YOGA FOR IMPROVING SLEEP QUALITY AND QUALITY OF LIFE OF OLDER ADULTS IN A WESTERN CULTURAL SETTING

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……*Jonathan Halpern*…….

Jonathan S. Halpern

Dedication

In loving memory of my father Professor Yeheskel Halpern, Dr. Wilson Wan and Ciril Kunstelj.

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Glossary of terms

Abbreviated progressive muscle relaxation (APRT): An abbreviated form of the progressive muscle relaxation technique (PMR)

Adho mukha svanasana: Downward facing dog pose.

Alpha rhythm: "*An EEG pattern consisting of trains of sinusoidal 8-13 Hz activity"* (AASM, 2007, p. 59).

Ardha chandrasana: Half moon pose.

Ardha kurmasana: Tortoise pose.

Ardha pavana muktasana: Half wind removing pose.

Ardha shalbhasana: Half locust pose.

Ardha chandrasana: Half moon pose.

Apnoea: "*An interruption of airflow lasting at least 10 seconds in adults or the equivalent of two breaths in children"* (AASM, 2007, p. 59).

Asana: The third limb of in Patanjali's eightfold path of yoga ('Ashtanga yoga') translated as postures, poses, and stable sitting positions.

Ashtanga vinyasa yoga: A dynamic system of yoga introduced to the west in the early 20th century by K. Pattabhi Jois. This form of yoga incorporates a series of 51 traditional static yoga poses linked by a 'Vinyasa', which is a dynamic connective flow of poses that links a series of static asanas.

Ashtanga yoga: The eightfold path of yoga as outlined by Patanjali. The eight branches include: Yama, Niyama, Asana, Pranayama, Pratyahara, Dharana, Dhyana and Samadhi.

Autonomic nervous system (ANS): The part of the peripheral nervous system that mainly controls subconscious involuntary functions such as heart rate, digestion, respiration rate etc.

B. K. S. Iyengar: A contemporary yoga master. Founder of 'Iyengar yoga'.

Balasana: Child's pose.

Bastrika pranayama: Bellows breathing technique - breathing forcibly in and out through the nose in equal proportions.

Beta rhythm: "*An EEG rhythm consisting of 13-30 Hz activity"* (AASM, 2007, p. 59).

Bhujangasana: Cobra pose.

Bikram Choudhury: A contemporary yoga master founder of 'Bikram yoga'

Bikram yoga: A system of yoga synthesized by Bikram Choudhury from traditional hatha yoga techniques and introduced in the west in the early 1970s. Bikram beginners yoga class incorporates a series of 26 postures and two short breathing exercises, and is ideally practiced in ambient temperature of 105°F (40.5°C), which is approximately 3.5° C - 4°C above normal body temperature, and in humidity of 40%. Practicing yoga in such conditions results in an aerobic effect as well as considerable sweating.

Bitilasana: Cow pose

Body mass index (BMI): A measure of obesity calculated by dividing body mass (in Kg) by the square of the height (in meter) as follows: BMI = body mass/$(\text{height})^2$

Chakra: A wheel-like vortex energetic centre which, according to yogic philosophy and traditional Indian medicine, are believed to exist on the surface of the human energy body. The seven main chakras are positioned along the body from the top of the head to the base of the spine.

Continuous positive airway pressure (CPAP): A device used for treating sleep apnea by maintaining a constant, continuous positive airway pressure to help keep the airway open.

Cyclic meditation (CM): A yogic practice introduced by Swami Vivekananda and the Vivekananda yoga anusandhana samsthana (VYASA). CM is based on cycles of static yoga postures followed by supine relaxation periods of several minutes each.

Delta rhythm: "*An EEG rhythm consisting of 1-4 Hz activity"* (AASM, 2007, p. 59).

Dharana: The sixth limb of Patanjali's eightfold path of yoga, translated as concentration, keeping the mind focused.

Dhyana: The seventh limb of Patanjali's eightfold path of yoga translated as meditation, contemplation, reflection, awareness.

Electrocardiogram (ECG): Recording of the electrical activity of the heart.

Electroencephalogram (EEG): Recording of the electrical activity of the brain.

Electromyogram (EMG): Recording of muscle electrical potentials.

Electromyogram (EOG): "*Recording of eye movements by means of changes in the electrical potentials between the retina and the cornea"* (Pollak et al., 2010, p 77).

Gamma-amino butyric Acid (GABA): A key neurotransmitter distributed throughout the central nervous system and also believed linked to the endocrine system and affecting sleep quality.

Garurasana: Eagle pose.

Goolf Chakra: Ankle rotations.

Guru: A spiritually evolved teacher, who can dispel ignorance and illusion from the mind of a devotee/disciple.

Hatha yoga: An ancient yoga system aiming at purifying the entire physical body using Shatkarma (cleansing practices), Asana (posture), Pranayama (breath/energy regulation), Mudra, (hand gestures) Banda (energy locks) and Dharana (focus/concentration).

Heart Rate Variability (HRV): The variability of the time interval between consecutive heart beats.

Hypopnea: "*A specified reduction in airflow lasting at least 10 seconds in adults or the equivalent of two breaths in children"* (AASM, 2007, p. 59).

Ida nadi: One of the main energy channels which, according to yogic philosophy, runs on the left side of the spine intersecting various chakras on the way.

K complex: "*An EEG event consisting of a well delineated negative sharp wave immediately followed by a positive component standing out from the background EEG with total duration ≥0.5 second, usually maximal in amplitude over the frontal regions"* (AASM, 2007, p. 59).

Kapalabati: A rapid breathing technique done with more emphasis on exhalation.

Kosha: A layer or sheath in Sanskrit. Yogic philosophy describes five sheaths, sometimes likened to layers of an onion that surround the soul, including a physical layer, a pranic (energetic) layer, an instinctive thinking/emotional layer, a mental/intellectual layer and a super- consciousness layer.

Kriya: Activity, dynamic yogic practice.

Kriya yoga: An ancient yoga system reintroduced by Mahavatar Babaji and his disciple Lahiri Mahasaya in the 19th century and popularised by Paramhansa Yogananda. It includes several levels of Pranayama and intended to facilitate accelerated spiritual development.

Kundalini: Retained energy or potential energy/ consciousness in human beings.

Kundalini yoga (KY): A yoga practice based on a philosophy expounding the awakening of potential energy and inherent consciousness within the human body and mind. KY as introduced by Yogi Bhajan utilises meditation and breathing techniques, Mudras and some postures.

Kurmasana: Tortoise Pose.

Manibandha Chakra: Wrist rotations.

Mantra: A subtle sound vibration, that aims through repetition at expanding one's awareness or consciousness.

Marjaryasana: Cat pose.

Matsyendrasana: Half lord of the fish pose**.** A spine twisting pose.

Mindfulness based stress reduction (MBSR): a non sectarian program established by Jon Kabat-Zinn at the University of Massachusetts Medical School. It aims at cultivating mindfulness, defined as a moment-to-moment non-judgmental awareness of mental and emotional processes and states, and using it as a tool for self transformation and self healing.

Mudra: A symbolic (hand) gesture used for directing energetic and spiritual focus

Nadi: Subtle energy channels which, according to yogic philosophy, run throughout the body.

Nadi Shodana Pranayama: Alternate nostril breathing' or 'balanced breathing'.

Nidra: Sleep in Sanskrit

Niyama: The second limb of in Patanjali's eightfold path of yoga which deals with fixed observances, values and precepts.

Oxygen desaturation: A drop below normal (90%) in the amount of oxygen in blood haemoglobin.

Oxygen desaturation index (ODI): A measure of mean oxygen desaturation

Padmasana: Lotus pose; a seated meditative pose.

Paschimottanasana: A seated forward bend pose.

Patanjali: Author of the 'Yoga Sutra' who systematised the eightfold path of yoga.

Pattabhi Jois: Contemporary yoga master who promoted the 'Ashtanga vinyasa yoga' method.

Pavana muktasana: Wind removing pose.

Periodic leg movements of sleep (PLMD): Movements of the legs during sleep occurring with a specified frequency, duration and amplitude.

Pingala nadi: One of the main energy channels, which, according to yogic philosophy, runs on the right side of the spine intersecting various chakras on the way.

Prana: A vital energy force, which, according to yogic philosophy, sustains life and creation.

Pranayama: The fourths limb of Patanjali's eightfold path of yoga that deals with regulation of breath for cultivating and regulating energy flow in the body.

Progressive muscle relaxation (PMR): A relaxation technique based on sequential tensing and relaxing of various muscle groups. PMR was introduced by American physician Edmund Jacobson in the early 1920s.

Pratyahara: The fifth limb of Patanjali's eightfold path of yoga that deals with withdrawal of the senses and directing them inwardly.

Qi Gong: Also called Qigong or Chi kung is a Chinese Mandarin term describing diverse methods of physical and mental training for health, martial arts and spiritual advancement. Qi can be interpreted as energy, breathing or air and Gong as a method for achieving results. Together they can be translated as an energy cultivation practice method.

Raja yoga: Translated from Sanskrit as 'royal yoga' or royal union. Deals mainly with cultivation of the mind using meditative techniques leading towards deeper self knowledge with the ultimate goal of achieving spiritual liberation. Raja yoga was first described in the 'Yoga Sutra' of Patanjali.

Rapid Eye Movements (REM): "*EOG events consisting of trains of conjugate, irregular, sharply peaked eye movements with an initial deflection usually lasting <500 milliseconds"* (AASM, 2007, p. 59).

Respiratory effort related arousal (RERA): "*A sequence of breaths lasting at least 10 seconds which does not meet criteria for an apnea or hypopnea and is characterised by increasing respiratory effort leading to an arousal from sleep"* (AASM, 2007, p. 59).

Samadhaya: The eighth limb of Patanjali's eightfold path of yoga. Deals with absorption of consciousness in the self, profound meditation, super-consciousness

Samadhi: see Samadhaya

Sankalpa: Spiritual resolve.

Savasana: Corpse pose; a supine relaxation pose.

Shalbhasana: Locust pose.

Shatkarma: Yogic purification practices for cleaning the upper digestive system and colon, nasal passages, eyes etc.

Shushumna: The central subtle energy channel, which according to yogic philosophy flow along the spinal column

Siddhasana: A meditative sitting pose.

Skandh Chakra: Shoulder rotations

Sleep efficiency: The ratio between the actual sleep time in a sleep episode to the total available time for sleep.

Sleep spindle: "*An EEG event consisting of a train of distinct waves with frequency 11-16 Hz (most commonly 12 – 14 Hz) with a duration ≥0.5 seconds, usually maximal in amplitude over the central regions"* (AASM, 2007, p. 59).

Slow Wave Sleep (SWS): SWS is the deepest sleep phase and is characterised by low frequency high amplitude brain waves that are 75 microvolt or greater as observed on EEG recordings.

Sudarshan kriya yoga (SKY): A contemporary yoga system formalised by Sri Sri Ravi Shankar, founder of the Art of living Foundation. The SKY protocol incorporates a

sequence of specific yogic breathing techniques, including ujai pranayama, bastrika pranayama, and a special SKY breathing sequence.

Swastikasana: The 'auspicious' pose; a meditative pose similar to Siddhasana.

Tadasana: Mountain pose.

Theta rhythm: "*An EEG rhythm consisting of 4-8 Hz activity"* (AASM, 2007, p. 59).

Tiryaka tadasana: Swaying palm tree pose

Transcendental meditation (TM): A meditation technique, using internal silent mantra repetition. The TM technique and TM movement were introduced in India in the mid-1950s by Maharishi Mahesh Yogi (1914–2008) and has achieved worldwide reach since the 60's.

Ujai pranayama: A kind of yogic breathing technique which produces a light sonorous sound.

Utkatasana: Squatting pose.

Uttankoormasana: Tortoise pose.

Vajrasana: The 'thunderbolt' pose.

Virasana: Hero's pose.

Vinyasa: Breath-synchronized yogic movement sequence.

Vipassana meditation: Vipassana (Insight - in Pali) in the Buddhist tradition refers to insight into the true nature of reality. Vipassana meditation is an insight or mindfulness method of self-observation which focuses on the close interconnection between mind and body. This interconnection is experienced directly by paying close attention to the physical sensations throughout the body. Vipassana Meditation in the tradition of Sayagyi U Ba Khin as taught by S.N. Goenka and hundreds of his assistant teachers runs regular courses worldwide.

Virabhadrasana: Warrior's pose

Yama: The first limb of Patanjali's eightfold path of yoga; translated from Sanskrit as moral injunctions, self restraint, and abstention.

Yoga Nidra: Yogic sleep - in Sanskrit**;** a deep relaxation and meditation technique aiming to bring the body to a state of complete rest and the mind to a state of full awareness.

Zen meditation: Zazen in Japanese - is the core practice in Zen Buddhism. Specific choice of practice methods depend on the specific school with the first step usually involving a concentration practice for anchoring the mind by observing or counting the breath or by focusing on an area below the navel. The two main schools of Zen Buddhism differ in main meditation method. In the Rinzai school pondering of 'Koans' is the main practice method where as in the Soto school whole-hearted sitting (Shikantaza) is the main practice method. Koans are stories, dialogues, questions or statements that cannot be understood by rational thinking and intended to lead the practitioner to transcend the rational mind. Shikantaza, on the other hand, is a practice of alert attention/mindfulness in which the mind does not attach to any particular thoughts or objects, but rather involves observing whatever arises in the mind without attaching to it.

Abbreviations

AASM	American Academy of Sleep Medicine
AHI	Apnea Hypopnea Index
AI	Apnea Index
ANOVA	Analysis of Variance
ANS	Autonomic Nervous System
APRT	Abbreviated Progressive Muscle Relaxation
ASA	Australasian Sleep Association
BMI	Body Mass Index
BP	Blood Pressure
CBT	Cognitive Behavioural Therapy
CM	Cyclic Meditation
CNS	Central Nervous System
CPAP	Continuous Positive Airway Pressure device
CSA	Central Sleep Apnea-Hypopnea syndrome
DASS	Depression Anxiety Stress Scale
DI	Desaturation Index (see ODI)
DSPL	Daily Sleep and Practice Log
ECG	Electrocardiogram
EDS	excessive daytime sleepiness
EEG	Electroencephalogram
EKG	see ECG
EOG	Electro-oculogram
ERS	European Respiratory Society

ESS	Epworth Sleepiness Scale
FDA	United States Food and Drug Administration
GABA	Gamma-amino butyric Acid
HI	Hypopnea index
HR	Heart Rate
HRV	Heart Rate Variability
IHG	Isometric Hand Grip
ITT	Intention to Treat
IYTA	Israel Yoga Teachers Association
KSS	Karolinska Sleepiness Scale
MANOVA	Multivariate Analysis of Variance
MAP	Multivariable Apnea Prediction index
MBCT	Mindfulness-Based Cognitive Therapy
MBSR	Mindfulness-Based Stress Reduction
MM	Mindfulness Meditation
ODI	Oxygen Desaturation Index
OSA	Obstructive Sleep Apnea
OSAHS	Obstructive Sleep Apnea Hypopnea Syndrome
OT	On Treatment
PMR	Progressive Muscle Relaxation
POMS	Profile of Mood States
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
QoL	Quality of Life

RAI	Respiratory Arousal Index
RDI	Respiratory Disturbance Index
REM	Rapid Eye Movement
RERA	Respiratory Effort Related Arousal
RMIT	Royal Melbourne Institute of Technology
SE	Sleep Efficiency
SF36	Short Form health survey (36 items)
SDB	Sleep-disordered breathing
SKY	Sudarshan Kriya Yoga
SOL	Sleep Onset Latency
SPO2	Blood oxygen saturation
SRBD	Sleep Related Breathing Disorder
SWS	Slow Wave Sleep
SZMC	Shaare Zedek Medical Centre
TST	Total Sleep Time
TTB	Total Time in Bed
VU	Victoria University
WASO	Wake After Sleep Onset
WLC	Waiting List Control
YHC	Yoga High Compliance subset group
YI	Yoga Intervention
YLC	Yoga Low Compliance subset group

Summary

The aging process is associated with a significant increase in the prevalence of sleep disorders, the most common being insomnia and obstructive sleep apnea (OSA). Insomnia is associated with daytime sleepiness and drowsiness, reduced attention, poor memory, slowed reaction time and reduced problem solving capacity. Insomnia is recognised as a major cause of morbidity in the elderly population and is associated with diminished mental and physical health and reduced quality of life along with increased likelihood of nursing home placement and increased risk of accidents and falls. Effective management of insomnia may contribute significantly to the reduction of morbidity and enhancement of quality of life in older adults and may help reduce health resource utilisation and related costs. While sedative-hypnotic drugs are currently the main form of treatment of insomnia, they have limited effectiveness and are associated with various side-effects, including daytime drowsiness, that further increase the risk of accidents and falls in the elderly population. Therefore, finding alternative, safe, non-drug interventions is highly desirable.

Yoga encompasses a wide range of practices, including physical exercises, breath exercises, meditation exercises and relaxation exercises. Studies have shown that yoga provides various physical and mental health benefits including reduction of stress, anxiety, depression, somatic and mental hyper-arousal. These conditions have been found to be strongly associated with insomnia. Therefore, the present study hypothesised that yoga intervention may be of benefit in alleviating geriatric insomnia.

Little research has been published (by early 2007) on yoga as an intervention for improving sleep quality and quality of life of older people in a western cultural setting. A single study conducted in India (Manjunath & Telles, 2005) and two studies

conducted in Taiwan (Chen et al., 2008; Chen et al., 2009; Chen et al., 2010) have shown yoga improved some subjective sleep quality and quality of life measures of older people. The aim of the present study was to fill a gap in existing research and evaluate whether a yoga intervention could improve sleep quality and quality of life of elderly people living in a western cultural setting. The study was designed to be ecologically valid in that it followed current clinical guidelines regarding patients presenting with insomnia complaints and included typical older people presenting with insomnia symptoms and excluded those presenting with other morbid or co-morbid conditions which may affect sleep.

A mixed 'waiting list control' (WLC) study design (n =74) was used, with participants' ages ranging from 60 to 87 (M = 74.4, SD = 7.1). The yoga intervention included two weekly classes incorporating physical and meditative yoga, and daily home practice of meditative yoga for 12 weeks. Subjective measures included reliable and valid self-reported questionnaire that assessed sleep quality, mental health and physical health. These included the Pittsburgh Sleep Quality index (PSQI), Karolinska Sleepiness Scale (KSS), Epworth Sleepiness Scale (ESS), Multivariate Apnea Prediction Index (MAP), Profile of Mood States (POMS), Depression Anxiety and Stress Scale (DASS), Short Form Health Survey (SF36) and daily sleep and practice logs. Objective measures were derived from sleep studies conducted using portable monitoring in each of the participants' home environment.

Overall, the results indicate that yoga appears to be a safe, easy to implement, and well accepted, non-drug intervention for insomnia in the elderly in a western urban cultural setting. Practicing yoga for at least 25 minutes a day for twelve weeks improved most aspects of subjective sleep status and many aspects of psychological and emotional

well being. Practice compliance was found to be an essential factor in the outcomes of the yoga intervention with high practice compliance being related to improvements in sleep quality measures and some quality of life measures. In comparison to the control group the treatment group showed significant improvements in a range of subjective factors including overall sleep quality (p=.011), sleep efficiency (p=.045), sleep latency (p=.004), sleep duration (p=.042), self assessed sleep quality (p=.002) and fatigue (p=.010). Improvements were also seen in general well being (p=.008), overall mental health (p=.009), depression (p=.019), anxiety (p=.011), stress (p=.022), tension (p=.044) anger (p=.005), vitality (p=.053) and daily function in physical (p=.035), emotional (p=.043), and social (p=.030) roles. Improvements were also seen in some objective sleep quality factors including duration of the deep sleep (SWS) stage (p=.042). However, no significant change was found in the use of sedative-hypnotic medications in the study population.

It was also found that obstructive sleep apnea (OSA) appears to be present to varying degrees in the majority of elderly people presenting with insomnia symptoms. While the results suggest that yoga practice did not result in a significant change in OSA status, the presence of co-morbid OSA reduced the efficacy of yoga in improving overall sleep quality and sleep efficiency and some aspects of quality of life including carrying out daily roles and social function, but did not affect most other measures. Currently, recommended diagnostic procedures for patients presenting with insomnia symptoms appear inadequate to diagnose OSA reliably in elderly patients presenting with insomnia symptoms, suggesting they should be screened for OSA using objective sleep studies prior to prescribing sedative-hypnotics.

Chapter 1. Introduction

1.1 Overview

The following review is intended to provide background on key issues related to the present study's aims, design, intervention, outcome measures, analysis and discussion of results. It is not intended to serve as a comprehensive review of aging, sleep, sleep disorders, sleep study technology or yoga. Accordingly, this review will briefly describe sleep physiology, sleep staging and sleep disorders. The review will then focus mainly on insomnia and geriatric insomnia. The review will more briefly discuss obstructive sleep apnea (OSA) and geriatric OSA due to the part OSA played in the present study. The review will also discuss traditional polysomnography versus novel methods for sleep analysis such as those used in the present study. Next, the review will briefly discuss yoga from ancient and modern perspectives in order to facilitate better understanding of the considerations guiding the design and implementation of the intervention. Since the body of evidence on some aspects of yoga is relatively limited in scope and quality, the review will also include some evidence on related practices that are aligned with yogic techniques incorporated in the present study. The review will then focus on aspects of yoga relevant to the intervention applied in the present study.

1.2 The aging population

In most developed countries the population is aging. A decline in fertility and a 20 year increase in average life span over the second half of the 20th century combined with elevated fertility over the 20 years following World War II (nicknamed the "Baby Boom"), will increase the proportion of those aged 65 years and over during 2010-2030 (Centers for Disease Control and Prevention [CDC], 2003). Thus, the average life span

is expected to increase by an additional 10 years by 2050 worldwide (CDC, 2003) with the number of older Americans being expected to reach 71 million, or roughly 20% of the US population by 2030 (CDC, 2007). Like that of most developed countries, Australia's population is ageing rapidly. From mid 1989 to mid 2009, the proportion in the population of those aged 65 years and over increased from 11.0% to 13.3% (Australian Bureau of Statistics [ABS], 2009) while the proportion of the Australian population aged less than 15 years old is projected to fall from 19.3% at June 2006 to 15.0% at June 2056 and the proportion of the population aged 65 years and over is projected to grow from 13.3% at June 2006 to 26.4% at June 2056 (Australian Government -Department of Health and Aging [DHA], 2006).

Elderly populations suffer from a much higher proportion of chronic diseases, ensuing disabilities and reduced quality of life (CDC, 2003). In the US it is estimated that 80% of older adults have at least one chronic health condition (CDC, 2007) and 50% have at least two chronic health conditions (CDC, 2003). Thus, amongst Americans aged 65 and over it is estimated that 55% of women and 42% of men have arthritis, 58 % of women and 43% of men have high blood pressure, 27 % of women and 38% of men have heart disease, 21 % of women and 24% of men have cancer, 30 % of women and 42% of men have trouble hearing, 19 % of women and 15% of men have trouble seeing (Federal Inter-agency Forum on Aging Related Statistics [FIFARS], 2010) and 30% of post menopausal women have osteoporosis (Office of the Surgeon General, US Dept. of Health and Human Services [OSG], 2004).

The prevalence of sleep disorders including insomnia and obstructive sleep apnea (OSA) also increases significantly with age (Wolkove et al., 2007; Ancoli-Israel et al., 1991). The growing elderly population increases demands on public and private health

systems and social services. This translates to increased healthcare related costs, and requires increasing infrastructure and human resources to maintain healthcare systems and public health programs (CDC, 2003).

1.3 Sleep

1.3.1 Defining Sleep

"The Principles and Practice of Sleep Medicine" defines sleep as follows:

"According to a simple behavioural definition, sleep is a reversible behavioural state of perceptual disengagement from and unresponsiveness to the environment. It is also true that sleep is an amalgam of physiologic and behavioural processes. Sleep is typically (but not necessarily) accompanied by postural recumbence, behavioural quiescence, closed eyes and all the other indicators one commonly associates with sleeping" (Carskadon, M. A. & Dement, W. C., 2010, p. 16).

1.3.2 Sleep physiology and staging

Sleep timing is closely related to the circadian rhythm, a roughly 24-hour cycle affecting many life processes. Sleep timing is regulated by a light-entrainable circadian pacemaker located in the suprachiasmatic nucleus of the hypothalamus portion of the brain (Dijk & Duffy, 1999) which regulates the secretion of melatonin from the pineal gland. Melatonin's primary physiological function is to convey information related to the light/darkness cycle to body physiology and there is evidence that melatonin plays an important part in the coupling of circadian rhythms, especially core body temperature and sleep-wake rhythms (Claustrata, et al., 2005).

Sleep staging criteria has been standardized and is described in "*A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human*

Subjects" (Rechtschaffen and Kales, 1968) and more recently revised and updated by the American Academy of Sleep Medicine (AASM) in the "*AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications"* (Iber et al., 2007). Sleep cycles several times during the total sleep period. Each cycle usually lasts 90 minutes on average in young adults (Pollak et al., 2010, p. 156) and includes Non Rapid Eye Movement (NREM) sleep followed by Rapid Eye Movement (REM) sleep (Pollak et al., 2010, pp. 228-229). Non-rapid eye movement (NREM) sleep constitutes approximately 80% of total sleep time and has been subdivided by Rechtschaffen & Kales into four stages, NREM stages 1, 2, 3 and 4 (Silber et al., 2007). NREM Stage 1, sometimes called the somnolence or drowsy stage, is a transitional period between sleep and wakefulness where sleep is light, and slow rolling eye movements may often be detected. It constitutes about 4 -5 percent of total sleep time and is characterised by medium amplitude combination of alpha (8-13 Hz) and low frequency (2-7 Hz) brain waves, (Pollak et al., 2010, p. 228; Silber et al., 2007). NREM stage 2 constitutes about 45 -50 percent of total sleep time. In NREM stage 2 the sleep becomes deeper and brain activity shows typical superimposed wave formations called K-complexes and sleep spindles (Pollak et al., 2010, p. 228; Silber et al., 2007). K-complexes are well-defined negative sharp wave-forms, followed immediately by a slower positive complex with the total duration of at least 0.5 seconds (Silber et al., 2007). Sleep spindles are bursts of 11-16 Hz (usually 12-14 Hz) waves that occur for at least 0.5 seconds (Silber et al., 2007). NREM Stages 3 and 4 are referred to jointly as Slow Wave Sleep (SWS) or deep sleep. SWS is the deepest sleep phase and is characterised by low frequency high amplitude brain waves that are 75 microvolt or greater as observed on EEG recordings (Pollak et al., 2010, p. 228; Silber et al., 2007).

The SWS stage is believed to contribute to the restorative processes that occur during sleep and is related to Growth Hormone (GH) secretion with studies reporting a linear relationship between amounts of SWS and amounts of GH secretion (Van Cauter et al., 1998) and a close temporal and quantitative relationship between GH secretion and SWS (Gronfier et al., 1996; Holl, 1991). Furthermore, pharmacologically stimulated SWS sleep, via oral administration of gamma-hydroxybutyrate (GHB), an effective stimulant of SWS in normal subjects, has been shown to result in increased GH secretion (Van Cauter et al., 1998; Van Cauter et al., 1997; Gronfier et al., 1996). Studies have also shown that selective suppression of SWS in healthy adults results in marked decreases in insulin sensitivity without adequate compensatory increase in insulin release, leading to reduced glucose tolerance and increased diabetes risk (Tasali et al., 2008). In rats the occurrence of SWS, but not REM sleep nor wakefulness, was associated with higher rates of protein synthesis throughout the brain, suggesting that SWS favours the restoration of cerebral proteins (Ramm & Smith, 1990).

REM sleep is characterised by rapid eye movements, a reduction or inhibition of voluntary muscle tone and increased mixed frequency low voltage brain activity, (Pollak et al., 2010, pp. 228-229; Silber et al., 2007). Most dreaming occurs during REM sleep and dreams are more vivid and more easily recalled then those which may occur in non-REM sleep (Pollak et al., 2010, p. 68),

The American Academy of Sleep Medicine (AASM) Visual Scoring Task Force has proposed a modified sleep staging scheme: Stage W (Wakefulness), Stage N1 (equivalent to NREM 1 sleep stage), Stage N2 (equivalent to NREM 2 sleep stage), Stage N3 (equivalent to SWS), and Stage R (equivalent to REM sleep) (Silber et al., 2007). The AASM Task Force found no evidence to indicate validity or biological

significance in subdividing the SWS stages into the two separate stages NREM 3 and NREM4 (Silber et al., 2007). The results of the objective sleep staging measures used in the present study have been further simplified and divide sleep into light sleep (which includes stages N1 and N2), SWS and REM.

1.3.3 Sleep studies

Polysomnography (PSG), also referred to as a sleep study, is a comprehensive multi-parameter recording of changes in various physiological variables that occur during sleep and is used to measure, diagnose or exclude, various sleep disorders (Pollak et al, 2010, pp. 176-178). In full traditional PSG these variables usually include brain electrical activity, eye movement, muscle electrical activity, electrical activity of the heart, limb movements, oral and nasal airflow, respiratory related movement (chest and abdominal movement), blood oxygen saturation and body position (Pollak et al, 2010, pp. 176-178; American Psychiatric Association [APA], 2000, p. 598). These variables are measured using non-invasive sensors that are applied to the patient's body and connected via leads to a central computerised monitoring and recording system. The technologies used to measure and analyse these signals are:

1. Electroencephalography (EEG) for measuring, recording and evaluating the brain's spontaneous electrical activity.
2. Electrooculography – (EOG) for measuring, recording and evaluating the resting potential of the eye retina.
3. Electromyography (EMG) - for measuring, recording and evaluating the electrical activity produced by skeletal muscles.
4. Electrocardiography (ECG or EKG) for measuring, recording and evaluating the electrical activity of the heart.

5. Peripheral Pulse Oximetry for measuring, recording and evaluating the oxyhaemoglobin saturation level in the patient's blood.

(Pollak et al, 2010, pp. 176-178; APA, 2000, p. 598).

Traditional Polysomnography (PSG) is generally conducted overnight in a sleep laboratory facility which is monitored by one or more sleep scientists. This is still the preferred method for diagnosis of sleep disorders, but it is expensive, inconvenient for patients, and because demand is greater than available supply, there are long waits and delays in diagnosis (Morgenthaler et al., 2010). In the United States the average time from a referral to a sleep specialist to a diagnosis in a sleep lab is 2-10 months (Morgenthaler et al., 2010). Therefore, PSG is gradually being supplemented or replaced by other techniques that allow at-home measurements, including portable monitoring and actigraphy (Morgenthaler et al., 2010).

Portable monitoring often has fewer data channels than traditional PSG, but is easier to use and interpret, and reduces costs by at least 35% compared to PSG (Morgenthaler et al., 2010). While the accuracy and reliability of portable monitoring technologies have been questioned in the past (Flemons et al., 2003), recent advancements have significantly narrowed the performance gap between traditional PSG and portable monitoring. For example, the positive predictive value of PSG in patients with moderate to severe obstructive sleep apnea is 99%, compared with 95% for portable monitoring (Morgenthaler et al., 2010). This constitutes a modest reduction in diagnostic power which is counterbalanced by portable monitoring's timeliness, greater patient comfort and substantial cost reduction (Morgenthaler et al., 2010). The Portable Monitoring Task Force of the American Academy of Sleep Medicine has published specific guidelines for the use of unattended portable monitoring in the diagnosis of obstructive sleep apnea in adult patients (Collop et al., 2007). According to

these guidelines portable monitoring should be performed only in conjunction with a comprehensive sleep evaluation supervised by a certified sleep medicine practitioner. Portable monitoring can be used as an alternative to PSG for diagnosing OSA in patients with a high pre-test probability of moderate to severe OSA but it is not considered appropriate for the for general screening for OSA of *asymptomatic* populations or the diagnosis of OSA in patients with significant comorbid medical conditions that may degrade its accuracy (Collop et al., 2007). Portable monitoring has been used in the present study (see section 3.4.3.2).

Actigraphy is occasionally used in conjunction with a sleep logs and a medical interview for the limited aim of studying sleep-wake patterns and circadian rhythms. It is based on assessing movement, most often of the wrist, using an actimetry sensor mounted in a wrist-watch like package, to measure and record gross motor activity. The data are then downloaded to a computer system for further analysis. Actigraphy offers significant cost reduction compared to PSG and simple portable monitoring and also allows longer monitoring periods (Littner et al., 2003; Thorpy et al., 1995; Morgenthaler et al., 2007).

Studies of Electrocardiography (ECG) and Heart Rate Variability (HRV) signal analysis have culminated in the development of mathematical algorithms for extraction of variables related to sleep directly from HRV data itself. These studies have shown that HRV analysis by time-dependant spectral analysis can be used to give information on the function of the autonomic nervous system (ANS) - specifically shifts between sympathetic and parasympathetic dominance and associated sleep stages (Baharav et al., 1995; Keselbrener et al., 1996). Later studies have revealed features related to awakenings and sleep stages can be extracted from HRV analysis (Ehrhart et al., 2000; Shinar et al., 2001; Shinar et al., 2003a; Shinar et al., 2006). Research has further demonstrated that ECG waveform analysis can be used to extract features related to

body position and EMG (Shinar et al., 2003b; Shinar et al., 1999). Extensive research into the link between obstructive sleep apnea (OSA) and the ECG has resulted in the development of algorithms for detecting OSA by analysing ECG data (Moody et al., 1985; Shinar et al., 2000; Baharav et al., 2001; Baharav et al., 2004; Dorfman-Furman et al., 2005). These studies have provided the theoretical foundation for the development of a comprehensive computerised sleep analysis system which enables using a smaller number of data channels, thus significantly reducing costs and enabling rapid, reliable and consistent analysis within a short time span (Hypnocore, 2010) (Used in the present study. See section 3.4.3.3).

1.4 Sleep disorders

A sleep disorder (somnipathy) is a condition that leads to a disruption of sleep patterns or abnormal behaviour occurring during sleep, or excessive daytime sleepiness and/or fatigue. The cause can be physiological, psychological, environmental or lifestyle factors. The disorder can be primary or secondary (i.e., a by-product of another primary disorder) or co-morbid (i.e., independent of, and coexisting with, another disorder). There are at least 77 recognised separate sleep disorders (Pollak et al., 2010, p. 212). The most prevalent are insomnia, sleep apnea, restless leg syndrome (RLS) and narcolepsy (Pollak et al., 2010, p. 212). The exact number of sleep disorders varies depending upon the particular classificatory system used. The main classificatory systems can be found in the "*International Classification of Sleep Disorders (ICSD-2)"* published by the American Academy of Sleep Medicine (American Academy of Sleep Medicine [AASM], 2005), the sleep disorders section of the "*Diagnostic and Statistical Manual of Mental Disorders"* (DSM IV-TR) published by the American Psychiatric Association (APA, 2000, pp. 597-661), and the non-organic sleep disorders section (general classification no.F-51) of the "International Classification of Disease" (ICD-

10) published by the World Health Organisation (WHO, 2007). In general sleep disorders can be divided into three major categories: Dyssomnias, Parasomnias and secondary sleep disorders, which may be caused by another primary medical or psychiatric disorder or by substance abuse (APA, 2000, pp. 597; The National Center for Biotechnology Information [NCBI-Mesh], 2010). Dyssomnias are primary disorders of sleep initiation or sleep maintenance or excessive sleepiness and are therefore mainly characterised by a disturbance in quantity, quality or timing of sleep (APA, 2000, pp. 598). They include primary insomnia, primary hypersomnia, narcolepsy, breathing related sleep disorders (BRSD) and circadian rhythm disorders (APA, 2000, p. 599). Insomnia is characterized by persistent difficulty falling asleep and/or difficulty maintaining sleep and/or non-restorative sleep (APA, 2000, p. 599), where as hypersomnia is characterized by excessive daytime sleepiness (APA, 2000, p. 604). Dyssomnias in general can be further subdivided into three subcategories based on the cause of the disorder, namely, intrinsic (i.e., the cause is from within the body), extrinsic (i.e., an environmental or pathogenic cause external to the body), and disturbances of the circadian rhythm (AASM, 2001, p. 15), which is the clock driven 24-hour cycle of physiological, biochemical and behavioural processes (Pollak et al., 2010, pp. 51-52). Parasomnias are abnormal movements or behaviours occurring during sleep. They are caused by inappropriate activation of the autonomic nervous system (ANS), motor system or cognitive process during sleep or sleep-wakefulness transition stages (APA, 2000, pp. 630-631). Parasomnias can be subdivided into arousal disorders (that include sleepwalking, sleep terrors and confusional arousals), sleep-wake transition disorders (that include sleep starts, sleep talking, nocturnal leg cramping and rhythmic movement disorders), parasomnias of REM sleep (such as nightmares, sleep paralysis, REM sleep behaviour disorder etc.) and a fourth group of all other non specific parasomnias (that

include sleep bruxism, primary snoring, sleep enuresis, abnormal swallowing syndrome etc.) (Pollak et al., 2010, pp. 51-52).

1.5 Insomnia

1.5.1 Insomnia - overview

Insomnia is mainly characterised by difficulty to initiate or maintain sleep, or complaints of non restorative sleep, which cause significant daytime impairment or distress (APA, 2000, p. 599). Insomnia is the most prevalent, chronic sleep disorder in the general population and has potentially serious consequences (Mai & Buysse, 2008; Harvey, 2001; Ford & Kamerow; 1989; NIH, 2005, p. 3). Insomnia has been considered both a symptom and a disorder in its own right. This distinction may have affected how it has been treated both in research and in clinical settings (Mai & Buysse, 2008).

Insomnia has a major impact on individuals and society. Chronic insomnia is associated with reduced quality of life, fatigue, mood disturbances, occupational performance impairment and decreased productivity (Mai & Buysse, 2008). Chronic insomnia sufferers may have poorer general health and use health care services more often (Mai & Buysse, 2008; Simon & Vonkroff, 1997). Also, they are more likely to be involved in industrial and motor vehicle accidents (Mai & Buysse, 2008; Chilcot & Shapiro, 1996; Balter & Uhlenhuth, 1992). All these consequences result in considerable direct and indirect economic burden (Mai & Buysse, 2008; Walsh & Englehardt, 1999; Chilcot & Shapiro, 1996).

The aging process is associated with physiological changes which may affect sleep (Van Someren, 2000). Diminished subjective sleep quality is one of the most frequent health complaints in the elderly (Prinz, 1995) with more than 80 percent reported having experienced some sleep disturbance and 50 percent reported frequent

occurrence of sleep disturbances (Foley et al., 1995, as cited in Schneider, 2002). In the elderly, undiagnosed and untreated insomnia may cause impaired daily function and reduced quality of life and is a risk factor for accidents and falls (Ancoli-Israel, 2009; Vitiello, 1999; Ancoli-Israel & Ayalon, 2006; Brassington et al., 2000; Byles et al., 2000). Benzodiazepines are currently the preferred pharmacologic intervention for insomnia (Holbrook et al., 2000a) but their use is associated with increase in adverse events (Nowell et al., 1997) and existing data supports only short-term use (Soldatos et al., 1999; Kupfer & Reynolds, 1997; Holbrook et al., 2000b; Nowell et al., 1997; Bain, 2006).

1.5.2 Insomnia - definitions and main classifications

Insomnia is a complex condition associated with diverse complaints including difficulty initiating and/or maintaining sleep and/or early morning final awakening and/or non-restorative sleep resulting in daytime fatigue, low energy, distress, functional, and social or work-related impairment. Insomnia may be transient, intermittent or chronic, lasting days or years. It can manifest as a primary, co-morbid or secondary disorder, resulting from another medical or psychiatric disorder or from drug use/abuse. It can be triggered by stressful personal circumstance or environmental factors. The symptoms may manifest separately or in various combinations (Pollak et al., 2010, pp. 111-114). All these factors have resulted in a lack of uniformity in definition and diagnostic criteria for insomnia.

The definition and diagnosis of insomnia are guided by the "*International Classification of Sleep Disorders*" (ICSD-2) by the American Academy of Sleep Medicine (AASM, 2005), the "*Diagnostic and Statistic Manual of Mental Disorders*" - (DSM IV-TR) by the American Psychiatric Association (APA, 2000), and the

"*International Classification of Disease*" (ICD-10) by the World Health Organisation (World Health Organisation [WHO], 2007). These classification systems differ somewhat in defining insomnia with regards to emphasis on frequency, severity, duration and consequences of symptoms (Mai & Buysse, 2008; Ohayon, 2002). Mai & Buysse summarise the three main definitions of insomnia as follows:

1. **"*ICSD-2 Main Criteria and definitions for Insomnia:***

   1. A complaint of difficulty initiating sleep, difficulty maintaining sleep, or waking up too early or sleep that is chronically unrestorative or poor in quality. In children, the sleep difficulty is often reported by the caretaker and may consist of observed bedtime resistance or inability to sleep independently.
   2. The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.
   3. At least one of the following forms of daytime impairment related to the night-time sleep difficulty is reported by the patient: fatigue or malaise; attention, concentration, or memory impairment; social or vocational dysfunction or poor school performance; mood disturbance or irritability; daytime sleepiness; motivation, energy, or initiative reduction; proneness for errors or accidents at work or while driving; tension, headaches, or gastrointestinal symptoms in response to sleep loss; concerns or worries about sleep" (Mai & Buysse, 2008).

2. ***"DSM-IV-TR Criteria for Primary Insomnia:***

1. The predominant complaint is difficulty initiating or maintaining sleep, or non-restorative sleep, for at least one month.

2. The sleep disturbance, or associated daytime fatigue, causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

3. The sleep disturbance does not occur exclusively during the course of Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia.

4. The disturbance does not occur exclusively during the course of another mental disorder (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, a delirium).

5. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition" (Mai & Buysse, 2008).

3. ***"ICD-10 Criteria for Nonorganic Insomnia***

A condition of unsatisfactory quantity and/or quality of sleep, which persists for a considerable period of time, including difficulty falling asleep, difficulty staying asleep, or early final wakening. Insomnia is a common symptom of many mental and physical disorders, and should be classified in addition to the basic disorder only if it dominates the clinical picture. (Mai & Buysse, 2008)"

The definitions above touch upon various aspects of insomnia, some of which deserve further deliberation, namely: relationship to other disorders (secondary

insomnia), duration (transient, intermittent or chronic insomnia), symptoms and effects. These will be discussed in the following sections.

