74) minutes, and SE% improved by 9.91% (CI, 8.09% to 11.73%), and changes seemed to be sustained at later time points.

No adverse outcomes were reported. CBT-i is an effective treatment for adults with chronic insomnia, with clinically meaningful effect sizes.

The findings did not reveal significant gender bias to the intervention. Both male and female participants who were introduced to cognitive behaviour technique and those exposed to relaxation technique hada significant reduction in their insomnia.This means that CBT is just as effective for the male as it was for the female participants. Similarly, relaxation technique was also found to be as effective in improving insomnia condition of the male subjects asthe female subjects. This findings, although, corroborate Lami, et al. (2016) report in their study on "Gender Differences in Patients with Fibromyalgia Undergoing Cognitive-Behavioral Therapy for Insomnia", that both groups showed significant improvements in sleep quality, and also other researchers (e.g., Edinger et al. 2001; 2009; Fornal–Pawłowska & Szelenerger 2013), who reported significant improvement in all sleep parameters at post intervention for both male and female subjects exposed to cognitive behaviour technique, and relaxation exercise (Ahmed & Younis 2014; Deora & Anthony 2013), it differ in other aspect as Lami, et al. (2016) observed differential treatment response between sexes. Male group exhibited significant changes at post-treatment in sleep disturbances compared to the female. A claim this study could not establish. One reason could be the small sample size of six males and six females used. Usually, smaller sample have lower tendency or power of detecting significant effect. Other reasons could be severity of the insomnia condition of the participants. Most of them have subthreshold to moderately severe level of insomnia, which is quite different from the chronic or very severe insomniac participants used by Lami, et al.

(2016). In addition, while Lami, et al.study was on comorbid insomnia, this study was conducted on students with primary sleep complain.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

5.1 Summary

The major goal of the study was to assess the efficacy of cognitive behaviour and relaxation technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria. To create a pathway for the study, five research objectives and corresponding research questions were used as guide. The specific hypotheses stated and tested in this study include; (i). there is no significant effect of cognitive behaviour technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria (ii) there is no

significant effect of relaxation technique oninsomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria(iii) there is no significant differential effect of cognitive behaviour and relaxation technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria (iv) there is no significant differential effect of cognitive behaviour technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria (v) there is no significant differentialeffect of relaxation technique on insomnia condition of male and female students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria.

To achieve the above, related literatures were consulted which offer insight the concept of insomnia and previous similar studies. Previous studies offer explanation about the efficacy of CBT and RT in treatment of insomnia of different sample. Some theories that were used to offer insight as regards to the insomnia condition include; 3ps theory of insomnia, selective attention bias, cognitive theory, hyper-arousal theory of insomnia, and sleep-wake regulation theory of insomnia. The basic assumption of 3Ps theory is that insomnia occurs acutely in relation to both predisposing (trait) and precipitating (state) factors and occurs chronically in relation to perpetuating or maintaining factors. Thus, an individual may be prone to insomnia due to trait characteristics, experience acute episodes because of precipitating events, and have chronic insomnia owing to a variety of perpetuating factors.Selective attention bias theory posits that inappropriate arousal may then lead to selective attention to sleep-related cues (implicit or explicit) and increased explicit intention and effort to sleep, which further inhibit normal sleep-related de-arousal. While, cognitive theory belief that thinking negative about oneself or situation affect the mood and in turn

affect sleep, hyper-arousal theory of insomniaposit that increased sensory processing is thought to interfere with the ability to initiate sleep.

The study used aquasi-experimental design involving pretest posttest, control/treatment groupbecause it helps in instances where it is difficult for the researcher to have adequate control over extraneous variable. It also allows participants to be measured on an outcome variable both before and after the intervention of interest with the hope that if the intervention is effective, outcome scores should improve compared to control or baseline outcome while, scores will hold steady if the intervention has no effect. Thirty-six (36) participants who were identified with primary insomnia after pretest administration of Insomnia Severity Index structured on a five points response format, and have agreed to participate were divided into three groups of twelve each and used for the study with two groups given intervention and one used as control group. Participants with secondary insomnia were excluded from study. They were identified with the help of Insomnia Screening Questionnaire. After intervention ({CBT = sleep time schedule, stimulus control, cognitive strategy, and education about sleep hygiene} and {RT = deep breathing exercise, progressive muscle relaxation, guided visualization and mindfulness sleep induction technique}), which lasted for a period of six weeks, participants were posttestedusing the Insomnia Severity Index, and were given a comprehensive manual containing step by step treatment delivery to help them in case of relapse. They were made to understand that, treatment gains are most often maintained or improved over time. That, relapse rates will be small provided all things remain equal, and that they continue good habits that aid sleep. For ethical reasons, control group were also given the treatment handout to use it as a self-help method to improve their insomnia condition.

The data collected at pre and post intervention stages were sorted and subjected to analysis using descriptive statistic for the research question and one-way between group analysis of covariance statistics for the hypotheses and the results were presented in tables in chapter four. All null hypotheses were tested at the 0.05 significance level. The result of analysis reveals a significant effect of cognitive behaviour technique in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria, suggesting that when compared to control group, subjects exposed to CBT show significant decreaseof about 65.6% in their insomnia condition. There was also a significant effect of relaxation technique in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria. When compared to control group, the outcome suggests that subjects exposed to RTachieved an average of 45.6% decrease or improvement in their insomnia condition at post intervention level. There was a significant differential effect of cognitive behaviour technique and relaxation technique in reducing insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria. The outcome suggests that when compared to 45.6% decreased in insomnia among subjects that were exposed to relaxation technique, those exposed to CBTshow significant decrease or improvement of about 65.6% in their insomnia condition at post intervention stage. However, no significant differential effect of cognitive behaviour technique in reducing insomnia of male and female students ofAhmadu Bello University, Zaria, Kaduna State, Nigeria was found. This suggests that CBT was just as effective for the male as it was for the female. Similar finding was also observed for subjects in relaxation technique group as no statistically significant differential effect of relaxation techniquein reducing insomnia of male and female students of Ahmadu Bello

University, Zaria, Kaduna State, Nigeria was found, thus suggesting that RT was as effective for the male subjects, so it was for the female.

5.2 Conclusion

Based on the outcome of the study, it was concludedthatsix weeks intervention using cognitive behaviour technique reduces insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria. Relaxation techniquealso successfullyreducesinsomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria.Comparatively, students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria exposed to cognitive behaviour technique had higher remission rate of about 65.6% when compared tothose exposed to relaxation techniquewho reported a success rate of 45.6%.Lastly, the study concluded that, male and female student's responsiveness to CBT and RT were successful and both interventions do not discriminate on the bases of gender.

5.3 Contributions to Knowledge

In assessing the efficacy of cognitive behaviour and relaxation technique on insomnia among students of Ahmadu Bello University, Zaria, Kaduna State, Nigeria,it was established that:

i. Students of Ahmadu Bello University, Zaria exposed to cognitive behaviour technique had a reduced insomnia ($p = 0.001$)
ii. Students of Ahmadu Bello University, Zaria exposed to relaxation technique had a reduced insomnia ($p = 0.002$)

iii. Students of Ahmadu Bello University, Zaria exposed to cognitive behaviour technique had a higher remission on their insomnia compared to those exposed to relaxation technique ($p = 0.002$)

iv. No difference in responsiveness between male and female students of Ahmadu Bello University, Zaria exposed to Cognitive behaviour ($p = 0.752$),

v. Both male and female students of Ahmadu Bello University, Zaria exposed to relaxation techniques had reduced insomnia ($p = 0.559$)

5.4 Recommendations

Based on the findings of the study, the following recommendations were made;

i. Psychologists, counsellors, other practitioners on insomnia related matters should be encouraged touse cognitive behaviour technique as an intervention to helpyoung adults' university students who are having sleep difficultyto resolve such difficulty and consequently regain control over their sleep.

ii. The university health center should be encouraged to collaborate with psychologists and counsellors to utilize relaxation techniquesas an intervention for insomnia condition to helpyoung adults' universitystudents with sleep difficulty achieve relaxation at bedtime and initiate sleep with ease.

iii. While both interventions reduced insomnia, psychologists and counsellors are recommended toexplore cognitive behaviour technique as a more viable option and first line of intervention to help students manage their sleep difficulty since it shows good level of effectiveness compared to relaxation technique.

iv. Cognitive behaviour technique should be used by school psychologists and counsellors, and other practitionersto help male and female students with complain of sleep difficultyto enhance their sleep quality and daytime performance.

v. School psychologists, counsellors, and teachers should use relaxation techniqueto help both male and female students with insomnia to manage their condition since the intervention is not gender bias.

5.5 Suggestions for Further Studies

This study is by no means exhaustive, it is suggested that future study should be undertaken to determine:

i. Efficacy of cognitive behaviour technique and stimulus control instruction in managing insomnia among diploma, postgraduate, or undergraduate students.

ii. Efficacy of cognitive behaviour and relaxation technique onuniversity students with severe insomnia, and other psychological related problems such as grief, disruptive behaviour, cruelty, mood swings, depression, and inattentiveness in school.

iii. Same techniques should be applied to assess their effectiveness on insomnia among primary and secondary school level students.

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Appendices

RESEARCH PARTICIPANT CONSENT FORM

The essence of the study has been explained to me, and I have been given the opportunity to ask questions about this research. I understand the aims, and agree to participate. I was also assured of confidentiality of any information I gave, and Iunderstand that my participation is voluntary so, I can withdraw from the study at any time.

_____ _____

Sign date

Instruction: Please complete the following questions; these questions ask for some details about you in order to help organize the information for quality improvement purposes. Please, you may answer all the questions and remember not to tick more than one box per question.

i. What is your gender? (Please check one box)

☐ Male
☐ Female

ii. What is your age range?