1.5.3 Primary, secondary and co-morbid insomnia

Insomnia can manifest as a primary disorder, a co-morbid disorder or secondary disorder (Mai & Buysse, 2008; Harvey, 2001). Chronic insomnia may coexist with other chronic medical or psychiatric conditions and may relate to these conditions in various ways (Ancoli-Israel, 2006). Sleep complaints may be a symptom of a primary disorder, such as congestive heart failure or conversely, may be a factor in the etiology of other disorders such as diabetes mellitus (Ancoli-Israel, 2006). The prevalence of insomnia is higher amongst patients with other chronic conditions than in the general population and evidence suggests that secondary insomnia which is a symptom of chronic disorders tends to be more severe and persistent than insomnia which is not a consequence of another chronic disorder (Ancoli-Israel, 2006)

The distinction between primary, secondary and co-morbid insomnia has importance in both clinical and research settings as it affects diagnosis, treatment strategy, study design and result analysis. The DSM-IV definition of primary insomnia (see above) centres on difficulty in initiating and/or maintaining sleep, or unrestorative sleep, lasting at least one month and resulting in daytime impairment or distress which does not occur exclusively together with other medical or psychiatric or substance use/abuse problems. A diagnosis of primary insomnia requires sleep disturbance to be the primary complaint (APA, 2000, p. 599), while secondary insomnia is a consequence of another sleep disorder, medical or psychiatric condition, or the use/abuse of drugs (Pollak et al., 2010, pp. 111-113). Secondary insomnia has to be etiologically related to a primary condition which predates the onset of the insomnia (Lichstein, et al., 2001;

Pollak et al., 2010, pp. 111-113). Co-morbid insomnia, although co-existing with another condition (i.e., medical or psychiatric condition, sleep disorder or substance use/abuse), is not etiologically related to it (Lichstein, et al., 2001). Following these definitions, diagnosis of primary or co-morbid insomnia would indicate a need to treat it directly. On the other hand, it has often been implied that the causative primary disorder should be treated effectively first in order to alleviate a secondary type insomnia (Lichstein, et al., 2000). However, studies suggest that secondary insomnia can also be treated successfully by direct intervention, and therefore patients suffering from secondary insomnia should not be excluded from direct insomnia treatment (Lichstein et al., 2000). Furthermore, the discrimination between secondary insomnia and co-morbid insomnia often relies mainly on evidence provided by the patient with regards to the course of the disorder and this may result in misdiagnosis (Lichstein et al., 2000; Lichstein et al., 2001).

1.5.4 Insomnia differentiation by duration

Insomnia can also be differentiated based on the time scale as transient, intermittent or chronic insomnia defined as follows:

1. Transient insomnia –occurs from one day to several weeks
2. Intermittent insomnia – is transient insomnia which occurs occasionally
3. Chronic insomnia – occurs most nights over a one month period

(NIH, 2003; Schneider, 2002)

Transient and intermittent-transient insomnia may be triggered by fluctuating environmental conditions (e.g., extreme temperatures, noise, light and other disturbances), transitory life style factors and sleep hygiene factors (e.g.,- work-related, travel-related, holiday-related events), stress caused by transitory life circumstances, and transitory side-

effects of drug use/abuse (Pollak et al., 2010, pp. 112-114; APA, 2000, pp. 599-603). The cause of chronic insomnia is frequently a combination of factors, including physiological and mental health disorders, regular drug use/abuse, chronic stress, and other sleep/wake cycle disorders (Pollak et al., 2010, pp. 112-113; Schneider, 2002).

1.5.5 Insomnia differentiation by symptoms

The following night-time symptoms may manifest separately or in combination:

1. Difficulty in initiating sleep
2. Difficulty in maintaining sleep, frequent awakenings, and difficulty resuming sleep
3. Difficulty in resuming sleep after awakening early in the morning

Accordingly, insomnia can be further classified based on symptom dominance as sleep onset insomnia, sleep maintenance insomnia, and terminal/early morning insomnia (NIH, 2003, p. 5; Schneider, 2002). This classification may have clinical relevance as sleep onset difficulty is often associated with anxiety and/or stress and/or heightened nocturnal mental or physiological arousal (Schneider, 2002; APA, 2000, p. 599). Conversely, sleep maintenance difficulty may be related to a range of psychiatric or physiological conditions such as depression, chronic stress, chronic pain, illnesses and also life style /sleep hygiene factors, which may affect circadian rhythms (e.g., shift work) (Pollak,et al., 2010, p. 112; APA, 2000, p. 600; Schneider, 2002).

Insomnia may result in various daytime symptoms including fatigue, lack of energy, sleepiness, irritability and mood disturbances, reduced concentration and reduced ability to perform various tasks (APA, 2000, p. 600; Schneider, 2002). These symptoms are not used to diagnose insomnia on their own as they may also be

associated with other conditions such as obstructive sleep apnea (OSA) (APA, 2000, p.617).

1.5.6 Diagnosis of insomnia in clinical settings

In clinical settings insomnia is primarily diagnosed by a clinical evaluation based on a systematic review of medical history, psychiatric history, substance use history and sleep history. This information may be acquired by patient interview, review of medical history and by administering medical/psychiatric questionnaires, a sleepiness assessment tool, sleep logs, measures of subjective sleep quality, daytime function, and quality of life and psychological assessment scales (AASM, 2010; Schutte-Rodin et al., 2008; Mai & Buysse, 2008; Littner et al., 2003) (All of these are used in the present study. See section 3.4.2). According to the 2003 practice parameters established by the American Academy of Sleep Medicine (AASM), PSG is not indicated for the routine evaluation of transient insomnia, chronic insomnia, or insomnia associated with psychiatric disorders. However, PSG is indicated in cases when a sleep-related breathing disorder (SRDB) or periodic limb movement disorder (PLMD) is suspected, or when initial diagnosis is inconclusive, initial treatment fails, or precipitous arousals occur with violent or injurious behaviour (Littner et al., 2003; Schutte-Rodin et al., 2008).

1.5.7 Prevalence and risk factors of insomnia

A study by the National Sleep Foundation found about one-third of Americans reported some type of sleep problem with approximately 25 percent reporting occasional insomnia and nine percent reporting sleep difficulties every night. This study is consistent with other national studies (Ancoli-Israel & Roth, 1999). Estimates of the prevalence of insomnia in the general population vary greatly from 10 percent to up to

40 percent, depending on what diagnostic criteria and definitions are used (Mai & Buysse, 2008; Roth, 2007; Buysse et al., 2005; Holbrook et al., 2000a; Montgomery, 2002). This is demonstrated by a review of 50 insomnia studies based on data collected in various representative community-dwelling samples that compares insomnia prevalence estimates using four different definitions: (1) insomnia symptoms; (2) insomnia symptoms with daytime consequences; (3) sleep dissatisfaction; and (4) insomnia diagnoses with prevalence estimates using these four definitions found to be as follows:

1. Approximately one-third of a general population presented with *at least one* of DSM-IV insomnia diagnostic criteria
2. Adding the criteria of daytime consequences of insomnia reduces the prevalence estimate to between 9 and 15 percent.
3. Using the sleep dissatisfaction criteria yields an estimate of between 8 and 18 percent of the general population.
4. Finally, using a precise/stringent process based on all DSM-IV classification criteria, corresponding to a clinical diagnostic procedure, yields prevalence estimate of six percent for the general population (Ohayon, 2002).

Similarly, a more recent study estimated insomnia prevalence of approximately 30 percent based on several population-based studies with a variety of adult samples from different countries with diagnostic criteria of *one or more* of the following symptoms of insomnia: Difficulty initiating sleep, difficulty maintaining sleep, waking up too early and in some studies the additional requirement of non restorative or poor quality sleep. However, by adding a diagnostic requirement of perceived daytime impairment or distress which was caused by the sleep disturbance, prevalence estimate dropped to 10 percent (NIH State-of-the-Science Conference Statement, 2005, as cited in Roth, 2007). Finally,

when using the stricter DSM-IV diagnostic criteria, estimates again dropped to approximately six percent (Roth, 2007).

An important factor affecting insomnia prevalence estimation is the distinction between primary and secondary insomnia, since many people suffering from insomnia also suffer from various other conditions, including other sleep disorders, medical conditions, psychiatric conditions, and conditions related to substance use/abuse. Insomnia has been associated with arthritis, cancer, kidney disease, pulmonary disease, hypertension, cardiovascular disease, stroke, dementia, asthma, sleep apnea, narcolepsy, neurological conditions (e.g., Parkinson's disease, Restless Legs Syndrome), endocrine system conditions (e.g., diabetes, hyperthyroidism, hypothyroidism), anxiety, depression, and mental illnesses, such as schizophrenia, and substance abuse disorders (Buysse et al., 2005; Lichstein et al., 2001; Schneider, 2002). A study based on data from the US National Ambulatory Medical Care Survey of Office Based Physicians (1989 - 1990) found only 31 percent of insomnia patients were diagnosed with primary insomnia; 30 percent also suffered depression; 20 percent also suffered from other mental disorders, and 19 percent also suffered from other medical conditions (Radecki & Brunton, 1993, as cited in Holbrook et al., 2000a).

Insomnia has also been associated with demographic and social factors. Higher rates of insomnia complaints were found in women (Ohayon, 2002) with a higher frequency of insomnia being reported in post menopausal women and in the elderly (65 year of age and older) (Schneider, 2002; Holbrook et al., 2000a). Elderly females in particular report sleep disturbances more frequently than elderly males. This may be related to some extent to postmenopausal hormonal changes such as estrogen deficiency which has been suggested to contribute to sleep problems and is often experienced

during the peri and post menopausal period and increases with age (Vitiello et al., 2004; Moe, 1999).

Studies have also found a strong association between various psychological conditions and insomnia. Recent research suggests an extensive overlap between anxiety and insomnia among the elderly is a result of a common vulnerability to negative emotionality and at times may act as risk factors for each other (Magee, & Carmin 2010). A review on determinants of geriatric insomnia, based on 18 relevant studies, concluded that bad sleepers had significantly higher scores for anxiety, neuroticism and depression than good sleepers and that anxiety and depression correlated positively with insomnia and negatively with sleep duration. In addition, depression, anxiety or neuroticism were often better predictors of insomnia than health indicators such as perceived health and number of prescribed drugs. These findings imply that there is a stronger association between primary geriatric insomnia and psychological factors than between primary geriatric insomnia and lifestyle or health indicators (Beullens, 1999).

The strong association between psychological factors, arousal and insomnia was further demonstrated in a large study of a randomly selected sample from the general population (n=3600, duration - one year) that revealed that anxiety, depression, arousal and beliefs in the long-term negative consequences of insomnia, were significantly related to the maintenance of insomnia (Jansson & Linton, 2007).

An association has been found between chronic pain and insomnia. A study of close to 19,000 individuals, representative of approximately 206 million Europeans, concluded that chronic pain is as important as mood disorders in contributing to insomnia. Chronic pain was associated with a worsening of insomnia as measured by a

greater number of insomnia symptoms, more severe daytime consequences and a more chronic insomnia condition (Ohayon, 2005). Another study found that 53 percent of chronic pain patients had scores suggestive of clinical insomnia on the Insomnia Severity Index versus three percent of subjects with no pain. Affective pain was also revealed as a significant predictor of insomnia severity (Tang et al., 2007).

History of insomnia is a risk factor for insomnia with 31 percent reporting insomnia at first interview also reporting insomnia at one year follow-up (Ford & Kamerow, 1989). Family history of insomnia is another risk factor with close to 27 percent reporting insomnia having at least one relative with insomnia complaints (Bastien et al., 2000).

With age, the prevalence of various sleep disorders increases. Sleep disorders such as primary and secondary insomnia, snoring and obstructive sleep apnea (OSA) occur more frequently in the elderly population (Wolkove et al., 2007). Objective sleep studies reveal sleep impairment, including increased wakefulness, arousal from sleep and decreased slow wave sleep (SWS) duration, even in healthy elderly (Prinz, 1995). Research has also shown that with age the ability to sleep decreases but not the need for sleep (Ancoli-Israel, 1997) and that diminished subjective sleep quality is one of the most frequent health complaints in the elderly (Prinz, 1995). Often, sleep disorders in the elderly are overlooked and undertreated by physicians and the complaints or symptoms attributed to coexistent medical or psychiatric conditions (Wolkove et al., 2007).

In a National Institute of aging study of 9000 elderly 65 year of age and older, more than 80 percent reported having experienced some sleep disturbance and 50 percent reported frequent occurrence of difficulty falling asleep, or difficulty

maintaining sleep or waking up too early, or needing a daytime nap, or daytime fatigue. Common complaints, confirmed by objective sleep studies, included sleep fragmentation, decreases in total sleep time, and increases in daytime sleepiness (Foley et al., 1995, as cited in Schneider, 2002). Using the broad diagnostic criteria of difficulty in initiating or maintaining sleep, insomnia affects up to half of elderly over 65 with the prevalence being higher in elderly women than in elderly men (Moan, 1992). A German survey of 330 elderly patients aged over 65 years treated in general practice, applied DSM-III-R diagnostic criteria and found 23 percent had severe insomnia, 17 percent had moderate insomnia and 17 percent had mild insomnia with more than 80 percent of the patients reporting chronic insomnia for one to five years or longer (Hohagen et al., 1994).

In institutionalised elderly, sleep disorders severity and frequency is even greater than in the general elderly population (Ancoli-Israel, 2009). Institutionalization of the elderly by itself is associated with reduced sleep quality. A study reported that 83 percent of residents in 'low care' housing considered themselves as poor sleepers compared to 60 percent living independently (Middlekoop et al., 1994). Several environmental and life style factors that have been shown to affect circadian rhythms and degrade sleep quality, such as low levels of sunlight exposure and lack of physical activity (Dijk & Duffy, 1999) are more common in 'aged care' facilities (Hood et al., 2004). In addition, longer than average time in bed, typical of aged care institutions, increases sleep fragmentation (Fetveit & Bjorvatn, 2002). Anxiety and depression, often associated with transition from one's home into an aged care facility, may also have a significant negative effect on sleep (Middlekoop et al., 1994; Sukegawa, 2003).

1.5.8 Insomnia - aetiology and pathogenesis

Primary psycho-physiological insomnia is thought to develop because of learned associations that have a detrimental impact on sleep (Pollak et al., 2010, p.183). It has been considered as either a predominantly psychological condition or as a predominantly physiological condition. Typically life stresses cause an increased level of tension and agitation which may manifest physiologically as muscular tension and vasoconstriction (Pollak et al., 2010, p.183) and/or manifest psychologically as an over concern about the inability to fall asleep, which creates a vicious circle of increased difficulty in falling asleep that in turn increases the preoccupation and concern about not being able to fall asleep (Pollak et al., 2010, p.183).

The physiological hyper arousal model theory is often used to explain a possible cause of insomnia on the physiological level. This theory postulates that a constant elevated level of alertness is a major cause of sleep difficulties. It has been supported by several studies using a range of subjective self-reported hyper-arousal scales and objective measures. These showed a tendency towards physiological hyper-arousal in individuals with chronic insomnia. This tendency may in turn be aggravated by other factors (e.g., psychiatric disorders or other sleep disorders) also associated with insomnia (Stepanski et al., 1988; Pavlova et al., 2001). Insomnia patients scored higher than normal sleepers on self reported hyper-arousal total scale score (Pavlova et al., 2001). Insomnia sufferers have also been found to have an elevated metabolic rate (measured by overall oxygen consumption) compared with normal sleepers (Bonnet & Arand, 2003). Other studies have shown that inducing higher physiological arousal levels for a week (by means of high caffeine intake) in normal sleepers also produced insomnia symptoms. However, inducing disturbed sleep in normal sleepers did not result in increased physiological arousal levels (Bonnet & Arand, 2003; Bonnet &

Arand, 1997) indicating that hyper arousal results in insomnia but that insomnia does not result in hyper-arousal.

Primary insomnia is associated with altered brainwave activity. Increased EEG beta wave activity has been found in subjects with primary insomnia compared to subjects with secondary insomnia and normal sleepers and this increased beta activity was negatively associated with the perception of sleep quality (Perlis et al., 2001). A study comparing the EEG frequency spectra of normal sleepers to those sub-typed subjective insomnia sufferers (with relatively long total sleep time coupled with relative underestimation of sleep time) and to those sub-typed objective insomnia sufferers (with relatively short actual total sleep time) found lower delta NREM EEG activity and higher alpha, beta and sigma (sleep spindles) NREM EEG activity in subjective insomnia patients, but not in objective insomnia patients compared to normal sleepers. There were no differences amongst the groups for REM EEG activity (Krystal et al., 2002). In addition, a significant correlation was found between NREM EEG frequency spectral indices and sleep complaints in patients with subjective insomnia, which may reflect heightened autonomic arousal during sleep (Krystal et al., 2002).

In addition to altered EEG activity, insomniacs have been found to exhibit greater pre-sleep Central Nervous System (CNS) arousal than normal sleepers (Jacobs et al., 1993). A study using functional neuro-imaging, found increased brain glucose metabolism both during sleep and wake states in insomnia patients compared to normal sleepers indicating that insomnia is associated with greater brain metabolism (Nofzinger et al., 2004). Several studies have also found that neuro-endocrine and clinical similarities exist between primary insomnia and major depressive disorder, related to abnormal corticotrophin releasing factor (CRF) activity. CRF hyperactivity seems to

mediate the hyper-arousal seen in primary insomnia. These findings imply hypothalamic-pituitary-adrenal (HPA) axis and CRF over activity in both disorders (Roth et al., 2007).

1.5.9 Insomnia and aging

Typically, the following changes in sleep may occur with aging:

1. A phase advance in the normal circadian sleep cycle (i.e., going to sleep earlier and waking up earlier).
2. Total nocturnal sleep time decreases (6.5 hours average for people over 60 compared to 7-8 hours in younger adults).
3. Delayed sleep onset.
4. Decrease in slow wave sleep (SWS) phase duration.
5. Decrease in rapid eye movement (REM) phase duration.
6. Decrease in arousal threshold.
7. Sleep becomes more fragmented with increased frequency of arousals and awakenings.

   (Wolkove et al., 2007; Holbrook et al., 2000a; Reidel et al.,1995; Reidel & Lichstein, 1998; Schneider, 2002; Prinz, 1995).

The aging process is associated with physiological changes which may affect sleep. With age, a lack of input to the suprachiasmatic nucleus (SCN), which is the brain's biological clock, may accelerate de-activation of neurons involved in the generation of circadian rhythm or output of this rhythm. These alterations appear to contribute strongly to sleep problems (Van Someren, 2000). This is supported by evidence of an age-related decline in production of melatonin. Melatonin has several claimed neurobiological roles, namely -an anti-ageing agent; a free-radical scavenger; a

regulator of circadian rhythm; and an endogenous sleep-inducer and it is suggested that a decline in melatonin production may also affect sleep quality (Rikkert & Rigaud, 2001). Circadian rhythms in the elderly also become more affected by daytime napping and low levels of exposure to sunlight, which are both more prevalent in the elderly population (Van Someren, 2000) and there is a decrease in arousal threshold in the elderly which in turn increases the sensitivity to environmental factors such as noise, light, temperature and results in higher probability of sleep disturbance occurring (Bliwise, 2000, as cited in Ellis et al., 2002)

Ageing is associated with sleep phase alterations. A meta analysis of 65 studies representing 3,577 subjects aged 5 years to 102 years, concluded that sleep efficiency, percentage of slow-wave sleep (SWS), percentage of REM sleep, and REM latency all significantly decreased with age, while sleep latency (SOL), percentage of stage 1 NREM sleep, percentage of stage 2 NREM sleep, and wake after sleep onset (WASO) significantly increased with age (Ohayon et al., 2004).

In addition to age-related physiological changes, which contribute to a higher prevalence of insomnia amongst the elderly, the elderly may also be more vulnerable to the effects of poor sleep hygiene and the effects of medications, including those prescribed for sleep problems and for other co-morbid medical conditions that are also more prevalent with age (Reidel & Lichstein, 1998; Schneider, 2002). The physiological and metabolic changes associated with aging affect the pharmacokinetics of drugs (Higbee, 2000) and may increase the time taken to metabolise stimulants such as coffee so that caffeine consumption after 2 PM in the afternoon is associated with longer duration of insomnia in the elderly (Ellis et al., 2002). With aging, total body fat also increases so that fat-soluble drugs, such as benzodiazepines have an increased volume

of distribution and decreased clearance rates (Schneider, 2002). In addition, drug metabolism may be altered due to impaired oxidation, reduction and hydroxylation. Reduced hepatic blood flow and glomerular filtration may also result in decreased drug excretion from the body. These changes result in increased central nervous system (CNS) sensitivity to effects of depressants (Schneider, 2002).

1.5.10 Insomnia - prognosis

Despite its high prevalence, insomnia symptoms may be attributed to various co-morbid conditions and insomnia is often misdiagnosed and may persist untreated for years, long after the original causative factors have resolved. Patients with primary insomnia, with typical onset at early adulthood, often do not present for treatment until middle adulthood. As a result, patients often resort to various self-help remedies including over the counter medications, alcohol, 'natural products', and various unproven treatments (Morin et al., 2006; Wolkove et al., 2007; APA, 2000, pp. 601-602; AASM, 2001, p. 29).

Findings regarding the prognosis of insomnia are inconclusive, however if left untreated insomnia is likely to become chronic. In a prospective study with a cohort of 13,563 participants aged 45 to 69 years, neither insomnia complaints nor hypnotic use predicted increased mortality over a 6.3 years period (Phillips & Mannino, 2005). On the other hand, a review of 16 studies reporting an association between insomnia and sleeping pills usage and mortality, concluded that the evidence linking insomnia and/or hypnotic consumption and mortality is suggestive but inconclusive and additional higher quality research is necessary (Nuhic & Kramer, 2007). Another review of insomnia epidemiological research, reported that sleep medication use is predictive of mortality but the evidence for insomnia as a risk factor for mortality is inconclusive

(Taylor et al., 2003). A large study (n=3445) of patients suffering at least one of five physician-identified chronic conditions (hypertension, diabetes, congestive heart failure, myocardial infarction, or depression) found 16 percent had severe insomnia and 34 percent had mild insomnia at baseline. A follow-up conducted after two years found 59 percent of patients with mild insomnia and 83 percent of patients with severe insomnia at baseline still suffered sleep problems (Katz & McHorney, 1998).

1.5.11 Insomnia's impact

1.5.11.1 Insomnia's impact on general quality of life

Comorbid insomnia may have a major negative impact on quality of life and overall functioning (Ancoli-Israel, 2006). A study of 1913 German adults found that quality of life was rated as bad in 22 percent and good in 28 percent of severe insomniacs compared to three percent (bad) and 68 percent (good) in normal sleepers (Hajak et al., 2001). Several Studies have compared the quality of life of insomnia patients to control groups of individuals with no sleep complaints using SF-36 questionnaire. This questionnaire measures general health and quality of life using eight scales representative of mental, physical and social functioning, physical and emotional well-being (SF36 was used in the present study; please refer to section 3.4.2.7 and to appendix 5.7). Patients with severe insomnia had lower scores on all eight scales of the SF-36 than those with mild insomnia or good sleepers while patients with mild insomnia had lower scores than good sleepers on all eight scales of the SF-36. Overall, the insomnia group had lower mean scores on all SF-36 subscales compared with the control group indicating impairment across diverse aspects of quality of life (Zammit et al., 1999; Leger et al., 2001). Insomnia has also been shown to be independently associated with poorer health-related quality of life comparable to that found with

chronic disorders such as congestive heart failure and clinical depression (Katz & McHorney, 2002).

1.5.11.2 Insomnia's impact on mental health

Insomnia is associated with a range of psychiatric disorders, and it is estimated that 40% of insomnia patients have a comorbid psychiatric condition, although the exact nature of the relationship has not been established. Insomnia is a risk factor for the development of anxiety disorder, depression and other psychiatric disorders (Ancoli-Israel, 2006; Ford & Kamerow, 1989; Breslau et al., 1996) as well as being a key symptom for various psychiatric disorders - depressive disorders in particular (Ford & Kamerow, 1989; McCall, 2001; Riemann et al., 2001). In a review of eight epidemiological studies, a very strong correlation was found between insomnia and later development of depression within one to three years, implying insomnia symptoms on their own have predictive value of depression onset in later years (Riemann & Voderholzer, 2003). Another review of epidemiological studies found that insomnia was consistently predictive of depression, anxiety disorders, other psychological disorders, alcohol abuse/dependence, drug abuse/ dependence, and suicide, indicating insomnia is a risk factor for these conditions (Taylor et al., 2003). Furthermore, a study showed that psychiatric history has been found to be closely related to the severity and chronicity of existing insomnia and also that chronic insomnia may be a residual symptom of a past mental disorder putting the patient at a higher risk of relapse (Ohayon & Roth, 2003). Results from a study using epidemiological data from a survey of over 10,000 community dwelling adults in the US suggest that uncomplicated insomnia is associated with an increase in a risk for a first onset of major depression, panic disorder, and alcohol abuse in the following year. These results further suggest that insomnia, even in

the absence of psychiatric disorders, was associated with increased utilisation of general medical and mental health services for emotional problems (Weisman et al., 1997).

1.5.11.3 Insomnia's impact on physical health

Insomnia is associated with a range of medical disorders with chronic insomnia patients reporting a higher frequency of various medical conditions compared to normal sleepers including heart disease (21.9% vs. 9.5%), high blood pressure (43.1% vs. 18.7%), neurologic disease (7.3% vs. 1.2%), breathing problems (24.8% vs. 5.7%), urinary problems (19.7% vs. 9.5%), chronic pain (50.4% vs. 18.2%), and gastrointestinal problems (33.6% vs. 9.2%) (Taylor et al., 2007). On the other hand, people suffering the following medical conditions also reported a higher frequency of chronic insomnia than those not suffering from these medical conditions: heart disease (44.1% vs. 22.8%), cancer (41.4% vs. 24.6%), high blood pressure (44.0% vs. 19.3%), neurologic disease (66.7% vs. 24.3%), breathing problems (59.6% vs. 21.4%), urinary problems (41.5% vs. 23.3%), chronic pain (48.6% vs. 17.2%), and gastrointestinal problems (55.4% vs. 20.0%) (Taylor et al., 2007).

In a cross-sectional study of 30,397 participants aged 18 years and older involved in the 2005 US National Health Interview Survey a positive association was found between both shorter and longer sleep durations and cardio vascular disease (CVD) suggesting that sleep duration may be an important marker of CVD (Sabanayagam, & Shankar, 2010). This is supported by a 12 year prospective study of 1870 subjects in Sweden which found an association between difficulty falling asleep and death from coronary artery disease in men but not in women (Mallon et al., 2002). There is further evidence to suggest that sleep complaints may actually be a factor in the etiology of some disorders, such as diabetes mellitus (Ancoli-Israel, 2006).

1.5.11.4 Socio-economical impact of insomnia

Insomnia is associated with considerable socio-economical impact as sleep disturbances may impact daytime activities and often result in daytime fatigue, memory deficits and other cognitive deficits, which may have substantial impact on daytime functioning in critical tasks such as driving and work, resulting in an increase in work absenteeism and accidents (some with catastrophic consequences) (Chilcott & Shapiro, 1996). Studies in the US and in France found insomniacs had double the rate of work absenteeism, took more sick leaves, showed reduced work performance and more frequent work-related accidents in comparison to good sleepers. Insomniacs also reported poor work related self-esteem, less job satisfaction, and reduced efficiency at work, compared with good sleepers (Leger et al., 2002; Leger et al., 2006; Novak et al., 2004). A study in France found that drivers with insomnia had a higher road accident rate and a threefold greater risk of having two or three serious road accidents compared to good sleepers (Leger et al., 2006).

Insomnia is also associated with increased health care utilisation and it is reported that compared to good sleepers, people with insomnia consult physicians more often, report more medical problems, consume more medications, have more emergency visits to hospitals and are hospitalised twice as often (Leger et al., 2002; Novak et al., 2004). Insomnia is also associated with greater functional impairment, reduced productivity, and higher health care utilization among primary care patients (Simon & Von Korff, 1997).

Insomnia results in substantial direct and indirect costs to individuals, the health care system and society. Estimates of insomnia's economic impact vary widely depending on the methodology used. In general, two main cost categories are analysed -

direct and indirect costs. Direct costs are those mostly related to application of medical intervention for treating insomnia and its side effects and consequences. These include costs of treatment, examinations, medicines, hospitalizations and other medical costs due to increased morbidity and mortality, depression related to insomnia, and increased insomnia related alcohol consumption. Indirect costs are related to disorders and/or conditions induced as consequences of insomnia including costs resulting from death, accidents, reduced productivity, increased absenteeism etc. (Drummond et al., 2005; Ozminkowski et al., 2007). A US study of 138,820 younger adults (aged 18 - 64) and 75,558 elderly (aged 65 and above) suffering from insomnia, with equivalently-sized control groups, revealed average direct and indirect costs for younger adults with insomnia over a six months period were about $1,253 greater than for good sleepers. For the elderly, direct costs were about $1,143 greater for insomniacs than for good sleepers (Ozminkowski et al., 2007). An estimation of direct health care costs of insomnia in 1995 in the US was $13.9 billion. Total cost for medications alone was $1.97 billion, of which less than half was for prescription medication. Health care services for insomnia totalled $11.96 billion, 91% of which was for nursing home care (Walsh & Engelhardt, 1999). Direct health care costs of insomnia in 1995 in France was estimated to be $2.07 billion (Leger et al., 1999). An estimate of the total annual cost of insomnia in the US in 1996 was between $92.5 and $107.5 billion US dollars (Chilcott & Shapiro, 1996). A more conservative estimate of total direct, indirect and related costs of insomnia in the US in 1994 was between 30 to 35 billion US dollars (Stoller, 1994).

1.5.11.5 Insomnia's impact on older adults

In the elderly, undiagnosed and untreated insomnia may cause impaired daily function and reduced quality of life. It is associated with diminished attention,

concentration, memory, reaction time, problem solving, cognition and overall decreased sense of well-being. It is also a risk factor for accidents and falls (Ancoli-Israel, 2009; Vitiello, 1999; Ancoli-Israel & Ayalon, 2006; Brassington et al., 2000; Byles et al., 2000). Falls are the leading cause of injury-related visits to emergency departments in the US and the main cause of accidental deaths in elderly patients (65 years and older) (Fuller, 2000). More than 90 % of hip fractures occur as a result of falls, the majority in individuals 70 and older. One third of community-dwelling elderly and 60 percent of nursing home residents fall each year (Fuller, 2000). The mortality rate for falls increases significantly with age across genders, racial and ethnic groups, with falls accounting for 70% of all accidental deaths in elderly aged 75 years and older (Fuller, 2000). Therefore, it is not surprising that diminished sleep quality is associated with higher morbidity and mortality rates in the elderly population (Hays et al., 1996; Ancoli-Israel, 2009). Sleep disturbances are often a factor in the decision taken by family members to move an older adult into an aged care facility and are associated with an increased likelihood of nursing home placement (Pollak et al., 1990; Pollak & Perlick, 1991) Taken together the research findings presented above emphasise the urgent need to address insomnia in the elderly population.

1.5.12 Treatments for insomnia

1.5.12.1 Overview

Due to the diverse nature and causation of insomnia, a variety of treatment approaches are used. In some cases, treating a primary physiological or mental disorder may result in improvement of the secondary sleep disorder (insomnia), while in other cases, treating the secondary sleep disorder (insomnia) symptoms may improve the primary disorder (Ancoli-Israel, 2006). Interventions for insomnia fall into two main

categories: pharmacological and non-pharmacological. Pharmacotherapy can be carried out on the symptomatic management level or by specific diagnosis and treatment. Pharmacological intervention relies mostly on sedative hypnotic drugs and in some cases on antidepressants. Benzodiazepines are most often prescribed and to a lesser extent non-benzodiazepines sedative agents (Kramer, 1999). Non-pharmacological treatments for primary and secondary insomnia include various combinations of cognitive behavioural therapies (CBT), various forms of relaxation training, phototherapy (Petit et al., 2003) and also several less researched interventions for sleep disturbance such as exercise and acupuncture.

1.5.12.2 Pharmacological treatment for insomnia

Benzodiazepines are currently the preferred pharmacologic intervention for insomnia. They are widely used because to date, no other class of drugs has been proven superior to benzodiazepines in terms of benefit to risk ratio (Holbrook et al., 2000a). Studies have revealed short-term treatment (2-4 weeks) of chronic insomnia using benzodiazepines or Zolpidem (a non-benzodiazepine hypnotic) produced reliable improvements, most notably, a significantly increased total sleep duration (Nowell et al., 1997). However, benzodiazepine treatment was also associated with an increase in adverse events, particularly daytime drowsiness and dizziness or light-headedness (Nowell et al., 1997). Several meta-analyses suggest intermediate and long-term pharmacotherapy for insomnia is associated with diminished benefits, tolerance, and insomnia rebound effects (Soldatos et al., 1999; Nowell et al., 1997). Furthermore, existing data supports only short-term use as data on long-term usage and consequences is lacking (Soldatos et al., 1999; Kupfer & Reynolds, 1997; Holbrook et al., 2000b; Nowell et al., 1997; Bain, 2006).

One meta-analysis using data from 45 randomised, controlled trials, representing a total of 2672 patients, (including 15 studies of patients over 65 years of age), concluded the overall benefit of benzodiazepines appears to be minor because they do not provide a major advantage over placebo in the treatment of insomnia. This meta-analysis also found that benzodiazepines use was often associated with adverse cognitive effects. The authors therefore recommended non-pharmacological interventions for insomnia (Holbrook et al., 2000b). Another study found sleep medication use was predictive of mortality although it did not suggest a direct causative link (Taylor et al., 2003).

Melatonin (N-acetyl-5-methoxytryptamine), is produced by the pineal gland in response to reduced photo-input. The dim light melatonin onset (DLMO) is a standard marker for the human circadian pattern (Kramer, 1999) and plays a role in regulation of the circadian cycle, and other physiological processes. Melatonin is a broad-spectrum antioxidant and a potent free radical scavenger with a particular role in protection of nuclear and mitochondrial DNA and a preventive role in oncogenesis (Altun & Ugur-Altun, 2007; Reiter et al., 2001). Newer melatonin based medications mimic physiological pattern of the endogenous hormone by prolonged release (PR) throughout the night. A study of PR melatonin intervention in primary insomnia patients aged 55 years and older has shown significant improvement in sleep latency and quality of sleep, as well as next morning alertness with no evidence of abuse potential or rebound insomnia or withdrawal effects following treatment discontinuation. This study also found a low incidence of adverse events, most side-effects of minor severity, and no evidence of impairment of cognitive and/or psychomotor skills, compared to placebo (Lemonie et al., 2007; Zisapel, 2009).

In case of co-morbid depression and insomnia, anti-depressants and sleep hygiene manipulation may serve a dual role. As described earlier, sleep disturbances are associated with depression and are typical of depressed patients. Various studies have demonstrated that in depressed patients there is reduction of slow wave sleep (SWS) and disinhibition of REM sleep. Most of the effective antidepressant agents have been found to suppress REM sleep. In addition, manipulations of the sleep-wake cycle, such as sleep deprivation or a phase advance of the sleep period, have been shown to alleviate depressive symptoms. These findings imply a strong bi-directional relationship between sleep, sleep modification, and depression (Riemann et al., 2001). However, antidepressants have been found to have variable effects on sleep and some antidepressants seem to worsen sleep in patients with depression (Jindal & Thase, 2004).

Benzodiazepines have respiratory depressant effects and may worsen sleep-related breathing disorders (SRDB). Long-term use may cause complete obstructive sleep apnea (OSA) in heavy snorers or short repetitive central sleep apnea in patients with recent myocardial infarction and for this reason benzodiazepines use is contraindicated in patients with sleep apnea (Guilleminault, 1990; Wagner et al., 1998). Non-benzodiazepines drugs for insomnia cause minimal respiratory depression, and therefore may be safer than benzodiazepines in patients with respiratory disorders (Wagner & Wagner, 2000).

1.5.12.3 Non-pharmacological treatments for insomnia

1.5.12.3.1 Introduction

Due to pharmacotherapy's limitations and side effects, a wide range of alternative therapies for treating insomnia have been tried. An AASM appointed expert task force

reviewed the scientific evidence on non-pharmacological interventions for insomnia from 1999- 2006 (Morgenthaler et al., 2006a). The task force found psychological and behavioural interventions to be effective in the treatment of both chronic primary and secondary insomnia. Specifically, stimulus control therapy, relaxation training, cognitive behavioural therapy, sleep restriction therapy, multi-component therapy (that does not include cognitive therapy), biofeedback and paradoxical intention were found to be individually effective interventions for chronic insomnia (Morgenthaler et al., 2006a). However, psychological and behavioural interventions require the involvement of a health care professional, are usually administered individually and are therefore costly in terms of health-care utilisation. On the other hand, like yoga, relaxation and physical exercise, also discussed below, are more applicable to both individual self-help practice, as well as group practice and arguably, this makes them more cost-effective and widely accessible than most other non-drug treatments for insomnia. Furthermore, relaxation and exercise share some common elements with meditative and physical yoga exercises respectively (see sections 1.5.12.3.9 and 1.5.12.3.13) and therefore evidence on their effect on sleep quality and quality of life is also reviewed below. Overall, there is some evidence on the positive effects of some relaxation techniques and some types of exercise on sleep quality however evidence on their effect on geriatric insomnia is lacking and more research is required.

1.5.12.3.2 Cognitive behavioural therapy (CBT)

Cognitive behavioural therapy (or CBT) refers to a wide range of psychotherapeutic methods aimed at resolving dysfunctional emotions, behaviours and cognitions. CBT in the broad sense may refer to any combination of cognitive therapies and behavioural therapies. A review of 16 meta-analyses supports the efficacy of CBT for diverse psychological and psychosomatic disorders (Butlera et al., 2006). A systematic review on the efficacy of cognitive behavioural therapy (CBT), bright light,

and physical exercise for treating insomnia in older adults reported that CBT only had a mild effect on sleep problems in older adults, most markedly in sleep maintenance insomnia and evidence on the efficacy of bright light and exercise interventions was too limited to enable drawing any conclusions (Montgomery & Dennis, 2004). A systematic review of seven studies of CBT intervention for primary insomnia concluded CBT was superior to any single-component therapy such as stimulus control, relaxation training, and sleep hygiene education, however more research was required to determine optimal multi therapy configuration (Wang et al., 2005)

1.5.12.3.3 Behavioural therapy

Behaviour therapy, or behavioural modification, is a form of psychotherapy that focuses on treatment of human behavioural disorders by reinforcing acceptable behaviour and suppressing of undesirable behaviour (The Columbia Electronic Encyclopaedia, 2007). A review of 12 studies of non-pharmacological interventions for treating insomnia in community-dwelling older adults reports behavioural approaches have produced reliable and durable therapeutic benefits including improved sleep efficiency and continuity and enhanced satisfaction with sleep patterns with reduced dependence on hypnotics. This review further reports that stimulus control and sleep restriction are more effective compared to relaxation methods (Morin et al., 1999 a).

1.5.12.3.4 Sleep restriction

Sleep restriction is a behavioural therapy method reported as one of the most effective non-pharmacological interventions for insomnia (Hastings & Long, 1994; Holbrook et al., 2000a; Smith et al., 2002). Sleep restriction is especially suited for insomniacs who tend to spend extended periods in bed, trying to fall asleep with limited success. The aim of this approach is to increase sleep efficiency (the ratio between

actual sleep time and total time spent in bed) (Morin et al., 1999a; Spielman et al., 1987; Glovinsky & Spielman. 1991, pp. 49-63; Hoche et al., 2001). Initially sleep restriction is often achieved by delaying bed time, which may result in mild sleep deprivation. As sleep latency and frequency of awakening decrease, bed time may be adjusted to earlier timing. The rationale behind this method is that extended time in bed leads to fragmented sleep and perpetuates insomnia. The advantage of sleep restriction is that it can be implemented easily compared to other interventions although patient compliance can be difficult (Morin et al., 1999a) and it may result in daytime sleepiness in elderly patients.

Sleep restriction protocol normally includes preliminary filling out preliminary sleep diaries (for at least 2 weeks) and restricting bedtime is then restricted to the average estimated Total Sleep Time (TST). The patient then continues to enter data into sleep diaries which are to monitor sleep efficiency. Total time in bed is modified in order to keep sleep efficiency in the range of 80–90% and is reduced by 15–20 minutes when sleep efficiency exceeds 90%. In contrast, it is decreased by 15–20 minutes when sleep efficiency is below 80% (some recommend 75% for elderly). Thus, total bedtime is thus adjusted weekly until an ideal steady state is reached but this should not fall below 5 hours. Short daytime naps are permissible especially in the early stages of therapy (Glovinsky & Spielman, 1991).

1.5.12.3.5 Stimulus control

Stimulus control is a behavioural therapy method which aims at creating an association between the bed/bedroom and rapid sleep onset by avoiding any non-sleep related activities in the bedroom (Morin et al., 1999a). The protocol generally incorporates several rules that need to be followed by the patient including:

1. Going to bed only when feeling tired.
2. Using the bed and bedroom for sleep (and possibly sex) but not for reading, watching TV, eating, drinking or pursuing mental activities (such as studying or lying awake and worrying about not being able to fall asleep).
3. Leaving the bedroom if sleep onset does not occur within 15–20 min. and returning to the bedroom only when feeling sleepy again.
4. Repeating step no. 3 again if sleep onset does not occur within 15–20 min. Repeating this procedure again throughout the night when necessary.
5. Getting up at the same time every morning regardless of amount of sleep achieved during the previous night (using an alarm clock for this purpose if necessary).
6. Avoiding daytime napping.

The advantage of this method is its relative simplicity, which enables family physicians and other health professionals to administer it with ease (Petit et al., 2003).

1.5.12.3.6 Sleep hygiene education

Poor sleep hygiene is a common cause of primary insomnia and good sleep hygiene is considered an essential component of insomnia treatment strategy (Morin et al., 1999a). Sleep hygiene education aims at teaching the patient how to modify lifestyle and environment to improve sleep quality (Morin et al., 1999a); however there is not enough evidence to support using it on its own to treat insomnia (Chesson et al., 1999). One study found sleep hygiene education resulted in improved sleep continuity and depth and better mood in the morning; however it also found sleep restriction therapy improved sleep efficiency more than sleep hygiene education (Hoch et al., 2001). Sleep hygiene education advice may typically include:

1. Avoiding caffeine, nicotine and alcohol, especially later in the day.

2. Avoiding heavy meals within two to three hours of bedtime.
3. Avoiding fluid consumption after dinner to reduce night-time urination frequency.
4. Avoiding excessive stimulation after 5 PM (noisy exciting places or situations).
5. Using the bed for sleep only and not for relaxation, reading, nor watching TV.
6. Establishing a routine for getting ready to go to bed.
7. Winding down and relaxing before going to bed.
8. Getting all the worrying done before going to bed and postponing remaining issues to the next day.
9. Maintaining comfortable bedroom temperatures; use enough but not too many blankets.
10. Trying to sleep in a quiet room or using earplugs.
11. Trying to keep the room dark; otherwise, using a sleep mask if maintaining a dark room is not possible.
12. Keeping fixed wakeup and bedtime schedule seven days a week.
13. Avoiding daytime naps as much as possible and if a nap is required – then napping before 3 PM and for no longer than one hour
14. Exercising regularly but not in the evening before going to bed

(Petit et al., 2003)

1.5.12.3.7 Paradoxical intention

Paradoxical intention aims at removing performance anxiety associated with falling asleep by asking the patient to try to remain awake as long as possible in bed in a darkened room without engaging in any other activity or turning on the lights. Paradoxical intention is supported empirically by the American Psychological Association (Morin et al., 1999) and it has the advantage that it can be administered with ease by family physicians (Chesson et al., 1999). Studies of paradoxical intention

however, all focus on sleep onset insomnia and have variable results that suggest that it is less effective than relaxation or stimulus control treatments (Morin et al., 1999).