☐ 18 – 22 ☐ 23 – 27 ☐ 28-32 ☐ 33+

iii. What is your marital status? (Please check one box)

☐ Married
☐ Single
☐ Divorce

iv. What is your personal monthly income? (Please check one box)

☐ <₦20,000 ☐ ₦20 – 29,000 ☐ ₦30 – 39,000 ☐ ₦40 – 49,000 ☐ ₦50,000+

v. How much in monetary valuewould you estimate your sleep difficulty cost you? (Please check one box)

☐ Nothing ☐ <₦30,000 ☐ ₦31 – 60,000 ☐ ₦61 – 90,000 ☐ ₦91,000+

vi. We want to know how you are faring with your schoolwork. Please, be honest by ticking the appropriate response. Howgood doyou think youare at schoolwork now, compared to other of your colleagues?**(Please check one box)**

☐ Excellent, I am probably one of the very best

☐ Above average

☐ Average

☐ Below average

☐ Poor, I am probably one of the worst

APPENDIXI

INSOMNIA SEVERITY INDEX (ISI)

Thinking about your CURRENT(i.e., last2 weeks) insomnia problem(s): CIRCLE the number that best describes your answer for each question.

Insomnia symptoms	*None*	*Mild*	*Moderate*	*Severe*	*Very severe*
1. Difficulty falling asleep	0	1	2	3	4
2. Difficulty staying asleep	0	1	2	3	4
3. Problemwaking up too early	0	1	2	3	4

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

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

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

Not at all Noticeable	ALittle	Somewhat	Much	VeryMuch Noticeable
0	1	2	3	4

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

Not at all Worried	ALittle	Somewhat	Much	VeryMuch Worried
0	1	2	3	4

7. To whatextent do you consider your sleepproblem to INTERFEREwith your daily functioning(e.g. daytime fatigue,mood, ability to function at work/daily chores, concentration, memory, mood, etc.)?

Not at all interfering	ALittle	Somewhat	Much	VeryMuch interfering
0	1	2	3	4

Adopted from Author: Bastien, Vallieres and Morin (2001).

INSOMNIA SCREENING QUESTIONNAIRE (ISQ)

Please complete the following questions; these questions ask for some details about you. Please, you may answer all the questions and remember not to circle more than one box per question. Your identity and information provided will be treated with utmost confidentiality.

		Circle the best answer				
s/n	**Over the past month;**	Never	Rarely	Occasionally	Most nights/days	Always
1	Do you have any medical condition that disrupts your sleep?	1	2	3	4	5
2	Have you lost interest in hobbies or activities?	1	2	3	4	5
3	Do you feel sad, irritable, or hopeless?	1	2	3	4	5
4	Do you feel nervous or worried?	1	2	3	4	5
5	Do you think something is wrong with your body?	1	2	3	4	5
6	Are you a shift worker or is your sleep schedule irregular?	1	2	3	4	5
7	Are your legs restless and/or uncomfortable before bed?	1	2	3	4	5
8	Have you been told that you are restless or that you kick your legs in your sleep?	1	2	3	4	5
9	Do you have any unusual behaviours or movements during sleep?	1	2	3	4	5
10	Do you snore?	1	2	3	4	5
11	Has anyone said that you stop breathing, gasp, snort, or choke in your sleep?	1	2	3	4	5
12	Do you have difficulty staying awake during the day?	1	2	3	4	5

Adopted from Alberto Health and Wellness(2007).

*Scoring:*Subjects who answer 4-5 on any question1 – 12likely suffer from comorbid insomnia.

Diagnostic Domains:

1. Medical condition Q1
2. Psychiatric Disorders: Q2-5
3. Circadian Rhythm Disorder: Q6
4. Movement Disorders: Q7-8
5. Parasomnias Q9
6. Sleep Disordered Breathing (Sleep Apnea): Q10-11
7. Narcolepsy Q12

APPENDIX II

RECOMMENDATIONS FOR SLEEP IMPROVEMENT

Insomnia is a sleep complain characterized by *difficulty falling or staying asleep long enough to feel refreshed the next morning, even when there is enough time to sleep*. Alternatively, it is an *experience of poor sleep quality*. This condition may affect students' ability to stay awake during the day thereby influencing their academic activities negatively.

This treatment handout contains various techniques, which you may find helpful and beneficial to aiding your sleep at night. Note that the effectiveness of the techniquesis solely dependent on your interest and commitment to practicing and or adhering to them (instructions). As you continue to practice or adhere to the instructions provided, it is hoped that your sleep condition will improve over time. Please, do not "surrender" when first attempt fails to work for you. Remember, you may need a little time to train your body to adjust to the new pattern so endure a bit. Remember, taking substance to aid sleep may not be a "bad idea" to some people but don't you think it will be better for people to train themselves to sleep naturally rather than resorting to substance use and abuse and consequently becoming a substance addict?

Stimulus Control Instruction

The aims of SCI are to strengthen the bed and bedroom as cues for sleep, to weaken them as cues for behaviors that are incompatible with sleep, and to develop a consistent sleep-wake pattern. Please, adhere to the instructions strictly to the best of your ability;

1. Lie down to go to sleep only when you are sleepy.

2. Do not use your bed for anything except sleep; that is, do not read contents that are mentally engaging, watch television, eat, or worry in bed.

3. If you find yourself unable to fall asleep, get up and go into another room or get something not mentally engaging to do. Stay up as long as you wish and then return to the

bedroom to sleep. Remember the goal is to associate your bed with falling asleep quickly! If you are in bed more than about 20 minutes without falling asleep and have not gotten up, you are not following this instruction.

4. If you still cannot fall asleep, repeat step 3. Do this as often as is necessary throughout the night.

5. Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body acquire a consistent sleep rhythm, sleep wake cycle.

6. Do not nap during the day because it will complicate your sleep problem at night.

Sleep Restriction Technique

The essence of thismethod is to limitstheamountof timespentinbedtotheamountoftimespentsleeping.By creating a temporary, mild state of sleep deprivation, this method helps to bring about a faster sleep onset and greater sleep continuity and quality.

Administration

If you are someone who stay in bed for 8 hours per night but is only sleeping for 6 hours each night, you may limit the time in bed to 6 hours of your "sleep window." This window of time is then altered later on according to your efficiency. Calculate: Sleep efficiency = ([total sleep time/total time in bed] x 100%). The goal is for you to achieve sleep efficiency of around ≥85%. If sleep efficiency exceeds 90% at any day, you should add an additional 15 to 20 minutes of time in bed per night.

If sleep efficiency is less than 80%, decrease the sleep window by 15 to 20 minutes per night. Please note that since daytime drowsiness can be a side effect of this technique, the sleep window should not be reduced below 5 hours per night, regardless of sleep efficiency. Please note also that SRT has a couple of paradoxical aspects to it. One paradox is that a person who report, "Not getting enough sleep" are, in essence, being told to "sleepless." The other paradox occurs over the course of treatment. With continued practice, you may find

that it is difficult to stay awake until the prescribed hour. SRT is thought to be effective for two reasons. First, it prevents you from coping with insomnia by extending sleep opportunity. This compensatory strategy, while increasing the opportunity to get more sleep, produces a form of sleep that is shallow and fragmented. Second, *the initial sleep loss that occurs with SRT is also thought to increase the homeostatic pressure for sleep, which in turn produces shorter sleep latencies, less wake after sleep onset, and higher sleep efficiency.*

Sleep Hygiene Education

The instructions here are ones that show good level of effectiveness in previous studies. Please, follow these instructions strictly; hopefully,you will begin to enjoy a good night rest.

1. **Sleep only as much as you need to feel refreshed during the following day.**

If you restrict your time in bed, it will help consolidate and deepen your sleep. Do not sleep excessively. Get up at your regular scheduled time the next day, no matter how little you slept.

2.Get up at the same time each day, 7 days a week.

A regular wake time in the morning leads to regular times of sleep onset and helps to set your "biological clock."

3. **Exercise regularly.**

Schedule exercise times so that they do not occur within 3 hours of when you intend to go to bed. Exercise makes it easier to initiate sleep and deepen sleep.

4.Dress your bed well and ensure your bedroom is comfortable and free from light and noise.

A comfortable, noise-free sleep environment will reduce your difficulty falling asleep and the likelihood that you will wake up during the night. Noise that does not awaken you may also disturb the quality of your sleep.

5.Make sure that your bedroom is at a comfortable temperature during the night.

Excessively warm or cold sleep environments may disturb your sleep.

6.Do not go to bed hungry.

Hunger may disturb sleep. A light snack at bedtime (especially carbohydrates) may help sleep, but avoid greasy or "heavy" foods that will increase the need for nighttime trips to the toilet.

7.Avoid excessive liquids (drinks/water) in the evening.

Reducing liquid intake will minimize the need for nighttime trips to the bathroom.

8.Cut down on all caffeine products.

Caffeinated beverages and foods (coffee, tea, and cola, and chocolate) can cause difficulty falling asleep, awakenings during the night, and shallow sleep. Even caffeine early in the day can disrupt nighttime sleep.

9.Avoid alcohol, especially in the evening.

Although alcohol helps tense people (those emotionally disturbed) fall asleep more easily, it causes awakenings later in the night once the effect wears out.

10.Smoking may disturb sleep.

Nicotine is a stimulant. Try not to smoke during the night when you have trouble sleeping.

11.Don't take your problems to bed.

Plan some time earlier in the evening for working on your problems or planning the next day's activities. Worrying may interfere with initiating sleep and produce shallow sleep.

12.Do not *try* to fall asleep.

This only makes the problem worse. Instead, turn on the light, leave the bedroom, and do something different like reading a book. Do not engage in stimulating activity. Return to bed only when you are sleepy.

13.Put the clock under the bed or turn it so that you cannot see it.

Clock watching may lead to frustration, anger, and worry which interfere with sleep.

14.Avoid naps.

Staying awake during the day helps you to fall asleep at night.