1.5.12.3.8 Cognitive therapy

Cognitive therapy is based on the premise that emotional disturbances (e.g., anxiety and depression) are a direct consequence of dysfunctional, maladaptive beliefs and thought patterns which affect how individuals interpret and react to external events; therefore, by identifying and challenging these beliefs and thought patterns, patients can learn to modify them, and minimise associated emotional disturbances (Corey, 2009, pp. 288-289).

It has been suggested that pre-sleep cognitive activity of insomniacs can be distinguished from that of good sleepers as being more focused on worries, problems and environmental noises and that insomniacs are more likely to think about sleeplessness or daily events (Harvey, 2000). Insomniacs have also been found to have higher negative beliefs and attitudes about sleep than good sleepers (Carneya et al., 2010).

When applied to sleep disturbances, cognitive therapy aims at identifying detrimental beliefs and attitudes about sleep and then challenging, modifying or replacing them with beliefs more conducive to better sleep (Morin et al., 1999a). The therapist normally prepares the patient by explaining the relationships between cognition, affect and behaviour and the therapy's main objectives. Dysfunctional maladaptive beliefs and attitudes are then identified and replaced (Petit et al., 2003). Cognitive therapy may help empower patients and give them a sense of control over the disorder. However it requires considerable time and professional involvement that family physicians may not be able to provide. There is also insufficient evidence to

support using cognitive therapy to treat insomnia on its own (Chesson et al., 1999) yet, cognitive therapy may be effective as part of multifaceted intervention strategy (Morin et al., 1999a) and several studies consider cognitive therapy an essential component of insomnia treatment strategy that may be especially beneficial for elderly chronic insomniacs (Morin et al., 1999a; Edinger et al., 1992).

1.5.12.3.9 Relaxation therapies

A broad range of relaxation methods can be used to improve sleep (Morin et al., 1999a) although some lack evidence to support their efficacy (Petit et al., 2003). Various muscle relaxation methods such as progressive muscle relaxation (PMR) (Freeman, 2009, pp. 130-133; Cancini et al., 1983), and autogenic training (Linden, 1994; Stetter et al., 2002) methods aim at reducing somatic arousal. Most relaxation methods can be self-administered, thus reducing health resource utilisation and involve teaching patients how to voluntarily and sequentially relax tension from muscle groups in order to induce physiological relaxation (Pollak et al., 2010, p. 181). Progressive muscular relaxation therapy (PMR), later named Jacobson Progressive Relaxation Therapy (JPRT) (Freeman, 2009; pp. 130-132) is the name first given to a technique developed in the 1930s by American physician Edmund Jacobson for reduction of stress and anxiety (Freeman, 2009; p. 131). JPRT involves observing muscular tension in a specific part of the body, learning to relax the tension away and then observing the difference before and after the relaxation. This process is applied systematically to all major muscle groups in the body (Freeman, 2009; p. 131) and may take up to 90 days to complete as it focuses on a single muscle group and a single body position in each one hour session. Limb position is modified from session to session in order to learn to work with subtler levels of tension with the ultimate goal of developing an ability to

unconsciously monitor and eliminate somatic tensions (Freeman, 2009; p. 131). The original process seemed too time consuming to many and various shortened versions of progressive muscle relaxation have been developed, often too short to allow intense and detailed observation as intended by Jacobson (Freeman, 2009; p. 132). A shorter form of progressive relaxation named Abbreviated Progressive Relaxation Techniques (APRT) was developed by Wolpe in the 1950s (Freeman, 2009; pp. 132-133). In APRT relaxation of several of the 16 major muscle groups may be practiced in a single session, thereby requiring a maximum of 10 sessions to complete the entire process (Freeman, 2009; pp. 132-133). Autogenic training is a form of self relaxation procedure that trains the practitioner to focus on sensations such as heaviness and warmth in various body parts with the aim of eliciting a psycho-physiological relaxation response (Stetter & Kupper, 2002; Pollak et al., 2010, p. 31). The yogic relaxation/meditation methods used in the present study, included a yogic muscle relaxation component and a yogic meditative awareness component, in which practitioners focus on opposing body sensations, such as heat and cold (see sections 1.7.12, and 3.3.10) and therefore research findings on the effect of PMR, APRT and autogenic training are arguably of relevance to the present study. Unlike PMR and APRT, the yogic relaxation technique incorporated in the present study can be learned and executed within a single one hour session (see sections 3.3.10.4),.

A review of 29 studies found favourable outcomes of Abbreviated Progressive Muscle Relaxation (APRT) interventions compared to psychotherapy outcomes. APRT delivered on an individual basis in conjunction with providing APRT training tapes to subjects for self-practice had the strongest association to favourable outcomes. The treatment duration and number of sessions were also positively associated with improvement (Carlson & Hoyle, 1993). A meta-analysis of three studies found positive

effects of autogenic training intervention versus control for a range of conditions including functional sleep disorders, tension headache/migraine, mild-to-moderate essential hypertension, coronary heart disease, asthma, pain disorder, Raynaud's disease, anxiety disorders and mild-to-moderate depression (Stetter & Kupper, 2002). Another study that compared effects of a single session of PMR, meditation and control found participants in the meditation and PMR groups decreased more in cognitive, somatic, and general state anxiety than controls with the PMR group having the greatest reduction in somatic anxiety (Rausch et al., 2006).

Several underlying physiological mechanisms may be involved in relaxation techniques including modulation of the autonomic nervous system (ANS) and endocrine system. A study comparing relaxation training, beta-adrenergic blockers and sham training found reduced excitatory autonomic response to mental and physical stimulation, and increased vagal activity in both the relaxation and beta blocker intervention groups compared to a sham intervention group (Lucini et al., 1997). Another study indirectly demonstrated relaxation training increased endorphin production by using the opioid receptor blocking drug Naltrexone which resulted in counteracting the diastolic pressure reducing effects of relaxation training in response to mental stress (McCubbin et al., 1996).

1.5.12.3.10 Multi component therapy

Multi component therapy for insomnia combines several of the above interventions (Jacobs et al., 1993) with specific protocols being individually tailored to suit the patient. The American Psychological Association consider multi component therapy as probably efficacious (Morin et al., 1999a) and multi-component therapy has

been shown to produce better results than no treatment, but it is not always more effective than stimulus control or sleep restriction on their own (Morin et al., 1999a).

Multifactor behavioural interventions combining sleep restriction, modified stimulus control, and relaxation response training, has been found highly effective in moving individuals with chronic sleep-onset insomnia into the range of normal sleepers. The effect may have been achieved, in part, by reducing pre-sleep CNS arousal (Jacobs et al., 1993).

1.5.12.3.11 Phototherapy

The circadian cycle is usually 24 hours and is normally entrained to be aligned with the external environmental rhythm of day/night changes in light levels. Light is the chief stimulus responsible for coordinating the internal circadian rhythm with the environmental 24-hour light/dark cycle and is perceived by the retina which transoms signals via the retino-hypothalamic tract to the brain (Marieb & Hoehn, 2010, p. 566; Zisapel, 2001). Absence of adequate and timely light input may result in misalignment of the circadian clock with the environmental cycle and this may result in sleep disorders. These may include insomnias associated with the endogenous clock running slower or faster than the norm, or in an irregular rhythm; periodic insomnias caused by disturbances in light perception (e.g., in blind people); and transient insomnias due to life circumstances (e.g., shift-work sleep disorder), or travel (i.e., jet-lag) (Zisapel, 2001).

Phototherapy aims at advancing or delaying the inner circadian clock using exposure to bright light in specific dosages and time intervals, and high exposure avoidance at other times. For patients with delayed sleep-phase syndrome (DSPS), exposure to bright light in early morning and avoidance of bright light in the evening is

prescribed. Conversely, for patients with advanced sleep phase syndrome (ASPS), exposure to bright light in the evening is prescribed in attempt to delay sleep onset (Zisapel, 2001). Several studies suggest that exposure to bright light in the evening is beneficial in alleviating sleep maintenance insomnia in healthy elderly subjects (Campbell et al., 1995). Phototherapy involves administering light via light boxes or head mounted visors with an intensity f greater than 1000 Lux being recommended for the elderly (Zisapel, 2001).

1.5.12.3.12 Acupuncture

A recent Cochrane systematic review evaluating acupuncture for insomnia found that the available body of evidence included only a small number of randomised controlled trials with poor methodological quality and significant clinical heterogeneity. The reviewers therefore concluded that the available evidence is insufficient to support use of acupuncture as an intervention for insomnia, and that larger higher quality clinical trials are needed (Cheuk et al., 2007).

1.5.12.3.13 Exercise

Existing evidence indicates that exercise is associated with improvement in sleep quality in the normal population and that aerobic type exercise may improve insomnia symptoms. However, more research is required to understand the relationship between exercise types and sleep quality and identify the most suitable exercise interventions for insomnia in general and in the elderly insomniac population in particular.

Two meta-analyses examining the effects of exercise on sleep found that while the relative efficacy of specific exercise interventions for improving sleep in normal and clinical populations could not be established based on available data (Kubitz et al., 1996; Driver & Taylor, 2000), acute and chronic exercise increased slow wave sleep

(SWS) and total sleep time (TST) and decreased sleep onset latency (SOL), REM sleep latency and REM sleep duration. Moderating variables included sex, age, fitness level and exercise type, duration and timing (Kubitz et al., 1996; Driver & Taylor, 2000).

A Cochrane review of physical exercise intervention for primary insomnia, where 80% or more of participants were over the age of 60, found little high quality evidence on the relationship between exercise and insomnia symptoms. Reviewers concluded that exercise, although not appropriate for all elderly, may improve sleep and quality of life, but further research was required as these conclusions were based on a single relatively small study (n=43) (Montgomery and Dennis, 2002). A more recent study comparing aerobic exercise and resistance training as interventions for insomnia found acute moderate-intensity aerobic exercise reduced pre-sleep anxiety and improved sleep in patients with chronic primary insomnia while resistance training exercise did not (Passos et al., 2010). Conversely, a recent retrospective study of young adults (n=862, mean age= 24.67 +/- 5.91) using self reported questionnaires on perception of exercise level, fitness level, and sleep quality, concluded that perceived high physical fitness, but not exercise level itself, was associated with favourable scores for various sleep quality measures and suggested that it was the cognitive processes manifested by these self perceptions, rather than the exercise itself, that played a major role in sleep quality (Gerber et al., 2010).

1.5.12.4 Pharmacotherapy versus other interventions

Overall, evidence suggests that pharmacotherapy is effective for short term or periodic use and may be associated with adverse effects, while alternative treatments for insomnia are suitable for both short and long term use and should be attempted as first line of intervention, prior to prescribing hypnotics.

A comparative meta-analysis of 21 studies totalling 470 subjects of pharmacotherapy and behaviour therapy for persistent insomnia found that overall behaviour therapy and pharmacotherapy produce similar short-term treatment outcomes in primary insomnia (Smith et al., 2002). Another review concluded that non-pharmacological treatments such as stimulus control, sleep restriction, sleep hygiene education, cognitive therapy, multi-component therapy and paradoxical intention, used for primary and secondary insomnia in elderly patients, are feasible and effective alternatives to benzodiazepines (Petit et al., 2003). Vitiello (1999) concludes that despite being widely used, hypnotics are best suited for periodic use rather than for treatment of chronic sleep disorder symptoms and should be used only if sleep hygiene intervention as first-line therapy proves unsuccessful. In another study Holbrook et al. (2000 a) concluded that the overall benefit of benzodiazepines appears to be minor and is associated with adverse affects. They recommend trying non-pharmacological interventions for insomnia including sleep hygiene modifications and exercise. Another study found that in chronic insomniacs, the addition of medication to CBT produced added benefits during the acute phase, but the best long-term outcome was achieved when medication was discontinued during the maintenance phase of CBT (Morin et al., 2009). A study comparing behavioural and pharmacological interventions for late life insomnia (mean age 65) found that both were effective for the short term management of insomnia but improvements in sleep were better sustained with the behavioural intervention compared to pharmacotherapy (Morin et al., 1999b).

1.6 Sleep related breathing disorders (SRBD)

1.6.1 Introduction

The present study which aimed at examining the effectiveness of a yoga intervention for improving sleep and life quality in elderly people presenting with complaints of insomnia attempted to exclude people with OSA. Candidates were screened using a standard clinical procedure recommended for patients presenting with insomnia complaints in line with the 2003 practice parameters established by the American Academy of Sleep Medicine (AASM), where PSG is not indicated for the routine evaluation of transient insomnia, chronic insomnia, or insomnia associated with psychiatric disorders (Littner et al., 2003). Those suspected of suffering from other sleep disorders were excluded. Nevertheless, as described in section 4.6, additional participants were later diagnosed with previously undetected co-morbid obstructive sleep apnea (OSA). Therefore, SRBD and specifically OSA have been included in this review.

1.6.2 SRBD definitions and classifications

SRBD is a term which encompasses a group of disorders characterized by disturbed respiratory patterns during sleep, including pauses in breathing, or reduction in the volume of ventilation during sleep (Wolkove et al., 2007). These events are called apneas, hypopneas and RERAs. A hypopnea event is associated with very shallow or slow breathing, resulting in a reduction in volume of ventilation and a fall in the blood oxygen saturation level (Pollak et al, 2010, pp. 102-103). This is distinguished from an apnea event where there is a complete pause in breathing, lasting for at least 10 seconds (Pollak et al, 2010, p. 26). A more elaborate definition is used in the context of scoring

apnea and hypopnea events in a sleep study with a clinically significant apnea event being scored in an adult patient, when all of the following criteria are met:

1. *A drop in the peak excursion of oro-nasal thermal sensor* (signal) *by ≥ 90% of baseline*
2. *Duration of the event is ≥ 10 seconds*
3. *At least 90% of the apnea event duration meets the amplitude reduction criteria for apnea* (AASM, 2007, p. 45).

A clinically significant hypopnea event is scored in a sleep study of an adult patient when all of the following criteria are met:

1. *A drop in the peak excursion of oro-nasal thermal sensor* (signal) *by ≥ 30% of baseline*
2. *Duration of the event is ≥ 10 seconds*
3. *An oxygen desaturation of ≥ 4% from pre event baseline occurs*
4. *At least 90% of the hypopnea event duration meets the amplitude reduction criteria for* hypopnea (AASM, 2007, p. 46).

Alternatively, a clinically significant hypopnea event can also be scored in a sleep study when all of the following criteria occur:

1. *A drop in the peak excursion of oro-nasal thermal sensor* (signal) *of ≥ 50% of baseline*
2. *Duration of the event is ≥ 10 seconds*
3. *A desaturation of ≥ 3% from pre event baseline occurs associated with an arousal.*
4. *At least 90% of the hypopnea event duration meets the amplitude reduction criteria* (AASM, 2007, p. 46).

Increased respiratory effort that does not meet the criteria for hypopnea or apnoea may result in an arousal event termed a Respiratory Effort Related Arousal (RERA). An event is scored as a RERA if there is a sequence of breaths that lasts for at least 10 seconds and is characterised by increased respiratory effort and negative esophageal pressure (as manifested by the flattening of the nasal pressure waveform) that leads to an arousal from sleep and yet does not meet the criteria for apnoea or hypopnea (as specified above) (AASM, 2007, p. 45; Pollak et al., 2010, p. 194).

An apnea event is classified as obstructive, central or mixed type apnoea event depending on the presence or absence of respiratory effort (Pollak et al, 2010, p. 26) and is scored as an obstructive apnea event if it meets apnoea scoring criteria (as specified above) and there is continued or increased respiratory effort during the entire period of absent airflow (AASM, 2007, p. 45). It is scored as a central apnea event if it meets apnoea scoring criteria (as specified above) and respiratory effort is absent during the entire period of absent airflow (AASM, 2007, p. 45). It is considered a mixed apnea event if it meets apnoea scoring criteria (as specified above) and respiratory effort is absent in the first part of the event followed by a resumption of respiratory effort in the second part of the event (AASM, 2007, p. 45).

A task force by the American Academy of sleep medicine (AASM), the European Respiratory Society (ERS), the Australasian Sleep Association (ASA), and the American Thoracic Society, recommended the following classification of SRBD:

1. Obstructive Sleep Apnea Hypopnea Syndrome (OSAHS)
2. Central Sleep Apnea-hypopnea syndrome (CSA)
3. Cheyne-Stokes syndrome.

4. Some patients may have a complex sleep apnea syndrome for example OSAHS and CSA (AASM, 1999, as cited in Morgenthaler et al., 2006b).

Unlike OSAHS, which is caused by an obstruction to the air flow, CSA and Cheyne-Stokes syndrome are caused by an imbalance in the brain's respiratory control centres. In CSA this causes the patient to miss one or more breaths (Morgenthaler et al., 2006b). Cheyne-Stokes syndrome, on the other hand, is characterised by a regular crescendo and decrescendo fluctuation in respiratory rate and volume. This breathing pattern causes a wide fluctuation in blood oxygen and carbon dioxide levels that may trigger an arousal at the peak of the crescendo phase of this breathing pattern. This in turn may result in sleep maintenance insomnia with frequent awakenings (Pollak et al., 2010, pp. 48-49). OSAHS is a much more common disorder. A retrospective review of data of one month sample of patients referred to the Mayo clinic for objective studies in 2004 revealed the following distribution of apnoea cases: OSAHS – 84%, CSA - 0.4%, and15% of the cases presented with complex apnoea (Morgenthaler et al., 2006b).

A wider definition of obstructive SRBD disorders related to upper airway resistance includes snoring in addition to upper airway resistance syndrome (UARS), and Obstructive Sleep Apnea–hypopnea syndrome (OSAHS). Snoring is defined as loud upper airway breathing sounds in sleep, without episodes of apnea or hypoventilation, arousals or other evidence of sleep disturbance. Snoring is very common, occurring in up to 40-50% of adults aged over 65. Snoring is strongly associated with SRDB however many snorers do not have SRBD (Kushida, 2005; Netzer et al., 2003; AASM, 2001, pp. 195-196). And many clinicians regard SRBD as a spectrum or continuum of disorders so that snoring may gradually deteriorate into OSAHS as a consequence of additional risk factors or deterioration in existing risk factors (such as weight, alcohol

consumption, use of sedatives/hypnotics etc.) (Wolkove et al., 2007; Collop & Cassel, 2002; Guilleminiault et al., 1993; Lugaresi et al., 1994; Kushida, 2005).

1.6.3 Measuring and expressing SRBD severity

A range of indexes is used in conjunction with PSG data in order to diagnose and express sleep related breathing disturbances. The following are the most common currently in use:

1. The Apnea index (AI) expresses average apnea events per hour (Pollak et al., 2010, p. 26).
2. The Apnea Hypopnea index (AHI) combines apnea and hypopnea events per hour (Pollak et al., 2010, p. 26). Revision of scoring guidelines using this measures has been recommended as using different standard hypopnea definitions can yield considerable differences in the Apnea Hypopnea index (AHI) (Ruehland et al., 2009; Kushida et al., 2005).
3. The Respiratory Disturbance Index (RDI) has usually been used synonymously with AHI (Pollak et al., 2010, p. 193; AASM, 2001, p. 347).
4. Respiratory Effort Related Arousal index (RERA index) is the number of RERAs per hour (see glossary and section 1.6.2).

Other measures which may also assist in assessing severity level of SRDB include blood oxygen saturation levels (Pollak et al., 2010, p. 166) and number of arousals (AASM, 2001, p. 56).

1.6.4 Obstructive sleep apnea (OSA)

1.6.4.1 OSA - symptoms and diagnosis

OSA is the most common SRBD and is characterised by repeated episodes of complete cessation of breathing called apnea (defined above). It may also include partial reductions in ventilation volume called hypopnea (defined above) (Bassiri & Guilleminault, 2000). Other symptoms include, snoring, waking up gasping for air or feeling choked, frequent arousals from sleep associated with the apnea, dry throat or mouth upon awakening, morning headaches or nausea, mild to severe daytime sleepiness and decreased cognitive functioning (AASM, 2001, p. 57; Berg, 2008; Mendelson, 1987, p. 194). Currently the standard clinical diagnosis of OSA relies on full PSG (Berg, 2008). The evaluation of the severity of OSA relies on PSG data and information regarding daytime sleepiness levels with AHI in the range of 5–15 considered mild, AHI in the range of 15–30 considered moderate and AHI>30 considered severe. Sleepiness levels are also classified as mild (unwanted daytime sleepiness in situations requiring little attention), moderate (unwanted daytime sleepiness in situations requiring some degree of attention) or severe (uncontrollable daytime sleepiness causing significant daytime impairment) (Berg, 2008).

1.6.4.2 OSA - aetiology and pathophysiology

OSA is characterised by episodes of apnea and hypopnea. These are caused by the interaction between sleep-related changes in upper airway muscle function and subtle narrowing of the oropharyngeal lumen. This results in an occlusion of one or more structures along the upper airways including the nose, palate and base of the tongue. This in turn results in the reduction or cessation of airflow. This phenomena is often most noticeable during the rapid eye movement (REM) sleep and may explain why

OSA often first progresses and is most severe during REM sleep (Bradley & Phillipson, 1985). Apnea and hypopnea episodes lead to decrease in blood oxygen levels (hypoxia) and increase in blood carbon dioxide level (carbocemia). Hypoxia may lead to asphyxia, a condition of deficient supply of oxygen to body tissues. Asphyxia may then lead to increased inspiratory effort. The combination of increased respiratory effort and collapse of the airway leads to creation of negative pressure (i.e., partial vacuum) within the thorax (Pepperell et al., 2002) which results in additional increase in inspiratory effort (Bradley & Phillipson, 1985). The increasing respiratory effort eventually leads to an arousal that results in a restoration of the muscle tone in the upper airway thus terminating the obstructive event (Bradley & Phillipson, 1985). There is some evidence that the increased respiratory effort itself and not the hypoxia or hypercapnia is the main factor that acts as the stimulus for the arousal from sleep (Gleeson et al., 1990). The arousal usually involves going into a lighter stage of sleep or partial awakening and once the normal breathing is restored, blood gases levels normalise, and the patient may go into a deeper sleep again. However, this again leads to an apnea event and a vicious circle is set up that may repeat itself hundreds of times during the night, leading to significant disruption of sleep and degrading of sleep quality (Bradley & Phillipson, 1985). The recurrent episodes of nocturnal asphyxia followed by recurrent arousals from sleep also induce various secondary physiological responses, such as increased sympathetic tone and elevated blood pressure that may eventually produce clinical cardiovascular, hemodynamic, and neuropsychiatric symptoms associated with OSA (Bradley & Phillipson, 1985; Douglas, 2002).

The exact pathophysiology of OSA is not fully understood. However, various studies have shown both anatomical and neurological abnormalities in OSA patients. Various imaging techniques have revealed that compared to normal subjects, OSA

patients have enlarged tongues and soft palates and inferiorly positioned hyoid bones, retro-positioned mandibles and maxillae, increased mandibular plane angle, increased facial height, narrow posterior airway spaces and shorter cranial base (Low et al., 1996; Hochban & Brandenburg 1994; Battagel & L'Estrange,1996). Obese OSA patients have additional structural abnormalities which are usually absent in non-obese OSA patients. A study found that compared with the controls, non-obese OSA patients had aberrations of the cervico-craniofacial skeleton while obese OSA patients also had abnormalities of upper airway soft tissue, head posture and position of the hyoid bone (Tangugsorn et al., 2000).

The fact that OSA patients are able to maintain unobstructed airway in the waking state indicates that anatomical abnormalities are only partially responsible and suggests an existence of a neuromuscular component. EMG studies have shown that in OSA patients the pharyngeal dilator muscles are more active in the waking state compared to normal subjects (Mezzanotte et al., 1992). The activity of these muscles is reduced shortly after sleep onset, indicating they receive input from brainstem neurons involved in sleep regulation. In apnea patients, this may lead to the collapse of the pharyngeal airway (Jordan & White, 2008). It has been suggested that the initial sleep onset reduction in upper airway muscle activity is due to loss of a 'wakefulness' stimulus, rather than to loss of responsiveness to negative airway pressure, and that this stimulus may be greater in the OSA patient than in healthy controls. Furthermore, this increased stimulus in the waking state may be a compensatory mechanism for overcoming pharyngeal abnormality (Fogel et al., 2005; Mezzanotte et al., 1992). Other studies suggest that chronic intermittent hypoxia/asphyxia contributes to the pathophysiology of SRDB by reducing upper airway muscle endurance and selectively impairing pharyngeal dilator EMG responses to physiological stimulation, and in this way

perpetuating a vicious circle of airway muscle collapse and hypoxia/asphyxia (Bradford et al., 2005).

1.6.4.3 Risk factors for OSA

OSA risk factors include obesity, anatomical upper airway abnormalities, age, gender, smoking, excessive alcohol intake, use of muscle relaxants, nasal congestion, estrogen depletion in menopause and family history (Young et al., 1993; Young et al., 2002a; Berg, 2008). Population based studies reveal that the risk of OSA in males is two to three times greater than in females (Young et al., 1993; Young et al., 2002).Obesity, is probably the single most predictive factor for OSA, observed in 60% of patients with OSA in developed countries **(**Strobel & Rosen, 1996). Obesity is usually expressed in body mass index (BMI), which is calculated by dividing body mass (in Kg) by the square of the height (in meter) as follows: BMI = body mass/ (height) $^{2.}$ A strong correlation exists between BMI, OSA and excessive daytime sleepiness (EDS) in professional drivers (Dagan, et al., 2006) and in the elderly the best predictors of sleep-disordered breathing were found to be BMI, falling asleep at inappropriate times, male gender, alcohol consumption within two hours of bedtime and daytime napping (Ancoli-Israel et al., 1991).

1.6.4.4 Prevalence of OSA

Population-based epidemiologic studies have revealed a high prevalence and wide spectrum of severity for undiagnosed obstructive sleep apnea (OSA). The prevalence of mild to moderate OSA was found to be particularly high (Young et al., 2002b). A study conducted to estimate the prevalence of undiagnosed sleep-disordered breathing among adults aged 30 to 60 years evaluated by PSG, used data extracted from the Wisconsin Sleep Cohort Study, a longitudinal study of the natural history of cardiopulmonary

disorders of sleep. This study estimated the prevalence of SRBD (defined as an AHI score of 5 or higher) as 9% for women and 24% for men (Young et al., 1993). A large study (n=1741) of men and women between the ages of 20 and 100 revealed that, for clinically defined sleep apnea (AHI ≥ 10 with daytime symptoms), men had a prevalence of 3.9% and women 1.2%. In premenopausal women the prevalence of sleep apnea was found to be 0.6% and in postmenopausal women with hormone replacement therapy (HRT) 0.5% while in postmenopausal women without HRT the prevalence of sleep apnea was 2.7%. These results reveal male gender and menopause as significant risk factors for sleep apnea, and that in women HRT is associated with reduced risk (Bixler et al., 2001).

With age, upper airway muscles weaken and lose tone during sleep, predisposing the patient to airway obstruction. This may lead to snoring and obstructive sleep apnea (Wolkove et al., 2007). Indeed, the frequency of sleep apnea increases with age and is substantial amongst the elderly. A survey of 461 randomly selected elderly aged over 65 in the US found 24% had Apnea index (AI) ≥ 5 and 62% had Respiratory Disturbance Index (RDI) ≥ 10 (Ancoli-Israel et al., 1991). In another study of 1389 community dwellers aged 60 to 70 years, 49.5% of subjects reported snoring, and 10.8% reported breathing stoppages during sleep. Both disorders were significantly higher in males and those with high BMI (Dealbarto et al., 1996).

1.6.4.5 Impact of OSA

A consistent association exists between OSA, including mild OSA, and significant morbidity. Undiagnosed OSA, with or without symptoms, is independently associated with increased likelihood of hypertension, cardiovascular disease, and stroke and these conditions are all leading causes of morbidity in adults. In addition, a consistent

association exists between OSA and between daytime sleepiness, drowsiness, motor vehicle accidents, and diminished quality of life (Young et al., 2002b).

Multiple studies support the hypothesis that intermittent hypoxia induced by OSA, and not sleep fragmentation, causes persistent hypertension and that the long-term consequences of chronic intermittent hypoxia may also include cerebral and coronary vascular problems, developmental and neuro-cognitive deficits, and neuro-degeneration; however more research is required to better understand the underlying mechanisms and the relation to OSA (Neubauer, 2001). OSA-induced frequent arousals cause sleep fragmentation which results in excessive daytime sleepiness (EDS) (Pepperell, et al. 2002). Both nocturnal hypoxia and sleepiness seem to contribute to various deficits shown in various cognitive functions including memory, general intellectual tasks and executive psychomotor tasks (Bedard et al., 1991). EDS may result in periods of 'micro sleep' lasting several seconds, causing lapses of attention, which may result in motor vehicle accidents (Williamson et al., 2000).

1.6.4.6 Treatments for OSA

Various interventions are used to treat OSA including weight loss, continuous positive airway pressure (CPAP), pharyngeal surgery, medication and oral appliances (Berg, 2008). In obese patients, a long term solution also involves weight reduction strategies; however, continuous positive airway pressure (CPAP) is generally considered the treatment of choice. There is growing evidence on the efficacy of oral appliances (OA) but these are mainly indicated when CPAP cannot be tolerated since CPAP seems more effective than OA in most cases. Evidence to date does not support drug therapy and surgical procedures as interventions for OSA (Berg, 2008; Busetto et al., 2005; Smith & Lasserson, 2009; Lim et al., 2006; Smith et al., 2006).

Weight loss is recommended as it significantly reduces OSA in obese patients (Shochat & Pillar, 2003) and a strong association has been shown between OSA and obesity (Young et al., 1993; Ancoli-Israel et al., 1991). The main mechanism by which weight loss helps reduce OSA seems to be reduction in adipose tissue deposited adjacent to the pharyngeal airway. The volume of this tissue is related to the presence and degree of OSA (Shelton et al., 1993) with obese patients having significantly lower pharyngeal cross-sectional areas compared to non obese male control subjects (Busetto et al., 2005). Indeed, weight loss in obese OSA patients has been shown to be associated with a marked decrease in the pharyngeal adipose tissue volume (Shelton et al., 1993), and an increase in the size of the upper airway passage (Busetto et al., 2005), fewer apneas and hypopneas, reduction of oxygen desaturation and sleep fragmentation (Shelton et al., 1993). Even a weight reduction of 15% may substantially increase the pharyngeal cross-sectional area and substantially improve the severity of OSAS in very obese patients (Busetto et al., 2005).

The most commonly recommended and applied intervention for OSA is a continuous positive airway pressure (CPAP) device designed to deliver positive air pressure, usually, via a nose mask. The increased pressure keeps the airway unobstructed (Shochat & Pillar, 2003). A recent Cochrane systematic review concluded CPAP can be an effective treatment for OSA but compliance is the main problem with this intervention because many people cannot tolerate it, or do not use it every night. Attempts to improve compliance have included various mechanical design modifications. However, none has led to significant increases in compliance (Smith & Lasserson, 2009).

Oral appliances (OA) are designed to keep the upper airway open by either advancing the lower jaw forward or by keeping the mouth open during sleep. A Cochrane systematic review of OA for OSA found that there is increasing evidence to suggest that OA improved subjective sleepiness and sleep disordered breathing compared with a control. When comparing effectiveness of OA to CPAP, the latter appears to be more effective in improving sleep disordered breathing and OA therapy is considered appropriate for patients with mild symptomatic OSA, and those unwilling or unable to tolerate CPAP therapy. Further research is required on the effectiveness of OA for OSA patients with more severe symptoms of sleepiness (Lim et al., 2006).

Surgical intervention has had some reported success in treating OSA but a Cochrane systematic review of surgery for OSA suggests that for mixed levels of AHI, with mostly moderate daytime sleepiness, current evidence does not support the use of surgery in sleep apnea/hypopnea syndrome and that long-term follow-up of patients who undergo surgical correction of upper airway obstruction is required (Sundaram et al., 2005). Furthermore, in the elderly, there is an increased risk of surgery related complications (Shochat & Pillar, 2003).

Pharmacotherapy has been proposed as an alternative to CPAP in some patients intolerant of CPAP with mild to moderate sleep apnea. Pharmacotherapy may work in several ways to reduce the severity of OSA, including an increase in the tone in the upper airway dilator muscles, an increase in ventilatory drive, a reduction in the proportion of REM sleep, an increase in cholinergic tone during sleep, a reduction in airway resistance and a reduction in surface tension in the upper airway. A recent Cochrane review of pharmacotherapy for OSA concluded that to date there is insufficient evidence to recommend drug therapy in the treatment of OSA and suggests

that better matching of drugs to individual patients according to their dominant OSA mechanism is required (Smith et al., 2006).

Positional therapy, which involves avoiding the supine sleeping position, may be partially effective in patients who usually have apnoea events mainly in that position Evidence is lacking however, regarding the prevalence of positional apnoea (Shochat & Pillar, 2003).

Currently there is a lack of good quality evidence on the use of alternative methods for treating OSA. A single small study (n=25) of Didgeridoo (an ancient wind instrument developed by indigenous Australians of northern Australia) playing as alternative treatment for OSA in adult OSA patients with moderate OSA and snoring, reported that four months of private practice with a didgeridoo instructor resulted in significant reduction in AHI and significant improvement in subjective sleep quality measures compared to control. The suggested mechanism of action was improving muscle tone and reducing collapsibility of the upper airways (Puhan et al., 2006).

1.6.4.7 The relationship between OSA and insomnia

A recent review points to an association between insomnia and sleep disordered breathing (SDB) that has mostly been overlooked by researchers and medical professionals in the last few decades (Beneto et al., 2009). The review further reports on findings from several studies that had estimated the frequency of insomnia in OSA patients to be between 24.2% and 54.9% with recent data reporting a frequency of between 42% and 54.9% (Beneto et al., 2009). Reviewers conclude that patients with co-morbid SDB and insomnia are characterised by three main features including significant presence of psychological and psychiatric disorders, more sleep disruptions and an association with other sleep disorders such as the restless leg syndrome (RLS)

(Beneto et al., 2009). Reviewers also suggest a hypothetical metabolic/endocrine link between OSA and insomnia whereby OSA results in autonomic modulation leading to activation of the hypothalamic–pituitary–adrenal (HPA) axis which then causes increased sleep fragmentation. This in turn increases the secretion of cortisol which leads to further deterioration in sleep quality and so on. However reviewers suggest more research is required to understand the interaction and bi-directional relationship between OSA and insomnia (Beneto et al., 2009).

Findings of several studies suggest that there is a stronger association between OSA and sleep maintenance insomnia and early morning awakening type insomnia than between OSA and sleep onset insomnia (Beneto et al., 2009). In a study of 157 predominantly male OSA patients, 42% were found to have at least one insomnia symptom. However, insomnia subtypes differed in daytime sleepiness measures, Apnea Hypopnea Index (AHI) and Arousal Index (AI). The sleep onset insomnia subtype group had significantly lower AHI and AI and lower daytime sleepiness while the sleep maintenance insomnia subgroup had similar AHI and AI levels to those of OSA-only subjects and also had more severe daytime sleepiness levels than those found in the sleep onset insomnia subtype (Chung, 2005). The association between OSA and insomnia may also depend on the degree/severity level of OSA. A retrospective study of 105 patients, referred to PSG for OSA evaluation, reported a strong positive correlation between OSA and insomnia symptom severity (Smith et al., 2004). Conversely, another similar retrospective study of 255 patients suggests that insomnia is not strongly associated with SDB. This study found insomnia complaints more prevalent in patients with AHI<10 than in those with AHI≥10 and no association between AHI, Desaturation index (DI) and the severity of insomnia symptoms in a subset of 228 patients with significant SDB (Krell & Kapur, 2005).

The differentiation between SDB patients with or without insomnia complaints has clinical relevance as SDB patients with insomnia complaints were found to have a significantly higher frequency of psychiatric disorders, cognitive-emotional symptoms, physical symptoms and mental symptoms that further degraded sleep quality. These patients also reported greater use of sedative hypnotic drugs (Krakow et al., 2001). Another study of 99 elderly with and without insomnia found that those with comorbid insomnia and SRBD had the greatest functional impairment including significantly lower daytime functioning and slower reaction time (Gooneratne et al., 2006).

The overall higher prevalence of both OSA and insomnia in the elderly population (see sections 1.5.9 and 1.6.4.3) also increases the likelihood of concurrent OSA and insomnia as well as the likelihood of undiagnosed OSA to be present in elderly presenting with insomnia complaints. One study reported a high prevalence of undiagnosed sleep apnea in elderly insomniacs that had previously undergone an interview to screen for sleep apnoea. This study reported an AHI >5 in 43 percent and an AHI >15 in 29 percent of study population. Since OSA may be a confounding factor in studies on insomnia, this study suggested using PSG as a screening tool when recruiting older adults for research on insomnia (Lichstein et al., 1999).

The implications of the above findings in relation to the screening process utilised in the present study are discussed in section 3.2.2.

1.7 Yoga

1.7.1 Introduction

The use of complementary therapies, including mind-body practices, is becoming increasingly prevalent in western cultural settings. One of the most popular mind-body methods is yoga. Yoga encompasses diverse philosophical systems and practice

methods that have evolved mainly in the Indian subcontinent since ancient times. Yoga practices may include various combinations of physical exercises, breathing exercises, relaxation and meditation exercises, as well as devotional and lifestyle practices. Studies have shown yoga may provide various physical health and mental health benefits and may reduce stress, anxiety, somatic and mental hyper-arousal, that have all been shown to be strongly associated with primary insomnia. Up until early 2007, when the present study was initiated, there had been little published research on the use of yoga for improving quality of sleep in older people within western cultural settings.

Yoga Nidra, (translated from Sanskrit as 'yogic sleep'), is a special, yet simple and accessible form of yogic guided meditation technique, that incorporates several meditation and relaxation components derived from ancient yogic and Tantric practices. In Yoga Nidra the practitioner enters into a state that approximates sleep, while maintaining alertness and awareness throughout the entire process. Despite its suggestive name and its simplicity there is no published research on Yoga Nidra's effect on geriatric insomnia.

There are however, a number of published studies on the effect of various non-yogic meditation techniques, such as Transcendental meditation (TM) (Ospina et al., 2007), Zen-Buddhist meditation (Chiesa, 2009; Chiesa & Serretti, 2010; Ospina et al., 2007), Vipassana meditation (Chiesa & Serretti, 2010; Chiesa & Serretti, 2009) and various contemporary forms of mindfulness meditation (Ospina et al., 2007; Chiesa & Serretti, 2009). Although these methods are not yogic in the limited sense, they have much in common with yogic meditation techniques in general and with Yoga Nidra in particular. Various studies have also been conducted on the effect of non-yogic contemporary muscle relaxation techniques (Carlson & Hoyle, 1993) as well as

stretching (Thacker et al., 2004; Herbert & Gabriel, 2002; Andersen, 2005; Shrier, 2004) and isometric training techniques (Millar et al., 2007; Peters et al., 2006; Taylor et al., 2003). These methods have arguably been influenced by and have much in common with similar yogic exercises. Due to the similarities, research on these non-yogic techniques may help obtain better insight into the mechanisms underlying yogic practices and their potential benefits. Therefore, they are reviewed briefly within this context. The present study's yoga protocol has incorporated yogic physical, relaxation and meditation exercises and the protocol design process was based on traditional and contemporary yogic texts and the existing scientific evidence on the effect of various yogic practices as well as similar non-yogic techniques.

1.7.2 Use of yoga and complementary and alternative medicine (CAM)

The use of complementary and alternative medicine (CAM) including mind-body practice methods such as yoga is on the rise. The 2007 National Health Interview Survey (NHIS) published by the National Center for Complementary and Alternative Medicine (NCCAM) and the National Center for Health Statistics of the Centers for Disease Control and Prevention (CDC) reported 38.3% of US adults have used CAM in the past 12 months (compared to 36% in the 2002 survey) (Barnes et al., 2008). CAM use among adults was greater among women, people with higher levels of education and higher incomes. Deep breathing, meditation, yoga, progressive relaxation and guided imagery were among the ten most common CAM therapies used by adults and their use has increased from 2002 to 2007 as follows: Deep breathing exercises from 11.6% to 12.7%, meditation from 7.6% to 9.4%, massage therapy from 5% to 8.3%, and yoga from 5.1% to 6.1% (Barnes et al., 2008). Anxiety/depression and insomnia were among the ten conditions for which CAM was most frequently used (4.5% and 2.2% respectively) (Barnes et al., 2008). A 1998 US survey estimated 15.0 million adults

(7.5% of US adult population) had used yoga at least once in their lifetime and 7.4 million (3.8%) during the previous year. Yoga was used both for general wellness (64% of yoga practitioners) and for specific health conditions (48% of yoga practitioners) such as neck and back pain (21% of yoga practitioners). Factors independently associated with yoga usage were female gender, metropolitan area dwelling, education level above high school, use of other CAM modalities and age, with the age group of 34-53 reporting highest use. 90% of respondents thought yoga was very or somewhat helpful and 76% did not report spending money related to their yoga practice (Saper et al., 2004).

1.7.3 What is yoga?

In western cultural settings the word 'Yoga' is often associated with physical yoga exercises. However, the term 'Yoga' has a much wider context. Yoga is a Sanskrit word derived from the root "Yuj" that has various connotations including binding, joining, attaching, yoking, harnessing and focusing the mind. From ancient times the root "Yuj" has also been used in spiritual practice context, often to denote the control of the mind and the senses. It can also be translated as union, usually referring to union between man and the divine (Iyengar, 2001, p. 1; Feuerstein, 2000, p. 342).Yoga may encompass diverse psycho- physical practices associated with various cultures, spiritual traditions and body cultivation methods (Butera, 2006, p. 202). Yoga philosophy deals with physical, mental and spiritual wellness. It views the body and the mind holistically as one; whereby physical health depends on mental health and mental health depends on physical health (Butera, 2006, p. 202).Yoga instruction may include physical practices, breathing exercises, relaxation and meditation practices (Butera, 2006, p. 202), as well as internal and external hygiene and cleansing practices (Yogendra, 2003, chap. III - XII).