15. Take a shower every night before going to bed to sleep

16. Remember to turn off your phone or put it on silence

Technique for Managing Intrusive Thoughts at Bedtime

This technique highlights the importance of changing physiologically and emotionally arousing thought content that is preventing or keeping you from sleep. First, you will need to identify a category of positive thought (topic of thought content) compelling enough to

maintain your attention at bedtime. For instance, an individual may think about new good grades in a course or a scene from his or her favorite television programs. Now, this chosen thought category would likely be emotionally and physiologically non- arousing like negative contents. Therefore, you need to become absorbed in that positive category at bedtime and upon waking up during the evening.

If negative/worrisome thought content creep in to your mind while on bed, just let go of or distract yourself from that thought content. Shrug off all negative thought at bedtime. Please, do not engage in thought that is emotionally, and physiologically compelling. The essence is to develop learned associations between a specific positive thought and sleep.

Please remember that it is important to commit yourself to *letting go* of all negative thoughts at bedtime because they will not allow you to sleep. Speak to yourself "This is the time for sleep, the time for planning and solving problems is later." Give yourself positive coaching. Tell yourself; "There is no need for me to lie awake thinking', let me sleep, all troubles will sort themselves out".

How to Prevent Relapse and Maintain Treatment Gain

Remember "how your sleep problem gets started" and how some of your behaviour gets it maintained over time. As much as you can, avoid such behaviour that affect your sleep.

What to Do When Insomnia Recurs

If perchance the insomnia condition resurfaces, do not panic because brief periods are to be expected at times and are normal. When it persists, however, remember to re-engage in the technique that best work for you in the first place.You may also call back for assistance when think situations are getting out of your control.

Finally, please be inform that it is worth noting that treatment gains are most often maintained or improved over time. That, relapse rates will be small provided all things remain equal, and that you continue good habits.

RECOMMENDATIONS FOR SLEEP IMPROVEMENT

Insomnia is a sleep complain characterized by *difficulty falling or staying asleep long enough to feel refreshed the next morning, even when there is enough time to sleep*. Alternatively, simply put, it is an *experience of poor sleep quality*. This condition may affect students' ability to stay awake during the day thereby influencing their academic activities negatively.

This treatment handout contains various techniques, which you may find helpful and beneficial to aiding your sleep at night. Note that the effectiveness of the techniques is solely dependent on your interest and commitment to practicing and or adhering to them (instructions). As you continue to practice or adhere to the instructions provided, it is hoped that your sleep condition will improve. Please, do not "surrender" when first attempt fails to work for you. Remember, you may need a little bit of a time to train your body to adjust to it. Taking substance to aid sleep may not be a "bad idea" to some people but don't you think it will be better for people to train themselves to sleep naturally rather than resorting to substance use and abuse and consequently becoming a substance addict?

Deep BreathingTechnique

Deep breathing is calming to do either on its own or along with other relaxation techniques for sleep. In fact, most meditations will encourage you to focus on your breathing at some point. You can also draw on deep breathing techniques at any point during the day or night to help you stay calm.

Next time you feel yourself stressed, upset or angry, or unable to sleep, try doing some deep breathing and you should find it helps surprisingly quickly.

Now practice this, hopefully you will find it helpful.

Step by Step Deep Breathing Exercise

1. You can do this exercise while lying in bed

2. Take a minute or two to try to relax and release tension from your muscles.

3. **Close your eyes** and focus your attention on your breathing.

4. Place one hand on your stomach and one on your chest. See if they both rise when you breathe in or just one of them rises. You do not need to try to do anything in particular at this time; Just see which hand is rising and pay attention to it. This will tell you if you naturally breathe with your abdomen, chest or both.

5. Breathe in slowly through your nose for the count of 4 seconds if you can. Try to breathe in such a way that the hand on your stomach rises, and the hand on your chest only rises a little. This is called abdominal breathing, and what you should ideally try to do. You may find it tricky at first, but keep practicing and it will come in time.

6 Once you breathe in, hold your breath for 4 seconds, and then breathe out for 4. If 4 seconds is too much or too little, then you can adjust the time to suit you. Continue with this cycle. Counting 4 – 4 – 4 in your head as you breathe in and out.

7. Allow your thoughts to focus on your counting or the breath as the air gently enters and leaves yours nose and mouth.

8. If your mind wanders, gently bring your attention back to your breath.

9. Once you have learned how to breathe with your belly, you can remove your hands and place them down by your sides, and continue breathing slowly and deeply.

10. You could just keep doing it while you try to fall asleep.

Deep breathing is a highly effective technique in countering certain factors affecting your sleep. It will help;

- Relax your muscles.
- Slow your heart rate down.

- Slow down your respiratory rate.
- Centers you in the moment instead of thinking about past or future problems.

Alternatively, do "A Life Hack for Sleep, 60-second sleep hack": The 4-7-8 Breathing Exercise introduced by Dr. Michelle E. Gordon; Founder of Northern Westchester Surgical Associates General, acute and emergency surgery specialist.

- Exhale completely through your mouth, making a whoosh sound.
- Close your mouth and inhale quietly through your nose to a mental count of four. **4**
- Hold your breath for a count of seven. **7**
- Exhale completely through your mouth, making a whoosh sound to a count of eight. **8**
- This is one breath. Now inhale again and repeat the cycle three more times for four breaths.

You may probably not complete the cycle before sleep takes you away.

Guided Visualization Meditation

The idea in this exercise is to focus your attention on an image or story, so that your mind can let go of worries or thoughts that keep you awake.

Get into a comfortable position in bed. **Close your eyes and relax**. Begin to visualize a scene, memory, or story that you find calming. This is highly individual—find what works best for you by trying a few choices. The key is to find something that allows you to focus your attention and let go of other thoughts. Begin to create this scenario in your mind. Visualize all the details of the image or story, as slowly and carefully as you can. Any time you find your mind drifting to an unrelated thought (a worry about the day or a "must do" for tomorrow), acknowledge it and let it go. Turn your mind's eye back to your relaxing story. It's okay if this takes time before it works, each time you practice you will get better at it.

Remember to follow these additional tips if you have difficulty sleeping:

- Turn off electronics and or put your phone on silent. Try not to worry if you cannot fall asleep, and remind yourself that your body will eventually take over and help you sleep.
- If you are awake for more than 20 minutes in bed, move to a different part of the house (one without bright lights). Do something relaxing for a while, until you begin to feel tired and come back to bed.

Progressive Muscle Relaxation

Progressive muscle relaxation is a technique that involves tensing specific muscle groups and then relaxing them to create awareness of tension and relaxation. It is termed progressive because it proceeds through all major muscle groups, relaxing them one at a time, and eventually leads to total muscle relaxation.

Instructions:

Step 1. Assume a comfortable position. You may lie down. Loosen any tight clothing. Close your eyes and be quiet.

Step 2. Assume a passive attitude. Focus on yourself and on achieving relaxation in specific body muscles. Tune out all other thoughts.

Step 3. Tense and relax each muscle group as follows:

- Forehead - Wrinkle your forehead; try to make your eyebrows touch your hairline for five seconds. Relax.
- Eyes and nose - Close your eyes as tightly as you can for five seconds. Relax.
- Lips, cheeks and jaw - Draw the centers of your mouth back and grimace for five seconds. Relax. Feel the warmth and calmness in your face.
- Hands - Extend your arms in front of you. Clench your fists tightly for five seconds. Relax. Feel the warmth and calmness in your hands.

- Forearms - Extend your arms out against an invisible wall and push forward with your hands for five seconds. Relax.
- Upper arms - Bend your elbows. Tense your biceps (the muscle at the front of your upper arm) for five seconds. Relax. Feel the tension leave your arms.
- Shoulders - Shrug your shoulders up to your ears for five seconds. Relax.
- Back - Arch your back off the floor for five seconds. Relax. Feel the anxiety and tension disappearing.
- Stomach - Tighten your stomach muscles for five seconds. Relax.
- Hips and buttocks - Tighten your hip and buttock muscles for five seconds. Relax.
- Thighs - Tighten your thigh muscles by pressing your legs together as tightly as you can for five seconds. Relax.
- Feet - Bend your ankles toward your body as far as you can for five seconds. Relax.
- Toes - Curl your toes as tightly as you can for five seconds. Relax.

Step 4. Focus on any muscles, which may still be tense. If any muscle remains tense, tighten and relax that specific muscle three or four time.

Step 5. Fix the feeling of relaxation in your mind. Resolve to repeat the process again.

Remember, people respond differently to various activities. Some feel pleasant or refreshed, and others feel calm and relaxed after an activity like this one. Some people notice little change the first time, but with practice, their control increases - as well as the benefits. If you practice this activity, your relaxation should increase.

Mindfulness Technique

Begin with abdominal breathing

- Place one hand on your chest and the other on your abdomen. When you take a deep breath, the hand on the abdomen should rise higher than the one on the chest. This insures that the diaphragm is expanding, pulling air into the bases of the lungs. (Once you have this mastered, you do not have to use your hands).

- Take a slow deep breath in through your nose for a count of 3-4 and exhale slowly through your mouth for a count of 6-7. (Exhalation should be twice as long as your inhalation).
- This diaphragmatic breathing stimulates the vagus nerve, which increases the "relaxation response."
- Allow your thoughts to focus on your counting or the breath as the air gently enters and leaves yours nose and mouth.
- If your mind wanders, gently bring your attention back to your breath.
- Repeat the cycle for 8 breaths.
- After 8 breaths, change your body position and repeat 8 breaths

• After each 8 breathe cycle, change your body position in bed and repeat another 8 breaths.

• It is rare that you will complete 4 cycles of breathing and body position changes before falling asleep.

How to Prevent Relapse and Maintain Treatment Gain

Remember "how your sleep problem gets started" and how some of your behaviour gets it maintained over time. As much as you can, avoid such behaviour that affect your sleep.

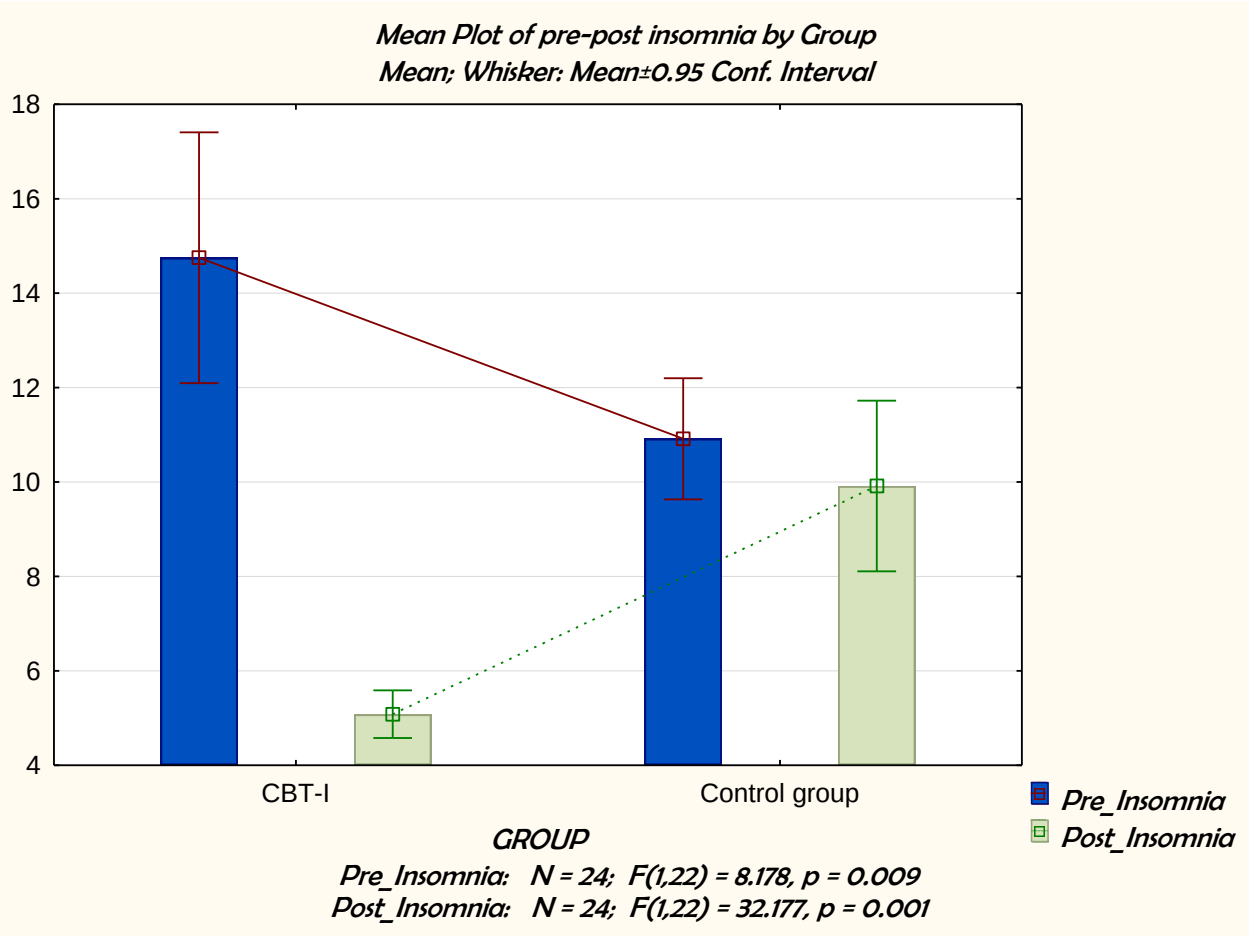
What to Do When Insomnia Recurs

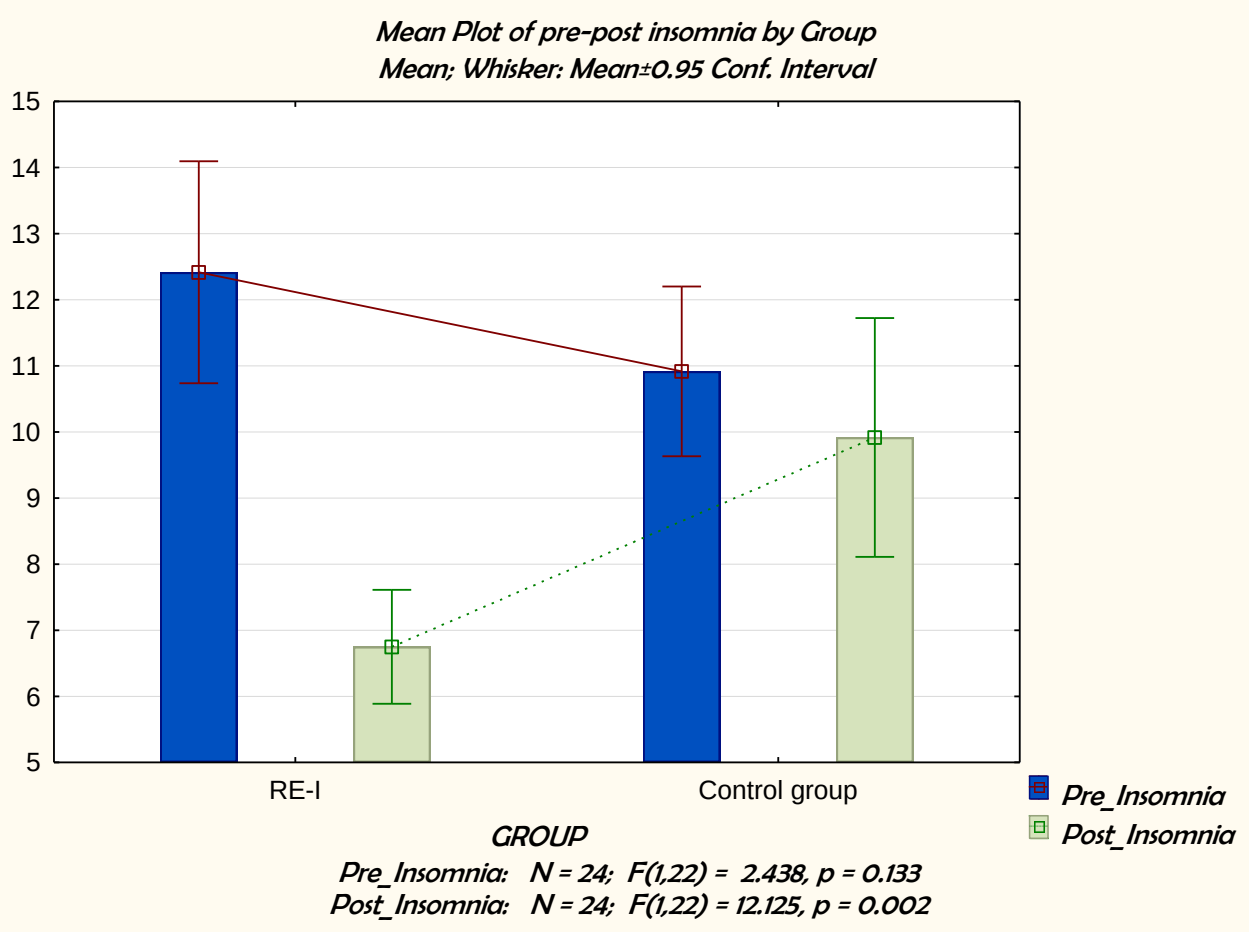
If perchance the insomnia condition resurfaces, do not panic because brief periods are to be expected at times and are normal. When it persists, however, remember to re-engage in the technique that best work for you in the first place. You may also call backfor assistance when you think situations are getting out of your control.

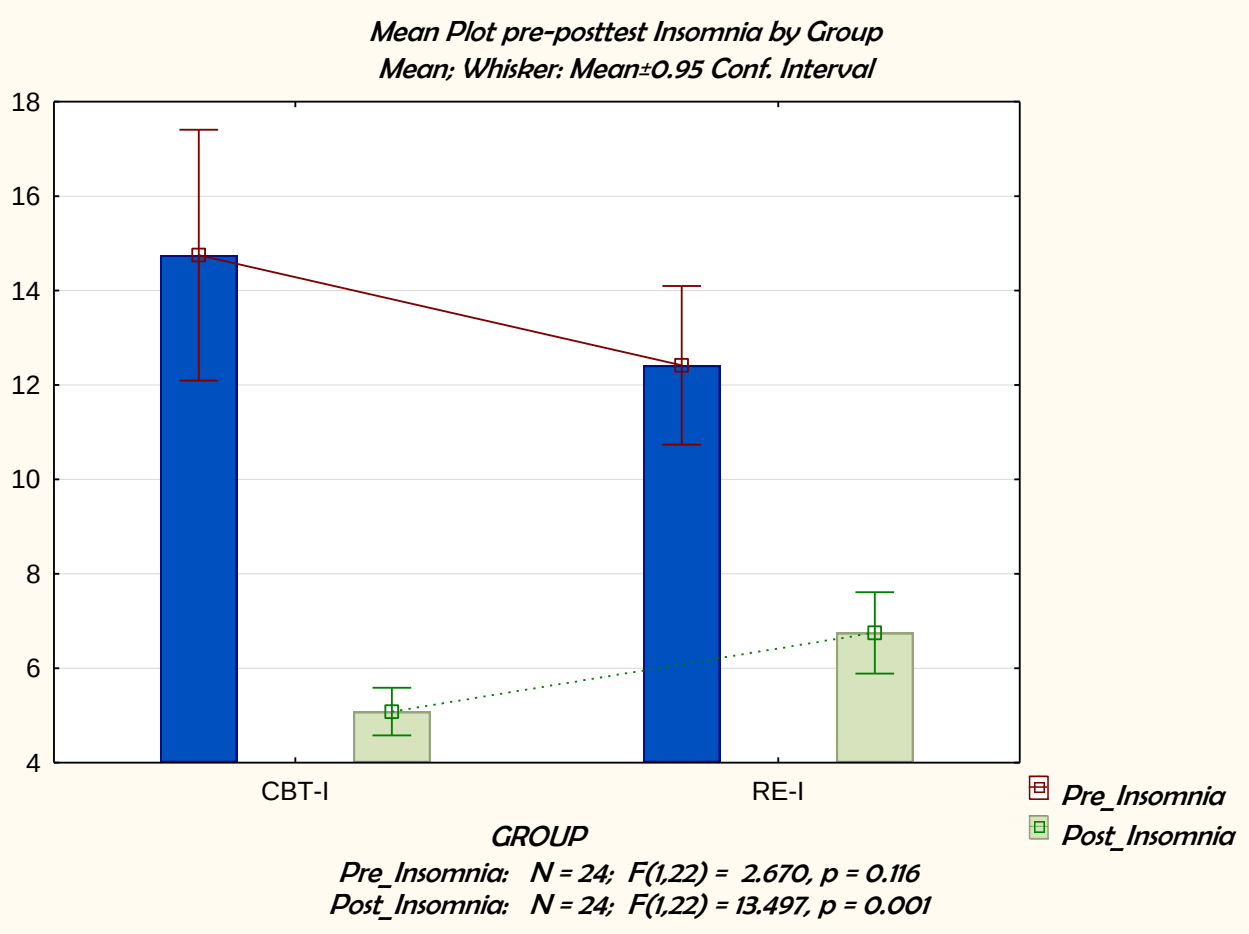
Finally, please be inform that it is worth noting that treatment gains are most often maintained or improved over time. That, relapse rates will be small provided all things remain equal, and that you continue good habits.