Yoga is one of six traditional systems of Indian philosophy (Iyengar, 2001, p. 1) and is thought to date back more than 4000 years (Feurstein, 1990, p. VII). Patanjali, the great Indian sage who according to tradition lived around 200 -500 BC, collated, systematised and codified yoga in his classical work the "*Yoga Sutra*" (translated from Sanskrit as yoga aphorisms or verses) (Feurstein, 1990, p. 3; Feurstein, 2000, p. 342; Iyengar, 2002, p. XVII). In the second verse of the of the "*Yoga Sutra*" Patanjali states that *"Yoga is the cessation of movement in the consciousness"* (*'Ýogah Citta Vrtti nirodah*' in Sanskrit) (Iyengar, 2002, p. 50; Feurstein, 1990, p. 26), or in other words, the goal of yoga according to Patanjali is the restraining of the fluctuations of the consciousness. In the 13th verse of the "*Yoga Sutra*" Patanjali states that *"Yoga practice is the steadfast effort to still these fluctuations"* (Iyengar, 2002, p. 63) and the goal is achieved through practice and dispassion which are the two poles of yoga practice (Feurstein, 1990, p. 34). Overall yoga provides the practitioner with various methods of understanding the functioning of the mind in order to gradually restrain its activity and achieve an undisturbed state of silence (Iyengar, 2002, p.62). Patanjali set out an eightfold path of yoga ('Ashtanga Yoga' in Sanskrit) for the practitioner to achieve this goal (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142). The eightfold path includes these eight components:

1. 'Yama' - Sanskrit term for moral injunctions, self restraint, abstention
2. 'Niyama' - Sanskrit term for fixed observances, values and precepts
3. 'Asana'- Sanskrit term for postures, poses, stable sitting positions
4. 'Pranayama' - translated as regulation of breath/energy
5. 'Pratyahara' - Sanskrit term for directing the senses inwardly
6. 'Dharana' – Sanskrit term for concentration, keeping the mind focused

7. ‘Dhyana’ - Sanskrit term for meditation, contemplation, reflection, awareness
8. Samadhaya - Sanskrit term for absorption of consciousness in the self, profound meditation, super consciousness (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142).

Several of these foundation yogic components have been incorporated into the yoga protocol of the present study (see section 3.3).

‘Hatha Yoga’ (Hathayoga in Sanskrit) is one of the major ancient yoga systems and the most prevalent form of yoga currently practiced in western cultural settings. Hatha yoga was introduced in India in the 15th century by the great yoga sage Svatmarama who consolidated his own experience and now lost ancient texts, in his key yoga text “*Hatha Yoga Pradipika*” (Svatmarama, 2002, p. IX). The Sanskrit word Hatha is a combination of ‘Ha’ (sun in Sanskrit) and ‘Tha’ (moon in Sanskrit). Some interpret this as alluding to the combination of solar and lunar energy, which are two main forms of energy described in yoga philosophy. These energies were believed to flow in the human mind-body and govern life. Hatha Yoga practice was believed to bring these two energies into balance (Maheshwarananda, 2009). In the 10th verse of “*Hatha Yoga Pradipika*” Svatmarama explains that hatha yoga is the foundation for any type of yoga practice, and also provides the means for curing the body from any pain and illness (Svatmarama, 2002, p. 4). In the opening verses of the “*Hatha Yoga Pradipika*”, Svatmarama states that hatha yoga is the first step leading to the heights of ‘Raja Yoga’ (Royal Yoga – in Sanskrit) (Svatmarama, 2002, p.1).

Traditional hatha yoga is subdivided into seven and sometimes eight limbs, which are similar, although not identical, to Patanjali’s eightfold path of yoga.. The great yoga

sages Patanjali, Svatmarama and Gheranda, author of the *"Gheranda Samhita"*, the most encyclopaedic of all root texts of hatha yoga (Gheranda, 2004, p. IX), are in agreement on the primary role of physical poses (Asana), breath regulation exercises (Pranayama), directing the senses inwardly (Pratyahara), meditation (Dhyana) and the ultimate goal of absorption of consciousness in the self or self realization (Samadhi) (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142; Svatmarama, 2002, p. X; Gheranda, 2004, pp. IX-XI). The first four of these components have become essential elements of contemporary yoga practice and will be discussed in more detail below to provide the background for the yoga protocol used in the present study (see section 3.3)

1.7.4 Asana

Asana practice is the third limb of the eightfold path of yoga as outlined in Patanjali's *"Yoga Sutra"* (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142; Feuerstein, 2000, p. 34). The Sanskrit word 'Asana' can be translated as sitting, a seat, or a sitting posture. In yoga practice asana originally referred to a posture or pose (Feuerstein, 2000, p. 34; Buhnemann, 2007, p. 17) used for prolonged meditation practice but was later developed within the framework of hatha yoga to refer to poses for building strength and suppleness and serving a range of therapeutic functions (Feuerstein, 2000, p. 34; Buhnemann, 2007, p. 20). Svatmarama explains that asana practice is the first step of hatha yoga as it builds physical strength, helps attain good health and assists in developing self control (Svatmarama, 2002, p. 8). Other traditional core yoga texts also view the asanas mainly as a foundation yogic practice intended to improve and maintain practitioners' well-being, flexibility, strength and vitality. Unlike some contemporary yoga schools classical texts do not view asana practice as an exclusive or primary yogic practice, but rather as a subordinate and preparatory practice intended to provide a good foundation for meditative practice by developing the ability

to remain in seated position for extended periods (Buhnemann, 2007, pp. 21- 22; Feuerstein, 2000, p. 34). Contemporary yoga master Iyengar further explains that in order to achieve body- mind integration, the body, which is the foundation for any practice, must be kept healthy (Iyengar, 2009, p. 204). Furthermore, because of the close connection between body and mind, they affect each other (Iyengar 2009, p. 228). Asana practice, therefore, affects not only the body but the mind as well (Iyengar 2009, p. 204).

The number of basic asanas mentioned in traditional texts varies greatly (Buhnemann, 2007, p. 25). Two seminal traditional hath yoga texts, the *"Hatha Yoga Pradipika"* and the *"Gheranda Samhitta",* mention the existence of 84 major poses (Svatwarama, 2002, p. 16; Gheranda, 2004, p. 16). The *"Gheranda Samhitta"* further explains that out of these, 32 are useful and essential for building strength (Gheranda, 2004, p. 16). Yoga asanas can be classified as standing poses, sitting poses, twists, forward bends, back bends, arm balances, core strength postures, inversions, and restorative poses (Yoga Journal, 2010; Raub, 2002). The design of the present study's asana protocol strived to incorporate poses from most of the above categories and adapt them, where necessary, to suit older adults (see section 3.3.9).

Each of the poses is claimed to provide specific physiological and mental benefits such as building strength, flexibility and mobility of spine and joints, improving postural alignment, improving balance, toning internal organs, improving circulation, digestion and elimination, preventing or alleviating specific health conditions and calming the mind (Iyengar, 2001, pp. 39-353; Buhnemann, 2007, p. 20). However, there is currently a lack of high quality evidence to substantiate claims made regarding the

physiological effects or health benefits of specific poses and this remains an important subject for future research.

1.7.5 Pranayama

Pranayama is the fourth limb of the eightfold path of yoga as outline by Patanjali (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142; Feuerstein, 2000, p. 225). 'Pranayama' is a Sanskrit word constructed of two separate words, 'Prana' and 'Ayama'. 'Prana' can be translated as breath, respiration, life, vitality and energy. 'Ayama' can be translated as restraint, control and regulation. Put together 'Pranayama' means regulation of breath and/or energy (Feuerstein, 2000, p. 225; Iyengar, 2010, pp. 13-14). Yoga texts describe subtle energy channels called 'nadi' (tube or pipe in Sanskrit), through which the prana flows. The practice of pranayama is claimed to clear 'blockages' from these channels and thus achieve better flow of vital energy and improved health (Iyengar, 2010, p. 15). Pranayama is considered by core ancient and contemporary yoga texts an important tool for improving health, preventing and curing diseases and also for facilitating concentration and calming the mind as an essential foundation practice for higher spiritual practice, as in pranayama practice the breath and the mind are closely interlinked (Svatmarama, 2002, pp. 33 - 35; Iyengar, 2010, p. 14; Feuerstein, 2000, p. 225; Iyengar, 2006, p. 32).

Several key ancient and contemporary yoga texts caution that incorrect or untimely pranayama practice may be harmful. In the *"Hatha Yoga Pradipika"* Svatmarama maintains that only after asana practice is consolidated, self control is attained, and moderate suitable dietary practices adopted, the practitioner may commence practice of pranayama. He adds that correct and timely pranayama practice can cure disease, but conversely, improper and untimely practice may cause disease

(Svatmarama, 2002, pp. 36-37). Contemporary Yoga master B.K.S. Iyengar, in his encyclopaedic manual *"Light on Pranayama"*, emphasises that pranayama practice should only be attempted once the yoga asana have been mastered and that there can be no short cuts in this respect (Iyengar, 2010, p. 53; Iyengar 2006, p. 33). In addition, Iyengar explains that before attempting pranayama practice, the practitioner needs to learn correct use of the intercostal muscles and diaphragmatic muscles via proper practice of the asana and warns that premature or improper practice of pranayama may severely harm the practitioner's health (Iyengar, 2010, p. 54).

Despite the above mentioned cautions, some contemporary schools of yoga incorporate substantial pranayama practice at the beginner's level and have demonstrated various health benefits with no reported adverse effects. One such yogic practice, is 'Sudarshan Kriya Yoga' (SKY) formalised by Sri Sri Ravi Shankar (Art of Living Foundation [ALF], 2010; SKY, 2010). SKY protocol incorporates a sequence of classic yogic breathing techniques and a special SKY breathing sequence (Brown & Gerbarg, 2005a; Brown & Gerbarg, 2005b). A review of studies on SKY intervention does not report adverse events (Brown & Gerbarg, 2005a; Brown & Gerbarg, 2005b). Another yogic system which incorporates breathing techniques at the beginner's level is Kundalini Yoga (KY). Several studies using KY intervention have not reported any adverse effects (Shannahoff-Khalsa, 2004; Khalsa, 2004b). It seems that more research is required to establish the safety, efficacy and correct application of other pranayama practices. The present study's protocol was designed for an elderly population with no previous yoga experience and therefore a conservative cautious approach was taken and pranayama practice was excluded. However, awareness of natural unregulated breath was used as one of the main tools in the meditative component of the protocol (see section 3.3.10).

1.7.6 Pratyahara

'Pratyahara' is a Sanskrit word translated as withdrawal of the senses or directing the senses inwardly. It is the fifth limb of the eightfold path of yoga as outlined by Patanjali in his *"Yoga Sutra"*. Patanjali does not provide many details how to practice and achieve pratyahara (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142). Pratyahara is the connecting link between external aspects and internal aspects of yoga as withdrawing the five senses (i.e. tactile, taste, smell, auditory and visual) inwards and away from external objects and bringing them under control paves the way to concentration practices (Iyengar, 2001, pp. 25 -27).

1.7.7 Dharana

'Dharana' is a Sanskrit word translated as concentration, practice with continuous single pointed focus of the mind, binding the consciousness (Feuerstein, 200, p. 85). It is the sixth limb of the eightfold path of yoga as outlined by Patanjali in is his *"Yoga Sutra"* (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142). Patanjali explains that in order to counteract the multitude of distractions, the practitioner should resort to the practice of concentration on a single principle (Feuerstein, 1990, p. 47). Dharana is accompanied by increased sensory inhibition and slowing down of the thought process which precedes entering into a full state of meditation (Dhyana) (Feuerstein, 2000, p. 85). In the present study the breath has been used as the main internal object of concentration (see section 3.3.10.3).

1.7.8 Dhyana

'Dhyana' is the seventh limb of the eightfold path of yoga as outlined by Patanjali is his *"Yoga Sutra"* (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp.140-142) and is a Sanskrit word translated as meditation, contemplation or meditative state (Feuerstein,

2000, p. 88). Dhyana is the most common term both for the meditative state of consciousness and the yogic techniques by which to reach it (Feuerstein, 2006 and is a central advanced technique common to all yogic paths (Feuerstein, 2000, p. 88) as it constitutes a deepening of the preceding process of concentration, or one-pointed-mindedness (Dharana) (Feuerstein, 2006). Indeed, as outlined by Patanjali in his *"Yoga Sutra"*, all limbs of yoga are part of a general effort to restructure the practitioner's consciousness and all lead in the same direction. However, in Dhyana the inner restructuring of the mind is greatly enhanced, as it is considered the foundation for the ecstatic breakthrough to a state of 'Samadhi' (Feuerstein, 2006). According to Iyengar (2009, p. 160) Patanjali provides a variety of practice methods based on his understanding that meditation is not suitable or possible for all levels of practitioners. In the present study, the practice of Yoga Nidra was chosen as the main meditative practice. One of the considerations behind this choice was the fact that it incorporates a variety of practice tools to accommodate individual difference and practice levels (see section 3.3.10.5).

1.7.9 Samadhi

'Samadhi' is a Sanskrit word translated as absorption of consciousness in the self, profound meditation or super consciousness. It is the eighth limb of the eightfold path of yoga as outlined by Patanjali is his *"Yoga Sutra"* (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp. 140-142; Feuerstein, 2006). Ultimately, the traditional spiritual goal of meditation is 'Samadhi', the final ecstatic merging with the object of meditation, and breakthrough towards higher state of consciousness. Furthermore, the ultimate goal of 'Samadhi' is achieving self-realization (Feuerstein, 2006).

1.7.10 Objects of meditation in yogic meditation techniques

Yoga practitioners consider that anything can serve as an object of meditation, from the most gross to the most subtle, from the most mundane to the divine including things that are internal or external, concrete or abstract, real or imagined (Feuerstein, 2006). Patanjali, in the *"Yoga Sutra"* leaves the choice of meditation object open and accepts a wide range of meditation objects for focusing consciousness including body parts, planets and stars, various abstract concepts, feelings and processes (Feuerstein, 2006).

The *"Hatha Yoga Pradipika"* recommends meditating upon the inner sound ('Nada' in Sanskrit) (Feuerstein, 2006). The practice of 'Mantra-yoga' can be found in many spiritual traditions and incorporates regular and prolonged recitation ('japa' in Sanskrit) of a specific 'mantra'. Mantras are considered to be sacred sounds, syllables, words or word combinations with the best known example being the sacred syllable 'Om', which symbolizes the absolute and is often used in conjunction with other sounds or combination of sounds (Feuerstein, 2006).

In 'Guru-yoga', the object of meditation is a fully enlightened teacher ('sad - Guru' in Sanskrit). The disciple, who constantly keeps the Guru in his mind in thought, worship, and meditation, aims to align himself with the teacher and duplicate within himself the teacher's illumined state (Feuerstein, 2006).

The *"Gheranda-Samhita"*, distinguishes between sthula ("coarse"), jyotir- ("luminous"), and sukshma-dhyana ("subtle meditation"). In Sthula-dhyana the practitioner visualises a particular form, such as a guru or a deity, in great detail. In Jyotir-dhyana the practitioner contemplates on the Divine and visualizes it as a mass of light in specific energy centres of the body ('chakra' in Sanskrit). In Sukshma-dhyana

the practitioner meditates upon the awakened 'Kundalini' energy - an unconscious, instinctive or libidinal force claimed to be lying dormant coiled at the base of the spine (Feuerstein, 2006) and is a meditation object favoured by many Hatha-yoga and Tantric schools (Feuerstein, 2006).

The yoga nidra practice incorporated in the present study provides a range of meditation objects, including various parts of the body, the natural breath, opposing physical sensations and various visualised images (see sections 1.7.12 and 3.3.10.5).

1.7.11 Meditation and related practices - modern versus traditional perspectives

The English term meditation was derived from the Indo-European root 'med' whose main meaning was that of 'measuring'. Its Latin equivalent 'meditor' originally meant 'exercise' in general but then restricted to mental or spiritual exercise (Bader, 1990, pp. 25-26). The current common usage of the term meditation is somewhat broader than that represented by term 'Dhyana' in Patanjali's "*Yoga Sutra"* or in the "*Gheranda Samhita"*. The "*oxford online dictionary"* defines the verb to 'meditate' as: to "*focus one's mind for a period of time, in silence or with the aid of chanting, for religious or spiritual purposes or as a method of relaxation"* (Oxford Online dictionary, 2010). In a comprehensive analysis of recent meditation research Shapiro et al. (2003) define meditation as: *"A family of practices that train attention and awareness, usually with the aim of fostering psychological and spiritual well being and maturity"*. According to Shapiro et al. (2003) meditation achieves this aim by training the mind and increasing the amount of voluntary control over mental processes in such a way that they can be directed in beneficial ways, including the development of qualities of concentration, calm, joy, love, compassion, and heightened awareness. Heightened

awareness, in turn, results in better self knowledge and understanding of how one relates to the world. It also leads to a better knowledge of consciousness and reality. According to Shapiro et al. (2003) meditation methods are commonly classified as concentration type meditation or awareness type meditation, where concentration type practices are designed to focus awareness on a single object such as the breath or an internal sound (mantra in Sanskrit), and awareness type practices allow attention to be directed at a wider variety of objects Shapiro et al. (2003). It seems therefore, that the current use of the term meditation may include elements of the fifth, sixth and seventh limbs of Patanjali's eightfold path of yoga, namely, Pratyahara (withdrawal of the senses), Dharana (concentration) and Dhyana (meditation), as well as relaxation practices. In traditional yoga texts, on the other hand, meditation is considered an advanced stage which requires adequate preparation. Pratyahara leads to Dharana which leads to Dhyana which eventually may lead to Samadhi (Iyengar, 2002, pp. 31-32; Iyengar, 2002, pp. 140-142; Feuerstein, 2006). The Pratyahara, Dharana and Dhyana elements as well as a relaxation element are incorporated in the meditation techniques used in the present study (see section 3.3.10)

1.7.12 Yoga Nidra

'Yoga Nidra' (translated from Sanskrit as yogic sleep), is a form of yogic guided meditation technique. Yoga nidra originated in India from the ancient teachings of yoga and 'Tantra'- a philosophical system incorporating a vast array of spiritual practices and ritual forms aiming at attaining spiritual liberation (Miller, 2005a, pp. 3-4; Satyananda, 1976, pp. 1-3). Several yoga masters have revitalised the practice of yoga nidra during the 20th century, mainly Swami Sivananda and his disciples including Satyananda Swaraswati of the Bihar school of yoga, Swami Satchyananda of Integral Yoga, and swami Vishnudevananda of the Sivananda Yoga Vedanta centre; but also Swami Rama

of the Himalayan Institute and his direct disciple, Swami Veda Bharati of the Swami Rama Sadhaka Grama and Sri Brahamananda Swaraswati(Rammurti S. Mishra), an initiate of the Radhaswami school of Surrat Shabd Yoga and others (Miller, 2005a, pp. 3-4).

'Yoga Nidra' is usually performed lying supine and motionless in 'Savasana' (translated from Sanskrit as 'dead corpse pose') and mentally following a series of instructions read out by a yoga teacher (Satyananda, 1976, p. 69) (in person, or using an audio CD for home- practice. Both methods have been applied in the present study - see 3.3.10.2 -3.3.10.5). A single yoga nidra sequence may incorporate several yogic techniques including relaxation, withdrawal of the senses, concentration, and awareness/mindfulness, autosuggestion, and visualisation. Furthermore, it uses a range of meditation objects including the natural breath, various parts of the body, opposing physical sensations (e.g. hot and cold, heavy and light etc.) and images generated by inner visualisation (Satyananda, 1976, pp. 70-73). This allows tailoring a range of meditation protocols with varying duration and meditative emphasis to suit beginner to advanced students with different needs and abilities. In his book and accompanying CD *"Yoga Nidra: The Meditative Heart of Yoga"*, Miller has added psychological components (Miller, 2005, pp. 79-82) designed to help in dealing with negative feeling and emotions (Miller, 2005b, track 3), negative thought and belief patterns and negative self perceptions (Miller, 2005b, track 4).

Yoga nidra practice is claimed to provide many benefits including somatic and mental relaxation, improved memory, reduction of stress, anxiety, fear, anger, depression and insomnia, and also improved learning ability and memory (Satyananda, 1976, p. 2). Satyananda explains that in yoga Nidra, the practitioner enters a state which

constitutes some aspects of Patanjali's yogic elements of 'Pratyahara' (Satyananda, 1976, p. 2), 'Dharana' (Satyananda, 1976, p. 72), and in advanced levels of practice also 'Dhyana' (Satyananda, 1976, p. 72). According to Miller (2010, p.38) each of the components of yoga nidra, directs the practitioner's awareness to a different 'sheath' ('Kosha' in Sanskrit – see glossary) or yogic aspect of the mind-body phenomenon. Miller lists the following sheaths/layers/levels, and respective yoga nidra practice elements: On the physical level - awareness of physical sensations. On the energy level - awareness of breath and energy flow. On the emotional level - awareness of feelings and emotions. On the intellectual level - awareness of thoughts, beliefs and images. On the sensual level - awareness of joy, pleasure and desire. On the ego level - awareness of the witness or ego. On the natural blissful level - awareness of the changeless body. According to Miller, a yoga nidra session may focus on a single aspect, several aspects or all of the above aspects of the mind-body (Miller, 2010, p.38). Indeed, yoga nidra allows tailoring various protocols varying in duration and number of meditative components (Satyananda, 1976, pp. 81-150). Arguably, by using an external 'directive' in the form of the teacher's voice, the practitioner is given a chance to let go further and reach a deeper level of relaxation. All these aspects of yoga nidra make it a multifaceted and versatile meditation tool that is easy to learn and follow.

There are various variants of yoga nidra sequences (Satyananda, 1976, pp. 81-150; Miller, 2005b) and the yoga nidra protocol used in the present study was based on several Yoga Nidra protocols as taught by Swami Satyananda Swaraswati of the Bihar school of yoga (Satyananda, 1976, pp. 81-150). A typical protocol may include the following components in this order:

1. **Preparation**: the practitioner is instructed to lie down comfortably on a mat in the supine position, feet apart and palms facing upwards. If necessary, the body is covered with a blanket to keep warm and once comfortable, the practitioner is instructed to stay still until the end of the practice (if the supine position is not comfortable other horizontal or sitting positions are permissible). The practitioner is then instructed to focus on external sounds and direct his/her attention from sound to sound ('Antar Mouna' in Sanskrit) (Satyananda, 1976, pp. 69).
2. **Relaxation :** The practitioner is instructed to scan the entire body and feel if there is any tension in the muscles and then feel the tension dissolving away while relaxing the whole body (part by part or all at once) (Satyananda, 1976, pp. 142-143).This component resembles some western muscle relaxation techniques described earlier (see section 1.5.12.3.9).
3. **Resolve** ('Sankalpa' in Sanskrit): **T**he practitioner is instructed to make a wish for any positive change in his/her life, health, relationships etc. and this is repeated three times (Satyananda, 1976, p.70). This element of Yoga Nidra resembles 'autosuggestion' techniques as introduced by Emil Coue in the early 20th century (Coue, 2006, pp. 21-28), although in a more concise format.
4. **Rotation of consciousness:** The names of various body parts are called out by the teacher, sequentially in a rapid succession, from top to bottom and from right to left. The practitioner is instructed to direct his/her awareness to the body part called out and to mentally repeat the name of the body part (Satyananda, 1976, p.70) and focus on any sensation there (Miller, 2005b, track 1). In yoga nidra, the practitioner is passive and the rate and direction of the body scan is dictated by the teacher or the recording. Arguably, this allows the yoga nidra practitioner to let go further and achieve a deeper level of relaxation.

5. **Awareness of the contact between body and floor**. The practitioner is instructed to be aware of the contact points between body and floor, whole body at once or part by part (Satyananda, 1976, p.132; Satyananda, 1976, p. 148).
6. **Awareness of subtle body movements –** The practitioner is instructed to be aware of muscle twitches or subtle movements in the body in conjunction with the breath (Satyananda, 1976, p. 143).
7. **Breath awareness:** The practitioner is instructed to direct the awareness to the natural (uncontrolled) breath as it manifests in the movement of the navel, chest, throat, nostrils eyebrow centre (Satyananda, 1976, p.132). Usually this practice is done while mentally counting the breaths. For example, the practitioner maintains focus and counts down from a certain number to zero (or vice versa) (Satyananda, 1976, p. 71).
8. **Awareness of opposing sensations:** The practitioner is instructed to sense opposite sensations in the body, such as heat and then cold, lightness and then heaviness, painful and then pleasant sensation etc. (Satyananda, 1976, p. 72).
9. **Focusing on the 'inner space' (Chidakasha – in Sanskrit):** The practitioner is instructed to focus on the inner space between the eyebrows (with the eyes closed) and observe whatever appears there such as colours, patterns etc. (Satyananda, 1976, p. 132).
10. **Visualisations:** The practitioner is instructed to visualise a rapid sequence of objects such as natural scenery, building, flowers, people, etc. The practitioner tries to visualise them as vividly as possible as they are called out by the teacher (Satyananda, 1976, p. 72; Satyananda, 1976, p. 132).
11. **Repeating the Resolve:** The practitioner is instructed to repeat the initial resolve **(**a wish for any positive change in his/her life, health, relationship etc.) three times

(Satyananda, 1976, pp. 72-73). At this point in the meditation the practitioner is immersed deeper in the meditation, the mind is calmer and arguably the level of suggestibility to the resolve statement is increased.

12. **Movement in time:** The practitioner is instructed to mentally review/visualise the events which have occurred during the day (Satyananda, 1976, p. 72; Satyananda, 1976, p. 132).
13. **Completion:** The practitioner is instructed to gradually become aware of his/her body, the room and its surroundings and thereby finish the practice. (Satyananda, 1976).

In summary, it seems that yoga nidra represents a broad range of meditative yogic techniques and also fits Shapiro et al.'s (2003) contemporary definition of meditation. Yoga nidra's flexibility and range of meditative tools allow creating protocols of varying length and emphasis to suit beginner to advanced students. Yoga Nidra's well structured guided meditation format makes it accessible and applicable using audio CDs and MP3 players and thus advantageous for home-based self-practice.

1.7.13 Research on the effects and benefits of yoga practice

1.7.13.1 Introduction

There is a growing body of evidence on the effects and benefits of yoga practice; however, studies vary greatly in quality, methodology, yoga protocols, and populations. Most studies on yoga intervention employ composite protocols that incorporate several yogic practice elements (e.g. asana and relaxation or pranayama and meditation). A small number of studies examine the effects of a single yogic practice element on its own (i.e. asana, pranayama, relaxation or yogic meditation on their own). This section will include a general review on the effects of yoga interventions followed by a short

review on the effects of specific yogic practice components (i.e. asana, pranayama, relaxation or meditation). The section on the effects of yogic meditation techniques will also include a brief review of the effects of similar non-yogic meditation techniques that are aligned with yogic meditation techniques.

1.7.13.2 Research on effects of composite yoga practices

1.7.13.2.1 Introduction

Most published studies to date have investigated the effect of composite yoga intervention protocols that incorporate at least two yogic practice components. Often the relative proportion of each of the components is not clearly stated and/or the protocol is described only in very general terms. Identifying the effects of specific components of yoga (i.e. yoga poses, breathing, relaxation, meditation, chanting etc.) within a protocol and comparing protocols with different mixes of yogic practice elements needs to be addressed in future research.

1.7.13.2.2 Yoga's effect on sleep quality

A small number of studies have shown that yoga may improve a range of sleep quality measures with most studies investigating the effects on normal sleepers and not those suffering from a sleep disturbance. Most studies to date have only used subjective sleep quality measures and at the inception of present study (early 2007) only one published study investigated yoga as an intervention for improving sleep quality in an elderly population (Manjunath & Telles, 2005).

This study involved a six months trial conducted in Bangalore India, in collaboration with the Vivekanada Yoga research foundation. The study, which recruited healthy subjects from a single aged care facility and did not exclude normal sleepers or use objective sleep measures, compared self rated sleep quality outcomes of

normal subjects randomised into three groups: yoga intervention, traditional Indian medicine ('Ayurveda') and control. The study population consisted of 69 out of 120 residents of a single aged care facility and the inclusion criteria did not require a diagnosed or self reported sleep disturbance. A very extensive yoga intervention protocol, that is unlikely to be suitable for the lifestyle of elderly people in western cultural settings, was used. This protocol included two daily guided yoga sessions six days a week. The morning session protocol included 60 minutes of regulated breathing exercises ('Pranayama'), physical warm-up exercise ('Shilikarna Vayama'), physical yoga poses ('Asana'), and guided relaxation. The evening session protocol included devotional songs ('Bhajan'), lectures on the theory and philosophy of yoga and a practice of 'Cyclic Meditation' (CM), which incorporates cycles of physical yoga poses followed by supine rest. Results revealed that the yoga group, but not the control group, showed significant improvement in various subjective sleep quality measures including sleep latency, total sleep time and feeling refreshed in the morning (Manjunath & Telles, 2005).

Shannahoff-Khalsa describes several yogic meditation and breathing techniques with a specific effect on insomnia and other sleep disorders (Shannahoff-Khalsa, 2006, pp. 164-179; Shannahoff-Khalsa, 2004). Some of these techniques were applied in a study (n =20) of Kundalini Yoga (KY) as an intervention for primary and secondary insomnia. This study used an intervention which included breathing exercises (some in conjunction with inward silent mantra recitation) and a breath awareness meditation and incorporated an initial guided session followed by home-based self-practice sessions for eight weeks. The study results, derived from self-reported sleep-wake diary, revealed a significant pre- to post-intervention improvement in Sleep Efficiency (SE), Total Sleep Time (TST), Total Wake Time (TWT), Sleep Onset Latency (SOL), Wake time After

Sleep Onset (WASO), number of awakenings, and other sleep quality measures. (Khalsa, 2004b).

A study of Tibetan Yoga (TY) for supporting cancer patients included a weekly session of meditation, visualization and gentle postures over a period of seven weeks with 98% of the TY intervention group completing 2-3 sessions and 58% at least five sessions. The results showed significant improvement in various subjective sleep quality measures but no significant difference in various mental health measures in the intervention compared to the control group (Cohen et al., 2004).

Three studies have been conducted in Taiwan to measure the effect of a six months intervention of hatha yoga adapted for the needs of the elderly ("Silver Yoga"). Subjects included community dwelling elderly (two studies) and elderly living in assisted living facilities (one study). The intervention included gentle yoga postures, relaxation exercises and guided imagery. Results revealed a significant improvement in various subjective mental health and sleep quality measures (Chen et al., 2008; Chen et al., 2009; Chen et al., 2010a)

The association between autonomic hyper arousal and insomnia has been discussed above (see section 1.5.8). Three short studies have shown yoga practice may affect autonomic modulation and also improve sleep quality pointing to a possible mechanism by which yoga practice may contribute to improved sleep quality. These studies, which compared the immediate effects of Cyclic Meditation (CM) and relaxation in the yogic shavasana ('corpse') pose (SR) in normal subjects found a shift in sympatho-vagal balance in favour of parasympathetic dominance in the CM group but not the SR group. A significant decrease in oxygen consumption and breath rate and an increase in breath volume were seen in both groups but, the magnitude of change on

all three measures was greater following a CM session. Furthermore, the proportion of slow-wave sleep (SWS) increased significantly, whereas proportion of rapid-eye-movement (REM) sleep and the number of awakenings decreased in the night that followed practice in the CM group compared to the SR group. The CM group also showed an improvement in a range of self reported subjective sleep quality measures including feeling more refreshed in the morning, increase in sleep duration, and decrease sleep disruptions. (Patra, & Telles, 2010; Patra, & Telles, 2009; Telles et al., 2000).

1.7.13.2.3 Yoga's effect on stress, anxiety and depression

Stress, anxiety and depression may have a significant negative impact on quality of life. The association between stress, anxiety and depression and between insomnia was discussed above (see section 1.5.7). Several studies have shown yoga practice may alleviate stress, anxiety and depression pointing to a possible mechanism by which yoga practice may improve sleep quality in individuals suffering from stress, anxiety or depression and comorbid sleep disturbance. However additional high quality research is required.

A recent critical review of the effect of yoga on depression found eight studies with poor methodology but encouraging results and concluded that additional research is required (Uebelacker et al., 2010). Another systematic review of the effect of yoga on depression implied that yoga interventions may have some positive effects on depressive disorders but additional research is required due to the heterogeneity of intervention protocols and poor methodological quality (Pilkington et al., 2005). A systematic review of the effectiveness of yoga for the treatment of anxiety and anxiety disorders, found eight poor quality studies reporting positive results, and recommends

further better quality research focused on specific anxiety disorders. (Kirkwood et al., 2005).

A study of an Iyengar yoga intervention for stress reduction in 24 self referred females found that two weekly classes over a period of three months led to significant improvements on self reported measures of stress, psychological and physical well being outcomes. Salivary cortisol hormone was found to decrease significantly after participating in a yoga class and those complaining of back pain or headache prior to the intervention reported marked improvement in pain levels (Michalsen et al., 2005).

A study (n=57) comparing the immediate effects of a 22.5 minutes session of CM to a similar session of relaxation in shavasana (SR) found that both techniques improved memory scores immediately after and decreased anxiety with the practice of CM having a greater effect than SR (Subramanya & Telles, 2009).

A 12 months study (n=21) of yoga as an intervention for obsessive compulsive disorder (OCD) compared 'Relaxation Response' plus 'Mindfulness Meditation' to Kundalini Yoga (KY) which included breath synchronized Sanskrit mantra chanting, shoulder shrugs, gentle flexing and relaxing movements of the spine in the sitting position, focusing on the "third eye" (point between eyebrows), and regulated four part, very slow breath through the left nostril only. Results showed that the KY group had greater improvement on several related psychological measures. (Shannahoff-Khalsa, 2004)

A single study suggests that stimulation of Gamma-amino butyric Acid **(**GABA) production is a possible mediating mechanism by which yoga improves mood and anxiety. The study compared Iyengar yoga intervention to walking intervention for 60 minutes three times a week for 12 weeks in metabolically matched subjects and found

greater improvements in mood and anxiety measures and a nearly significant (p=.09) increase in acute thalamic GABA levels in the yoga group compared to the walking group with a positive correlation between improved mood and decreased anxiety and thalamic GABA levels. The researchers suggest a possible role of GABA in mediating the beneficial effects of yoga on mood and anxiety as pharmacologic drugs prescribed to improve mood and decrease anxiety also stimulate GABA production (Streeter et al., 2010).

1.7.13.2.4 Yoga's effect on pain and musculoskeletal health

Pain and physical discomfort may have a detrimental impact on sleep by causing increased sensory stimulation that may result in higher somatic arousal (see section 1.5.7 -1.5.8). Yoga practices have been found to improve musculoskeletal conditions and alleviate a range of musculoskeletal complaints and reduce pain and tenderness (Raub, 2002; Sherman et al., 2005; Garfinkel et al., 1994; Kolasinksi et al., 2005) and these findings, reported below, point to a possible mechanism by which yoga practices may improve sleep quality in individuals suffering from musculoskeletal pain.

A review of the effect of yoga practice on musculoskeletal status identified ten relevant studies and reported improvement in isokinetic muscle strength and endurance, general flexibility, and body composition (Raub, 2002).

A study of 101 adults with chronic low back pain compared three interventions: guided yoga classes, versus conventional therapeutic exercise classes versus home-work using a self-care book. Back-related function in the yoga group was significantly superior to the book and exercise groups at 12 weeks with the benefits persisting for several months as least (Sherman et al., 2005).

An eight week, one-class per-week, study of a yoga intervention for osteoarthritis of the hands found significant improvements in pain during activity, tenderness and finger range of motion in the intervention group compared to the control group (Garfinkel et al., 1994). A similar study of an Iyengar yoga intervention for osteoarthritis of the knee revealed significant improvements in pain level and physical function (Kolasinksi et al., 2005).

Yoga may also improve non-musculoskeletal physical pain. A three months study (n=72) of yoga intervention versus self care intervention for migraine headaches (without aura) found significant improvement in the yoga group compared to self care group in various measures related to headache intensity, pain rating index, affective pain rating index, total pain rating index, anxiety and depression scores, and use of symptomatic medication (John et al., 2007).

1.7.13.2.5 Yoga and common risk factors for chronic diseases

Yoga has been shown to reduce a range of common risk factors for chronic disease, including blood pressure (BP), elevated levels of cholesterol, triglycerides, oxidative stress, glucose and coagulation factors (Yang, 2007; Innes et al., 2005; Innes & Vincent, 2007).

A systematic review of yoga for common risk factors for chronic diseases found 32 relevant studies (between1980 and 2007). Reviewers report that overall yoga interventions appear to be effective in reducing body weight, blood pressure, glucose level and high cholesterol but that not enough data is available regarding long-term adherence and long term effects (Yang, 2007).

A systematic review by Innes et al. on yoga's effect on cardiovascular disease (CVD) and insulin resistance disorder (IRD) concludes that yoga may reduce various

IRD-related risk factors for CVD including glucose tolerance, insulin sensitivity, lipid profiles, anthropometric characteristics, blood pressure, oxidative stress, coagulation profiles, sympathetic activation, and cardio vagal function and suggests that yoga may improve clinical outcomes, and aid in the management of CVD and other IRD-related conditions. However, the poor quality of most studies prevented coming to more decisive conclusions (Innes et al., 2005).

A systematic review on the influence of yoga-based programs on risk profiles in adults with type 2 diabetes mellitus (DM), found 25 relevant studies that overall suggest beneficial changes in several DM risk indices, including glucose tolerance and insulin sensitivity, lipid profiles, anthropometric characteristics, blood pressure, oxidative stress, coagulation profiles, sympathetic activation and pulmonary function, as well as improvement in specific clinical outcomes (Innes & Vincent, 2007).

1.7.13.2.6 Yoga's effect on cardiopulmonary function

A review of yoga's effect on cardiopulmonary function identified 20 relevant studies. This review suggests that yoga interventions result in significant improvements in a range of parameters including lung function, cardiovascular endurance, improvement in work rate and reduction in oxygen consumption per work unit and improvement in exercise performance. Several of the reviewed studies also suggest that yoga practice may cause significant improvement in symptoms of chronic asthma and bronchitis (Raub, 2002).

1.7.13.3 Specific effects of asana practice

In general, the focus in asana practice is more on isometric strength training and stretching and less on aerobic cardiovascular training (Khalsa, 2004a) (see section 1.7.4). However, there is a paucity of good quality evidence on the specific effects and

underlying mechanisms of specific asana practices. Significant research has been conducted on effects and benefits of non-yogic stretching (Thacker et al., 2004; Herbert & Gabriel, 2002; Andersen, 2005; Shrier, 2004) and some research has also been done on the effects of cycles of isometric effort and rest periods (Millar et al., 2007; Taylor et al., 2003; Peters et al., 2006). Arguably, due to the similarities between the non-yogic application of these exercise elements and its application in yogic asana practice, the results of these studies may help shed some light on possible underlying mechanisms of yogic asana practice

There are currently few studies on the specific effects of the stretching component of yogic asana practice and few studies comparing the effects of yogic versus non-yogic stretching techniques. Furthermore, because yogic poses usually combine stretching and isometric effort (Khalsa, 2004a), both of these factors have to be taken into account in design of future studies on the effect of asana practice. Nevertheless, a number of studies have been conducted on the efficacy of stretching for improving sports performance and prevention of sports related injuries (Thacker et al., 2004; Herbert & Gabriel, 2002; Andersen, 2005; Shrier, 2004). Due to the similarities between the non-yogic application of these exercise elements and its application in yogic asana practice, the results of these studies may be relevant to yogic asana practice. Overall, it seems that existing evidence indicates that pre- or post-exercise session stretching does not significantly decrease the likelihood of injury and it may only result in very small reductions in post-exercise muscle soreness or recovery time, although stretching may improve some performance parameters (Thacker et al., 2004; Herbert & Gabriel, 2002; Andersen, 2005; Shrier, 2004). On the other hand, cycles of isometric effort and rest periods have been shown to provide a range of significant health benefits including

reduction in blood pressure, reduction in oxidative stress and increased vagal modulation (Millar et al., 2007; Taylor et al., 2003; Peters et al., 2006).

A comprehensive systematic review of 361 studies concluded that there is insufficient evidence to either support or oppose routine stretching before or after exercise to prevent injury in competitive or recreational sports (Thacker et al., 2004). Another systematic review concluded that stretching pre- or post-exercise session does not give protection from muscle soreness and stretching before exercising does not seem to reduce the risk of injury, and that findings are insufficient to determine the effects of stretching on sports performance (Herbert & Gabriel, 2002). A third systematic review reported that on average, stretching results in a reduction in subjective soreness of less than 2% during the 72 hours after exercise and that stretching provides injury risk reduction of only 5%. Reviewers concluded that the stretching protocols used in the reviewed studies do not meaningfully reduce lower extremity injury risk in study population (Andersen, 2005). Another systematic and critical review on the effect of stretching on athletic performance concluded that regular stretching improves force, jump height, and speed; however there is no evidence that it also improves running economy. Furthermore, an acute bout of stretching was not seen to improve force or jump height (Shrier, 2004).

Several studies have been conducted on the effects of isometric hand grip training (IHG) on various physiological parameters in hypertension patients. Intervention included IHG practice three days/week for eight weeks with each session consisting of several rounds of continuous isometric hand grip exercise at 30% of maximal voluntary contraction (MVC) for 2 minutes, separated by similar rest periods. Analysis of results amalgamated from three studies revealed significant decrease in both systolic and

diastolic blood pressure (BP) over time (5.7 and 3 mmHg reductions, respectively) with those participants with higher initial systolic pressure showing greater rates of BP decline (Millar et al., 2007). Other studies reveal similar results along with increased vagal modulation as demonstrated by a decrease in low frequency to high frequency area ratio of the power spectral analysis of heart rate variability (HRV) (Taylor et al., 2003) and significant reduction in markers of oxidative stress demonstrated by a significant (266%) decrease in exercise-induced oxygen centred radicals and a 61% increase in the ratio between resting whole blood glutathione to oxidized glutathione (Peters et al., 2006).

Interestingly, rounds of isometric effort followed by rest periods are commonly applied in several forms of yoga practice. One such example is Bikram Yoga in which each of the poses in the mat-based part of the basic protocol is followed by a complete relaxation in the 'shavasana' (corpse pose) (Bikram, 2000, pp. 110-188). Another example is the yogic practice of Cyclic Meditation (CM which incorporates cycles of static yoga postures followed by relaxation period of several minutes (Telles et al., 2000). Studies on the effects of CM are reported in detail above (see section 1.7.13.2.2). Both studies on the effects of CM and the effects of IHG revealed a shift in sympatho-vagal balance in favour of parasympathetic dominance, yet further research is required to determine whether the principle of alternating isometric effort and complete rest periods is involved in the effects of CM and other yogic practices. This can be done by comparing CM practice to similar but continuous asana practices, which do not include resting periods. Additional research can then be conducted to determine the ideal durations and ratios of rest periods to asana periods.