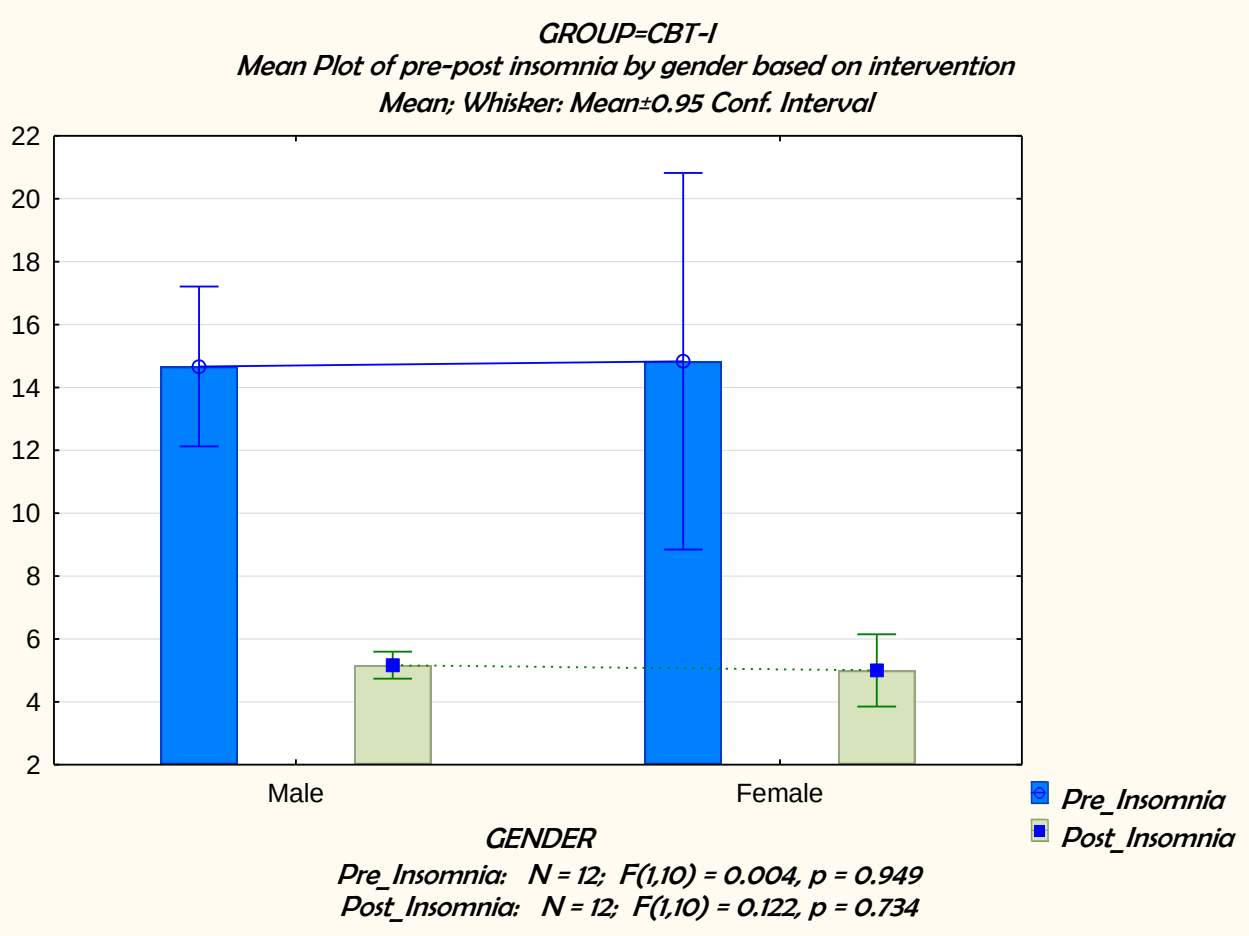
APPENDIX III

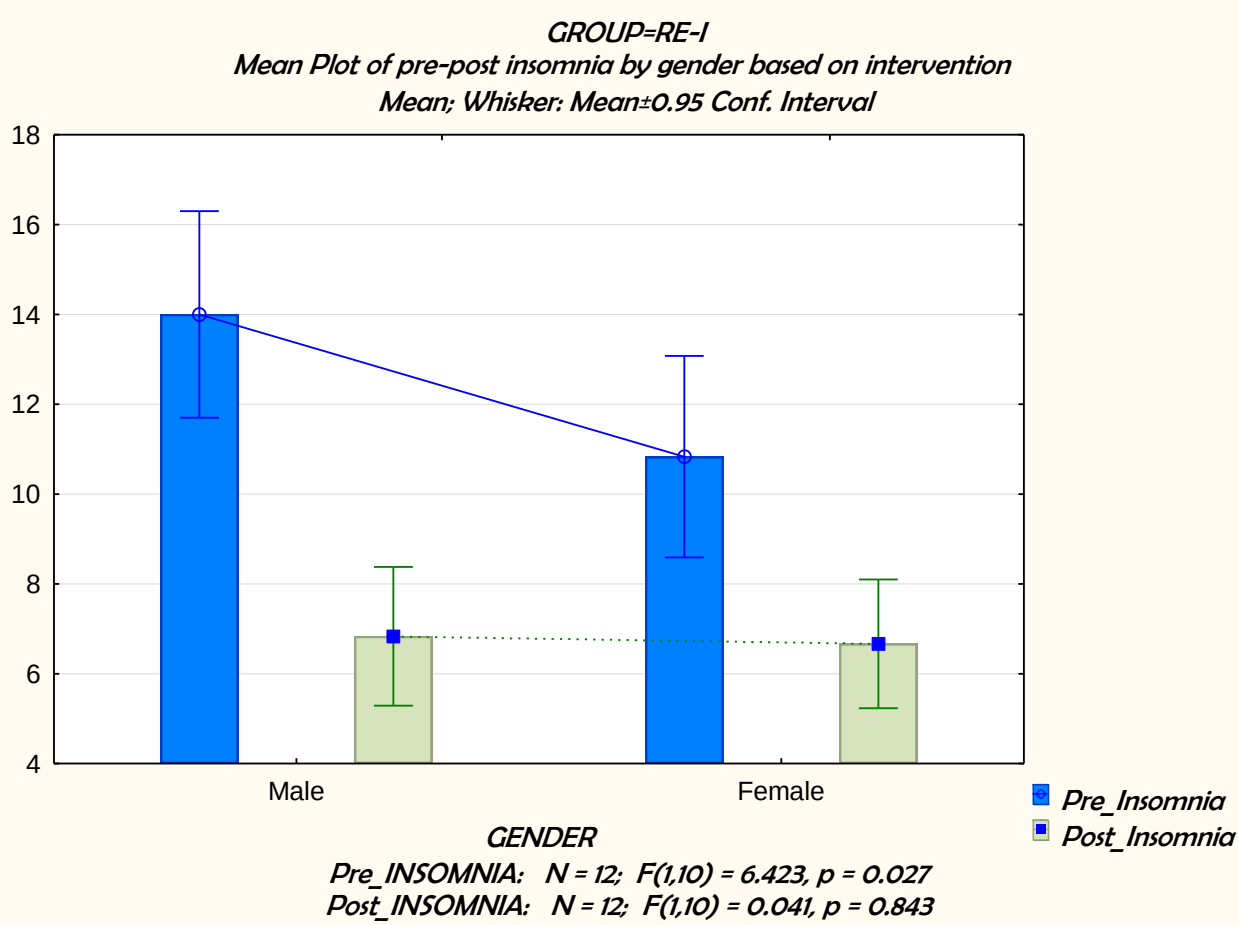
INTERACTION PLOT OF PRE – POSTTEST MEAN SCORES BY GROUP




Mean Plot of pre-post insomnia by Group
Mean; Whisker: Mean±0.95 Conf. Interval
15
14
13
12
11
10
9
8
7
6
5
RE-I
Control group
GROUP
Pre_Insomnia
Post_Insomnia
Pre_Insomnia: N = 24; F(1,22) = 2.438, p = 0.133
Post_Insomnia: N = 24; F(1,22) = 12.125, p = 0.002


Mean Plot pre-posttest Insomnia by Group
Mean; Whisker: Mean±0.95 Conf. Interval
18
16
14
12
10
8
6
4
CBT-I
RE-I
GROUP
Pre_Insomnia
Post_Insomnia
Pre_Insomnia: N = 24; F(1,22) = 2.670, p = 0.116
Post_Insomnia: N = 24; F(1,22) = 13.497, p = 0.001


GROUP=CBT-I
Mean Plot of pre-post insomnia by gender based on intervention
Mean; Whisker: Mean±0.95 Conf. Interval
22
20
18
16
14
12
10
8
6
4
2
Male
Female
GENDER
Pre_Insomnia
Post_Insomnia
Pre_Insomnia: N = 12; F(1,10) = 0.004, p = 0.949
Post_Insomnia: N = 12; F(1,10) = 0.122, p = 0.734


GROUP=RE-I
Mean Plot of pre-post insomnia by gender based on intervention
Mean; Whisker: Mean±0.95 Conf. Interval
18
16
14
12
10
8
6
4
Male
Female
GENDER
Pre_Insomnia
Post_Insomnia
Pre_INSOMNIA: N = 12; F(1,10) = 6.423, p = 0.027
Post_INSOMNIA: N = 12; F(1,10) = 0.041, p = 0.843

APPENDIX IV

SPSS SYNTAX AND ANALYSIS OUTPUT

```
DATASET ACTIVATE DataSet5.
DATASET ACTIVATE DataSet1.
UNIANOVA Post_INSOMNIA BY GROUP WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /CRITERIA=ALPHA(.05)
  /DESIGN=GROUP Pre_INSOMNIA GROUP*Pre_INSOMNIA.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
INTERVENTIO N	1.00	CBT	12
	2.00	Control group	12

Test of Homogeneity of regression slope output

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	141.874[a]	3	47.291	10.049	.000
Intercept	47.294	1	47.294	10.049	.005
GROUP	1.356	1	1.356	.288	.597
Pre_INSOMNIA	.673	1	.673	.143	.709
GROUP * Pre_INSOMNIA	1.708	1	1.708	.363	.554
Error	94.126	20	4.706		
Total	1586.000	24			
Corrected Total	236.000	23			

a. R Squared = .601 (Adjusted R Squared = .541)

```
UNIANOVA Post_INSOMNIA BY GROUP WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(GROUP) WITH(Pre_INSOMNIA=MEAN) COMPARE
ADJ(BONFERRONI)
  /PRINT=ETASQ DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=Pre_INSOMNIA GROUP.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
INTERVENTIO	1.00	CBT	12
N	2.00	Control group	12

Descriptive Statistics

Dependent Variable: Post_INSOMNIA

INTERVENTIO N	*Mean*	*Std. Deviation*	*N*
CBT	5.0833	.79296	12
Control group	9.9167	2.84312	12
Total	7.5000	3.20326	24

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Corrected Model	140.167[a]	2	70.083	15.357	.000	.594
Intercept	76.233	1	76.233	16.705	.001	.443
Pre_INSOMNIA	.000	1	.000	.000	.996	.000
GROUP	102.295	1	102.295	22.416	.000	.516
Error	95.833	21	4.563			
Total	1586.000	24				
Corrected Total	236.000	23				

a. R Squared = .594 (Adjusted R Squared = .555)

Estimated Marginal Means

INTERVENTION

Estimates

Dependent Variable: Post_INSOMNIA

INTERVENTIO N	*Mean*	*Std. Error*	*95% Confidence Interval*	
			Lower Bound	*Upper Bound*
CBT	5.082[a]	.672	3.685	6.479
Control group	9.918[a]	.672	8.521	11.315

a. Covariates appearing in the model are evaluated at the following values: Pre_INSOMNIA = 12.8333.

Pairwise Comparisons

Dependent Variable: Post_INSOMNIA

(I) INTERVENTION	*(J) INTERVENTION*	*Mean Difference (I-J)*	*Std. Error*	*Sig.*[b]	*95% Confidence Interval for Difference*[b]	
					Lower Bound	*Upper Bound*
CBT	Control group	-4.836*	1.021	.000	-6.960	-2.712
Control group	CBT	4.836*	1.021	.000	2.712	6.960

Based on estimated marginal means

**. The mean difference is significant at the .05 level.*

b. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable: Post_INSOMNIA

	Sum of Squares	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Contrast	102.295	1	102.295	22.416	.000	.516
Error	95.833	21	4.563			

The F tests the effect of INTERVENTION. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
DATASET ACTIVATE DataSet2.
UNIANOVA Post_INSOMNIA BY GROUP WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /CRITERIA=ALPHA(0.05)
  /DESIGN=GROUP Pre_INSOMNIA GROUP*Pre_INSOMNIA.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
INTERVENTION	1.00	RT	12
	2.00	Control group	12

Test of Homogeneity of regression slope output

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	63.845[a]	3	21.282	4.035	.021
Intercept	32.927	1	32.927	6.243	.021

GROUP	2.370	1	2.370	.449	.510
Pre_INSOMNIA	3.444	1	3.444	.653	.429
GROUP * Pre_INSOMNIA	.000	1	.000	.000	.993
Error	105.489	20	5.274		
Total	1836.000	24			
Corrected Total	169.333	23			

a. R Squared = .377 (Adjusted R Squared = .284)

```
UNIANOVA Post_INSOMNIA BY GROUP WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(GROUP) WITH(Pre_INSOMNIA=MEAN) COMPARE
ADJ(BONFERRONI)
  /PRINT=ETASQ DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=Pre_INSOMNIA GROUP.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
INTERVENTIO	1.00	RT	12
N	2.00	Control group	12

Descriptive Statistics

Dependent Variable: Post_INSOMNIA

INTERVENTION	*Mean*	*Std. Deviation*	*N*
RT	6.7500	1.35680	12
Control group	9.9167	2.84312	12
Total	8.3333	2.71336	24

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Corrected Model	63.844[a]	2	31.922	6.355	.007	.377
Intercept	34.319	1	34.319	6.832	.016	.245
Pre_INSOMNIA	3.677	1	3.677	.732	.402	.034
GROUP	63.447	1	63.447	12.630	.002	.376
Error	105.489	21	5.023			
Total	1836.000	24				
Corrected Total	169.333	23				

a. R Squared = .377 (Adjusted R Squared = .318)

Estimated Marginal Means

INTERVENTION

Estimates

Dependent Variable: Post_INSOMNIA

INTERVENTION	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
RT	6.620[a]	.665	5.237	8.002
Control group	10.047[a]	.665	8.665	11.429

a. Covariates appearing in the model are evaluated at the following values: Pre_INSOMNIA = 11.6667.

Pairwise Comparisons

Dependent Variable: Post_INSOMNIA

(I) INTERVENTION	(J) INTERVENTION	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
RT	Control group	-3.427*	.964	.002	-5.433	-1.422
Control group	RT	3.427*	.964	.002	1.422	5.433

Based on estimated marginal means

**. The mean difference is significant at the .05 level.*

b. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable: Post_INSOMNIA

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Contrast	63.447	1	63.447	12.630	.002	.376
Error	105.489	21	5.023			

The F tests the effect of INTERVENTION. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
DATASET ACTIVATE DataSet3.
UNIANOVA Post_INSOMNIA BY GROUP WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /CRITERIA=ALPHA(0.05)
  /DESIGN=GROUP Pre_INSOMNIA GROUP*Pre_INSOMNIA.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	N
INTERVENTION	1.00	CBT	12
	2.00	RT	12

Test of Homogeneity of regression slope output

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	19.262[a]	3	6.421	5.226	.008
Intercept	32.053	1	32.053	26.089	.000
GROUP	.345	1	.345	.281	.602

Pre_INSOMNIA	.957	1	.957	.779	.388
GROUP * Pre_INSOMNIA	2.483	1	2.483	2.021	.171
Error	24.572	20	1.229		
Total	884.000	24			
Corrected Total	43.833	23			

a. R Squared = .439 (Adjusted R Squared = .355)

```
UNIANOVA Post_INSOMNIA BY GROUP WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(GROUP) WITH(Pre_INSOMNIA=MEAN) COMPARE
ADJ(BONFERRONI)
  /PRINT=ETASQ DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=Pre_INSOMNIA GROUP.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
INTERVENTIO N	1.00	CBT	12
	2.00	RT	12

Descriptive Statistics

Dependent Variable: Post_INSOMNIA

INTERVENTIO N	*Mean*	*Std. Deviation*	*N*
CBT	5.0833	.79296	12
RT	6.7500	1.35680	12
Total	5.9167	1.38051	24

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Corrected Model	16.779[a]	2	8.390	6.512	.006	.383
Intercept	43.732	1	43.732	33.946	.000	.618
Pre_INSOMNIA	.112	1	.112	.087	.771	.004
GROUP	15.725	1	15.725	12.206	.002	.368
Error	27.054	21	1.288			
Total	884.000	24				
Corrected Total	43.833	23				

a. R Squared = .383 (Adjusted R Squared = .324)

Estimated Marginal Means

INTERVENTION

Estimates

Dependent Variable: Post_INSOMNIA

INTERVENTIO	*Mean*	*Std. Error*	*95% Confidence Interval*

N			*Lower Bound*	*Upper Bound*
CBT	5.059[a]	.337	4.358	5.761
RT	6.774[a]	.337	6.072	7.476

a. Covariates appearing in the model are evaluated at the following values: Pre_INSOMNIA = 13.5833.