In summary, additional high quality research is required in order to identify effects and benefits of asana practice. It seems that the reported benefits of asana practice cannot be attributed mainly to the stretching element involved. Isometric effort or a combination of isometric effort and rest periods may possibly play an important role in the overall effect of asana practice. Additional research is required to specifically isolate and examine various factors of asana practice including stretching, isometric effort, combinations of effort and rest periods, as well as other bio-mechanical aspects of asana practice. Future research may measure and compare the effects of specific asanas, asana combinations and various asana practice protocols. Furthermore, future research may examine if and how asana practice differs substantially from non-yogic stretching and isometric training practices.

1.7.13.4 Specific effects of pranayama practice

A relatively small number of studies have been published on the effect of pranayama breathing techniques performed on their own (i.e. not as part of a protocol which also includes yogic poses and/or meditation exercises). These studies, that are reported in detail below, have shown that pranayama exercises as well as device guided breathing which emulate some pranayama practices may bring various physical and mental health benefits including reduced anxiety, stress and depression, improved oxygen utilisation, oxidative status and immune system markers, reduced blood pressure and heart rate and modulation of sympathetic nervous system towards parasympathetic dominance. (Brown & Gerbarg ,2005a; Brown & Gerbarg, 2005b; Kjellgrenet al., 2007; Janakiramaiah et al., 2000; Sharma et al., 2008; Raju et al., 1994; Pramanik et al., 2009; Jain et al., 2005; Shannahoff-Khalsa, 1993; Grossman & Grossman, 2003; Grossman et al., 2001; Rosenthal et al., 2001; Schein et al., 2001; Meles et al., 2004). Currently, there is not enough evidence to enable any differentiation between the effects of various

pranayama practices. There is also a lack of evidence on the underlying physiological mechanisms of pranayama practice. Furthermore, although no adverse effects related to pranayama practice have been reported, more research is required regarding the safety of various types of pranayama practice at the beginner's level (see section 1.7.5).

A growing body of evidence exists on the effects of a contemporary yogic breathing method named Sudarshan Kriya Yoga (SKY) that incorporates a sequence of specific yogic breathing techniques, including 'ujai pranayama', 'bastrika pranayama' (see glossary), and a special SKY breathing sequence (Brown & Gerbarg ,2005a; Brown & Gerbarg, 2005b). A review on SKY research concluded that it has been shown to alleviate anxiety, depression, everyday stress, post-traumatic stress, and stress-related medical illnesses with the reviewers concluding that there is sufficient evidence to consider SKY as a beneficial, low-risk, low-cost addition to current treatment strategies for stress, anxiety, post-traumatic stress disorder (PTSD), depression, stress-related medical illnesses, substance abuse, and rehabilitation of criminal offenders. (Brown & Gerbarg, 2005a; Brown & Gerbarg, 2005b).

A six week controlled trial (n=103) examining the effect of 30 minutes of SKY practice daily versus an active control that included relaxing in an arm chair for the same period of time found reduced anxiety, depression and stress, and increased optimism in the SKY but not the control group as revealed by self reported questionnaires. Furthermore, the dropout rate was low and no adverse events were reported implying this intervention may be reasonably safe (Kjellgrenet al., 2007). A three week study (n=45,) compared SKY to electroconvulsive therapy (ECT) and Imipramine (IMN) treatment for melancholic depression and found significant improvement in all three groups although SKY and IMN were inferior to ECT

(Janakiramaiah et al., 2000). A one year pilot study (n=84), which compared SKY practice to non practicing controls found improved antioxidant status both at the enzyme activity level and RNA level in the SKY group which also had improved stress regulation and better immune status manifested by prolonged lymphocytes life span. These finding imply SKY practice may have a positive effect on immunity, aging, cell death, and stress regulation through transcriptional regulation (Sharma et al., 2008).

A two year study on the effect of pranayama on athletic performance showed that pranayama practice helps athletes achieve higher work rates with reduced oxygen consumption and without an increase in blood lactate levels along with significantly reduced resting blood lactate levels (Raju et al., 1994).

A relatively small number of studies have examined the effects of specific pranayama techniques. Most have shown breathing exercises may modulate breath rate, heart rate and other cardiovascular parameters. A small study (n = 39, age = 25-40 years) has shown that a single slow pace (respiratory rate of six breaths per minute) Bhastrika pranayama (see glossary) technique for five minutes reduces systolic and diastolic blood pressure significantly and heart rate slightly. (Pramanik et al., 2009). A small (n=20) eight week study on the effect of left and right single nostril breathing has also shown both acute and chronic decreases in respiratory rate (RR), heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBF) (Jain et al., 2005). Three similar earlier studies of forced unilateral nostril breathing (UFNS) (at rates of six breaths per minute or rates of 2-3 breaths per second) all showed that breathing via the right nostril increased HR compared to breathing via the left nostril (Shannahoff-Khalsa, 1993).

'Device guided breathing' is a breathing technique that uses a bio-feedback device to help regulate breath rate. This resembles some pranayama practices, which use internal counting to regulate breath rate and the duration of the four phases of the breath cycle (i.e. inhalation, post inhalation breath retention, exhalation and post exhalation breath retention). The device first measures the patient's current breath rate to establish a baseline and then uses two different tones prompting the patient to breathe in and out more slowly than the baseline with the exhalation longer than the inhalation. Although this method uses modern technology for regulating the breath, the results of these studies may shed some light on possible effects and benefits of similar pranayama exercises. Recent studies with hypertensive patients (n=268) have shown that device-guided breathing interventions used for 15 minutes daily for eight weeks results in reduction in systolic and diastolic blood pressure (BP) of 12.1 and 6.1 mmHg respectively, as compared to reduction of 7.6 and 3.4 mmHg respectively in an active control group that listened to relaxing music for the same duration. The BP reduction in the device group was significantly greater than a predetermined clinically meaningful threshold of 10.0, 5.0 and 6.7 mmHg (for systolic BP, diastolic BP and Mean arterial pressure respectively). The study authors concluded that the intervention proved effective, for reducing BP with no reported adverse effects, for hypertensives and mild hypertensives, in office and home settings (Grossman & Grossman, 2003; Grossman et al., 2001; Rosenthal et al., 2001; Schein et al., 2001; Meles et al., 2004).

1.7.13.5 Effects of yogic meditation and relaxation practices

A relatively small number of studies have been published on the effects of yogic meditation and relaxation techniques on their own (i.e. not as part of a protocol which also includes yogic breathing and/or yoga postures). Conversely, a substantial number of studies have been published on the effects of non-yogic meditation techniques.

Arguably, the broad definition of yogic meditation in Patanjali's *"Yoga Sutra"* may apply to non-yogic meditation methods if they share common principles and techniques. This section focuses on evidence on the effects of yogic meditation techniques, and the next section focuses on evidence on the effects of several similar non-yogic meditation techniques that may be of relevance to similar but less researched yogic practices.

Yoga nidra has played a major role in studies on yogic meditation techniques which have shown that yoga nidra meditation is associated with improvement in a range of psychological and physiological measures including self reported stress (Pritchard et al., 2010), anxiety, and well-being (Kamakhya, 2004), increased release of endogenous neurotransmitter and changes in brain EEG spectra. (Lou et al., 1999; Kjaer et.al, 2002) and improvement in symptoms of diabetes (Amita et al., 2009), while the practice of other yogic guided meditation and relaxation techniques has been shown to be associated with decreases in heart rate, breath rate, oxygen consumption, and a regulatory effect on the autonomic nervous system (ANS) (Vempati & Telles, 2002; Amita et al.,2009). These findings are reported in detail below.

Yoga nidra appears to assist in improving various subjective measures. A small study of yoga nidra practice consisting of daily half hour sessions for six months reported significant positive changes in practitioners' subjective measures of anxiety and well-being (Kamakhya, 2004 as reported by Jadhav & Havalappanavar, 2009) while another small study (n=22) found significant reductions in perceived stress in cancer patients and multiple sclerosis (MS) patients following six weeks of a yogic meditation program that incorporated a weekly 90 minute yoga nidra class with daily home-based practice using two audio CDs with two different meditation exercises that incorporated various meditative components including body scan; breath work; exploration of

sensations, emotions and thought patterns, moving back and forth between feeling and witnessing; and sitting in awareness (Pritchard et al., 2010).

A Positron Emission Tomography (PET) and EEG study comparing brain activity of experienced yoga teachers during yoga nidra meditation to resting states of normal consciousness found differential activity in parts of the brain responsible for imagery tasks during yoga nidra versus differential activity in parts of the brain responsible for executive attentional network during normal consciousness (Lou et al., 1999). Another PET study demonstrated a 65% increase in endogenous dopamine release in the ventral striatum and a concomitant increase in EEG theta activity during yoga nidra meditation with all participants reporting a decreased desire for action during yoga nidra, along with heightened sensory imagery, suggesting that yoga nidra meditation causes a suppression of cortico-striatal glutamatergic transmission (Kjaer et.al, 2002).

A three months study (n=41) of yoga nidra as an intervention for diabetes found that patients on yoga nidra plus oral hypoglycaemic regimen had better control of blood glucose levels and other symptoms associated with diabetes, compared to controls who were on a hypoglycaemic regimen alone (Amita et al., 2009).

A study of the ANS responses of 35 male subjects aged 20 – 46 to yoga based guided relaxation compared to yogic rest in the supine rest position found that both techniques were associated with similar decrease in heart rate and skin conductance with the guided relaxation techniques also being associated with significant increases in breath volume and decreases in oxygen consumption. Heart rate variability (HRV) spectral analysis further indicated a reduction in sympathetic activation during guided relaxation in subjects with hyper-activation at baseline but not in others, implying a regulatory effect of guided yogic relaxation practice on ANS (Vempati & Telles, 2002).

1.7.13.6 Effects of *non-yogic meditation methods*

A rapidly growing body of evidence exists on the effects of non-yogic meditation techniques, many of which are similar to various yogic meditation techniques. For the sake of brevity only the results of several major systematic reviews and meta-analyses are discussed as well as several studies on meditation intervention for insomnia. Overall, meta-analyses suggest that meditation practice may help reduce blood pressure, stress, anxiety, alcohol abuse and relapse of depression (Chiesa & Serretti, 2009; Chiesa, 2009; Chiesa, 2010). Studies have also, shown that meditation practice may affect changes in EEG spectra and brain physiology (Chiesa, 2010; Chiesa & Serretti, 2010). Similar findings have been reported above with regards to the effects of yogic meditation practices - yoga nidra in particular (see section 1.7.13.5). This further demonstrates the great affinity between yogic and non-yogic meditation techniques. Most of the meditation techniques included in the reviews are shown to provide some benefits, although to varying degrees. Most reviews do not make a clear distinction between meditation techniques, breathing techniques and other mind-body cultivation techniques, and often use general terms such as yoga and chi-kung. Thus it is not clear if references are made to yoga and Qi Gong practices in general or specifically to the meditative components within these vast and diverse practice systems.

A recent comprehensive systematic review of the effect meditation on health reviewed 813 predominantly poor-quality studies. A subset meta-analyses of 65 low-quality studies on the effects of meditation on hypertension patients showed that Transcendental Meditation (TM), Qi Gong and Zen Buddhist meditation (see glossary) significantly reduce blood pressure and that Yoga helps reduce stress but is no better than Mindfulness Based Stress Reduction (MBSR) in reducing anxiety in patients with cardiovascular diseases. A subset meta-analysis of 55 poor quality studies on the

physiological and neuropsychological effects of meditation practices indicates that some meditation practices produce significant changes in healthy participants. Reviewers state however, that solid conclusions cannot be drawn based on available evidence and better quality evidence is required (Ospina et al., 2007). Similarly, a recent Cochrane review of meditation therapy as an intervention for anxiety disorders found only a small number of adequate quality studies and could not draw conclusions on the efficacy of the intervention (Krisanaprakornkit et al., 2006).

A few studies indicate that TM is comparable to other kinds of relaxation therapies in reducing anxiety and that Kundalini Yoga (KY) does not demonstrate significant effectiveness in treating obsessive-compulsive disorders (OCD) compared to other relaxation and meditation methods. Furthermore, dropout rates in general appeared to be high (Krisanaprakornkit et al., 2006).

Several studies have found a significant increase in alpha and theta brain wave activity during meditation with neuroimaging studies showing that Mindfulness Meditation (MM) practice, including Zen meditation, Vipassana meditation and MBSR, is associated with activation of the prefrontal cortex (PFC) and the anterior cingulate cortex (ACC) and that long-term MM practice is associated with an enhancement of cerebral areas related to attention (Chiesa, 2010; Chiesa & Serretti, 2010). MBSR has also been found to be comparable to standard relaxation in stress reduction and reduced stress and anxiety in comparison to an inactive control (Chiesa & Serretti, 2009). Zen meditation is also associated with stress reduction and blood pressure reduction and increased antioxidant activity (Chiesa, 2009; Chiesa & Serretti, 2010) while Vipassana meditation has been found to reduce alcohol and substance abuse in prison populations (Chiesa & Serretti, 2010).

Several studies have found mindfulness based practices to be effective for treating primary insomnia. A single eight week study (n=30) compared mindfulness based stress reduction (MBSR) to pharmacotherapy (PCT) intervention for primary insomnia and found comparable large significant improvements in a range of subjective sleep quality measures and a significant objective improvement in SOL (Gross et al., 2011). Another study (n=21) of a combination of mindfulness meditation with cognitive behavioural therapy as an intervention for insomnia (CBT-I) revealed both acute and long term (12 months) improvements in a range of subjective sleep quality measures (Ong et al., 2009).

In summary, yoga nidra incorporates several meditative techniques, which are similar to the non-yogic meditation techniques reviewed above (also see section 1.7.13.5). Therefore, future research on yoga nidra and other yogic meditation techniques is required to examine whether they can also provide similar benefits.

1.7.14 Yoga practice for older adults

There is a growing awareness of the need to adapt yoga practice for the special needs and limitations of the elderly (Krucoff et al., 2010). A typical group of seniors is likely to have a wider range of physical abilities and health conditions than any other age group and various health conditions become more prevalent with age and need to be taken into consideration to ensure participants safety and well-being (Krucoff et al., 2010). For example, hearing difficulties become prevalent with age and yoga teachers need to take this into account when instructing elderly students. Symptoms of dizziness also become more prevalent with age affecting up to 38% of older adults (CDC, 2007) and this may lead to falls and injuries especially with poses requiring good balance (Krucoff et al., 2010). Osteoporosis and associated risk of fractures also becomes

prevalent with age especially amongst post menopausal women and this may require avoiding or modifying various yoga poses. The National Osteoporosis Foundation has recommended avoiding exercises such as sit-ups, stomach crunches and toe-touches and also avoiding twisting the spine to the point of strain and bending forward from the hip (National Osteoporosis Foundation [NOF], 2010). It has therefore been suggested that yoga teachers who work with the elderly should be vigilant and that emergency medical assistance should be sought if elderly yoga students suddenly becomes uncomfortable, dizzy, develop nausea, chest pain or shortness of breath (Krucoff et al., 2010). These aspects were all taken into consideration in design of yoga protocol for the present study (see section 3.3).

Increasingly special yoga programs are being developed for elderly populations. For example, a "Therapeutic Yoga for seniors" teachers training program was launched in 2007 by Duke University as part of integrative medicine facility. This program combines evidence based western medicine and traditional yogic teaching and includes yoga movement and meditation, health conditions common to the elderly and safety issues (Krocoff et al., 2010). Another example is Silver Age Yoga (SAY), a non- profit organization offering yoga classes and teachers training courses and promoting a system of hatha yoga developed for the elderly (SAY, 2010).

Three studies have been conducted in Taiwan measuring the effect of a six months intervention of hatha yoga adapted for the needs of the elderly ("Silver Yoga"). Subjects for these studies include community dwelling elderly (two studies) and elderly living in assisted living facilities (a single study). The interventions included gentle yoga postures, relaxation exercises and guided imagery. Results reveal significant improvements in various subjective mental health and sleep quality measures along with

objective physical fitness measures including body composition, cardiovascular-respiratory function, physical function and range of motion with improvements in fitness levels being associated with physical yoga exercises but not with meditative exercises (Chen et al., 2008; Chen et al., 2009; Chen et al., 2010a). Another six month Indian study (reported above), which examined subjective sleep quality outcomes of yoga versus 'Ayurveda' medicine in normal older adults, revealed significant improvements in various subjective sleep quality measures including sleep latency, total sleep time and feeling refreshed in the morning in the yoga group, but not the control group (Manjunath & Telles, 2005) (see section 1.7.13.2.2). In view of the encouraging results reported in these studies, additional research is required to examine how yoga can be adapted for the special needs and limitations of older adults in general and in western cultural settings in particular and the present study has aimed at filling this gap in the existing evidence.

1.7.15 'Western' exercise versus yoga

A recent review of 81 studies on yoga interventions, found ten studies that compared yoga outcomes to non-yogic ('western') exercise intervention outcomes in both healthy and unhealthy populations. Reviewers concluded that yoga interventions appeared to be equivalent or superior to exercise interventions in almost every health-related outcome measured except for those related to physical fitness (Ross & Thomas, 2010). A small (n = 34) twelve week study comparing Iyengar yoga practice to walking (mentioned above) found greater improvement in psychological factors in the yoga group compared to the walking group. Furthermore, positive correlations were reported between changes in mood scales and increase in GABA levels pointing to a possible mood modulating mechanism which is affected to a greater extent by yoga than by walking (Streeter et al., 2010). Additional research is required however, to compare

various types of yogic exercise protocols to various types of non-yogic exercise protocols.

1.7.16 Compliance in yoga and related practices

Relatively few studies have examined adherence in yoga practice and the 'dose response' relationship between yoga practice and the rate of change in physiological and psychological measures. More studies have examined adherence in non-yogic meditation practices. However, in most cases, it is not clear what the definition of 'good' adherence and compliance level is based on, especially in the case of home-based practice. Overall it seems that home-based practice compliance is somewhat lower than class compliance and that outcomes may be related to practice compliance levels (Flegal et al., 2007; Carmody & Baer, 2008)

Several studies indicate a good level of adherence to yoga interventions as well as non-yogic meditation based interventions both for class and home-based practice (Flegal et al., 2007; Carmody & Baer, 2008). Only a few studies have examined long-term adherence to yoga practice (Yang, 2007). A six month, three-armed study of a Iyengar yoga intervention versus exercise versus waiting list control of 135 generally healthy older adults (aged 65-85) found good adherence in both yoga and exercise interventions. Overall drop-out rate was 13% with class attendance scores being higher than home compliance scores in both groups and scores being higher in the yoga group than in the exercise group. For the yoga intervention group class attendance was 77% and home practice compliance was 64%, whereas for the exercise intervention, group class attendance was 69% and home practice compliance was 54% (Flegal et al., 2007). A four year study of a non-yogic meditation based intervention reported a high degree of adherence with the meditation technique, maintenance of improved status over time,

and a high degree of importance attributed to the training program (Kabat-Zinn et al., 1986). A further study on the effect of a mixed intervention incorporating body scan, yoga, and mindfulness sitting meditation found that the amount of time spent in home-based practice was significantly related to the extent of improvement in most aspects of mindfulness and some measures of physical symptoms and well-being (Carmody & Baer, 2008).

1.8 Implications for present and future research

The literature review above raises several points that are of special relevance to the present study:

1. It seems that relatively few studies have been published on yoga for the elderly population or on yoga as an intervention for insomnia. Even fewer studies have been published specifically on yoga intervention for improving sleep quality in a geriatric population. As discussed in section 1.7.13, two studies were conducted recently in Taiwan (Chen et al., 2009; Chen et al., 2010) and one in India (Manjunath &Telles, 2005). These studies did not specifically select participants with a history or present complaints of sleep disturbances. Furthermore, none of these studies used objective sleep quality measures, and were not conducted in a western cultural setting. Therefore, the present study was initiated in March 2007 with the aim of filling the gap in existing evidence, by studying the effects of yoga practice on insomnia and well being, of older adults in a western cultural setting, while incorporating both objective and subjective measures.
2. This review discussed various risk factors for insomnia including cognitive and physiological hyper arousal, stress, anxiety, depression and chronic pain

and a range of yogic practices have been shown to reduce these risk factors. Arguably, practices that affect these psychological factors may also indirectly help improve sleep quality and the present study aimed at examining these relationships more closely by comprehensively measuring both sleep quality and psychological outcomes.

3. Pranayama practice has been shown to provide various physiological and psychological benefits but ancient and some contemporary core texts recommend practicing pranayama only after a sufficient foundation of asana practice has been established. As a conservative precautionary measure the present study incorporated practices which include breath awareness but excluded practices which include breath regulation (i.e. pranayama practice)
4. There is evidence on the benefits of practices which incorporate cycles of isometric effort and relaxation, both in yogic and non-yogic context. The present study applied this evidence by incorporating a short period or relaxation following each of the yoga poses performed during yoga classes
5. Ancient texts point out that different yogic practice elements may be more suitable for different people. Yoga Nidra, is a yogic meditation which combines diverse meditative and relaxation elements, including somatic relaxation, breath awareness and breath counting, mindfulness of internal body sensations, objectless mindfulness, visualisations and positive suggestions. The diversity of meditative elements was one of the factors which weighed in favour of incorporating yoga nidra in the present study's protocol. Furthermore, the protocol also incorporated a yogic somatic relaxation practice and yogic breath counting/awareness practices separately to accommodate individual differences and to allow individuals to build

essential yogic practice elements to support the more complex yoga nidra practice

6. There is some evidence that rate of improvement is related to practice compliance level and that home-based practice compliance may be lower than class attendance. The present study took this evidence into consideration by recording the home based practice exercises on an audio CD in order to facilitate easier application and higher compliance.

In an analysis of meditation research Shapiro etc. al (2003) suggested specific recommendations, for future research on meditation including:

1. Differentiation between types of meditation to identify general, overlapping and specific effects of different types of meditation.
2. Recording frequency and duration of meditation to establish 'dose effect relationship' between meditation and its effects.
3. Long and short term follow-up assessment.
4. Research on long-term as well as short term meditators.
5. Comparing meditation to alternative attentional practices (e.g. playing a musical instrument).
6. Component analysis of meditation including factors such as belief and expectancy, postural, somatic, attentional, cognitive etc. with the aim of differentiating the effects and interactions of various factors.
7. Examination of interaction effects between meditation and a variety of relevant psychological, spiritual and clinical factors.
8. Development of subjective and objective mediating variables to determine those that account for the most variance in predicting change.

9. Collecting qualitative data on the subtlety, depth and overall experience of the meditation experiences as well as the interplay between subjective and objective factors.
10. Expanding meditation research from effects on symptoms reduction of mental and physiological conditions to effect on problem prevention and health enhancement and the transpersonal.
11. Research effect of meditation on traditional goals of meditation, such as the development of exceptional maturity, love and compassion, and lifestyles of service and generosity.

In the last seven years some of Shapiro et al.'s suggestions have been applied. Several studies and reviews have compared the effect of different meditation and relaxation techniques. Several studies have compared the effect of meditation to other alternative attentional activities such as listening to music for the same period.

In a more recent comprehensive review of meditation practices for health Ospina et al. (2007) suggest the following regarding future research on meditation:

1. Develop a consensus on a working definition of meditation applicable to a heterogeneous group of practices.
2. Systematically compare the effects of different meditation practices that research has shown to have promise.
3. Pay special attention to the appropriate selection of controls
4. Conduct more research on the "dose response" of meditation practices to determine appropriate study durations and to help standardize courses of therapeutic meditation.

5. Employ designs and analytic strategies that optimise the ability to make causal inferences (even if in some cases it requires the use of uncontrolled pre and post intervention designs).
6. Aim at using larger samples and concurrent controlled designs
7. Use disease-specific measures.
8. Provide clear descriptions of intervention components.
9. Better quality reporting by a wider dissemination and stricter enforcement of the CONSORT (Consolidated Standards of Reporting Trials) guidelines within the complementary and alternative medicine (CAM) community.

Although the recommendations above were given specifically in the context of meditation research, they may arguably apply to yoga research as well. The present study addresses some of the points mentioned above by:

1. Incorporating a daily practice log in order to establish a 'dose - response' relationship between duration and frequency of practice and the effects.
2. Supporting home-based practice with an audio CD as well as repeating the instructions and revising during class practice.
3. Using comprehensive objective and subjective measures specific to sleep disorders in general and insomnia in particular.
4. Incorporating various psychological, physiological, social, and quality of life measures in addition to specific sleep quality/disturbances measures.
5. Providing a very detailed description of all aspects of the intervention.
6. Striving to achieve the largest sample with available limited budget and human resources.

7. Selecting a suitable intervention for older adults that also represents a range of yoga practices practiced widely in western cultural settings.

Chapter 2. Study aims

2.1 Study aims

1. To examine the effectiveness of an integrated yoga intervention for improving quality of sleep in elderly people presenting with complaints of insomnia.
2. To examine the effectiveness of an integrated yoga intervention for enhancing mood and quality of life in elderly people presenting with complaints of insomnia.
3. To determine the suitability and acceptance of an integrated yoga intervention for elderly people living in a westernised culture.

2.2. Hypotheses to be tested

The following hypotheses were proposed and tested in the present study:

1. Integrated yoga intervention will significantly improve subjective and objective measures of sleep quality in elderly presenting with complaints of insomnia.

2. Integrated yoga intervention will significantly improve measures of both mood and quality of life in the elderly people presenting with complaints of insomnia.

3. That an integrated yoga intervention will be safe and acceptable for elderly people with complaints of insomnia living in a westernised cultural setting.

Chapter 3. Materials and methods

3.1 Study design considerations

3.1.1 Introduction

The present study utilised a mixed study design that developed during the course of the research which was constrained by the limited available resources (Please refer to study flowchart - Figure 4.1). Organisations and individuals made generous contributions by donating precious expertise and time and/or by allocating equipment and space (see acknowledgments). Nevertheless, it was still necessary to find the right balance between an ideal design, described below, and between a feasible design dictated by available resources. As the study progressed, through divine providence, generosity and good will of individuals and organisations, additional resources were made available. These enabled reassessing and modifying the original study design. However, additional resources only became available incrementally while the study was already in progress. These enabled the study design to be modified to a stronger design, but nevertheless not the ideal design that would have been possible had all resources been available or committed at the outset. Overall, resource limitations affected the total number of sleep studies that could be conducted, the number of participants that could be recruited and measured, the number of groups that could be compared, the total duration of the intervention period, the choice of control and randomisation as described in detail below.

3.1.2 Required resources

A great number of resources were required for successful implementation of the present study including:

- Certified yoga teachers to conduct yoga classes.

- Appropriate yoga practice venues.
- Physician(s) for conducting medical interviews and examinations of candidates/participants.
- Medical staff to assist with setting up, removing and collecting portable sleep monitoring equipment.
- Sleep scientists for analysing sleep studies.
- Administrative staff to assist with required paperwork at the medical centre
- Hospital rooms and facilities for screening and examining applicants and participants.
- Sleep monitoring equipment and related consumables for conducting sleep studies.
- Computer hardware and software for interfacing with portable sleep monitoring equipment, downloading, analysing and storing acquired data.
- Office supplies and equipment.
- Human resources and equipment for creating, recording, editing and duplicating audio CDs with meditative yoga exercises for the home self practice intervention component.
- Securing resources for advertising the study in local media, bulletin boards, community centres and medical centres throughout the Jerusalem metropolitan area.
- Securing resources for communicating with applicants, participants, physicians, hospital staff, yoga teachers, practice venue administrators etc.
- Means of transportation required for travel to various venues carrying necessary equipment.

3.1.3 Initial resources availability

The author was awarded a research fellowship from the Australia Israel Scientific Exchange Foundation (AISEF) to the total sum of 5000 AUD, and a travel grant from RMIT University to the total sum of 1000 AUD, to cover transport, insurance and accommodation expenses associated with a return trip from Melbourne, Australia to Jerusalem, Israel. Two mobile sleep labs were assignd for two continuous two to three weeks periods. This enabled conducting objective pre- and post-intervention objective measures with participants admitted to the study but did not allow screening the much larger overall number of applicants (see section 4.2.2) for SRBD and OSA in particular. Furthermore, since availability of traditional PSG facilities at the medical centre was extremely limited it was decided that it would be used only if diagnosis based on portable monitoring was not possible. Overall, the number of sleep studies that could be conducted at each milestone was limited to around 30. Furthermore, the number of milestones for taking objective measures was limited to two, namely, pre- and post-intervention measures. Availability of sleep physicians, sleep technicians and sleep scientists was also limited to specific days and hours. Initially, only two yoga teachers were available for a duration of 12 weeks, each able to teach one weekly class only. Also, only one practice hall was available for two weekly sessions for duration of 12 weeks.

3.1.4 Additional resources made available

Several positive though unforeseen developments occurred during the course of the study that led to reassessment and expansion of the original study design. Hundreds of phone enquiries continued to be received from potential participants well past the original application deadline. Applicants kept contacting the study's office weeks after the original participant quota for the study had already been reached, participants inducted and baseline measures taken. Also, four additional certified yoga teachers, affiliated with the Israeli

Yoga Teacher's Association, contacted the study office offering their help at no cost. An additional suitable yoga practice venue was offered at no cost for two weekly classes over a period of 12 weeks, and a third venue was later offered at a discount. Through the kindness of Dr. Cahan and Dr. Baharav two additional brand new portable sleep monitoring units were also made available. Dr. Cahan and Dr. Baharav kindly agreed to screen, process, analyse and diagnose additional applicants and participants. However, the rate at which it was possible to screen, process and induct additional applicants was slower due to other commitments of physicians, sleep scientists and technical staff and due to work load related to monitoring, acquiring, and processing data of the first group already in progress.

3.1.5 Ideal versus feasible design

The ideal strongest experimental design is a double blinded, randomised placebo controlled trial where subjects are assigned at random to treatment and placebo groups and both subjects and researchers are unaware of which treatment was given until the study has been completed (Sibbald & Roland, 1998). Furthermore, due to the nature of the intervention in the present study, it would arguably have been advantageous to incorporate at least four groups including a yoga intervention group, another active intervention (e.g. exercise or walking) group, a sham intervention group (discussed in more detail below) and a control group. That would have allowed better control for possible extraneous factors associated with participating in an intervention group (e.g., encountering fellow participants, being in contact with teachers, changes to daily and weekly routines, getting out of the house to go to practice sessions, etc.) as well as comparing effectiveness of the yoga intervention to another active intervention. All applicants would ideally be screened using both portable monitoring and standard clinical assessment. All participants would ideally be measured at pre-intervention, post –intervention and also at several pre-

determined, equally spaced milestones during the intervention phase in order to examine not only the final effect of the intervention, but also the rate of change for each of the measures over the course of the intervention period. A long intervention period would ideally be incorporated to reduce extraneous factors such as the rate at which individuals are able to acquire and assimilate new skills.

After consulting a substantial number of yoga masters and teachers (see acknowledgments), and taking safety considerations and available venues into account, a common consensus was reached that maximum class size should be around 30 participants, but ideally around 20 participants. Furthermore, a majority opinion among yoga masters and teachers consulted (see acknowledgments) was that a minimum of two weekly classes over 12 weeks was necessary in order to achieve tangible results, although several yoga teachers recommended periods of between 18 and 24 weeks.

The present study's budget constraints and resource availability timeline also affected the control design. A control is essential in studies with an evaluation of a treatment's effect on behaviour, performance or mood compared to baseline levels (Goodwin, 2010, p. 173; Goodwin, 2010, p. 267). There are several varieties of possible control designs including no-treatment, placebo control, waiting list control (WLC) and yoked control (Goodwin, 2010, pp. 267-275).

A no-treatment control is a typical and most straightforward type of control design for many types of studies while in drug trials a placebo control is often used (Goodwin, 2010, p. 267). However, in studies designed to evaluate a therapy aimed at alleviating a physical or mental health problem, these types of control design may give rise to ethical issues. For example, if the therapy is later found to have been effective at alleviating a health problem, some may argue that withholding therapy from the subjects in the

control group is unethical (Goodwin, 2010, pp. 269-270). This ethical problem may be avoided by using a WLC design, often used in studies intended for assessing the efficacy of various therapies or programs (Goodwin, 2010, p. 267). The WLC group is a no-treatment group of subjects suffering from the same problem as the subjects in the active intervention group. The WLC group subjects are given an opportunity to receive an equivalent treatment after having completed the control phase (Goodwin, 2010, p. 267). In the present study due to ethical considerations it was decided to incorporate the WLC principle in the design.

A 'placebo', typically used in drug intervention research, usually refers to an inactive pharmacological substance given to participants in the 'placebo' control group while an active pharmacological substance is given to subjects in the treatment group. The participants in the 'placebo' control do not know that they are receiving an inactive substance to prevent them from being subtly influenced by the knowledge that they are taking an inactive substance (Goodwin, 2010, p. 267). A placebo control design may sometimes be applied in procedure intervention research by using a 'sham' procedure or treatment (Sutherland, 2007) defined as "*An inactive treatment or procedure that is intended to mimic as closely as possible a therapy in a clinical trial. Also called placebo therapy"* (National Cancer Institute, 2010). Obviously a sham intervention must be one that had previously been shown to have no effect on research outcomes of interest. In the present study, a 'sham' intervention had been considered but rejected for the following reasons:

1. The yoga intervention used in the present study incorporated a combination of yogic relaxation, meditation and physical exercises. A suitable 'sham' protocol would have to be a comparable composite protocol that had already been validated

as having no effect on physical health, mental health and sleep quality. No such suitable ‘sham’ protocol has been identified.

2. A set of ‘non yogic’ stretches as a ‘sham’ physical yoga component was also considered. However, the known range of yoga stretches is vast and most ‘western style’ stretching techniques have commonalities with subsets of yogic stretches.
3. A ‘sham’ meditative audio CD, was also considered (e.g. classical music, new age music or natural sounds such as waves or waterfall sounds), however, listening to music while sitting can in itself be considered a form of meditative technique, that uses music or sounds as objects of meditation (see section 1.7.10) that may contribute to the reduction of ‘the fluctuations of the mind’ and therefore be related to the broad definition of the purpose of yoga and may possibly induce a meditative state in some participants (see section 1.7.3). A study (n=63) compared mental and physical states of a subjects that listened to Mozart music, subjects that listened to new age music and subjects that spent the same amount of time reading recrational magazines. The study reported that listening to Mozart's music induced more psychological relaxation and less stress than listening to New Age music or reading recreational magazines. Those who had listened to Mozart music also reported significantly higher levels of mental quiet, awe, wonder, and mystery, while those who had listened to New Age music reported slightly higher levels of feeling at ease/peace and feeling rested/refreshed (Smith & Joyce, 2004). People from different ethnic, cultural and educational backgrounds may arguably be affected differently by various types of music, and this in turn may affect their nervous systems differently. This could possibly have introduced an undesirable confounding factor to the study.

4. The efficacy of an intervention with a daily home-based practice component is affected significantly by the level of compliance (see sections 1.7.15 and 3.5.3). Using a 'sham' protocol, with a 'sham' audio CD, (e.g., with music) may possibly result in a different compliance profile (in frequency and/or duration of practice) compared to the intervention. This could have possibly introduced an additional confounding factor to the study.

Under ideal study conditions, randomisation is incorporated in study design. This is done in an attempt to equalise the composition of the control group and intervention group, in a way that would make them as similar as possible in all relevant characteristics, including possible confounding factors (Chatburn, 2011, p. 328). Each subject is allocated to either control or intervention group using the laws of statistical probability. This can be done by flipping a coin, drawing a subject identification number from a hat (Chatburn, 2011, p. 328) or by using random number generating software (Haahr, 2011). In the present study initial budget and resources limitations enabled recruiting only 31 participants. A randomised controlled approach would have resulted in two groups (intervention and control) of 15 to 16 participants each, and consequently resulting in reduced statistical power, defined as the "*probability of rejecting a false null hypothesis" (*Christensen et al., 2011, p. 267), or in simple terms the probability of correctly stating that the intervention produced an effect *(*Christensen et al., 2011, p. 267). A greater number of participants increases statistical power and vice versa *(*Christensen et al., 2011, p. 267). The risk of a substantial number of dropouts from an already small (n=15) intervention group was of special concern, as it would have reduced power and would have had a detrimental effect on quality of randomisation (Lachin, 2000).

Restricted resources also excluded the possibility of applying a 'crossover design' where 'group A' would undergo yoga intervention for 12 weeks followed by no-treatment

for 12 weeks and group ‘group B’ would undergo no-treatment for 12 weeks followed by yoga intervention for 12 weeks (Chatburn, 2011, p. 323), as that would have required to double the number of ‘yoga teacher hours’ and ‘venue hours’. Furthermore, since the yoga intervention involved participants acquiring meditative and relaxation skills, there was a strong possibility of a ‘carry over’ effect, defined as “*conditions from one experiment affecting subsequent experiment”* (Chatburn, 2011, p. 323) occurring in the group that would start with yoga intervention for 12 weeks and then cross over to no-intervention for 12 weeks (‘group A’). This carry over effect could happen in two ways: The yoga intervention could theoretically affect long term or even permanent psychological and/or physiological changes that would affect participants well into the no-treatment period that would follow the treatment period (in ‘group A’). Some crossover studies incorporate a ‘washout period’ between the two periods of intervention and no-intervention, intended to allow the effect of the first intervention to wear out (Chatburn, 2011, p. 323). This may arguably be more suited to a drug intervention then for a yoga intervention because in a drug intervention, traces of the drug are gradually cleared out of the body after patients stop taking them but in a yoga intervention, changes may theoretically be long lasting, and acquired yogic practice skills may persist well into the future. Another confounding factor could occur if some of ‘group A’ participants did not stop practicing yoga techniques during the 12 weeks of no-treatment following the crossover, despite being instructed to stop as would be required by study protocol. These participants might nevertheless continue practicing at home if they felt that the intervention had resulted in positive outcomes and worried that stopping to practice may result in deterioration in the progress they had made with regards to sleep quality or quality of life. It would be very difficult to monitor and detect such non-adherence to protocol.

Blinding is a method whereby the researchers do not know during the study the nature of the treatment (or no-treatment) a participant is receiving (Christensen et al., 2011, p. 497) and double blinding is a method whereby both researchers and participants do not know during the study the nature of the treatment (or no-treatment) a participant is receiving (Christensen et al., 2011, p. 500). In the present study each participant received a code number that was used to identify his/her subjective and objective data. In this way sleep physicians, scientists and technicians handling the data were blinded to the nature of treatment a participant had received. However, double blinding was not possible since no 'sham' intervention /placebo was used.

A follow-up of participants at a one year interval after the completion of the study had been considered. The purpose of the follow-up would have been to examine participants' sleep quality and quality of life a year later; to examine whether any significant changes in measures or trends had occurred and to compare these changes to changes that had occurred over the course of the study. Furthermore, a follow-up could have helped examine the percentage of participants that had continued to practice meditative yoga at home using the audio CD provided for the study and at what practice frequency level compared to the level that had been achieved during the study. Such findings could have helped shed more light on the long term effect of short duration yoga programs and may have helped formulate better protocols for such programs. Unfortunately and regrettably, due to limited funding and its affect on available resources and study timeline, no follow-up was possible.

After taking all the above into consideration, it was decided at the outset that the optimal strategy would be to assign all those admitted to the study to WLC group followed by active yoga intervention. The control phase would consist of 12 weeks of no-intervention. Pre- and post-WLC phase measures would be taken. After completing post

control phase measures *all* control phase *completers* would be contacted and given an opportunity to undertake a 12 week yoga intervention phase. Those who would accept the offer would be assignd to a 12 weeks yoga intervention phase after which post- yoga intervention measures would be taken. Using this method would enable using a smaller number of participants without losing statistical power. Obviously, this method does not allow random assignment of participants to treatment and control groups. The most effective control for the WLC study design was 'control by matching' (Christensen et al., 2011, p. 207). Matching can be an effective control method *provided* all the data required for matching participants is available (Christensen et al., 2011, p. 207). There are several matching techniques. Due to the initial small number of participants and the large number of variables, it was decided the best matching method would be matching by equating participants (Christensen et al., 2011, p. 211). There are various methods to equate participants. In the present study it was decided to use the most obvious method and equate each participant in the WLC phase to himself/herself in the subsequent yoga intervention phase. This matching technique can be very effective *provided no* major changes had occurred (Christensen et al., 2011, pp. 207 -208) within the participant or in the general environment from WLC baseline to the yoga intervention baseline. Possible changes may include significant changes in personal circumstances, physical health, mental health and general environmental conditions etc.

As mentioned above, at the initial study design stage, the likelihood of additional resources becoming available was seen as extremely low. Nevertheless, additional resources became available incrementally during the course of the study and additional applicants responded for weeks after the original application closing deadline (see section 3.1). Resource availability timeline and intermittent applicant inflow did not allow applying a randomised control design or a WLC design to *additional* participant intake

admitted to the study (n=43).It was therefore decided to assign all *additional* participants (n=43) to 12 weeks yoga intervention in the two separate venues that had become available and utilise the four additional yoga teachers that had become available. It was decide to use the existing WLC group as a control for all active yoga participants, as well as for itself, as originally intended (see section 3.1 &3.5.1). This resulted in a mixed experimental design combining, the original WLC experimental design (i.e., WLC completers versus themselves as subsequent yoga intervention participants) and an additional expanded experimental design (WLC versus *all* yoga intervention participants). The WLC design had a stronger control design and the expanded design involved a larger number of participants and therefore had greater statistical power. Arguably, combining the two designs provided a better design within the external constraints and limitations that had been described above.

In the present study a much higher proportion of women applied to join the study and consequently the study included 81 percent woman. An ideal study would have had an equal number of men and women but this would have required processing a much larger number of applicants which the study's limited resources and timeline did not allow.

3.2 Participants

3.2.1 Recruitment

Participants were recruited via an advertising campaign targeting elderly community-dwelling and independent retirement-dwelling men and women with complaints of insomnia. Advertisement campaign used a range of means and media in order to target as wide as possible population base throughout the Jerusalem metropolitan area (see section 3.2). Applicants were processed non-preferentially on a 'first come first serve' basis. The following advertisement methods were used (see appendix 12):

1. Advertisements in local community newsletters and papers.
2. Posters on bulletin boards in local community centres, retirement housing complexes (for independent dwelling), shopping centres, medical centres and outpatient clinics at main hospitals.
3. Leaflets were left in offices of local community centres and retirement housing complexes (for independent dwelling).
4. Phone calls were made to medical centres throughout metropolitan area, targeting general practitioners and asking for referrals of suitable candidates.
5. Introductory lectures were given jointly by sleep physicians and yoga teachers at community and retirement housing complexes. The lectures covered geriatric insomnia, yoga, the intervention used in the study, subjective and objective measures used in the study and general inclusion and exclusion criteria.
6. Word of mouth – many enquiries came from individuals who heard about the study from friends (or friends of friends) and relatives who had seen the advertisement.

The response to the modest low budget advertisement campaign was substantial. Many enquiries from prospective candidates or their relatives were received well after having completed the initial recruiting process of 31 participants.