Pairwise Comparisons

Dependent Variable: Post_INSOMNIA

(I) INTERVENTION	*(J) INTERVENTION*	*Mean Difference (I-J)*	*Std. Error*	*Sig.[b]*	*95% Confidence Interval for Difference[b]*	
					Lower Bound	*Upper Bound*
CBT	RT	-1.714*	.491	.002	-2.735	-.694
RT	CBT	1.714*	.491	.002	.694	2.735

Based on estimated marginal means

**. The mean difference is significant at the .05 level.*

b. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable: Post_INSOMNIA

	Sum of Squares	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Contrast	15.725	1	15.725	12.206	.002	.368
Error	27.054	21	1.288			

The F tests the effect of INTERVENTION. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
DATASET ACTIVATE DataSet4.
UNIANOVA Post_INSOMNIA BY GENDER WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /CRITERIA=ALPHA(0.05)
  /DESIGN=GENDER Pre_INSOMNIA GENDER*Pre_INSOMNIA.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
Sex	1.00	Male	6
	2.00	Female	6

Test of Homogeneity of regression slope output

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*
Corrected Model	.640[a]	3	.213	.272	.844
Intercept	16.953	1	16.953	21.607	.002
GENDER	.297	1	.297	.378	.556

Pre_INSOMNIA	.556	1	.556	.709	.424
GENDER * Pre_INSOMNIA	.251	1	.251	.320	.587
Error	6.277	8	.785		
Total	317.000	12			
Corrected Total	6.917	11			

a. R Squared = .093 (Adjusted R Squared = -.248)

```
UNIANOVA Post_INSOMNIA BY GENDER WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(GENDER) WITH(Pre_INSOMNIA=MEAN) COMPARE
ADJ(BONFERRONI)
  /PRINT=ETASQ DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=Pre_INSOMNIA GENDER.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
Sex	1.00	Male	6
	2.00	Female	6

Descriptive Statistics

Dependent Variable: Post_INSOMNIA

Sex	*Mean*	*Std. Deviation*	*N*
Male	5.1667	.40825	6
Female	5.0000	1.09545	6
Total	5.0833	.79296	12

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Corrected Model	.389[a]	2	.195	.268	.771	.056
Intercept	26.466	1	26.466	36.491	.000	.802
Pre_INSOMNIA	.306	1	.306	.422	.532	.045
GENDER	.077	1	.077	.106	.752	.012
Error	6.527	9	.725			
Total	317.000	12				
Corrected Total	6.917	11				

a. R Squared = .056 (Adjusted R Squared = -.153)

Estimated Marginal Means

Sex

Estimates

Dependent Variable: Post_INSOMNIA

Sex	*Mean*	*Std. Error*	*95% Confidence Interval*

			Lower Bound	Upper Bound
Male	5.163[a]	.348	4.377	5.950
Female	5.003[a]	.348	4.217	5.790

a. Covariates appearing in the model are evaluated at the following values: Pre_INSOMNIA = 14.7500.

Pairwise Comparisons

Dependent Variable: Post_INSOMNIA

					95% Confidence Interval for Difference[a]	
(I) Sex	(J) Sex	Mean Difference (I-J)	Std. Error	Sig.[a]	Lower Bound	Upper Bound
Male	Female	.160	.492	.752	-.953	1.273
Female	Male	-.160	.492	.752	-1.273	.953

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable: Post_INSOMNIA

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Contrast	.077	1	.077	.106	.752	.012
Error	6.527	9	.725			

The F tests the effect of Sex. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
DATASET ACTIVATE DataSet5.
UNIANOVA Post_INSOMNIA BY GENDER WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /CRITERIA=ALPHA(0.05)
  /DESIGN=GENDER Pre_INSOMNIA GENDER*Pre_INSOMNIA.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	N
Sex	1.00	Male	6
	2.00	Female	6

Test of Homogeneity of regression slope output

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	3.748[a]	3	1.249	.606	.630
Intercept	4.277	1	4.277	2.074	.188
GENDER	.469	1	.469	.227	.646
Pre_INSOMNIA	2.979	1	2.979	1.444	.264

GENDER * Pre_INSOMNIA	.758	1	.758	.368	.561
Error	16.502	8	2.063		
Total	567.000	12			
Corrected Total	20.250	11			

a. R Squared = .185 (Adjusted R Squared = -.121)

```
UNIANOVA Post_INSOMNIA BY GENDER WITH Pre_INSOMNIA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(GENDER) WITH(Pre_INSOMNIA=MEAN) COMPARE
ADJ(BONFERRONI)
  /PRINT=ETASQ DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=Pre_INSOMNIA GENDER.
```

Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	*N*
Sex	1.00	Male	6
	2.00	Female	6

Descriptive Statistics

Dependent Variable: Post_INSOMNIA

Sex	*Mean*	*Std. Deviation*	*N*
Male	6.8333	1.47196	6
Female	6.6667	1.36626	6
Total	6.7500	1.35680	12

Tests of Between-Subjects Effects

Dependent Variable: Post_INSOMNIA

Source	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*Sig.*	*Partial Eta Squared*
Corrected Model	2.990[a]	2	1.495	.779	.487	.148
Intercept	3.962	1	3.962	2.066	.184	.187
Pre_INSOMNIA	2.906	1	2.906	1.515	.250	.144
GENDER	.707	1	.707	.369	.559	.039
Error	17.260	9	1.918			
Total	567.000	12				
Corrected Total	20.250	11				

a. R Squared = .148 (Adjusted R Squared = -.042)

Estimated Marginal Means

Sex

Estimates

Dependent Variable: Post_INSOMNIA

			95% Confidence Interval	
Sex	**Mean**	**Std. Error**	**Lower Bound**	**Upper Bound**
Male	6.439[a]	.650	4.969	7.909
Female	7.061[a]	.650	5.591	8.531

a. Covariates appearing in the model are evaluated at the following values: Pre_INSOMNIA = 12.4167.

Pairwise Comparisons

Dependent Variable: Post_INSOMNIA

		Mean Difference			95% Confidence Interval for Difference[a]	
(I) Sex	**(J) Sex**	**(I-J)**	**Std. Error**	**Sig.[a]**	**Lower Bound**	**Upper Bound**
Male	Female	-.622	1.025	.559	-2.940	1.696
Female	Male	.622	1.025	.559	-1.696	2.940

Based on estimated marginal means

Insomnia Severity before Intervention * INTERVENTION Crosstabulation

			INTERVENTION			
			CBT	**RT**	**Control group**	**Total**
Insomnia Severity before Intervention	Subthreshold insomnia	Count	5	9	11	25
		% within intervention	41.7%	75.0%	91.7%	69.4%
	Moderate Insomnia	Count	7	3	1	11
		% within intervention	58.3%	25.0%	8.3%	30.6%
Total		Count	12	12	12	36
		% within intervention	100.0%	100.0%	100.0%	100.0%

Insomnia Severity after Intervention * intervention Crosstabulation

			INTERVENTION			
			CBT	**RT**	**Control group**	**Total**
Insomnia Severity after Intervention	absence of insomnia	Count	12	9	2	23
		% within intervention	100.0%	75.0%	16.7%	63.9%
	Subthreshold insomnia	Count	0	3	10	13
		% within intervention	0.0%	25.0%	83.3%	36.1%
Total		Count	12	12	12	36
		% within intervention	100.0%	100.0%	100.0%	100.0%

```
DATASET ACTIVATE DataSet2.
T-TEST GROUPS=GROUP(1 2)
  /MISSING=ANALYSIS
  /VARIABLES=Post_Insom1 Post_Insom2 Post_Insom3 Post_Insom4 Post_Insom5 Post_Insom6 Post_Insom7
  /CRITERIA=CI(.95).
```

T-Test

[DataSet2] C:\Users\MO VIC\Desktop\FILES\New folder\INSOMNIA\CBT VS RT.sav

Group Statistics

	INTERVENTION	*N*	*Mean*	*Std. Deviation*	*Std. Error Mean*
Difficulty falling asleep	CBT	12	.7500	.45227	.13056
	RT	12	.5000	.52223	.15076
Difficulty staying asleep	CBT	12	.7500	.45227	.13056
	RT	12	1.1667	.71774	.20719
Problem waking up too early	CBT	12	.4167	.51493	.14865
	RT	12	1.1667	.57735	.16667
SATISFIED/DISSATISFIED with sleep pattern	CBT	12	1.5833	.51493	.14865
	RT	12	1.7500	.45227	.13056
Noticeable impairment in term of quality of life	CBT	12	.4167	.51493	.14865
	RT	12	.5833	.66856	.19300
WORRIED/DISTRESSED about current sleep pattern	CBT	12	.5000	.52223	.15076
	RT	12	1.0000	.42640	.12309
INTERFERENCE with daily functioning	CBT	12	.6667	.49237	.14213
	RT	12	.5833	.51493	.14865

Independent Samples Test

		Levene's Test for Equality of Variances		*t-test for Equality of Means*						
									95% Confidence Interval of the Difference	
		F	*Sig.*	*t*	*df*	*Sig. (2-tailed)*	*Mean Difference*	*Std. Error Difference*	*Lower*	*Upper*
Difficulty falling asleep	Equal variances assumed	3.667	.069	1.254	22	.223	.25000	.19943	-.16359	.66359
	Equal variances not assumed			1.254	21.560	.223	.25000	.19943	-.16408	.66408
Difficulty staying asleep	Equal variances assumed	1.704	.205	-1.701	22	.103	-.41667	.24490	-.92455	.09122
	Equal variances not assumed			-1.701	18.546	.106	-.41667	.24490	-.93009	.09676
Problem waking up too early	Equal variances assumed	.382	.543	-3.358	22	.003	-.75000	.22332	-1.21315	-.28685
	Equal variances not assumed			-3.358	21.718	.003	-.75000	.22332	-1.21349	-.28651
SATISFIED/DISSATISFIED you with sleep pattern	Equal variances assumed	2.532	.126	-.842	22	.409	-.16667	.19784	-.57697	.24363
	Equal variances not assumed			-.842	21.640	.409	-.16667	.19784	-.57736	.24403
Noticeable impairment in term of quality of life	Equal variances assumed	1.365	.255	-.684	22	.501	-.16667	.24361	-.67187	.33854
	Equal variances not assumed			-.684	20.654	.501	-.16667	.24361	-.67379	.34046
WORRIED/DISTRESSED about current sleep pattern	Equal variances assumed	8.800	.007	-2.569	22	.018	-.50000	.19462	-.90363	-.09637
	Equal variances not assumed			-2.569	21.154	.018	-.50000	.19462	-.90457	-.09543
INTERFERENCE with daily functioning	Equal variances assumed	.607	.444	.405	22	.689	.08333	.20566	-.34319	.50986
	Equal variances not assumed			.405	21.956	.689	.08333	.20566	-.34324	.50991