3.2.2 Screening participants

The study was designed to be ecologically valid in that it included typical older people presenting with insomnia symptoms. Accordingly, the screening process followed current clinical diagnostic guidelines regarding patients presenting with sleep complaints, whereby insomnia is primarily diagnosed by a clinical evaluation based on a systematic medical, psychiatric, substance use and sleep history acquired by interviewing patients, reviewing their medical records and administering medical/psychiatric questionnaires,

sleepiness assessment tool(s), sleep logs, measures of subjective sleep quality, psychological assessment scales, daytime function, and quality of life scales (AASM, 2010; Schutte-Rodin et al., 2008; Mai & Buysse, 2008; Littner et al., 2003) (See sections 1.5.6 and 3.2.2). As discussed in section 1.5.6, according to the 2003 AASM practice parameters, PSG is not indicated for the routine evaluation of insomnia unless a breathing disorder or limb movement disorder (PLMD) is suspected (Schutte-Rodin et al., 2008; Littner et al., 2003).

On the other hand, as reported above a survey of 461 randomly selected elderly aged over 65 in the US has found 24 percent had Apnea index (AI) $\geqslant$ 5 and 62 percent had Respiratory Disturbance Index (RDI) $\geqslant$ 10 (Ancoli-Israel et al., 1991) (see section 1.6.4.4) and several studies have estimated the frequency of insomnia in OSA patients to be between 24.2 percent and 54.9 percent (Smith et al., 2004; Krakow et al., 2001; Chung, 2005; Krell & Kapur, 2005) (see section 1.6.4.7). Furthermore, one study reported a high prevalence of undiagnosed sleep apnea in elderly insomniacs that had previously undergone an interview to screen for sleep apnoea with an AHI >5 in 43 percent and an AHI >15 in 29 percent of the study population and suggested using PSG as a screening tool when recruiting older adults for research on insomnia (Lichstein et al., 1999). The screening process of the present study was intended to minimise OSA diagnosis false negatives by combining a systematic clinical examination by a sleep and respiratory physician in conjunction with psychological and sleep quality questionnaires including the Multivariate Apnoea Prediction index (MAP) that had been reported as useful for discriminating between patients with and patients without sleep apnoea in non sleep centre populations (Maislin et al., 1996; Maislin et al., 1995). Also, if any previously undiagnosed psychiatric or medical disorder, including OSA, or PLMD, was suspected, the patient was referred to further medical investigation, including a sleep study if necessary, and was

excluded from the study unless all additional medical investigations were completed and yielded negative results. The assumption that this screening process would be adequate to minimise OSA diagnosis false negatives was later disproven as reported in section 4.6 and this is discussed further in section 5.3.

The multi-stage screening process utilised in the present study included the following stages:

1. An initial phone interview
2. Mail-out of study information statement (see appendix 4.4) and medical forms (see appendixes 3.2-3.3) to potential participants and their personal physicians
3. Review of letters from personal physicians including patient's full medical history and the physician signed approval for patient's participation in yoga activity.
4. The completion of additional medical forms and subjective sleep quality (including MAP, PSQI, ESS and KSS) and psychological questionnaires at the medical centre
5. A structured interview by a sleep and respiratory physician at the medical centre
6. Signing of a consent form at the medical centre

3.2.3 Inclusion and exclusion criteria

3.2.3.1 Inclusion criteria

The following inclusion criteria were applied in participants for the present study:

1. Individuals – both male and female, 60 years of age and above.
2. Presenting with insomnia complaints, occurring at least three times a week, for at least one month (see section 3.5.3).
3. Willing to accept assignment procedure.
4. Able and willing to comply with all study protocols including: Regular attendance of intervention yoga classes; not engaging in any other mind-body activities during

the study and not starting any new exercise or recreational activities for the duration of the study. Continuation of well established regular activities (e.g. walking, swimming, playing bridge and other hobbies) was allowed.

5. Able to reach yoga practice venue independently or via public transport.
6. Able and willing to read, understand and fill out all forms and questionnaires
7. Able and willing to provide informed consent

3.2.3.2 Exclusion criteria

The following exclusion criteria were applied in screening participants for the present study:

1. Evidence of other primary sleep disorders by history and/or previous sleep study - such as untreated obstructive sleep apnea (OSA), NREM Parasomnias (e.g., restless leg syndrome [RLS], periodic leg movement [PLMD], confusional arousals, sleepwalking [somnambulism], sleep terrors, bruxism [teeth grinding] etc.) REM Parasomnias (e.g., REM sleep behaviour disorder, Catathrenia [breath holding and groaning] etc.)
2. Any medical condition known to affect sleep and/or mental state.
3. Chronic medical conditions which may affect sleep if uncontrolled, unstable or severe (e.g., diabetes, hypertension, ischemic heart disease, renal disease, urinary incontinence, prostate disease, etc.)
4. Recent major medical procedures (e.g., major surgery)
5. Severe chronic pain, caused by a primary health condition (e.g., musculoskeletal condition, cancer, fibromyalgia, arthritis, irritable bowel syndrome etc.) or by a primary pain disorder.
6. Malignant arrhythmia by ECG (e.g., ventricular tachycardia, heart block, atrial flutter, paroxysmal atrial tachycardia, etc. not controlled by medications)

7. *Any major* mental illness, dementia, major depression (by DSM-IV criteria) (APA, 2000).
8. Currently receiving psychiatric or psychological care
9. Reported current alcohol intake greater than two standard drinks daily (≥20 grams)
10. Reported current drug abuse (prescription and/or 'recreational')
11. Any health condition or mental condition which precludes gentle physical exercise or meditation exercise in judgment of study physician and/or personal physician
12. Planned life stressors (e.g., moving house, divorce, overseas travel etc.).
13. Engaged in *similar* mind-body activities over the last three months, or past history of *similar* mind-body activities (at least once per week for three months).
14. Note: use of sedative-hypnotics and/or relaxants was allowed if participant had been on a regular consumption pattern for at least 6 weeks. The present study examined changes in the consumption of these medications via the PSQI sleep medication subscale and via the daily sleep logs (see sections 3.4.2.3 and 3.4.2.8).

3.3 Intervention

3.3.1 General protocol design questions

The yoga intervention protocol was designed after reviewing ancient and contemporary yogic texts and commentaries (see section 1.7). Discussions were held with several yoga masters who generously gave of their time and knowledge (please refer to the acknowledgments). To facilitate optimal protocol design the following questions were asked at the outset:

1. Should the protocol be based on a specific yoga method/school or should it be a customised protocol which incorporates selected practices/components?

2. Should the protocol be based on class practice, home-based self practice or a combination of both? And if so what should the ideal ratio be?
3. What is the optimal number of weekly practice sessions?
4. What should the home self-practice component include and preclude?
5. What specific modifications are needed to adapt yogic practices to an elderly population in a western cultural setting?
6. What can be done to ensure the quantity and quality of home-based self-practice?

3.3.2 Protocol design guidelines

Yoga protocol requirements and priorities based on research aims and ethical guidelines:

1. Safe and suitable for the general elderly population.
2. Easy to understand, and apply by the elderly.
3. Suitable for western living conditions and life style.
4. Focuses on improving sleep quality and quality of life of elderly people presenting with complaints of insomnia.
5. Easy to apply by yoga teachers and participants
6. Designed to promote uniform and consistent practice by participants.

3.3.3 Protocol design considerations

1. The aging process is associated with physical decline and various common health conditions such as osteoporosis, hypertension, hearing loss, impaired vision, etc. Furthermore, the aging process may affect balance, coordination, muscular strength and response time (See section 1.2). These factors needed to be taken into account in the design of the yoga intervention protocol.

2. The present study had a limited budget. The yoga teachers were volunteers affiliated with the Israel Yoga Teacher's association and experienced in teaching general hatha yoga and not affiliated with any specific school or brand of yoga. Therefore, an intervention protocol that was unaffiliated with a specific yoga lineage was developed.
3. A customised protocol tailored specifically for the study aims would better represent yoga's potential future benefits for the population under study.
4. Two weekly classes were considered by the yoga masters consulted to be an optimal compromise: One weekly class was deemed insufficient to maintain continuity and to adequately support home practice, and three weekly classes might have been considered by many elderly people as an inconvenience or burden and may have resulted in lower attendance rates.
5. On the other hand, all yoga masters consulted were of the opinion that to achieve significant results within a 12 week period, two weekly classes may not be adequate and adding a home self practice component was believed to be necessary.
6. Of the eight 'limbs' of classical Yoga described in the ancient seminal text "*Yoga Sutra"* of Patanjali (Iyengar, 1996; Feurstein, 1990) (See sections 1.7.4 -1.7.9), the following limbs were found of relevance to present study: 'Asana'(postures), 'Pranayama' (breath control), 'Pratyahara' (withdrawal of sensory stimuli, direction of attention inwards), 'Dharana' (concentration on an object, controlling the mind not to be distracted by thoughts) and 'Dhyana' (meditation, uninterrupted flow of awareness). However, a key ancient text and a contemporary master are of the opinion that practice of 'Pranayama' (breath control), although an important tool for improving health, preventing and curing diseases, facilitating concentration and calming the mind, requires adequate preparation, including a good foundation in

asana practice. Furthermore, these sources believe that premature or improper practice of pranayama may harm the practitioner's health – specifically predisposing him/her to hypertension, respiratory disorders, and cardiovascular disorders (Svatmarama, 2002, pp. 36-37; Iyengar, 2010, pp. 53-54; Iyengar 2006, p. 33) (See section 1.7.5). Taking these opinions into consideration, it was concluded that for an elderly population with no experience of yoga practice, due caution should be taken and pranayama breathing exercises should be excluded from the study's yoga protocol. Nevertheless, the breath did play an important part in the protocol as two of the three meditative yoga exercises, incorporated some form of breath awareness, by using the breath as an object of concentration. Furthermore, in the physical yoga practice, while standing in tadasana ('mountain' pose), participants were asked to raise the arms while breathing in and lower them while breathing out, thus teaching basic breath – movement coordination.

7. Safety considerations also precluded unsupervised home practice of yoga poses. Therefore, it was decided that the home self practice component would include meditation and relaxation exercises only. Thus, 30 - 50 percent of net class time was assignd to meditation and relaxation exercises in order to support home practice and ensure its correct execution. The remaining 50 – 70 percent of net class time was assignd to physical asana practice.
8. Compliance and correct execution were considered the main challenges for home – based self-practice. To facilitate home practice an audio CD was designed and produced, which included instructions and three guided meditation/relaxation exercises. The CD was designed to facilitate uniform execution of exercises and also make self discipline easier.

9. Taking all above considerations into account, a protocol which was based mostly on meditative aspects of yogic practice seemed the most applicable.

3.3.4 Aims of physical yoga component

General aims for the physical yoga component were *to improve* the following mind-body aspects:

1. The ability to relax and concentrate
2. Mind-body awareness
3. Balance
4. Core strength
5. Posture and poise
6. Spinal mobility, flexibility and strength
7. Joint mobility and flexibility
8. Coordination
9. Blood circulation.

3.3.5 Aims of meditative yoga component

General aims for the meditative yoga component were to develop core skills of relaxation, concentration and awareness and more specifically to:

1. Develop an ability to consciously relax.
2. Develop an ability to consciously concentrate.
3. Develop some awareness of the mind's habitual thinking patterns.
4. Develop some ability to control/slow down the thinking process.
5. Develop awareness of physical sensations and physical tension.

3.3.6 Yoga practice duration, frequency and timing

The present study's yoga protocol was based on a combination of yoga classes and home-based self-practice. Practice time was assignd as follows:

1. **Intervention period:** 12 weeks
2. **Practice frequency:** Two yoga classes per week and home-based practice - recommended seven days a week (see item no. 5)
3. **'Practice unit':** a yoga class was considered as one 'practice unit'. For home-based practice – one guided meditative exercise (using an audio CD) plus time required to get ready for practice and then conclude the practice was considered one practice unit (net practice time was between 25 and 35 minutes).
4. **Yoga class duration:** Total class duration was one hour which included net practice time as well as giving instructions, questions and answers, taking attendance, collecting forms and logs. Actual net practice time was between 25 and 35 minutes (one 'practice unit')
5. **Recommended daily practice:** The recommended total daily practice was three 'practice units', seven days per week (a total of 21 practice units per week). Since each supervised class was considered one practice unit, on days that yoga classes were held, recommended home practice was two practice units (roughly 40 -50 minutes) and on other days, recommended home practice was three practice units (roughly 60 -75 minutes). Recommending participants practice seven days a week was intended to reinforce the general intention and suggestion of making yoga practice an integral part of the daily routine. Applying this recommendation was possible for all participants including orthodox Jews that observe the holy day of the Sabbath, as according to the Jewish tradition the Sabbath religious rituals and

customary restrictions end at nightfall (Israel Ministry of Tourism, 2010; Chabad, 2010).

3.3.7 General precautions during class

The following precautions were taken during yoga classes:

1. Participants were placed at a safe distance from other participants.
2. Unobstructed access and line of sight from yoga teacher to each participant was maintained
3. A first aid kit, telephone and water were available at all yoga venues.
4. Comfortable ambient temperature was maintained.
5. The classrooms were close to the toilets

3.3.8 Yoga class structure

Each of the yoga classes had the following general structure:

1. General welcoming of students by the teacher
2. The completion of an attendance log.
3. Physical yoga exercises
4. Meditative yoga exercises
5. General question and answer time
6. Discussion of any administrative issues

3.3.9 Physical yoga component

3.3.9.1 Introduction

The physical practice component included a sequence of yoga poses (asana) and movements. Standing, sitting, horizontal prone and supine poses were incorporated.

3.3.9.2 Cautions

The following cautions were given every class:

1. Participants were asked to make sure they understood the instructions before performing a pose and if not to raise their hand and ask the teacher for assistance.
2. Participants were asked not to do anything which they felt might compromise their health and safety.
3. Participants with a (controlled) blood pressure condition were asked not to raise their hands above their shoulders and not to bend their head below the chest (heart) level.
4. Participants with osteoporosis were asked to avoid postures that involve bending forward from the waist and avoid twisting to the point of strain.
5. Participants were asked to perform every exercise gently and gradually, and avoid straining.
6. Participants were asked to stop any exercise immediately and notify the teacher if they felt dizzy, short of breath, tired, with a chest pain, with nausea, generally unwell or injured.
7. Participants were asked not to leave class without teacher's permission.
8. Participants were asked to alert the teacher immediately if they did not feel well.
9. Teachers were instructed to have the entire class in their field of vision.
10. In case of any adverse effects teachers were instructed to stop class activity, ask other participants to rest, and attend to the participant with the problem immediately.

3.3.9.3 Alternative poses

An important protocol design issue was whether or not to provide alternative/modified poses for participants who could not, or were reluctant, to perform any particular pose. In community based yoga classes, alternative poses are offered regularly due to prevalence of physical limitations among individuals in the elderly population (see section 1.2.). Under ideal conditions, all participants would be able to perform all poses. However, the prevalence of musculoskeletal complaints, poor balance and individual discomfort, make this an unrealistic expectation in elderly cohorts. Two possible solutions were suggested: The first was to offer constrained participants alternative poses. The second solution was to ask participants who thought that they could not perform a particular pose, to rest until the next pose was called out. Both methods resulted in reduced uniformity of practice. However, it was considered that the former option offered higher a level of uniformity because the alternative/modified poses were designed to preserve the basic physiological principle (e.g., spinal twist, forward stretch etc.) and core benefits (e.g., flexibility, mobility, core strength, balance etc.) of the original pose.

The yoga teachers and masters who were consulted reported many elderly participants are reluctant to perform mat based poses because of difficulty or hesitation in getting down to the mat and getting up from the mat. There can be various reasons for this, including, leg and knee weakness, knee pains, poor balance etc. Thus most of the alternative poses were chair-based substitutes for mat-based poses. Chairs were also used for those who required support in standing poses.

3.3.9.4 Protocol

The total duration and number of poses in the physical yoga component of a class varied in accordance with the time assignd to explanations, questions and answers, and to the meditative component etc. Total duration of the physical yoga component normally ranged from 35 to 20 minutes. Number of repetitions for most exercises was one or two, but in some cases up to nine depending on the exercise and available time. Total duration in most poses was from 20 seconds up to 60 seconds depending on the pose. Each of the chair and mat based poses was followed by a relaxation period of approximately 20 seconds. The general sequence of the physical component was:

1. standing poses
2. chair based poses
3. Mat based poses (or chair based alternate poses).

The general sequence of mat based poses was:

1. a short relaxation in shavasana (corpse pose)
2. supine poses
3. prone poses
4. sitting poses
5. a short relaxation in Savasana (corpse pose)

Table 3.1 includes a detailed full sequence of the physical yoga component. The table lists the poses as well as alternative/modified poses. Appendix no. 9 provides photographs of all poses.

Please continue to the next page ➔

Table 3.1

Yoga poses sequence used in the protocol

No.	Sanskrit name	English name	type	base	Rep	Modification	comment
1	Tadasana	Mountain pose	STA		1		
1-ALT	Tadasana	Mountain pose	STA		1	MOD # 1	
2	Tadasana	Mountain pose	STA		1 - 2	MOD # 1A	
3	Tadasana	Mountain pose	STA		1 - 2	MOD # 1B	
4	Virabhadrasana II	Hero pose II	STA		1 - 2		RS, LS
4-ALT	Virabhadrasana II	Hero pose II	STA		1 - 2	MOD # 1	RS, LS
5	Bikram style Ardha-Chandrasana	Bikram style half moon pose	STA		1 - 2	MOD # 2	RS, LS
5-ALT	Ardha-Chandrasana	modified half moon pose	STA		1 - 2	MOD # 2A	
6	Adho Mukha Svanasana	downward facing dog pose	STA		1 - 2		
6-ALT	Adho Mukha Svanasana	downward facing dog pose	STA	CHR	1 - 2	MOD # 3	
7	Manibandha Chakra	Wrist rotations	SIT	CHR	3 -9		CW, CCW
8	Goolf Chakra	Ankle rotations	SIT	CHR	3 -9		CW, CCW
9	Skandh Chakra	shoulder rotations	SIT	CHR	3 -9		CW, CCW
10	Garurasana	Eagle pose	SIT	CHR	1 - 2	MOD # 4	RS, LS
11	Savasana	Corpse pose	SUP	MAT	1		
11-ALT		seated relax	SIT	CHR	1	MOD #11	
12	Ardha Pavana muktasana	Half Wind removing pose	SUP	MAT	1 - 2		RS, LS
12-ALT	Ardha Pavana muktasana	Half Wind removing pose	SUP	CHR	1 - 2	MOD # 5	RS, LS
13	Bhujangasana (easy version)	"Baby" Cobra Pose	PRN	MAT	1 - 2	MOD # 6	
13-ALT		standing baby cobra	SIT	WAL	1 - 2	MOD # 7	
14	Ardha shalbhasana	Half Locust Pose	PRN	MAT	1 - 2		
14-ALT		standing half Locust	STA	WAL	1 - 2	MOD # 7	
15	Marjaryasana	cat pose		MAT	2-4		
15-ALT	Marjaryasana	cat pose		CHR	2-4	MOD # 3	
16	Bitilasana	Cow pose		MAT	2-4		
16-ALT	Bitilasana	Cow pose		CHR	2-4	MOD # 3	
17	Marichyasana (easy version)	Spinal twist	SIT	MAT	1 - 2		RS, LS
17-ALT	Chair twist	Spinal twist	SIT	CHR	1 - 2	MOD # 8	RS, LS
18	Ardha-Kurmasana	Half Tortoise Pose	SIT	MAT	1 - 2	MOD # 9	
18-ALT	Ardha-Kurmasana	chair - Tortoise	STA	CHR	1 - 2	MOD # 10	
19	Balasana	Child's pose	SIT	MAT	1 - 2	MOD # 9	
19-ALT		Chair child pose	STA	CHR	1 - 2	MOD # 10	
20	Paschimottanasana	Seated forward bend	SIT	MAT	1 - 2	MOD # 12	
20-ALT		Chair –forward bend	SIT	CHR	1 - 2	MOD # 12	
21	Savasana	Corpse pose	SUP	MAT	1		
21-ALT		Chair – relax 1	SIT	CHR	1	MOD #11	
21-ALT		Chair – relax 2	SIT	CHR	1	MOD #11a	

Notes for Table 3.1:

1. The following abbreviations are used in table above:
   RS – right side, LS- left side, CW – clockwise, CCW – counter clockwise, STA- standing pose. SIT – sitting pose, PRN – prone pose, SUP- supine pose, MAT – mat based pose, CHR – chair based pose, WAL –using wall for support, MOD –modified pose, ALT – alternative

2. The following modifications have been applied or offered optionally:

MOD # 1 - Holding onto back of chair for balance

MOD # 1A –shifting weight to right, left, front, back while engaging abdominal muscles gently

COM #1B – raising hands while breathing in. Lowering hands while breathing out

MOD # 2 – when bending to right – right hand on hip and left hand over head and vice versa

MOD # 2A – same as MOD # 2 but with right hand on back of chair for support

MOD # 3 – holding seat of chair or top of back of chair and bending forward

MOD # 4 – in chair sitting position arms interlaced with left elbow below right and the legs – right knee over left knee and vice versa on the other side

MOD # 5 – in chair sitting position raise knee towards trunk and hug over or below knee

MOD # 6 – gentle upper back raise with neck in line with the spine, facing down, palms on floor next to armpits facing down

MOD # 7 – facing wall, hands on wall, weight forward slightly, back arched looking up

MOD # 7A – facing wall, hands on wall, weight forward slightly, back arched looking up. One leg extended back, off the floor

MOD # 8 when twisting to the right – left hand over right tight grabbing chair seat and right hand grabbing back of chair and vice versa

MOD # 9 – in case of knee restriction pose is executed with increased angle between calf and thigh (up to 90 degrees if needed)

MOD # 10 –standing and bending forward placing forehead and palms of hands on chair's seat.

MOD # 11 – sitting upright back to chair's back support, palms resting on thighs, feet flat on ground, close eyes and relax

MOD # 11a – sitting upright back to chair's back support, palms resting on thighs, legs extended forward on another chair, close eyes and relax

MOD # 12 – bending forward with palms resting on thighs for support. Legs together

MOD # 12a – bending forward sitting on a chair with legs extended on another chair

MOD # 13 – sitting while leaning forward with palms and head resting on back of another chair for support. Folded towel may be used under forehead

3.3.9.5 Poses and effects

Ancient and contemporary yogic texts asanas list various claimed physiological effects and benefits of yoga poses. Table 3.2 below lists claimed effects and benefits of selected poses in line with defined aims of the physical yoga component of the protocol.

Table 3.2

Yoga poses used in protocol - claimed effect and benefits

Pose No.	Sanskrit name	English name	Claimed benefits/ protocol aim
1	Tadasana	Mountain pose	Improve posture, strengthen legs
2	Tadasana	Mountain pose variation	Improve balance and core strength
3	Tadasana	Mountain pose variation	Develop Fuller breathing
4	Virabhadrasana II	Hero pose II	strengthen and stretch legs, ankles improves stamina
5	Bikram style Ardha-Chandrasana	Bikram style half moon pose	Improve Lateral spinal mobility, flexibility and strength, stretch shoulders
6	Adho Mukha Svanasana	downward facing dog pose	Improve circulation to brain, calms the mind, Strengthen arms and legs, Stretch the shoulders, hamstrings, calves, arches, and hands
7	Manibandha Chakra	Wrist rotations	Improve wrist mobility and flexibility
7	Goolf Chakra	Ankle rotations	Improve Ankle mobility and flexibility
8	Skandh Chakra	shoulder rotations	Improve shoulder mobility and flexibility
9	Garurasana	Eagle pose	Strengthen & stretch ankles, hips, shoulders, elbows and upper back
10	Savasana	Corpse pose	Relax whole body, resting pose
11	Ardha Pavana muktasana	Half Wind removing pose	Massage abdomen and remove wind, improve hip joint flexibility
12	Bhujangasana (easy version)	"Baby" Cobra Pose	Strengthen spinal muscles, stretch chest, shoulders, , buttocks, massage abdomen
13	Ardha shalbhasana	Half Locust Pose	Strengthen spinal muscles & buttocks, stretch chest, shoulders, buttocks, and backs of the arms and legs chest, abdomen, and thighs, massage abdomen
14	Marjaryasana	Cat pose	Counter Stretches the back torso and neck
15	Bitilasana	Cow pose	Stretches the back torso and neck gently
16	Marichyasana (easy version)	Spinal twist	Improve spinal and hip flexibility , mobility and circulation, Massage abdomen
18	Ardha-Kurmasana	Half Tortoise Pose	Relaxation pose. Calms the mind. Increases circulation to the brain and upper lungs, improve flexibility of hip joints, ankles and shoulder girdle
18	Balasana	Child's pose	Relaxation pose. Calms the mind. Increases circulation to the brain improve flexibility of hip joints, ankles , relieves back tension
19	Paschimottanasana	Seated forward bend	Stretches the spine, shoulders, hamstrings
20	Savasana	Corpse pose	Relaxes whole body, resting pose

Note for table 3.2:

The following references were used: Yoga Journal website Asana section (Yoga Journal, 2010), Asana Pranayama Mudra Banda (Swami Satyananda Saraswati, 1996), Light on Yoga (Iyengar, 2001); Yoga Wisdom and Practice (Iyengar, 2009), Bikram's Beginning Yoga Class (Bikram, 2000).

3.3.9.6 Uniformity of practice and quality control

Yoga teachers were certified by the Israel Yoga Teachers Association. Several pre-intervention meetings were held with the teachers to discuss the intervention class structure, pose sequences, modified poses, safety regulations, cautions and precautions, in order to ensure correct and uniform practice and participants' health and safety. Also, to ensure uniformity of practice, sheets containing pose sequences and photographs of all poses and modified poses were distributed to the yoga teachers. An investigator monitored all classes to ensure uniformity and adherence to protocol.

3.3.10 Meditative and relaxation yoga component

3.3.10.1 Overview

The Meditation/Relaxation protocol consisted of three basic meditative exercises designed to facilitate the development of basic mind-body meditative skills including relaxation, concentration, breath awareness and sensory awareness. These are intended to facilitate a state of mind which combines alertness, concentration, relaxation and mind-body awareness. Three basic mediation exercises were taught during yoga classes and were also included in the audio CD for home-practice:

1. Breath counting meditation
2. Muscular relaxation
3. Yoga Nidra

3.3.10.2 Audio CD for at-home self-practice

A pre- recorded audio CD containing all necessary instructions and three guided meditation sequences was provided to all participants. The recorded instructions allowed participants to review, revise, and refresh their memory whenever necessary. The recorded guided meditations facilitated uniformity in execution and were designed to facilitate better concentration and relaxation during practice by allowing participants to simply follow instructions rather than having to remember the sequence, decide what to do next or for how long to do each exercise. Furthermore, the voice of an experienced teacher, although from a recording and not in person, provided some simulation of class atmosphere and a feeling of being supervised and guided. The participants provided a lot of feedback on the content and form of the CD via space provided on their daily sleep and practice logs (DSPL).The majority of participants' comments were positive, indicating the CD fulfilled its intended objectives. There were some critical remarks but these tended to focus on the vocabulary and grammatical style used in the CD.

The audio CD for home self practice contained these five tracks:

1. Track 1 – instructions on how to use the audio CD
2. Track 2 – general instructions on yoga practice in the study
3. Track 3 –Breath counting meditation – 20 minute guided meditation sequence
4. Track 4 – Muscle relaxation practice - 20 minute guided meditation sequence
5. Track 5 –Yoga Nidra meditation- 20 minute guided meditation sequence'

3.3.10.3 'Breath counting meditation' exercise

The first meditative exercise incorporated in the audio CD was a natural breath counting meditation. It is similar to one of essential components incorporated in a typical yoga nidra sequence (Satyananda, 1976, p. 69).The aim of this exercise was to

prepare participants for full yoga nidra practice, by improving concentration and developing awareness of the natural breath cycle and also of the mind's tendency to get distracted by random thoughts. Because the ability to concentrate the mind leads to and is essential for success in any meditative technique (Yogendra, 2003, pp. 40-41; Feuerstein, 2000, p. 85) (see section 1.7.7), participants were taught the 'breath counting meditation' during the first week of the intervention and it was also the first of three meditative techniques included in the audio CD (see appendix 10.1 for a full CD sequence transcript.) It was recommended to conduct this exercise while sitting or lying down with the eyes closed. The practitioners were asked to simply direct their attention to the natural breath without altering it in any way. In order to facilitate breath awareness, the practitioners were instructed to observe the rising and falling of the abdomen, or as an alternative, observe the sideways expansion and contraction of the rib cage, or observe the flow of air via the nostrils. The practitioners were instructed to count each complete breath cycle as follows: The practitioner observes the inhalation phase followed by the exhalation phase. Towards the end of the exhalation phase the practitioner counts the current breath cycle number. The practitioner continues to count up (1, 2, 3 etc.) until the end of the exercise on the CD is reached. In case of losing concentration or forgetting to count, the practitioner was instructed to start counting up again from one. On the CD track for this exercise, instructions were given at the beginning of the exercise and then every few minutes a short repetition of the instructions was given and finally the end of the session was announced (See Appendix 10.1 for CD transcript).

3.3.10.4 Muscle relaxation exercise

As discussed above muscle relaxation techniques aim at training the practitioner to voluntarily relax muscular tension and affect physiological relaxation in order to

induce psychological relaxation utilising the linkage between body and mind (see section 1.5.12.3.9). Yoga also incorporates various muscle relaxation techniques which are also one of the components in most 'Yoga Nidra' protocols (see section 1.7.12). In current study a separate muscular relaxation exercise was incorporated to induce somatic relaxation and also as preparation for the full 'Yoga Nidra' practice. Participants were taught the muscle relaxation sequence, as recorded on the audio CD, during the second week of the intervention. In the muscle relaxation exercise used in the present study, practitioners were asked to assume a comfortable position, ideally the supine shavasana (corpse pose). The practitioner were then asked to direct his/her attention sequentially to muscle groups throughout the body starting from the feet, then calves, thighs, lower back, middle back, upper back, lower abdomen, chest, shoulders, upper arms, lower arms, palms and fingers, scalp, temples, back of the head, neck, forehead, eyebrows, cheeks, nose, mouth, chin, throat. Once awareness was focused on a particular body part, the practitioner was asked to quietly tell that body part "relax". Uttering the word "relax" by whispering or by silently moving the lips rather than by inward silent mental repetition. The word "relax" is used in this meditation as a Mantra (see section 1.7.10) with the intention of facilitating concentration and mind-body integration. This is repeated several times for each muscle group. If the practitioner finds it difficult to relax a particular muscle group, he/she is instructed to consciously contract that muscle group while breathing in and then consciously relax it while breathing out (See Appendix 10.2 for CD transcript.).

3.3.10.5 Yoga nidra exercise

There are various variants of 'Yoga Nidra' sequences. In the present study the yoga nidra protocol incorporated main components of yoga nidra transcripts as taught

by Swami Satyananda Swaraswati of the Bihar school of yoga (Satyananda, 1976, pp. 81- 150) as follows:

1. **Preparation** – the practitioner is instructed to lie down comfortably on a mat in the supine position, feet apart and palms facing upwards, cover the body with a blanket if necessary to keep warm and once comfortable stay still till the end of the practice. If the supine position is not comfortable other horizontal or sitting positions are permissible.
2. **Relaxation -** the practitioner is instructed to feel the tension in the muscles dissolving and relax the whole body (part by part or all at once)
3. **Resolve -** the practitioner is instructed to make a wish for any positive change in life, health, relationship etc.
4. **Rotation of consciousness –** Body parts are mentioned sequentially in a rapid succession from top to bottom and from right to left. The practitioner directs his/her awareness to the body part called out by the teacher (or audio recording) and tries to sense it and visualise it as vividly and clearly as possible. The sequence used in this protocol starts on the right side of the body: first finger, second finger, third finger, fourth finger, fifth finger, palm of the hand, back of the hand, wrist, forearm, inside of the elbow, back of the elbow, upper arm, shoulder, armpit, torso, hip, buttock, thigh, front of the knee, back of the knee, calf, shin, top of the foot, heel, sole, first toe, second, toe, third toe, fourth toe, fifth toe. The awareness is then directed to the left side of the body and a mirror image sequence is repeated. Awareness is then directed to the stomach, chest, lower back, middle back, upper back, back of the neck, scalp, forehead, eyebrows, eyes, cheeks, nose, nostrils, lips, inside the mouth, teeth, chin, and throat. Awareness is then directed to the entire lower part of the body from the

waist down. Then to upper part of the body from the waist up. Then to the entire right side of the body. Then to the entire left side of the body. Then to the entire back side. Then to the entire front of the body. Finally to the entire body.

5. **Breath awareness –** the practitioner directs awareness to the breath as it manifests in the movement of the abdomen, ribs, chest, throat, nostrils. In each of these body parts, the practitioner maintains focus and counts down from a certain number to zero (or vice versa). This component is similar to the breath counting meditation mentioned above.
6. **Awareness of sensations** – The practitioner senses opposite sensations in the body: Heat and then cold, lightness and then heaviness, painful sensations and then pleasant sensations etc.
7. **Focusing on the inner space –** The practitioner observes whatever appears in the inner space between the eyebrows (with the eyes closed).
8. **Visualisations –** The practitioner is instructed to visualise a sequence of objects, scenery, animals etc. which the practitioner tries to visualise as vividly as possible as they are called out in a rapid succession.
9. **Repeating the Resolve –** The practitioner is instructed to repeat the initial resolve three times **(**a wish for any positive change in your life, health, relationship etc.).
10. **Completion** – The practitioner is instructed to become aware of the body, the room, the surroundings, stretch gently, move the limbs gently, blink the eyes a few times and then get up slowly

Notes:

1. The full standard 'Yoga Nidra' protocol above may take between 40 to 45 minutes. In the home practice audio CD a shorter version was used which omitted steps 6 – 8, thus taking 25 minutes in total duration.

2. In some classes the full 'Yoga Nidra' protocol was used and in others - shorter versions, depending on time assignd for other exercises etc.

(See Appendix 10.3 for CD transcript.)

3.4 Outcome measures

3.4.1 Introduction

Both subjective and objective instruments were used to measure the study outcomes. Measures were taken during the ten day period pre-intervention and again during the ten day period post-intervention. Pre-intervention subjective measures were administered at the SZMC medical centre and post-intervention subjective measures were administered at the training locations (for the intervention group) or SZMC medical centre (for the control group). Subjective measures were derived from a range of reliable and valid self-reported questionnaires, eliciting information on sleep quality and disturbances, daytime sleepiness and function, psychological wellbeing, physical wellbeing, daily and social functioning. These measures included the Profile of Mood States –short form (POMS-SF), the Depression Anxiety Stress Scale –long form (DASS-42), the Pittsburgh Sleep Quality Index (PSQI), the Karolinska Sleepiness Scale (KSS), the Epworth Sleepiness Scale (ESS), the Multivariable Apnea Prediction Index (MAP), the Health Survey (SF36) and daily sleep and practice logs (DSPL). There were several overlaps in measures amongst these questionnaires, but the selected combination was deemed to provide a comprehensive set of measures. Where measures overlapped, the most specific and detailed measures were relied on. Furthermore, some redundancy

was incorporated intentionally by using two similar questionnaires for mood states, namely the POMS and the DASS. This was done primarily because the POMS incorporates six mood state scales (Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigour-Activity, Fatigue-Inertia and Confusion-Bewilderment) whereas the DASS has only three scales (depression, anxiety and stress). However, the DASS is more detailed and specific in itemising possible symptoms of depression, anxiety and stress. It has separate scales for anxiety and stress, whereas the POMS has a single combined scale for tension-anxiety. In addition the DASS gives a detailed situational description for each of the items (e.g., "I found myself in situations that made me so anxious I was most relieved when they ended") whereas, each of the POMS items consists of a single adjective (e.g., "tense", "anxious", etc.) and is therefore more prone to variation in respondents' interpretation of the precise meaning. Indeed, most queries from respondents while filling out the questionnaires were related to the POMS. However, the POMS does have the useful scales of fatigue and vigour which are not included in the DASS.

Daily Sleep and Practice logs (DSPL) were used during the study to provide daily information on sleep and dietary patterns, consumption of medications, including hypnotics and relaxants and home practice patterns. In addition, demographic (age, gender, marital status, weight, height) and medical questionnaires were used during pre-study recruitment stage to elicit medical and demographic information including medical history and diagnoses of any medical or psychiatric conditions, and also prescriptions of any medications (including hypnotics and relaxants).

Several studies have been conducted to validate Hebrew versions of the SF-36 (Lewin-Epstein et al., 1998), the PSQI (Shochat et al., 2007) and the POMS (Netz et al.,

2005). The Hebrew versions have been found appropriate for the intended purpose. There is a need to conduct validation studies for the Hebrew versions of the DASS, MAPS, KSS and ESS for diverse Hebrew speaking populations as the validity of the translated version cannot be automatically assumed. In the present study it was decided to use the Hebrew versions of KSS, ESS and MAPS due to the simple language and concepts used in these questionnaires. The significant overlap between DASS and POMS (see above) enabled comparing the results of overlapping subscales and they were found to be similar.

Objective measures were derived from an analysis conducted by a sleep scientist based on data recordings acquired during overnight sleep studies at participants' homes using portable monitoring equipment in conjunction with the Hypnocore sleep analysis system. Sleep studies were conducted on "regular" nights with respect to participants' sleep-wake schedules (i.e. not on nights during which special activities had been scheduled or anticipated) and sleep time was 'ad libitum'. Each participant was given a detailed explanation of the procedure on the day of the study. A brief explanation was also included in the consent forms (see appendix 4).

3.4.2 Subjective instruments used

3.4.2.1 The Karolinska Sleepiness Scale (KSS)

The Karolinska Sleepiness Scale (KSS) (see appendix 5.1) is a very simple frequently used self-rated questionnaire for evaluating subjective sleepiness. It asks the respondent to rate "How sleepy are your right now?" on a scale of 1 to 9 which ranges from (1) "not sleepy at all" to (9) "extremely sleepy-fighting sleep". The advantage of KSS is in its simplicity and the short time required to complete it. The main disadvantages are that it measures transient sleepiness rather than a usual or average

state and its lack of detail. Consequently, the scores may fluctuate according to quality or quantity of the last sleep period, time of day, and transient circumstances - making test conditions difficult to duplicate and thus possibly affecting measure stability. Furthermore, transient sleepiness may be affected by the act of responding to a questionnaire. A study that investigated whether verbal rating of sleepiness can itself affect sleepiness and performance revealed that the act of rating affects both subjective and EEG measures of sleepiness perhaps via the modest stimulation involved in this act (Kaida et al., 2007). However, the validity and reliability of the KSS have been established in several studies. A study revealed that the KSS score was closely related to EEG and behavioural variables, indicating a high validity in measuring sleepiness and concluded that the KSS ratings may be a useful proxy for EEG or behavioural indicators of sleepiness (Kaida, 2006). Another study of sleep deprived subjects found scores on the KSS showed high correlations with performance tasks (Gillberg, et al., 1994). In summary, the KSS is a useful and convenient tool for measuring transient sleepiness

3.4.2.2 The Epworth Sleepiness Scale (ESS)

The Epworth Sleepiness Scale (ESS) (see appendix 5.2) is a short self-rated questionnaire shown to provide a general level of daytime sleepiness. It asks the respondent to rate the likelihood of falling asleep in eight different common daily life situations on a scale of 0 to 3 as follows: 0 = would never doze, 1= slight chance of dozing, 2 = moderate chance of dozing, 3 = high chance of dozing. If respondents have not encountered a particular situation in daily life, they are asked to estimate the likelihood of falling asleep in that situation. The scores of all eight items are then summed to yield the total score. A total score between 0 and 9 is considered normal. A total score between 10 and 24 is considered to indicate excessive daytime sleepiness (EDS) that may require additional medical investigation (Johns, 1991).

In a study with 180 participants (30 normal and 150 patients with a range of sleep disorders) ESS scores were significantly correlated with sleep latency measured in the multiple sleep latency test (MSLT) and overnight polysomnography (Johns, 1991). In patients with obstructive sleep apnea (OSA), ESS scores were significantly correlated with the respiratory disturbance index (RDI) and minimum blood oxygen saturation (SaO2) whereas the ESS scores of normal snorers did not differ from controls (Johns, 1991). The ESS was also able to easily discriminate between normal subjects and patients suffering from various sleep disorders such as OSA , narcolepsy and idiopathic hypersomnia, demonstrating concurrent validity (Johns, 1991). A study analysing results from several previous studies of narcoleptic patients suffering EDS clearly showed that the ESS is more discriminating than the maintenance of wakefulness test (MWT) and the multiple sleep latency test (MSLT), which had previously been considered as the gold standard. The ESS was shown to have both a high specificity (100%) and sensitivity (93.5%) (Johns, 2000). In a one year study with more than 600 participants, the ESS was completed two times at a 12 months interval and was shown to be a stable measure of sleepiness over time in middle-aged adults (Knutson, 2006). A Spanish version of the Epworth Sleepiness Scale (ESS-Sp) was tested and validated in 345 patients with OSA and shown to be sensitive to post-treatment changes and level of severity and correlated with polysomnography variables (Chiner et al., 1999).

3.4.2.3 The Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index (PSQI) (see appendix 5.3) is a self-rated questionnaire designed specifically to measure sleep quality and sleep disturbances in clinical populations. The PSQI asks subjects to rate sleep quality and disturbances over the month preceding test administration. The PSQI questionnaire consists of 19 items eliciting information on usual sleep habits, nature of sleep disturbances, suspected

causes for sleep disturbances, use of sleep medication, overall sleep quality, daytime sleepiness, and vitality, and also includes an additional five items eliciting information from a bed-partner or roommate. The responses to the latter five questions are not used in the calculation of PSQI global and subscale-scores. They serve only to provide additional information which may be useful in a clinical setting (Buysse et al., 1989). The first 19 items are used to yield seven separate subscale scores (each ranging from 0 to 3), each calculated from related questionnaire items using simple algorithms. The seven subscale scores are then summed to yield a global PSQI score (ranging from 0 to 21). The seven subscales are: subjective sleep quality, sleep latency, sleeps duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications and daytime dysfunction. (Buysse et al., 1989).