```
GET
  FILE='C:\Users\MO VIC\Desktop\FILES\New folder\INSOMNIA\PAIRED TEST RT.sav'.
DATASET NAME DataSet3 WINDOW=FRONT.
T-TEST PAIRS=Pre_Insom1 Pre_Insom2 Pre_Insom3 Pre_Insom4 Pre_Insom5 Pre_Insom6 Pre_Insom7 WITH
    Post_Insom1 Post_Insom2 Post_Insom3 Post_Insom4 Post_Insom5 Post_Insom6 Post_Insom7 (PAIRED)
  /CRITERIA=CI(.9500)
  /MISSING=ANALYSIS.
```

T-Test RT

[DataSet3] C:\Users\MO VIC\Desktop\FILES\New folder\INSOMNIA\PAIRED TEST RT.sav

Paired Samples Statistics

		Mean	*N*	*Std. Deviation*	*Std. Error Mean*
Pair 1	Pre-Difficulty falling asleep	1.6667	12	.98473	.28427
	Po-Difficulty falling asleep	.5000	12	.52223	.15076
Pair 2	Pre-Difficulty staying asleep	2.0000	12	1.04447	.30151
	Po-Difficulty staying asleep	1.1667	12	.71774	.20719
Pair 3	Pre-Problem waking up too early	1.6667	12	1.23091	.35533
	Po-Problem waking up too early	1.1667	12	.57735	.16667
Pair 4	Pre-SATISFIED/DISSATISFIED with sleep pattern	2.4167	12	.51493	.14865
	Po-SATISFIED/DISSATISFIED with sleep pattern	1.7500	12	.45227	.13056
Pair 5	Pre-Noticeable impairment in term of quality of life	1.3333	12	.65134	.18803
	Po-Noticeable impairment in term of quality of life	.5833	12	.66856	.19300
Pair 6	Pre-WORRIED/DISTRESSED about current sleep pattern	1.5000	12	.90453	.26112
	Po-WORRIED/DISTRESSED about current sleep pattern	1.0000	12	.42640	.12309
Pair 7	Pre-INTERFERENCE with daily functioning	1.8333	12	.71774	.20719
	Po-INTERFERENCE with daily functioning	.5833	12	.51493	.14865

Paired Samples Correlations

		N	*Correlation*	*Sig.*
Pair 1	Pre-Difficulty falling asleep & Po-Difficulty falling asleep	12	-.354	.260
Pair 2	Pre-Difficulty staying asleep & Po-Difficulty staying asleep	12	.728	.007
Pair 3	Pre-Problem waking up too early & Po-Problem waking up too early	12	-.426	.167
Pair 4	Pre-SATISFIED/DISSATISFIED with sleep pattern & Po-SATISFIED/DISSATISFIED with sleep pattern	12	.098	.763
Pair 5	Pre-Noticeable impairment in term of quality of life & Po-Noticeable impairment in term of quality of life	12	.139	.666
Pair 6	Pre-WORRIED/DISTRESSED about current sleep pattern & Po-WORRIED/DISTRESSED about current sleep pattern	12	-.236	.461
Pair 7	Pre-INTERFERENCE with daily functioning & Po-INTERFERENCE with daily functioning	12	.533	.074

Paired Samples Test

		Paired Differences					*t*	*df*	*Sig. (2-tailed)*
		Mean	*Std. Deviation*	*Std. Error Mean*	*95% Confidence Interval of the Difference* *Lower*	*Upper*			
Pair 1	Pre-Difficulty falling asleep - Po-Difficulty falling asleep	1.16667	1.26730	.36584	.36146	1.97187	3.189	11	.009
Pair 2	Pre-Difficulty staying asleep - Po-Difficulty staying asleep	.83333	.71774	.20719	.37730	1.28936	4.022	11	.002
Pair 3	Pre-Problem waking up too early - Po-Problem waking up too early	.50000	1.56670	.45227	-.49543	1.49543	1.106	11	.293
Pair 4	Pre-SATISFIED/DISSATISFIED with sleep pattern - Po-SATISFIED/DISSATISFIED with sleep pattern	.66667	.65134	.18803	.25283	1.08051	3.546	11	.005
Pair 5	Pre-Noticeable impairment in term of quality of life - Po-Noticeable impairment in term of quality of life	.75000	.86603	.25000	.19975	1.30025	3.000	11	.012
Pair 6	Pre-WORRIED/DISTRESSED about current sleep pattern - Po-WORRIED/DISTRESSED about current sleep pattern	.50000	1.08711	.31382	-.19072	1.19072	1.593	11	.139
Pair 7	Pre-INTERFERENCE with daily functioning - Po-INTERFERENCE with daily functioning	1.25000	.62158	.17944	.85507	1.64493	6.966	11	.000

```
DATASET ACTIVATE DataSet2.
GET
  FILE='C:\Users\MO VIC\Desktop\FILES\New folder\INSOMNIA\PAIRED TEST CBT.sav'.
DATASET NAME DataSet4 WINDOW=FRONT.
T-TEST PAIRS=Pre_Insom1 Pre_Insom2 Pre_Insom3 Pre_Insom4 Pre_Insom5 Pre_Insom6 Pre_Insom7 WITH
    Post_Insom1 Post_Insom2 Post_Insom3 Post_Insom4 Post_Insom5 Post_Insom6 Post_Insom7 (PAIRED)
  /CRITERIA=CI(.9500)
  /MISSING=ANALYSIS.
```

T-Test CBT

[DataSet4] C:\Users\MO VIC\Desktop\FILES\New folder\INSOMNIA\PAIRED TEST CBT.sav

Paired Samples Statistics

		Mean	*N*	*Std. Deviation*	*Std. Error Mean*
Pair 1	Pre-Difficulty falling asleep	2.1667	12	1.02986	.29729
	Po-Difficulty falling asleep	.7500	12	.45227	.13056
Pair 2	Pre-Difficulty staying asleep	2.0833	12	.90034	.25990
	Po-Difficulty staying asleep	.7500	12	.45227	.13056
Pair 3	Pre-Problem waking up too early	1.9167	12	.99620	.28758
	Po-Problem waking up too early	.4167	12	.51493	.14865
Pair 4	Pre-SATISFIED/DISSATISFIED with sleep pattern	2.4167	12	.66856	.19300
	Po-SATISFIED/DISSATISFIED with sleep pattern	1.5833	12	.51493	.14865
Pair 5	Pre-Noticeable impairment in term of quality of life	1.8333	12	.57735	.16667
	Po-Noticeable impairment in term of quality of life	.4167	12	.51493	.14865
Pair 6	Pre-WORRIED/DISTRESSED about current sleep pattern	1.9167	12	.99620	.28758
	Po-WORRIED/DISTRESSED about current sleep pattern	.5000	12	.52223	.15076
Pair 7	Pre-INTERFERENCE with daily functioning	2.4167	12	1.08362	.31282
	Po-INTERFERENCE with daily functioning	.6667	12	.49237	.14213

Paired Samples Correlations

		N	*Correlation*	*Sig.*
Pair 1	Pre-Difficulty falling asleep & Po-Difficulty falling asleep	12	-.098	.763
Pair 2	Pre-Difficulty staying asleep & Po-Difficulty staying asleep	12	-.614	.034
Pair 3	Pre-Problem waking up too early & Po-Problem waking up too early	12	.251	.431
Pair 4	Pre-SATISFIED/DISSATISFIED with sleep pattern & Po-SATISFIED/DISSATISFIED with sleep pattern	12	.022	.946
Pair 5	Pre-Noticeable impairment in term of quality of life & Po-Noticeable impairment in term of quality of life	12	-.051	.875
Pair 6	Pre-WORRIED/DISTRESSED about current sleep pattern & Po-WORRIED/DISTRESSED about current sleep pattern	12	-.262	.411
Pair 7	Pre-INTERFERENCE with daily functioning & Po-INTERFERENCE with daily functioning	12	-.398	.201

Paired Samples Test

		Paired Differences							
					95% Confidence Interval of the Difference				
		Mean	*Std. Deviation*	*Std. Error Mean*	*Lower*	*Upper*	*t*	*df*	*Sig. (2-tailed)*
Pair 1	Pre-Difficulty falling asleep - Po-Difficulty falling asleep	1.41667	1.16450	.33616	.67678	2.15655	4.214	11	.001
Pair 2	Pre-Difficulty staying asleep - Po-Difficulty staying asleep	1.33333	1.23091	.35533	.55125	2.11542	3.752	11	.003
Pair 3	Pre-Problem waking up too early - Po-Problem waking up too early	1.50000	1.00000	.28868	.86463	2.13537	5.196	11	.000
Pair 4	Pre-SATISFIED/DISSATISFIED with sleep pattern - Po-SATISFIED/DISSATISFIED with sleep pattern	.83333	.83485	.24100	.30290	1.36377	3.458	11	.005
Pair 5	Pre-Noticeable impairment in term of quality of life - Po-Noticeable impairment in term of quality of life	1.41667	.79296	.22891	.91284	1.92049	6.189	11	.000
Pair 6	Pre-WORRIED/DISTRESSED about current sleep pattern - Po-WORRIED/DISTRESSED about current sleep pattern	1.41667	1.24011	.35799	.62874	2.20460	3.957	11	.002
Pair 7	Pre-INTERFERENCE with daily functioning - Po-INTERFERENCE with daily functioning	1.75000	1.35680	.39167	.88793	2.61207	4.468	11	.001