In a study of more than 600 participants the PSQI was administered twice, approximately one year apart and was found to be a stable measure of sleep quality in early middle-aged adults. A PSQI global score greater than 5 was classified as poor quality sleep (Knutson, 2006). Clinical and clinometric properties of the PSQI were assessed over an 18-month period comparing "good" sleepers and "poor" sleepers suffering various disorders. Acceptable measures of internal homogeneity and validity were obtained. A global PSQI score > 5 yielded a diagnostic sensitivity of 89.6% and specificity of 86.5% (Kappa =.75, $p \leq .001$) in distinguishing between good and poor sleepers, demonstrating concurrent validity and indicating the PSQI is a useful tool for psychiatric clinical practice and research (Buysse et al., 1989). A study reported reliability, validity, sensitivity and specificity of PSQI seven subscales/components as follows: Overall reliability coefficient for the seven PSQI components -0.83. Largest component-total correlation coefficient was found in habitual sleep efficiency and subjective sleep quality components (0.76 each). Lowest component-total correlation

coefficient was found in sleep disturbances component (0.35), possibly due to the large number of items in this component that may also be more susceptible to variation. Test-retest reliabilities for PSQI components ranged from 0.84 to 0.65. Global score test-retest reliability was 0.85 (Buysse et al., 1989). Polysomnography findings did not correlate well with all PSQI component scores. The discrepancy may be related to the fact that PSQI elicits self estimate of usual sleep quality over a one-month period, thus decreasing its sensitivity to daily variations (Buysse et al., 1989).

A Hebrew version of the PSQI (PSQI-H) has been administered to 450 patients from two sleep clinics and to 61 health subjects. The results showed that the PSQI-H had adequate reliability, good validity and is suitable for use as a standardised tool for the assessment of subjective sleep quality in clinical research with a Hebrew language speaking population (Shochat et al., 2007).

3.4.2.4 The Multivariable Apnea Prediction index (MAP).

The MAP is a self-rated questionnaire designed mainly to predict probability/risk of sleep apnea, based on self-assessed frequency of occurrence of various symptoms during the month preceding test administration. The questionnaire includes 13 questions on frequency of snorting, gasping, snoring, breathing cessations, frequent awakenings, movement during sleep, cataplexy upon awakening, difficulty falling asleep, jumpy or jerky legs, falling asleep during daily activities, and excessive daytime sleepiness. Respondents are asked to rate the frequency of each symptom on a scale of zero to four as follows: Never – 0; rarely/ less than once a week – 1; 1-2 times a week – 2; 3-4 times a week – 3; 5-7 times a week – 4; don't know/not sure – 0 (see appendix 5.4). The 13 items can be grouped into four separate component scores, by averaging the scores of items related to sleep-disordered breathing (items 1, 2 and 3), difficulty sleeping (items

4, 5, 6, 7,8) excessive daytime sleepiness (items 9, 10 and 11) and catatonia (items 12 and 13). The responses to items 1-3 are used in conjunction with data on body mass index (BMI), gender and age to predict apnea probability/risk using a logistic regression procedure.

MAP was assessed as a screening tool for sleep apnoea using questionnaire data from 928 patients presenting at three sleep disorders centres. Multiple logistic regressions using survey responses, age, gender and BMI were then used to estimate a multivariable apnea risk index. The survey was shown to be reliable in a subset of patients from one of the three sites with a test-retest correlation of 0.92. Survey data were then compared to RDI obtained from polysomnography studies. Receiver Operating Characteristic (ROC) curves was then used to assess the predictive ability of the MAP predictive procedure. Using all above risk factors resulted in an area of 0.79 under the ROC curve ($p < .0001$); using only the BMI risk factor resulted in an area of 0.73 and using only self-reported frequency of apnea symptoms resulted in an area of 0.70. Maislin et al. concluded that the MAP may be useful in clinical settings for discriminating between patients with and without sleep apnea (Maislin et al., 1995).

A validity study of the MAP in an elderly population compared MAP scores of relatively healthy older adults with (n=50 cases) and without (n=58) excessive daytime sleepiness (EDS) MAP values were compared to stratified polysomnography derived RDI values ($RDI<10$, $10 \leq RDI<20$, $RDI \geq 20$). ROC curves were utilised for quantitative analysis of the predictive utility of MAP. As an example, for a MAP cut point of 0.40 the sensitivity and specificity (95% C.I.) for $RDI>=20$ for EDS subjects were 0.61 and 0.72 respectively and for controls 0.56 and 0.80 respectively. Maislin et al. concluded

that the MAP may be useful in clinical settings for discriminating between patients with and patients without sleep apnoea in non sleep centre populations (Maislin et al., 1996).

In contrast to the above findings, a recent comprehensive meta-analysis of clinical screening tests for obstructive sleep apnoea (OSA) compared a range of tests and clinical procedures, including the MAP index, Berlin questionnaire, Kushida index and several others and found poor reproducibility and significant variability in accuracy in repeated validation studies of the same screening test and suggested this may result from an underlying heterogeneity in either the clinical presentation of OSA or in the tests' measured clinical elements (Ramachandran & Josephs, 2009). The study concluded that due to the significant rates of false-negatives, there is a high probability that most of the clinical screening tests will miss a significant percentage of patients with OSA and although it is possible to predict *severe* OSA with a high degree of accuracy by clinical methods, no single prediction tool functions as an ideal test (Ramachandran & Josephs, 2009).

3.4.2.5 The Depression Anxiety Stress Scale (DASS)

The DASS (see appendix 5.6) is a self-report questionnaire designed for both research and clinical applications. It consists of three separate scales that measure negative emotional states, including depression, anxiety and stress. These three components can also be summed to yield a global DASS score. The depression scale assesses dysphoria (i.e., unpleasant or uncomfortable mood), hopelessness, devaluation of life, self-deprecation, lack of interest/involvement, anhedonia (i.e., inability to experience pleasurable emotions from normally pleasurable life events), and inertia. The anxiety scale assesses autonomic arousal, skeletal muscle effects, situational anxiety, and subjective experience of anxiety. The stress scale assesses levels of chronic non-

specific arousal manifested by difficulty relaxing, nervous arousal, and being easily upset/agitated, irritable/over-reactive and impatient. There are two versions of DASS, namely the DASS42 with 42 items (and 14 items per scale), and the shorter version DASS21 with 21items (and 7 items per scale). Respondents are asked to rate the extent to which they have experienced each state over the past week using 4-point severity/frequency scale. Scores for depression, anxiety and stress scales are calculated by summing the scores of the items belonging to each subscale (Lovibond & Lovibond, 1995a).

All three DASS subscales (depression, anxiety and stress) have been shown to have high internal consistency and to provide meaningful discriminations in a variety of settings (Lovibond & Lovibond, 1995a). The psychometric properties of the DASS have been evaluated and compared to Beck Depression inventory (BDI) and the Beck Anxiety Inventory (BAI) using a normal population of 717 respondents. The DASS psychometric properties were found to be satisfactory and the factor structure was substantiated by both exploratory and confirmatory factor analysis. The DASS scales showed greater separation in factor loadings than the BDI and BAI. The DASS anxiety scale correlated 0.81 with the BAI. DASS depression scale correlated 0.74 with the BDI. Factor analyses implied that the main difference between BDI and the DASS depression scale is that the BDI includes various items such as weight loss, insomnia, somatic preoccupation and irritability, which do not discriminate between depression and other emotional states (Lovibond & Lovibond, 1995b). The psychometric properties of the DASS were evaluated in two large studies. In the first study (N = 437), the DASS subscales were shown to have excellent internal consistency and temporal stability. An exploratory factor analysis found high consistency with the factor structure previously found in nonclinical samples. Between-groups comparisons found DASS predictably

distinguished various anxiety and mood disorder groups (Brown et al., 1997). In the second study (N = 241), the conceptual and empirical latent structure of the DASS was corroborated by factor analysis. In addition, convergent and discriminate validity of the subscales was demonstrated by correlations between the DASS and other questionnaires and clinical rating measures of anxiety, depression, and stress (Brown et al., 1997).

There is some overlap between DASS and POMS (described next). They both include a depression subscale. However, DASS specifically focuses on three factors that are thought to play a major role in insomnia, namely, depression, anxiety and stress (see section 1.5.7), where as The POMS does not have specific stress and anxiety subscales but rather a more general tension subscale.

3.4.2.6 The Profile of Mood States (POMS)

The POMS is a psychological test designed to measure a person's transitory mood/emotional states. POMS has been used in more than 5000 studies in a wide range of research domains, demonstrating wide user acceptance (McNair et al, 2003as cited in Bourgeois et al., 2010). Each POMS item includes an adjective used to describe a positive or negative mood state. Respondents are requested to rate each item using a five-point scale ranging from 0 (not at all) to 4 (extremely) according to the degree that they have experienced that mood state or emotion in the week preceding test administration. The items are then grouped into six subscales. Subscale scores are obtained by summing the numeric responses of the items comprising each subscale. The higher the score, the higher the level of disturbance for the particular subscale, except for the vigour subscale where the opposite applies. The six subscales are: Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigour-Activity, Fatigue-Inertia and Confusion-Bewilderment. The six subscales are then summed to yield a global mood

disturbance score. When summing the subscale scores, the Vigour-Activity score is negatively weighted (i.e., subtracted from the sum of the other subscales) and all others positively weighted (McNair et al., 1981).

A study (n=479) examined the reliability and validity of the POMS with a population of older adults and concluded that older adults adopted the same underlying mood constructs as younger adults when responding to the POMS (Gibson, 1997). The POMS was able to distinguish between normal subjects and those suffering mood disturbances with an excellent internal consistency and a very good test retest reliability for the POMS subscales. The study found strong support for the POMS subscales concurrent validity and concluded that the POMS is a reliable and valid measure of mood states in older adults, although a considerable shift of normative values for POMS across the age spectrum had been noted (Gibson,1997).

The original long form POMS consisted of 65 items. A short version SV-POMS, (sometimes also referred to as POMS-SF) with the same six subscales but only 37 items was later introduced (Shacham, 1983). Other abbreviated POMS versions have been introduced more recently, including a 30-item abbreviated version (EPOMS) introduced in1991 by the Educational and Industrial Testing Service (EDITS) and several others (Bourgeois et al., 2010). A recent study evaluated several abbreviated versions of POMS, including the EPOMS and SV-POMS and others in relation to the full-scale POMS by analysing combined data from six studies conducted over four years that had used the full-scale POMS (N = 915, 439 men and 476 women, age range 17 to 34 years). Results showed that the abbreviated versions of POMS have comparable, and in some cases superior reliability, compared to the full-scale POMS and that the abbreviated versions of POMS are valid and applicable where the full-scale POMS has

been proven valid and applicable. Furthermore, because shorter forms substantially reduce administration time and are comparable to the full-scale POMS, their use is recommended (Bourgeois et al., 2010). A study (n=83) found correlation coefficients between the POMS-SF and the original POMS scales were all above .95, indicating the POMS-SF was suitable for estimating the original mood scale scores in the study's population of cancer patients (Shacham, 1983). Another larger study (n= 600) using data of both clinical and healthy samples revealed that internal consistency estimates for the POMS–SF subscales were very similar to those for the original long form POMS. In addition, correlation values between the global score and subscale scores on the POMS–SF and those on the original long form POMS were all greater than .95 (Curran et al., 1995). A more recent study examined the internal consistency, validity, and factor structure of the POMS-SF with a sample of 428 cancer patients. It reported Cronbach's alphas ranging from 0.78 to 0.91 for each of the six POMS subscales and for the global score. This study also incorporated correlational analyses with other questionnaires including the CES-D, the self-rated Karnofsky, the MOS SF-20 physical functioning, and the Bradburn positive and negative affect scales, which have shown the convergent and discriminant validity of the POMS-SF. Confirmatory factor analysis further supported the mood factor interpretation of the POMS items in the POMS-SF (Baker et al., 2002). Based on these findings it was decided to use POMS-SF in the present study (see appendix 5.6).

A study of a Hebrew version of a 28 item POMS showed valid psychometric properties but concluded that further studies in various populations were needed (Netz, 2005).

There is some overlap between POMS and DASS as they both include a depression subscale. However, POMS also has additional useful subscales including anger, confusion, vigour and fatigue which DASS does not have. Therefore, the present study incorporated both POMS-SF and DASS.

3.4.2.7 The SF -36 health survey

The SF-36 (see appendix 5.7) is a multi-purpose, short-form self-reported health survey. A second version -SF36 Ver. 2.0 has been developed later (Ware, 2010). The SF-36 has become the tool of choice for measuring health status (Dexter et al., 1996). It is a generic measure that can be used across age, disease, or treatment group and has been proven useful in a wide range of general and specific populations, in comparing the relative burden of diseases, and differentiating health benefits from a wide range of interventions (Ware, 2010; Ware & Gandek, 1998; McHorney et al., 1994). Experience using SF-36 has been documented extensively in close to 1500 articles and reports including reports of close to 200 randomised controlled trials (Ware et al., p. 6, 2002). The SF36 consists of 36 items, the responses to which are summed to yield eight subscale-scores of functional health and wellbeing (physical functioning, role limitations due to physical problems, role limitations due to emotional problems, general mental health, general health perceptions, bodily pain, and vitality). These eight health-related concepts were selected from 40 concepts incorporated in the survey used in the Medical Outcomes Study (MOS) and thus enabled far fewer questions (Ware et al., 2000). The eight SF36 scales represent the most frequently measured concepts in commonly used health surveys and those most affected by disease and intervention (Ware et al., p. 6, 2002; Ware et al., 1995). Condition specific symptoms are not included in the SF-36 due to the fact that it is a generic measure (Ware, 2010). The eight scales' scores may also be grouped into two main categories; physical health and mental

health and summed accordingly to yield two category summary scores, physical health summary (PCS) and mental health summary (MCS) respectively. All subscale scores can be also be summed to yield a SF36 global health score (Ware, 2010). The present study relied mainly on the detailed POMS and DASS questionnaires for psychological measures and the SF36 was intended mainly to measure general health and well-being, physical function, physical role, and social function.

The SF-36 has undergone rigorous psychometric analysis both in the US and elsewhere to establish scale reliability, precision, and validity (Hays and Stewart 1990; McHorney et al., 1994; Ware & Gandek, 1998). With rare exceptions all SF-36 items have been found to correlate substantially with their respective hypothesized scales (Ware & Gandek, 1998). In the vast majority of published reliability estimates, SF-36 measures exceeded the minimum standard of 0.70 recommended for measures that are used in group comparisons. A summary of 15 studies showed that most measures exceeded 0.80 (Ware & Gandek, 1998). Reliability estimates for physical and mental summary scores exceed 0.90 in most studies. These trends in reliability for the SF-36 scales and summary measures have also been replicated across 24 patient groups of diverse socio-demographic characteristics and conditions (Ware & Gandek, 1998). The content validity of the SF-36 has been found comparable to that of other widely used generic health surveys. The eight SF-36 subscales were found to address the most frequently measured health concepts thus demonstrating content validity in both physical health and mental health measures (Ware & Gandek, 1998). Relative to the much longer MOS measures, that the SF-36 was designed to reproduce, the SF-36 scales have been found to have roughly 80–90% empirical validity for physical and mental health concepts. On the other hand, some of the long-form measures, that the SF-36 had been compared to, require five to ten times longer time to complete, implying

that the SF-36 provides a practical alternative to much longer measures for measuring physical or mental health status in group level comparisons (Ware & Gandek, 1998).

A validation study of a Hebrew version of the SF36 concluded that it provided an appropriate measure of general health status and that the translation into the Hebrew language did not diminish its qualities (Lewin-Epstein et al., 1998).

3.4.2.8 Daily Sleep and Practice Logs (DSPL)

A sleep log is a 24 hour recording of a person's sleep-wake pattern (Pollak et al., 2010, p. 221). Participants were asked to complete a daily sleep and practice log (DSPL) (see appendix 5.8). The DSPL was designed to collect self reported information about daily sleep and wake patterns, eating patterns, yoga practice patterns and usage of hypnotics and relaxants. The DSPL data was used in conjunction with class attendance records to calculate participants' global compliance to treatment. The DSPL allows collecting detailed daily information. However, it was observed that many participants considered it a significant inconvenience, which may have affected compliance and accuracy in filling it out. Furthermore, some participants commented that they couldn't remember exact sleep onset time and/or number and duration of awakenings. Others mentioned that trying to remember and note these details increased their stress levels and affected their sleep quality. In order to prevent DSPL data entry becoming a distracting and confounding factor, participants were asked to do the best they could and not to worry whether they would remember all the details in the morning. In order to avoid these confounding factors more emphasis was given in sleep quality analysis on pre- and post-intervention PSQI measures while the DSPL was used mainly for corroborating the PSQI data, monitoring and calculating practice compliance and

receiving timely feedback from participants about various issues relevant to management of the intervention.

3.4.2.9 Summary of subjective measures

Following is a summary of subjective measures obtained from above subjective instruments in the present study:

1. KSS – sleepiness (now) score
2. ESS – sleepiness (in various situations) score
3. PSQI – global score
4. PSQI – subjective sleep quality subscale score
5. PSQI – sleep latency subscale score
6. PSQI – sleep duration subscale score
7. PSQI – sleep efficiency subscale score
8. PSQI – sleep disturbances subscale score
9. PSQI – sleep medication subscale score
10. PSQI – daily dysfunction subscale score
11. MAP – derived apnea prediction score
12. DASS – global score
13. DASS – depression subscale score
14. DASS – anxiety subscale score
15. DASS – stress subscale score
16. POMS – global score
17. POMS – tension subscale score
18. POMS – depression subscale score
19. POMS – anger subscale score

20. POMS – fatigue subscale score
21. POMS – confusion subscale score
22. POMS – vigour subscale score
23. SF36 – global score
24. SF36 –physical function subscale score (PF)
25. SF36 – limitation in physical role subscale score (RP)
26. SF36 – body pain subscale score (BP)
27. SF36 – general health subscale score (GH)
28. SF36 – vitality subscale score (VT)
29. SF36 – social functioning subscale score (SF)
30. SF36 – limitation in emotional role subscale score (RE)
31. SF36 – mental health subscale score (MH)

3.4.3 Objective outcome measures

3.4.3.1 Introduction

In the present study portable sleep monitoring was used in conjunction with a computerised sleep diagnostic system. Portable monitoring allowed sleep studies to be conducted in participants' natural home environment and facilitated conducting pre- and post-intervention measures in a short period of time, because there was no reliance on the availability of sleep clinic laboratory facilities. By using four mobile sleep labs concurrently it was possible to conduct 20 to 24 sleep studies per week. Using a computerised sleep diagnostic system operated by a sleep scientist to analyse the data allowed consistent and timely derivation of all polysomnography variables required to examine pre- to post-intervention change in main sleep measures. With regards to OSA, diagnosis of existence and severity level of OSA was done by a sleep physician, after

reviewing sleep study analysis results and medical data and then interviewing the participant and conducting a physical examination.

3.4.3.2 The Embletta portable monitoring system

The present study used the Embletta mobile sleep recording system, produced by Embla systems, Denver CO, USA (Embla Systems, 2010). The Embletta is fully compliant with US Centres for Medicare & Medicaid Services (*CMS*) and the American Academy of Sleep Medicine (AASM) recommendations for portable monitoring (Embla Systems, 2010). It has been used in over half million sleep studies worldwide, and has been chosen by the American Sleep Medicine Foundation (ASMF) for use in their study on portable monitoring in the diagnosis and management of OSA (Embla Systems, 2010). The Embletta is battery powered with an internal memory that is able to store the amount of data generated during a full sleep study. It is easy to use, light weight and compact, and attaches to a belt system with the wiring connecting the sensors to the Embletta directly, allowing participants to independently get out of bed during the night, and go to the toilet, if required, without having to disconnect any of the wires or sensors (Embla Systems, 2010). In the present study the Embletta was used to record ECG, pulse oximetry and body position/actigraphy data. The data was downloaded in the morning to a central computer system for analysis and diagnosis by a sleep scientist and a sleep physician.

3.4.3.3 The HC1000P sleep diagnosis system

In the present study the HC1000P sleep analysis system, produced by Hypnocore, Yehud, Israel (Hypnocore, 2010) was used to assist in analysing the data recorded by the Embletta (see above). The HC1000P provides full analysis of sleep architecture, movements, body position changes, respiratory disturbances, and oxygen saturation as

well as information on Autonomic Nervous System (ANS) function and balance, thus providing all information related to insomnia and OSA. The HC1000P performs these tasks by analysing continuous ECG and pulse oxygen saturation data, acquired using traditional PSG or portable monitoring, from a sleeping patient (Hypnocore, 2010), thus simplifying sleep diagnostic process by eliminating the need to record multiple physiological variables (Hypnocore, 2010). The HC1000P performs Heart Rate Variability (HRV) analysis by time-dependant spectral analysis of instantaneous Inter Beat Interval (IBI) of the ECG signal using various algorithms based on numerous scientific studies (Moody et al., 1985; Keselbrener et al., 1996; Ehrhart et al., 2000; Shinar et al., 2000; Shinar et al., 2001; Shinar et al., 2003; Shinar et al., 2006; Shinar et al., 2003; Shinar et al., 1999; Baharav et al., 1995; Baharav et al., 2001; Baharav et al., 2004; Baharav et al., 2009; Dorfman-Furman, 2005) (see section 1.3.3). The ECG is considered the best and most robust signal for deriving the HRV data (Chatlapalli, 2004; Piotrowski, & Rozanowski, 2010; Hypnocore, 2010). This presents a substantial advantage when conducting unmonitored sleep studies in the home environment as the occurrence of poor quality ECG or pulse oximetry signals is relatively low compared to other signals, such as EEG. The interpretation of the results provided by the HC1000P is performed by a sleep scientist and supplemented with clinical information obtained from the patient by a qualified sleep physician. Analysis and diagnosis is labour and time efficient compared to traditional PSG (Hypnocore, 2010). This allows a faster, cost-effective, reliable and consistent diagnosis with multiple patients within a short time period thus providing significant advantage in research settings such as the present study where a substantial number of sleep studies needed to be performed within a short period of time and then analysed reliably and consistently. Furthermore, this technology was capable of providing all the necessary data for the present study where main

emphasis was on the pre- to post-intervention changes in main sleep features and main insomnia and OSA measures.

The HC1000P system has been validated extensively and approved by both the Federal Drug Administration (FDA) and the Conformité Européene (CE) approval processes for medical technologies (CE, 1993; FDA, 2008a; FDA, 2008b). Its output was found essentially equivalent to traditional PSG results. This was statistically confirmed via retrospective analysis of a large number of cases from a clinical study (FDA, 2008; Hypnocore, 2010). A multicentre retrospective validation study used randomly selected, standard PSG recordings from a cohort Chronic Fatigue Syndrome study at the CDC and from consecutive patients referred for PSG at Share Zedek Medical Center in Jerusalem (Cahan et al., 2008). The PSG data had been manually scored according to Rechtschaffen & Kales and AASM criteria. The ECG and pulse oximetry signals of this PSG data were separately and blindly analysed by the HC1000P system. No significant differences were found between sleep architecture and RDI derived using manual PSG analysis and the HC1000P analysis. Specifically no significant differences were found between ECG and PSG derived values of TST (391.5+62.8 vs. 387.7 ± 31.6 minutes), SE (84.6% ± 10.4% vs. 82.7% ± 4.5%), wake (54.3 ± 39.5 vs. 58.5 ± 19 minutes), NREM (322.1 ± 52.5 vs. 319.0 ± 29.8 minutes), and REM sleep (72.9 ± 33.8 vs. 68.7 ± 25.2 minutes). There was also a strong correlation (correlation coefficient of R=0.92) between RDI values derived using the HC1000P and using standard manual analysis with a 98.0% match-up. Cohen's Kappa was 0.96, and both specificity and sensitivity were 0.98 (Cahan et al., 2008).

The HC1000P system has the following limitations:

1. It is not suitable for measuring patients with arrhythmia and pacemakers.

2. Diagnostic capabilities are limited to the two most common sleep disorders; insomnia and OSA.
3. HC1000P uses direct measurement of ECG and pulse oximetry and indirect measurement of all other traditional channels, by extracting the data mathematically from the ECG. In traditional PSG all signals (ECG, EEG, EMG, EOG, oral and nasal airflow) are measured directly (Hypnocore, 2010).

As discussed above these limitations were not considered to be significant in the present study, which focused on examining pre- to post-intervention changes in main sleep features, and was mainly interested in changes in measures related to insomnia and co-morbid OSA.

3.4.3.4 Sleep data recording quality control

Since portable monitoring was conducted unsupervised in participants' homes, great care was taken while setting up the equipment and connecting the leads and sensors in order to prevent potential technical faults and subsequent loss of data. Recording signal quality was verified using special software when downloading the data to the central computer the following morning. Only good quality recordings were accepted. If a technical fault had occurred during the night (e.g. loose leads, detached sensors etc.) or a poor signal quality insufficient for performing a reliable sleep analysis was detected, the recording was conducted again on one of the following nights. Due to limited resources, only one additional measurement was allowed per person per milestone. If the second measurement also failed to yielded good quality signal, the data was excluded from the analysis. In some cases data quality allowed reliable derivation of most but not all measures. In such cases only measures that could be derived reliably were included.

3.4.3.5 Summary of objective variables

Sleep was measured using the Embletta device and the subsequent analysis of the sleep data was done using the HC1000P system. This process resulted in an output of a large number of variables. However, for the purpose of the current study only the variables listed below were considered essential and subsequently included in the statistical analyses. The variables derived and scored using the HC1000P program were also further validated by a sleep technician who was independent from the study and later review by a sleep physician.

The following variables were used as objective measures in statistical analysis:

1. **Sleep onset latency (SOL)** - time period measured from bedtime to the beginning of first stage of sleep, either REM or non-REM sleep (Pollak et al., 2010, p. 221).
2. **Total sleep time (TST)** - The total amount of actual sleep time in a sleep period equalling to the total sleep period minus the wake time period. Total sleep time consists of the total of all REMs and non-REMs in a sleep period (Pollak et al., 2010, p. 251).
3. **Total time in bed (TTB) –** total period spent in bed including wake and sleep periods
4. **Wake after sleep onset (WASO) -** total time that is scored as a wakefulness state in a PSG recording occurring between sleep onset time and the final wake-up time (Pollak et al., 2010, p. 261).
5. **Light sleep duration** – total durations of all non SWS NREM. Equivalent to the total of stage one and stage two NREM sleep.
6. **SWS duration** – total durations of all SWS stage. Typically comprises sleep stages NREM 3 and NREM 4 combined together (Pollak et al., 2010, p. 235).

7. **REM sleep duration**– total durations of REM sleep stage.
8. **Slow Wave Sleep (SWS) latency -** time period measured from sleep onset to the first appearance of SWS.
9. **REM latency -** time period measured from sleep onset to the first appearance of stage REM sleep (Pollak et al., 2010, p. 192).
10. **Oxygen saturation (SPO2)** –indirect measurement using infrared sensor reflecting oxygen saturation level in the blood (Pollak et al., 2010, p. 166).
11. **Respiratory Disturbance Index (RDI) –** mean number of respiratory events that disturb sleep per hour (Pollak et al., 2010, p. 193).
12. **Oxygen Desaturation Index (ODI3) –** mean number of blood oxygen desaturations >3% per hour
13. **Sleep efficiency (SE) –** is the ratio of the amount of actual sleep to amount of time available for sleep during the sleep episode (Pollak et al., 2010, p. 217) calculated by dividing the total sleep time (TST) by total time in bed (TTB)

3.5 Data analysis

3.5.1 Comparators

As discussed in section 3.1 the study design was constrained by the limited resources available. This resulted in a mixed experimental design combining, an initial WLC design component with a better quality control, and a subsequent expanded design component involving a larger number of participants and therefore with a greater statistical power and arguably, combining the two designs, with WLC participants serving as control in both designs, provided an optimal design within the context of the external constraints and limitations described above. Regular contacts were made with WLC participant by phone on a fortnightly basis.

In the course of the WLC phase 5 of 31 participants had dropped out. At the end of the WLC phase, 26 participants were available for post-WLC-phase measures. After taking the post-WLC -phase measures all WLC completers were contacted and offered to participate in a yoga group (YI). 16 participants accepted the offer and joined a subsequent YI and 10 participants declined the offer.

The wait list control group (WLC) served a dual purpose. First, WLC served as a control group for all participants in yoga intervention (YI) (see section 3.1.5). In addition, the 16 WLC completers that accepted the offer to join a subsequent YI phase were found to be a good matching control to themselves and were used in an additional analysis (see sections 3.1.5 & 3.5.2).

All applicants wishing to participate in the study were told in advance they would be assigned to either a control group or an intervention group. This information was also stated in the consent form, which they were requested to sign when joining the study. The 'waiting list control' period was 12 weeks which was identical to the 12 week intervention period. The measures for the control and intervention groups were identical. These included subjective measures (self reported questionnaires) and objective measures (sleep studies) taken at baseline – pre-control/intervention phase and at post- control/intervention phase and daily sleep logs.

Both intervention and control groups were asked to continue their daily routine normally. For example, if a participant had a habit of walking his/her dog twice a day, going swimming twice a week, going to a concert once a week, playing bridge once a week, going to social functions etc. then he/she was asked to continue doing so. However all participants, both intervention and control, were asked not to start any new activity whether physical, mental, spiritual or social during the study period. The rationale behind

these guidelines was that any change in a person's routine, either by adding a new regular activity or by suspending a regular activity would be a confounding factor.

3.5.2 Statistical strategies and methods

This section describes statistical strategies and methods that were used to analyse the data derived from subjective and objective measures. The most common strategy for analysing treatment efficacy in similar studies is the 'intention to treat' (ITT) analysis (also called "intent to treat"). ITT is based on initial treatment intent. It yields information regarding potential effects of an intervention using data from all participants assignd to a particular group, whether or not they actually completed or even received the treatment. The ITT strategy is intended to avoid bias, which may be caused by confounding artifacts such as compliance (Chatburn, 2011, p. 325). For example, participants with a more serious health disorder tend to drop out at a higher rate, and therefore using only completers' data may distort the overall effect of the intervention. Furthermore, using only completers' data may degrade randomisation (Lachin, 2000). However, ITT strategy may be applied most effectively only if there is complete outcome data for all randomised participants including dropouts (Chene, 1993; Lachin, 2000) as dropping subjects with missing outcomes and ignoring compliance information can be biased for the intention-to-treat effect (Frangakis et al., 1999). A contrasting form of analysis is the 'per-protocol (PP) analysis' also called 'on treatment' (OT) analysis'. It is based on treatment actually administered and the potential effects of a specific intervention under specific conditions. In some cases it is used to avoid the dilution of the treatment effect (Chene, 1993; Lachin, 2000). In 'Efficacy subset analysis' a subset (or subsets) of the patients who received the treatment of interest regardless of initial randomisation, is selected; some subjects and observable subject data may be excluded from the analysis based on information obtained post-randomisation. However, this may introduce a potential bias, such as the inflation in

Type I Error, even when the null hypothesis is true (Lachin, 2000). Subset analyses is often associated with problems of multiplicity and limitations in numbers of patients studied (Simon, 1982).

In the present study both ITT and OT approaches were used in order to reduce bias and confounding artefacts on one hand, and on the other hand obtain a fair representation of the intervention's effect on study outcomes by avoiding dilution of the treatment effect that may result from significant variability in compliance. For both analyses strategies, mixed analysis of variance (ANOVA) and multivariate analysis of variance (MANOVA) methods were used. The between subjects factor was group and the within subjects factor was time (Pre-intervention versus post-intervention). In ITT analyses the groups were Intervention (YI) versus control. In OT analyses the groups were high compliance intervention participants (YHC) versus low compliance intervention participants (YLC) versus control participants.

A third supplementary analysis strategy was used to support the findings of the two main analyses above. The third method involved using a matching technique which can be an effective technique to equate groups where randomisation is not possible, provided adequate information is available to enable good matching (Christensen et al., 2011, p. 207) (see section 3.1.5). In this case high quality matching was obtained by matching a group of subjects to themselves with the 16 WLC phase completers that joined subsequent YI phase being matched to *themselves*. The ensuing control and intervention groups in this third analysis were smaller (n=16) resulting in reduced power. However the quality of the control was higher than in the two main analyses. A paired samples t test was used to compare the mean measure values of pre- and post WLC phase measures to the post YI measure values.

Correlational analyses were also used to examine the relationship between the degree of co-morbid OSA and pre- to post-intervention changes in the dependent variables. This was done in order to identify measures that may have been affected by the presence of co-morbid OSA component and to determine the direction and extent of the potentially confounding co-morbid OSA component.

In the majority of participants a high degree of conformity was found between the DSPL and responses to PSQI items. As described above more emphasis was given in sleep quality analysis to pre- to post-intervention change in PSQI measures while the DSPL was used mainly for corroborating PSQI data, deriving practice compliance scores, and receiving timely feedback from participants about various issues relevant to management of the intervention. Therefore, the PSQI scores were used for statistical analysis of changes in sleep quality.

3.5.3 The compliance factor

Defining and measuring compliance is more complex with a yoga intervention than in a typical medical intervention that requires taking medication or undergoing a medical procedure. In both yoga and medical interventions participants may be required to remember to follow a set of instructions, take certain actions, attend intervention sessions when required etc. However, in the present study the yoga intervention included a substantial unsupervised home self-practice component that required comprehension of instructions, concentration, patience, perseverance and mental acceptance of the process and its challenges. These factors may vary significantly from individual to individual, are difficult to measure, and yet may have a significant impact on timing, frequency, duration and quality of practice. Furthermore, home environments may also vary and affect these variables. To reduce variability all participants were

provided with a pre-recorded audio CD, containing all necessary instructions and guided meditation sequences. The CD was designed to increase uniformity in duration and quality of practice by simulating a teacher-led guided meditative yoga session. However, the audio CD on its own may not have a significant effect on choice of timing and frequency of practice. Recommendations were made regarding optimal and minimal level of practice but participants' personal choice determined to what extent to comply with these recommendations. Also, since there were three guided meditation exercises on the CD, a participant may have chosen to perform one, two, three or more exercises per session, which would result in practice duration varying further. Duration may also have been affected, if for some reason, a participant while practicing at home decided to stop practicing before reaching the end of a particular exercise.

In the present study analysis of class attendance records and self-reported daily sleep and practice logs (DSPL), revealed considerable variability in both home-based practice compliance and to a lesser extent in class attendance (see results section 4.3). Variability in compliance implies that participants are getting the intervention in varying 'dosages'. In randomised control trials (RCTs) poor participant compliance may result in the reporting of 'false negative' results and erroneous conclusions (Kehoe et al., 2008). These implications underscore the importance of taking compliance into account in the analyses.

Measuring compliance was affected by the limitations described above. It was decided to rely only on the number of 'practice units' performed and to differentiate between two levels of compliance only (low and high). Due to the relatively small overall participant number, using three levels of compliance (i.e., - low, medium, high) or more, would have resulted in reduced group size and reduced statistical power. The

number of practice units was derived from self-reported daily sleep and practice logs (DSPL) in conjunction with class attendance records kept for all yoga classes as follows:

1. Two levels of compliance were defined (low and high).
2. A single home-based meditative exercise was scored as one practice unit (an average duration of 25 to 30 minutes of preparation plus practice time.
3. A single yoga class was also scored as one practice unit (actual practice duration equivalent to total class time *minus* time taken for introductions, explanations, instructions, questions and answers, conclusions, administrative issues etc. – roughly 25 to 35 minutes. (See section 3.3).
4. Daily score was calculated by summing up scores for all home and class practice units performed on that day
5. Global personal compliance score was calculated by summing up all daily scores and dividing by the number of intervention days.
6. Participants below median compliance score were classified as 'low compliance' participants (YLC). Participants equal to or above the median were classified 'high compliance' participants (YHC). This delineation resulted in three groups:YLC (N=29), YHC (N=30) and wait list control - WLC (N=31) of similar size for most measures (as group size may vary from measure to measure due to omissions in completing subjective measures by some participants or due to poor/variable signal quality in individual overnight sleep studies).

There is a question whether high and low compliance participants may have different psychological characteristics that may affect subjective perception of intervention outcomes. The term "demand characteristics" which was originated by Orne (1959 refers to the totality of cues and mutual role expectations inherent in a

psychological experiment or therapy that influence behaviour and/or self-reported outcomes. According to Orne (Orne, 1959, pp. 469-470), the behavioural impact of "demand characteristics" will vary with the extent to which they are perceived, and also with the motivation and ability of the participant to comply. Applying this theory to a yoga intervention (or any other 'self help' method) one may argue that the same psychological characteristics contributing to higher yoga practice compliance may also lead to a different perception of yoga intervention outcomes. However, this theory is yet to be studied specifically for yoga or similar interventions. Furthermore, variability in compliance may also be affected by other factors, such as individual, family, social and environmental circumstances. In other words, personal traits are only one of various factors which contribute to participant's compliance level. As an example, two female participants had noticeable fluctuations in their compliance levels during the present study With both noting in their DSPL that their respective husbands' deteriorating health and hospitalisation was the cause for their decrease in home practice compliance. Another example was that some participants used private transportation to get to classes while others used public transportation. Public transportation users' class attendance was affected more frequently by bad weather.

3.5.4 The OSA factor

A multi stage screening process designed to include participants presenting with complaints of insomnia and exclude various medical and/or psychiatric conditions which may affect sleep was used. Nevertheless, despite this process, 69% of participants were diagnosed post study, as having some level of co-morbid obstructive sleep apnea (OSA) (see section 4.6). Presence of co-morbid OSA is a potential confounding factor in the present study, since both OSA and insomnia have both been shown to be associated with reduced sleep quality and diminished mental health, physical health and

quality of life in general., In order to determine whether and to what extent the presence of OSA may have had a confounding effect, further analysis was conducted as follows:

1. The changes pre- to post-intervention in measures' scores were calculated and defined as new variables.
2. Pearson's correlational analyses were used to determine whether there was an association between co-morbid OSA presence or degree and these new variables (see section 4.6).
3. Pearson's Chi Square tests were used to determine whether there was an association between co-morbid OSA presence or degree and compliance level (see section 4.6).
4. In order to find out why MAP apnea prediction score had not been effective in predicting OSA in the study population, Pearson's Chi Square tests were used to determine whether there was an association between co-morbid OSA presence or degree and between MAP derived apnea predictions score (see section 4.6)

3.5.5 Handling missing data

All dropouts were contacted but regrettably declined to make themselves available for post intervention (or post control measures) and were excluded from the analysis.

In some cases, objective measures could not be derived reliably from recording data due to poor signal quality and a second data recording was conducted within the next few days. Limited resources allowed conducting only one additional recording per subject per milestone (see section 3.4.3.5). In several other cases data quality allowed deriving most but not all objective measures. OSA diagnosis was only made based on adequate and reliable data.

Great care was taken to ensure complete filling out of self reported questionnaires. Nevertheless, in some cases, it was later revealed that a subject had failed to respond to a certain questionnaire item. As described in section 3.4.2 subjective measures are calculated based on questionnaire items and consequently missing reposes to questionnaire items may have prevented calculating scores for specific subjective measures in some cases.

For ANOVA analyses, cases with missing measure scores were excluded from any specific single design in which the missing value had occurred while correlations were computed using pair wise deletion of missing data based on the number of pairs with non-missing data (UCLA Statistics Consulting Group, 2011).

As described in section 4.2, a total of 74 subjects were included in the study. 31 were assigned to WLC with five dropping out and 26 remaining (31-5=26). 16 WLC completers agreed to participate a in subsequent YI with all 16 completing the YI. The additional 43 subjects recruited to the study were assigned directly to YI with two dropping out and 57 remaining (16+43-2=57). For every single measure without any data losses there could be a maximum of 83 data 'cases' (26+57=83). Due to reasons explained above, the actual number of 'cases' fluctuated with the mean number of 'cases' per subjective measure being 73 (88%) and per objective measure being 70 (84%).

3.5.6 Testing for assumptions underlying parametric testing

The subjective and objective dependent variables measured pre- and post-intervention were subjected to the tests of the assumptions underlying ANOVA and MANOVA. To test the assumptions of normality all data were subjected to visual inspections of frequency distributions and also Kolmogorov-Smirnov and Shapiro-

Wilks tests of normality. Given the sample size, emphasis was placed on visual inspection of the stem and leaf plot and histograms over the inferential tests of the assumption. Although several of the variables exhibited some degree of skew, none of these deviations was considered large enough to warrant transformation. Homogeneity of variance was tested using Levene's test. Where violation of homogeneity of variance assumption was identified, the suggested power transformation was applied to pre- and post-interventions measures of the variable. The transformed variables were again tested for homogeneity. Where the recommended transformation did not result in acceptable homogeneity, another transformation was carried out. Table 3.3 lists the variables that showed a violation of homogeneity of variance and with the recommended power figure for transformation or other successful transformation:

Table 3.3

Variables which violated homogeneity of variance and suggested power transformation

Variable	**Recommended/ suitable power transformation**	**var. type**
KSS score	None found	subjective
PSQI medication subscale score	None found	subjective
POMS global score	None found	subjective
POMS depression score	(-2) power transfor. of (POMS depression +100)	subjective
SF36 social function score	None found	subjective
SF36 emotional role score	None found	subjective
Number of position changes	(-.36) power transformation	objective
Number of awakenings	(-1) power transformation	objective
RDI	(-1.939) power transformation	objective
SWS latency	(-1) power transformation of (SWS latency +1)	objective
SWS percentage	None found	objective
Light sleep percentage	None found	objective

3.6 Procedure and study milestones

1. Research proposal was submitted and approved at RMIT University.
2. Ethics committee approval at RMIT University.
3. Ethics ('Helsinki') committee approval at Shaare Zedek Medical Centre.
4. Advertisement campaign in community papers, bulletin boards, medical centres etc.
5. Initial screening phase: telephone interviews.
6. Mail-out of detailed information about the study and medical forms and questionnaires to candidates and their personal physicians.
7. Second screening phase: detailed review of medical forms and history/records received from applicants' personal physicians.
8. Candidates fill out detailed questionnaires and medical forms at the medical centre.
9. Third screening phase: review of detailed questionnaires and medical forms.
10. Fourth screening phase: Structured examination by a sleep & respiratory physician at the medical centre.
11. Taking pre-intervention measures of the WLC group.
12. Start of WLC phase – 12 weeks.
13. Additional participants screened and assigned over several months in line with resources availability.
14. Pre intervention measures taken – for all additional participants.
15. All additional participants assigned to yoga intervention (YI).
16. Post intervention measures taken from WLC complete after12 weeks.
17. All WLC completers offered to enrol in (YI)..
18. WLC completers assigned to a 12 week (YI)..
19. Post intervention measures taken from (YI) participants upon completion of 12 week YI phase.

20. Completion of analysis of all sleep studies.
21. All participants invited to attend post study medical appointments at the medical centre.
22. Post study medical reports sent to participants' personal physicians .

Chapter 4. Results

4.1 Introduction

The present study hypothesised that an integrated yoga intervention would significantly improve sleep quality and quality of life (QoL) in elderly people presenting with complaints of insomnia. It also hypothesised that an integrated yoga intervention would be safe and acceptable for elderly people in a western cultural setting. This chapter contains a summary of the results including study demographic data; results related to acceptance and compliance, QoL, sleep quality; findings on prevalence and severity of OSA among participants and the impact OSA had on measures in present study. The significance and implications of the results are discussed in depth in the discussion chapter. Most of the results are reported in concise tabular format. Appendix 11 contains a more detailed reporting format. Please refer to it if needed.

4.2 Study demographics

4.2.1 Introduction

This section includes the present study's demographic data from the recruitment phase, through screening, pre- and post- intervention measures and data analysis phases. It also reports on dropouts and the reasons given for dropping out.

4.2.2 Study flow demographics

1. **First screening phase**: 458 phone interviews were conducted with people who responded to the advertisement campaign.
2. Based on phone interviews results, 247 letters were sent out to applicants who passed initial screening and agreed to have the forms mailed to them. Mail out

included detailed information about the study, a medical form and an approval form to be filled out and signed by applicant's personal physician.

3. **Second screening phase**: Of the 247 forms mailed out -126 were received back and reviewed. 21 applicants were excluded based on review of the medical forms. Invitation letters to attend a third screening phase at the medical centre were sent to 105 responders who passed second screening phase.
4. **Third screening phase**: 105 people filled out detailed questionnaires and were interviewed at the medical centre. After reviewing the questionnaires, 10 people were excluded and 95 who fit inclusion/exclusion criteria were given an appointment with a sleep physician at the Shaare Zedek medical centre
5. **Fourth screening phase:** 95 people attended an appointment with a sleep physician at the Shaare Zedek medical centre. 16 were excluded based on exclusion/inclusion criteria and five declined to sign the consent forms and thus could not be admitted to the study. In total - 74 participants were included in the study.
6. The first 31 participants to be admitted to the study were assigned to a waiting list control (WLC).
7. 26 participants completed the WLC phase
8. 16 WLC completers accepted an offer to continue to a subsequent yoga intervention (YI) phase while 10 WLC completers declined the offer.
9. Additional participants admitted over the following months (n= 43) were all assigned to YI
10. In total 458 applicants were processed, 232 did not meet inclusion criteria, 152 decline to join, 74 joined the study and 67 completed the study.

The study flowchart in Figure 4.1 below illustrates study progression, including, number of applicants, participants, dropouts and completers in control and intervention during all phases of the study.

Please go to next page →

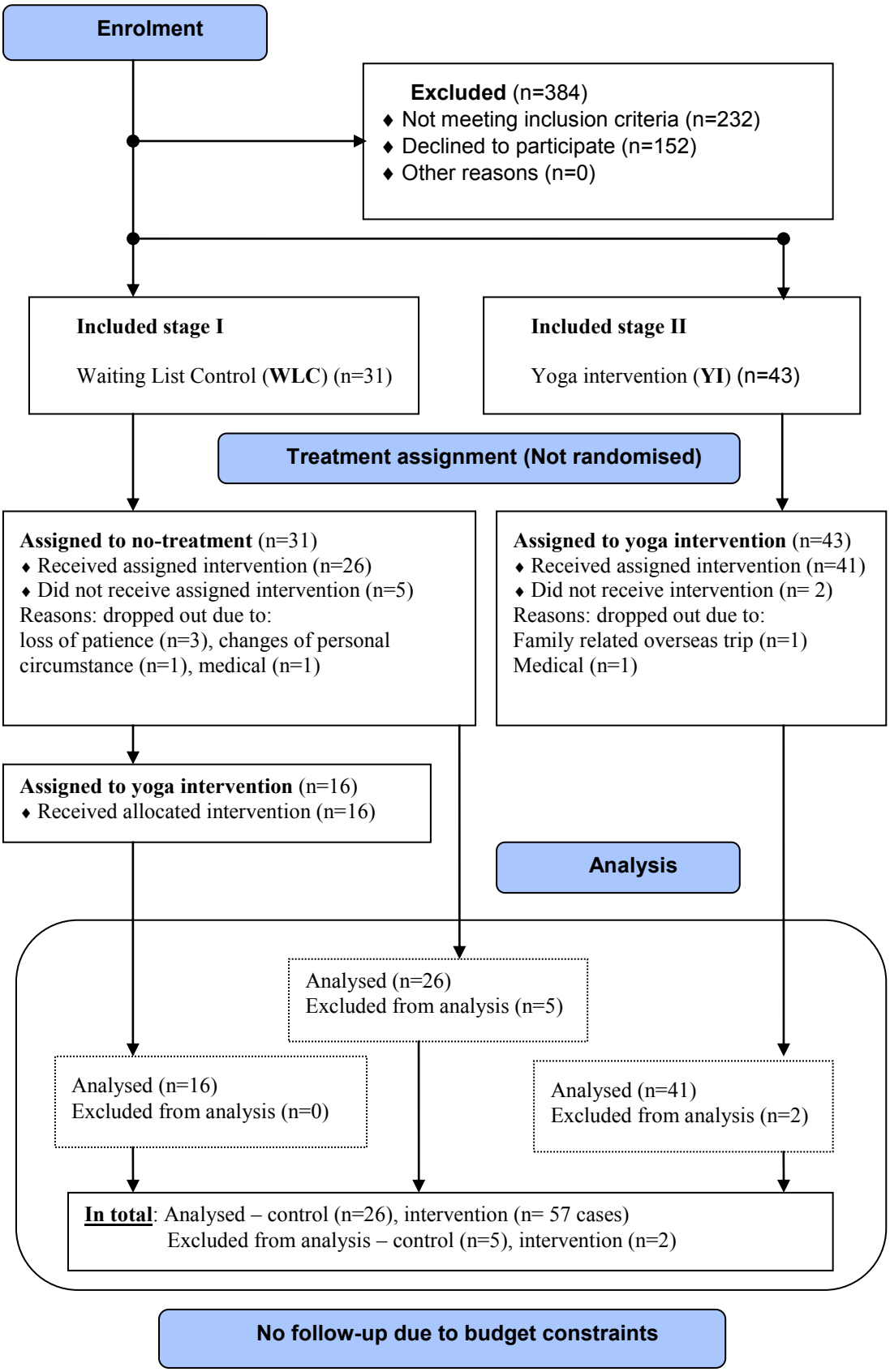

Figure 4.1 *Study flowchart*

4.2.3 Demographics characteristics of participants

1. **Age** - participants' ages ranged from 60 to 87, (M = 74.4, SD = 7.1)
2. **Gender** - 81% of participants were women and 19% men
3. **Marital status** - 45% of participants were married, 55% widowed, divorced or single.
4. **Housing status** - 60% living within the general community, 40% living in independent retirement housing
5. **Ethnic and religious background** – for ethical and privacy reasons, information regarding ethnicity, religion, culture, political views or political affiliations was not collected, *however*, observations made during the interview process revealed that participants *w*ere from a wide range of ethnic backgrounds (including Jewish 'Ashkenazi' and 'Sephardic', European and middle eastern) and religious backgrounds (secular, traditional, conservative, and modern orthodox), country of birth (Israeli born, and immigrants from Middle Eastern countries, Eastern Europe, Russia, Western Europe, North America and South America). At least three participants were holocaust survivors.
6. **Locality** - Approximately 70% of participants lived within 7 Km radius of the medical centre and 30% in other locations throughout the greater Jerusalem metropolitan area. Practice venues and medical centre were easily accessible by public and private transport with ample parking facilities available. However, venue location may have influenced some candidates' final decision whether to participate or not.

Table 4.1 below demonstrates a high degree of similarity of demographics characteristics between the control and intervention groups.

Table 4.1

Demographic characteristics of control and intervention groups

	group	control	intervention
Age		M=71.26 , SD=6.77	M=74.66 , SD=7.39
Gender	**male**	84%	81%
	female	16%	19%
Marital status	**married**	52%	45%
	single / widowed	48%	55%
Number		N=31	N=74

4.2.4 Participants, completers and dropouts

Table 4.2 below includes numbers of participants, dropouts, and completers in the waiting list control (WLC) group, intervention (YI) group, and in total. 'Total participants' refers to all applicants who passed the screening process and were assigned to a group (WLC or YI). 'Completers' refers to those who did not drop out and underwent both pre- and post- intervention or control phase measures.

Table 4.2

Participants, dropouts, and completers

Category	Started	completed	Dropouts	Dropout rate
Total Yoga intervention (YI)	59	57	2	3.4 %
Waiting list control (WLC)	31	26	5	16.1 %
Control completers assigned to intervention.	16	16	0	0%
Total participants	74	67	7	9.5 %

4.2.5 Dropouts and reasons given

As shown in table 4.2 above there were a total of seven mid phase dropouts; five were from the WLC group and two from the YI group.

The reasons for dropping out of the intervention group were: (1) Medical reasons: One subject complained of dizziness and vertigo at home (not during yoga practice) and was removed from the study and referred to detailed medical examinations that revealed BP fluctuations and irregular consumption of related medication. (2) Family reasons: One subject was forced to travel overseas due to unforeseen family circumstances. The reasons for dropping out of WLC were: (1) Medical reasons: One subject dropped out due to self reported deterioration in health status ('not feeling well') (2) Personal reasons: Three subjects dropped out due to 'running out of patience' or 'losing interest in the study' (3) Family reasons: One subject dropped out due to change of personal circumstances related to a close family member. None of the dropouts were able and/or willing to attend post- phase measures. Numbers of dropouts are summarised in table 4.3 below.

Table 4.3
Numbers of dropouts and reasons given

Reason given	**Total**	**WLC**	**YI**
Medical	2	1	1
Personal	3	3	
Family related issue	2	1	1
Total	7	5	2

4.2.6 A note on WLC subsequent assignment to YI

As described and illustrated above, 31 participants were assigned to the WLC group. Of these - 5 (16%) dropped out during the WLC phase. The remaining 26 (84%) completed their 'post control' measures and were then contacted and offered to participate in a yoga intervention (YI) group. 16 of the 26 WLC completers (62%) accepted the offer and were assigned to YI. The remaining 10 who completed the control phase (38%) declined the offer. The reasons given for declining to continue to YI were: (1) inconvenient timing of yoga intervention classes (five participants), (2) changing personal /family circumstances (three participants), (3) and loss of patience or interest (two participants).

4.3 Safety and acceptance of yoga practice

Hypothesis three, which stated that "*an integrated yoga intervention will be safe and acceptable for elderly people with complaints of insomnia living in a westernised cultural setting*", was tested using the present study's demographics, compliance and adverse reports data. Yoga as an intervention for geriatric insomnia generated much interest within the target community. As reported above, the response to a low budget and very modest advertisement campaign was substantial as 458 enquiries were received within several months.. The control group had a much higher dropout rate (16.1%) than the intervention group (3.4%) (See tables 4.1 and 4.2). Participants were encouraged to practice a total of three practice units per day (equivalent of 60 – 75 minutes net practice including class net practice and/or self practice), for seven days a week, but in case this was not possible, then they were advised to complete a minimum of one practice unit per day (see section 3.3.6). Class attendance, expressed in percent of classes attended, was: Mdn=72.0 %, M=63.7%, SD=30.6% and global compliance score (incorporating class and home practice), expressed in practice units per day was: Mdn=.96, M=1.34, SD=1.54. In other words, median

compliance was close to the requested minimum of one practice unit per day but only 10% of participants practice compliance level was equal to or greater than the recommended three practice units per day.

Overall, adherence to instructions during yoga classes was good with the graded yoga poses variations enabling most participants to follow instructions and complete the full sequence in most classes. A small number of reported events where participants decided to abstain from a particular exercise occurred mostly during the first two weeks of the intervention period.

As reported above there were two dropouts from the intervention group both unrelated to the yoga intervention. One participant dropped out due to spells of dizziness and vertigo (not during yoga classes) and further medical examinations revealed BP fluctuations and irregular use of related medications. The other participant dropped out due to unforeseen personal/family circumstances. No other adverse effects were reported throughout the study by participants, yoga teachers, personal physicians or study physicians. (See tables 4.1 and 4.2)

Overall, the study's adverse event data, demographic data and compliance data reported above shows that yoga intervention *as applied in present study* is a safe, well accepted and applicable intervention for elderly people with complaints of insomnia living in a westernised cultural setting. However, data also shows that compliance in general and home practice compliance in particular was considerably lower than what had been recommended to participants. In other words despite good acceptance of yoga, most participants found compliance with self-practice at home - challenging.

4.4 Quality of life results

4.4.1 Quality of life results – overview

Hypothesis two, which stated that: "*an integrated yoga intervention will significantly improve measures of both mood and quality of life (QoL) in elderly people presenting with complaints of insomnia"* was tested using the POMS DASS and SF36 instruments.

Overall, significant improvements ($p<.05$) were found in many QoL measures with ITT ANOVA analysis in the yoga intervention (YI) group but not in the control group, and with OT ANOVA analysis in the YHC but not in YLC group or control. Specifically, significant improvements ($p<.05$) were found in the YI group but not in control group in the global scores of the DASS ($p=.010$), POMS ($p=.09$) and SF36 ($p=.008$), and also in the following mental and physical health subscales: depression ($p=.019$), stress ($p=.020$), fatigue ($p=.010$), emotional role function ($p=.043$), physical role function ($p=.035$), vitality ($p=.053$) and also in social function ($p=.030$). Furthermore, significant improvements ($p<.05$) were found in the YHC group but not in YLC group in the global scores of the DASS ($p=.002$), POMS ($p=.09$) and SF36 ($p=.008$), and also in the following mental and physical health subscales: depression ($p=.003$), stress ($p=.008$), anxiety ($p=.011$), tension ($p=.044$), anger ($p=.005$) and also in social function ($p=.001$). Significant deterioration was seen in the control only in emotional role limitation ($p=.035$).

In summary the results show that yoga intervention resulted in improvement in many aspects of QoL. Improvement was strongly related to practice compliance level as evidenced by the significant improvement in YHC compared to YLC and control in most measures where improvement had been seen.

4.4.2 Depression Anxiety Stress Scale (DASS)

Scores on the DASS were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in tables 4.4 and 4.5. ITT analyses revealed a significant ($p \leq .05$) improvement in the YI group but not in the control group in the depression, and stress subscale scores and also in the global DASS score. In addition, OT analyses revealed a significant ($p \leq .05$) improvement in the YHC group but not in the YLC and control groups, in the anxiety, depression and stress subscale scores and also in the global DASS score, indicating that high compliance to yoga practice was vital to improvement in these psychological measures.

Table 4.4

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for Depression Anxiety Stress Scale (DASS)

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
DASS	control	21	23.48 (23.30)	19.86 (16.28)	1.61	1,71	.21	.022
Global	YI	52	21.87 (17.32)	17.06 (14.03)	7.03	1,71	.010	.090
DASS	control	21	6.00 (6.86)	5.38 (5.43)	.34	1,72	.56	.005
Depression	YI	53	5.34 (6.23)	3.74 (4.75)	5.77	1,72	.019	.074
DASS	control	22	7.00 (6.60)	6.68 (5.80)	.12	1,76	.73	.002
Anxiety	YI	56	7.07 (5.72)	5.96 (4.96)	3.66	1,76	.060	.046
DASS	control	22	10.50 (11.08)	8.91 (7.65)	1.35	1,73	.25	.018
Stress	YI	53	9.53 (7.90)	7.43 (7.27)	5.64	1,73	.020	.072

Table 4.5
Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for Depression Anxiety Stress Scale (DASS)

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
DASS	YLC	23	20.04 (19.21)	19.04 (13.08)	.14	1,70	.71	.002
Global	YHC	29	23.31 (15.86)	15.48 (14.77)	10.78	1,70	.002	.13
score	control	21	23.48 (23.30)	19.86 (16.28)	1.67	1,70	.20	.023
DASS	YLC	24	5.08 (7.41)	4.88 (5.91)	.05	1,71	.83	.001
Depression	YHC	29	5.55 (5.18)	2.79 (3.34)	9.69	1,71	.003	.12
subscale	control	21	6.00 (6.86)	5.38 (5.43)	.35	1,71	.55	.005
DASS	YLC	26	6.19 (5.64)	6.15 (4.10)	.002	1,75	.96	<.001
Anxiety	YHC	30	7.83 (5.78)	5.80 (5.67)	6.79	1,75	.011	.083
subscale	control	22	7.00 (6.60)	6.68 (5.80)	.12	1,75	.73	.002
DASS	YLC	24	8.71 (7.86)	8.00 (7.13)	.30	1,72	.59	.004
Stress	YHC	29	10.21 (8.01)	6.97 (7.48)	7.50	1,72	.008	.094
subscale	control	22	10.50 (11.08)	8.91 (7.65)	1.36	1,72	.24	.019

4.4.3 Profile of Mood States (POMS)

Scores on the POMS were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in tables 4.6 and 4.7 below. ITT analyses revealed a significant ($p \leq .05$) improvement in the YI group but not

in the control group in the depression, and fatigue subscale scores and also in the global POMS score. OT analyses revealed a significant ($p \leq .05$) improvement in the YHC group but not in the YLC and control groups, in the tension and anger subscale scores and also in the global POMS score.

Table 4.6

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for POMS

Variable	**group**	**N**	**M (SD)-pre**	**M (SD)-post**	**F**	**df**	**p**	**η2**
POMS	control	19	16.16 (25.65)	11.21 (20.86)	1.81	1,64	.18	.028
Global *	YI	47	11.96 (19.10)	5.68 (13.58)	7.21	1,64	.009	.10
Depression	control	21	4.67 (6.37)	4.10 (4.71)	.50	1,68	.48	.007
**	YI	49	3.47 (4.11)	2.35(3.24)	4.54	1,68	.037	.063
Depression	control	21			.30	1,68	.59	.004
(transformed)	YI	49			5.14	1,68	.027	.070
Tension	control	22	6.14 (7.03)	5.41 (4.50)	.58	1,69	.45	.008
	YI	49	5.29 (4.86)	4.24 (4.09)	2.61	1,69	.11	.036
Anger	control	20	4.70 (4.93)	5.15 (4.16)	.41	1,67	.52	.006
	YI	49	4.33 (4.20)	3.24 (3.00)	3.31	1,67	.073	.047
Fatigue	control	22	4.59 (3.71)	4.73 (2.80)	.04	1,72	.84	.001
	YI	52	4.96 (3.06)	3.79 (2.62)	7.08	1,72	.010	.089
Confusion	control	22	2.95 (3.00)	3.45 (2.63)	.69	1,70	.41	.010
	YI	50	3.45 (3.24)	2.82 (2.47)	2.72	1,70	.10	.037
Vigour	control	21	8.33 (3.84)	10.52(8.86)	2.20	1,69	.14	.031
	YI	50	9.94 (7.24)	10.00 (5.20)	.007	1,69	.93	<.001

Notes: * Homogeneity assumption not satisfied for this variable. No suitable transformation found

** Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable

Table 4.7

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for POMS

Variable	group	N	M (SD)-pre	M (SD)-post	F	df	p	η2
POMS	YLC	21	10.05(17.27)	5.90 (12.46)	1.40	1,63	.24	.022
Global	YHC	26	13.50 (20.67)	5.50 (14.65)	6.45	1,63	.014	.093
Score *	control	19	24.89 (24.45)	11.21 (20.85)	1.80	1,63	.18	.028
Depression	YLC	22	3.18 (4.33)	2.18 (3.98)	1.60	1,67	.21	.023
subscale	YHC	27	3.70 (4.00)	2.48 (2.56)	2.92	1,67	.09	.042
**	control	21	4.67 (6.37)	4.10 (4.71)	.50	1,67	.48	.007
POMS	YLC	22			1.96	1,67	.17	.028
Depression	YHC	27			3.12	1,67	.082	.044
(transformed)	control	21			.29	1,67	.59	.004
POMS	YLC	23	4.52 (4.73)	4.35 (3.20)	.034	1,68	.85	.001
Tension	YHC	26	5.96 (4.97)	4.15 (4.81)	4.21	1,68	.044	.058
subscale	control	22	6.14 (7.03)	5.41 (4.50)	.58	1,68	.45	.008
POMS	YLC	23	3.48 (3.62)	3.17 (2.62)	.22	1,66	.64	.003
Anger	YHC	26	5.08 (4.59)	3.31 (3.35)	8.28	1,66	.005	.11
subscale	control	20	4.70 (4.93)	5.15 (4.16)	.41	1,66	.52	.006
POMS	YLC	25	4.60 (3.19)	3.40 (2.36)	3.51	1,71	.065	.047
Fatigue	YHC	27	5.30 (2.96)	4.15 (2.84)	3.47	1,71	.067	.047
subscale	control	22	4.59 (3.71)	4.73 (2.80)	.04	1,71	.84	.001
POMS	YLC	24	3.29 (3.33)	3.04 (2.20)	.19	1,69	.67	.003
Confusion	YHC	26	3.65 (3.20)	2.62 (2.73)	3.50	1,69	.066	.048
subscale	control	22	2.95 (3.00)	3.45 (2.63)	.69	1,69	.41	.010
POMS	YLC	24	8.04 (5.19)	8.96 (5.85)	.44	1,68	.51	.006
Vigour	YHC	26	11.69 (8.45)	10.96 (4.40)	.30	1,68	.58	.004
subscale	control	21	8.33 (3.84)	10.52(8.86)	2.20	1,68	.14	.031

Notes:

* Homogeneity assumption not satisfied for this variable. No suitable transformation found

** Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable

4.4.4 Health Survey short form (SF36)

Scores on the SF36 were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in tables 4.8 and 4.9. ITT analyses revealed a significant ($p \leq .05$) improvement in the YI group but not in the control group in the SF36 global score, role limitation due to physical factors, role limitations due to emotional factors, vitality and social functioning. OT analyses revealed a significant ($p \leq .05$) improvement in the YHC subset group but not in the YLC subset group and control group in the global SF36 score and social functioning subscale score. In addition, significant deterioration was seen in the control group only in the role limitations due to emotional factors subscale score.

Table 4.8

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for SF36

Variable	group	N	M (SD) - pre	M(SD) - post	F	df	p	η2
SF36	control	19	68.16 (11.25)	67.82 (11.69)	.03	1,61	.87	<.001
Global	YI	44	66.21 (11.87)	69.90 (12.73)	7.54	1,61	.008	.11
PF *†	control	24	81.81 (11.22)	79.79 (14.41)	.63	1,77	.43	.008
	YI	55	75.05 (19.40)	76.64 (20.40)	.91	1,77	.34	.012
RP †	control	23	65.22 (37.49)	57.61 (39.48)	1.26	1,71	.26	.08
	YI	50	54.33 (37.95)	64.17 (35.60)	4.64	1,71	.035	.061
BP †	control	23	65.78 (22.77)	64.96 (25.23)	.033	1,76	.86	<.001
	YI	55	67.25 (23.46)	67.04 (22.05)	.006	1,76	.94	<.001
GH †	control	20	59.99 (19.45)	59.80 (14.05)	.005	1,71	.94	<.001
	YI	53	61.41 (16.85)	62.57 (17.70)	.51	1,71	.47	.007
VT †	control	20	58.25 (17.50)	61.50 (18.36)	.83	1,70	.37	.012
	YI	52	59.33 (17.35)	63.65 (17.84)	3 86	1,70	.053	.052
RE *†	control	22	86.36 (26.54)	68.18 (39.14)	4.61	1,68	.035	.064
	YI	48	61.11 (40.29)	72.92 (35.57)	4.25	1,68	.043	.059
SF *†	control	23	81.52 (21.93)	75.00 (22.61)	2.36	1,77	.13	.03
	YI	56	77.45 (21.93)	83.70 (21.57)	4.91	1,77	.030	.060
MH †	control	20	67.00 (17.60)	69.65 (15.75)	.42	1,70	.52	.006
	YI	52	70.92 (15.92)	71.65 (17.22)	.35	1,70	.56	.005

* Homogeneity assumption not satisfied for this variable. No suitable transformation found., PF – physical function, RP – physical role limitation, BP – physical pain, GH – general physical health, VT – vitality, RE – emotional role limitation, SF – social function, MH- mental health,

Table 4.9

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for SF36

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
SF36	YLC	19	61.68 (9.67)	64.81 (11.88)	2.33	1,60	.13	.04
Global	YHC	25	69.65 (12.39)	73.73 (12.19)	5.23	1,60	.030	.08
score	control	19	68.16 (11.25)	67.82 (11.69)	.03	1,60	.87	<.001
SF36	YLC	25	75.31 (19.71)	76.82 (21.52)	.37	1,76	.55	.005
PF *†	YHC	30	74.83 (19.40)	76.50 (19.79)	.54	1,76	.47	.007
	control	24	81.81 (11.22)	79.79 (14.41)	.63	1,76	.43	.008
SF36	YLC	21	50.00 (38.73)	61.11 (38.04)	2.45	1,70	.12	.034
RP †	YHC	29	57.47 (37.75)	66.38 (34.25)	2.18	1,70	.14	.030
	control	23	65.22 (37.49)	57.61 (39.48)	1.26	1,70	.26	.08
SF36	YLC	25	62.88 (25.23)	63.44 (21.76)	.017	1,75	.90	<.001
BP †	YHC	30	70.90 (21.63)	70.03 (22.21)	.048	1,75	.83	.001
	control	23	65.78 (22.77)	64.96 (25.23)	.033	1,75	.86	<.001
SF36	YLC	25	56.94 (17.11)	57.58 (17.17)	.072	1,70	.79	.001
GH †	YHC	28	65.39 (15.86)	67.02 (17.24)	.53	1,70	.47	.007
	control	30	59.99 (19.45)	59.80 (14.05)	.005	1,70	.94	<.001
SF36	YLC	24	55.21 (14.78)	58.40 (15.51)	.96	1,69	.33	.014
VT †	YHC	28	62.86 (18.89)	68.15 (18.73)	3.08	1,69	.083	.043
	control	20	58.25 (17.50)	61.50 (18.36)	.83	1,69	.37	.012
SF36	YLC	19	47.37 (38.99)	63.16 (41.41)	2.92	1,67	.09	.04
RE *†	YHC	29	70.11 (39.18)	79.31(27.33)	1.54	1,67	.22	.02
	control	22	86.36 (26.54)	68.18 (39.14)	4.57	1,67	.035	.06
SF36	YLC	26	77.40 (19.69)	75.96 (26.20)	.13	1,76	.72	.002
SF *†	YHC	30	77.50 (20.86)	90.42 (13.80)	12.09	1,76	.001	.13
	control	23	81.52 (21.93)	75.00 (22.61)	2.36	1,76	.13	.03
SF36	YLC	24	67.17 (14.67)	66.12 (16.41)	.08	1,69	.78	.001
MH †	YHC	28	74.14 (16.49)	77.82 (17.50)	1.12	1,69	.29	.016
	control	20	67.00 (17.60)	69.65 (15.75)	.42	1,69	.52	.006

Notes: * Homogeneity assumption not satisfied for this variable. No suitable transformation found.

†, PF – physical function, RP – physical role limitation, BP – physical pain, GH – general physical health, VT – vitality, RE – emotional role limitation, SF – social function, MH- mental health,

4.4.5 Comparing QoL of WLC matched to themselves as YI

Results of quality of life measures revealed statistically significant pre- to post-intervention improvements in most psychological measures including stress (p=.001), anxiety (p=.018), depression (p=.005), tension (p=.028), anger (p=.014), fatigue (p=.004) and confusion (p=.023). A significant pre- to post-intervention improvement was also seen in the SF36 social function subscale score (p=.002). Results also revealed a trend of pre- to post-intervention improvement in all SF36 physical health measures although, none were statistically significant. Arguably, the reduced power due to the small sample size (n=16) may have resulted in fewer significant results than those seen in ANOVA analyses that compared the entire control group (n=26) to the entire intervention group (n=59) as reported above. Nevertheless, these results support the results of ANOVA analyses above in demonstrating that yoga intervention improved various sleep quality and quality of life measures. The results are shown in table 4.10 below.

Table 4.10
QoL scores of WLC subset– matched to themselves as YI subset. Paired samples t test.

Measure	N	M (SD) - pre	M (SD) - post	t	df	p
DASS global score	15	32.37 (21.30)	15.47 (8.85)	3.95	14	.001
DASS stress	15	14.10 (8.93)	7.13 (4.82)	4.09	14	.001
DASS anxiety	16	8.87 (5.12)	7.56 (3.44)	2.66	15	.018
DASS depression	16	8.59 (6.82)	3.94 (3.11)	3.28	15	.005
POMS global score	14	27.07 (18.80)	13.50 (10.34)	2.18	13	.048
POMS tension	15	17.036 (21.79)	5.28 (10.22)	2.45	14	.028
POMS depression	14	5.32 (5.67)	2.21 (2.66)	2.075	13	.058
POMS anger	15	5.13 (3.86)	3.07 (2.58)	2.79	14	.014
POMS fatigue	15	4.90 (2.89)	2.80 (2.18)	3.41	14	.004
POMS confusion	15	3.40 (2.53)	2.07 (1.79)	2.55	14	.023

POMS vigour	14	8.96 (3.34)	9.79 (4.06)	-.75	13	.47
SF36 physical function	15	81.26 (15.11)	85.11 (16.77)	-1.73	14	.10
SF36 physical role	16	64.06 (36.76)	70.31(40.02)	-1.52	15	.15
SF36 physical pain	16	65.87 (22.20)	71.50 (20.67)	-1.51	15	.15
SF36 general health	15	63.22 (13.60)	67.03 (15.24)	-1.41	14	.18
SF36 vitality	16	61.41 (16.10)	67.19 (17.41)	-1.17	15	.26
SF36 social function	16	76.17 (20.57)	90.62 (14.79)	-3.79	15	.002
SF36 emotional role	16	72.92 (29.11)	85.42 (24.25)	-1.69	15	.11
SF36 mental health	16	66.53 (12.16)	69.25 (22.89)	-.54	15	.60
SF36 global score	15	68.69 (10.00)	73.37 (10.37)	-1.74	14	.10

4.5 Sleep quality results

4.5.1 Overview

Hypothesis one, which stated that: "*an integrated yoga intervention will significantly improve measures of sleep quality in elderly presenting with complaints of insomnia*" was tested using the results of the subjective and objective sleep quality measures reported in detail below. Overall, significant improvements ($p<.05$) were found in most subjective measures corresponding to the insomnia diagnostic criteria discussed above (see section 1.5.2) with ITT ANOVA analysis in the yoga intervention (YI) group but not in the control group, and with OT ANOVA analysis in the high compliance yoga participants subset group (YHC) but not in the low compliance yoga participants subset group (YLC). Specifically, significant improvements ($p<.05$) were found in the intervention group but not in the control group in the subjective measures of sleep efficiency ($p=.045$), sleep duration ($p=.042$) and self assessment of sleep quality ($p=.002$). Significant improvements were seen in subjective measures of sleep latency measures for both control ($p=.004$) and intervention ($p=.012$) groups. Significant improvements ($p<.05$) were found in the YHC subset group but not in YLC subset group in subjective sleep efficiency ($p=.012$), sleep

duration ($p<.001$), and self assessment of sleep quality ($p=.012$). Analyses revealed significant ($p\leq.05$) deterioration in the control group but not in the YI group in KSS sleepiness score ($p=.01$). No significant ($p<.05$) change was seen in any of the corresponding direct objective measures for insomnia (namely, SOL, TST, WASO and SE) in any of the analyses. However, ITT multivariate analysis (MANOVA**)** revealed a significant change ($p=.024$) in the insomnia multivariate which comprised of these four measures (i.e., SOL, WASO, TST and SE) in the YI group, but not in the control group. A significant decrease was seen in total time in bed (TTB) in the YLC only ($p=.045$). No significant changes were seen in other insomnia related objective measures. No significant change was seen in any of the groups with consumption of sleep related medications. A significant ($p=.042$) increase of 11.5% in SWS duration, and a significant change ($p=.022$) in the SWS multivariate (comprised of SWS duration and latency) was revealed in the YHC subset group only. The results did not reveal a significant change in other objective measures including OSA related measures.

In summary the results show that yoga intervention resulted in improvement in most aspects of subjective sleep quality and also in the duration of SWS phase and in the objective insomnia related multivariate. Improvement was strongly related to practice compliance level as evidenced by the significant improvement in YHC and lack of change in YLC in the majority of the measures mentioned above. The discrepancies between subjective and objective measures related to insomnia are discussed in detail in the discussion section (see section 5.5). The results did not reveal a significant change in OSA related measures.

4.5.2 Subjective sleep quality measures

4.5.2.1 Pittsburgh Sleep Quality Index (PSQI)

Scores on the PSQI were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in tables 4.11 and 4.12 below. ITT analyses revealed a significant ($p \leq .05$) improvement in the yoga group but not in the control group in the global sleep quality score and also in subjective sleep quality assessment, sleep duration and sleep efficiency subscale scores. The only exception was found in the sleep latency subscale score, where both yoga and control groups improved significantly. In addition, OT analyses revealed a significant ($p \leq .05$) improvement in the YHC group but not in the YLC group in the in the global sleep quality score and also in the subjective sleep quality assessment, sleep latency, sleep duration and sleep efficiency subscale scores, indicating that high compliance with yoga practice was vital to improvement in sleep quality seen in the yoga intervention group.

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Table 4.11

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for PSQI

Variable	group	N	M(SD)- pre	M(SD)-post	F	df	p	η^2
PSQI	control	21	10.14 (3.21)	10.00 (3.08)	.057	1,64	.81	.001
Global	YI	45	9.82 (3.49)	8.67 (3.62)	6.84	1,64	.011	.097
Sleep	control	25	1.84 (.62)	1.72 (.54)	.59	1,78	.44	.008
Quality	YI	55	1.60 (.65)	1.27 (.52)	9.83	1,78	.002	.11
Sleep	control	22	2.45 (.67)	2.00 (.93)	6.70	1,62	.012	. 099
Latency	YI	42	1.86 (.98)	1.48 (1.02)	8.72	1,62	.004	.12
Sleep	control	23	2.04 (.82)	2.26 (.75)	1.87	1,73	.17	.025
Duration	YI	52	2.00 (.99)	1.77 (.85)	4.29	1,73	.042	.055
Sleep	control	14	1.50 (1.29)	1.86 (1.10)	1.41	1,53	.24	.026
Efficiency	YI	41	1.41 (1.22)	1.05 (1.09)	4.23	1,53	.045	.074
Sleep	control	22	1.27 (.46)	1.27 (.63)	.00	1,70	1.00	<.001
Disturbance	YI	50	1.34 (.56)	1.26 (.49)	.87	1,70	.35	.012
Sleep *	control	26	1.77 (1.18)	1.73 (1.15)	.050	1,79	.82	.001
Medication	YI	55	1.38 (1.41)	1.33 (1.41)	.22	1,79	.64	.003
Sleep	control	24	.92 (.58)	.88 (.68)	.06	1,72	.80	.001
Dysfunction	YI	50	.80 (.76)	.74 (.63)	.28	1,72	.60	.004

Note: * Homogeneity assumption not satisfied for this variable. No suitable transformation found

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Table 4.12

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post-intervention change by group (YLC, YHC, control) results for PSQI

Variable	group	N	M(SD)- pre	M(SD)-post	F	df	p	η2
PSQI	YLC	18	9.17 (3.94)	9.72 (4.03)	.74	1,63	.39	.012
Global	YHC	27	10.26 (3.14)	7.96 (3.22)	18.90	1,63	<.001	.23
score	control	21	10.14 (3.21)	10.00 (3.08)	.057	1,63	.81	.001
Sleep	YLC	25	1.56 (.71)	1.28 (.46)	3.23	1,77	.076	.04
Quality	YHC	30	1.63 (.61)	1.27 (.58)	6.66	1,77	.012	.08
subscale	control	25	1.84 (.62)	1.72 (.54)	.59	1,77	.44	.008
Sleep	YLC	17	1.94 (1.03)	1.82 (1.07)	.35	1,61	.56	.006
Latency	YHC	25	1.80 (.96)	1.24 (.93)	11.56	1,61	.001	.166
subscale	control	22	2.45 (.67)	2.00 (93)	6.70	1,61	.012	. 099
Sleep	YLC	23	1.74 (1.10)	1.87 (.87)	.67	1,72	.41	.009
Duration	YHC	29	2.21(.86)	1.69 (.85)	13.38	1,72	<.001	.16
subscale	control	23	2.04 (.82)	2.26 (.75)	1.87	1,72	.17	.025
Sleep	YLC	18	1.28 (1.23)	1.22 (1.00)	.044	1,52	.83	.001
Efficiency	YHC	23	1.52 (1.24)	.91 (1.16)	6.75	1,52	.012	.11
subscale	control	14	1.50 (1.29)	1.86 (1.10)	1.41	1,52	.24	.026
Sleep	YLC	21	1.24 (.54)	1.33 (.48)	5.3	1,69	.47	.008
Disturbance	YHC	29	1.41 (.57)	1.21 (.49)	3.49	1,69	.066	.048
subscale	control	22	1.27 (.46)	1.27 (.63)	.00	1,69	1.00	<.001
Sleep *	YLC	25	1.44 (1.39)	1.40 (1.41)	.052	1,78	.82	.001
Medication	YHC	30	1.33 (1.45)	1.27 (1.48)	.174	1,78	.68	.002
subscale	control	26	1.77 (1.18)	.73 (1.15)	.050	1,78	.82	.001
Sleep	YLC	22	.91 (.81)	.95 (.65)	.71	1,71	.79	.001
Dysfunction	YHC	28	.71 (.71)	.57 (.57)	.89	1,71	.35	.012
subscale	control	24	.92 (.58)	.88 (.68)	.06	1,71	.80	.001

Note: * Homogeneity assumption not satisfied for this variable. No suitable transformation found.

4.5.2.2 Karolinska Sleepiness Scale (KSS)

Scores on the KSS were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (intervention, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in table 4.13 and table 4.14. Analyses revealed significant ($p \leq .05$) deterioration in the control group but not in the yoga intervention group or any of its subsets,

Table 4.13

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for KSS *

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
KSS	control	24	2.79 (1.35)	4.00 (1.93)	7.04	1,76	.01	.083
	YI	54	3.02 (1.51)	2.96 (1.53)	.033	1,76	.86	<.001

Note: * Homogeneity assumption not satisfied for this variable. No suitable transformation found.

Table 4.14

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for KSS *

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
KSS *	YLC	24	2.96 (1.46)	3.46 (1.93)	1.20	1,75	.28	.016
	YHC	30	3.07 (1.57)	2.57 (.97)	1.51	1,75	.22	.020
	control	24	2.79 (1.35)	4.00 (1.93)	7.04	1,75	.01	.086

Note: * Homogeneity assumption not satisfied for this variable. No suitable transformation found.

4.5.2.3 Epworth Sleepiness Scale (ESS)

Scores on the ESS were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in table 4.15 and table 4.16. No significant change was seen in any of the groups.

Table 4.15

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for ESS

Variable	**group**	**N**	**M (SD) - pre**	**M (SD) - post**	**F**	**df**	**p**	**η2**
ESS	control	26	7.00 (3.67)	7.85 (3.47)	1.97	1,80	.16	.024
	YI	56	6.80 (3.88)	7.49 (3.83)	1.61	1,80	.21	.020

Table 4.16

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for ESS

Variable	**group**	**N**	**M (SD) - pre**	**M (SD) - post**	**F**	**df**	**p**	**η2**
ESS	YLC	26	6.08 (4.23)	6.69 (4.42)	1.04	1,79	.31	.013
	YHC	30	7.43 (3.49)	7.78 (3.58)	.60	1,79	.44	.008
	control	26	7.00 (3.67)	7.85 (3.47)	1.97	1,79	.16	.024

4.5.2.4 Consumption of hypnotics and relaxants

Sleep related medication consumption did not change significantly pre to post intervention in any of the groups. No significant pre to post change was seen in PSQI (sleep related) medication subscale score in ITT analysis or OT subsets analysis in any of the groups (see above). Analysis of sleep related medication consumption revealed that 50.9 percent of participants self reported using these medications pre-intervention and 47.4 percent self reported using them post-intervention. Analysis of daily logs and medical questionnaires revealed that among sleep medication users 68 percent used sedative-hypnotics, 12 percent used relaxants and 20 percent used both.

In summary: Yoga practice did not affect sleep medication consumption

4.5.2.5 Comparing WLC matched to themselves as YI

As discussed in section 4.2, the first 31 participants recruited to the study were assignd to a WLC. Five participants dropped out during the control phase and the remaining 26 participants completed the 12 weeks control phase. Subjective and objective measures were taken pre- and post- control phase. The 26 WLC *completers* were then offered to participate in a 12 week yoga intervention (YI). Of these, 16 accepted the offer and 10 declined. An additional analysis was conducted using paired samples t tests to comparing the mean WLC measure scores to the post- YI phase measure scores in these 16 participants who served as a well matched control to themselves.

Results of subjective sleep quality measures revealed a trend of pre- to post-improvement in *all* measures (with the exception of the apnoea probability score). However *significant* improvement was seen only in the PSQI global score (p=.037), sleep latency subscale score (p=.003) and sleep duration subscale score (p=.037). The results are shown in table 4.17.

Table 4.17

WLC subset group – matched to themselves as YI subset group. Paired samples t test. Subjective Sleep quality measures scores.

Measure	N	M (SD) - pre	M (SD) - post	t	df	p
KSS	16	3.44 (.96)	2.94 (1.00)	1.62	15	.13
ESS	16	7.78 (2.83)	7.31 (3.38)	.61	15	.55
PSQI Global score	15	9.80 (2.53)	8.07 (3.77)	2.31	14	.037
PSQI subjective sleep quality	16	1.53 (.34)	1.37 (.62)	1.00	15	.33
PSQI sleep latency	14	2.00 (.71)	1.36 (.93)	3.63	13	.003
PSQI sleep duration	15	2.17 (.70)	1.73 (.88)	2.69	14	.017
PSQI sleep efficiency	14	1.18 (1.12)	1.07 (1.33)	.27	13	.79
PSQI sleep disturbances	16	1.34 (.51)	1.19 (.40)	1.32	15	.21
PSQI medication	16	.78 (1.12)	.75 (1.24)	1.74	15	.86
PSQI daytime dysfunction	16	1.03 (.72)	.69 (.70)	1.70	15	.11
MAP – apnoea probability	15	.35 (.26)	.44 (.34)	-1.09	14	.29

4.5.3 Objective sleep quality measures

4.5.3.1 Objective sleep quality measures - ANOVA analyses

Scores of the objective sleep study variables were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in table 4.18 and table 4.19 below. OT analysis revealed a significant (p=.042) increase in the

duration of the Slow wave sleep (SWS) phase in YHC subset group but not in other groups with the mean total SWS duration in YHC increasing by 13.9 minutes per night (13 percent increase). In addition, OT analysis also revealed a significant (p=.045) reduction in the total time spent in bed (TTB) in the YLC subset group but not in other groups. The mean total TTB in YLC decreased by 38.4 minutes per night (8.9 percent decrease). No other significant changes were seen in any of the measures.

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Table 4.18

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, YI) results for objective sleep study variables.

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
TTB †	control	23	402.3 (91.43)	416.30 (73.98)	.50	1,69	.48	.008
(minutes)	YI	49	424.85 (70.33)	414.04 (78.31)	.61	1,69	.44	.009
TST †	control	23	355.91 (80.92)	363.78 (62.06)	.20	1,68	.65	.003
(minutes)	YI	47	375.32 (57.24)	367.74 (74.57)	.38	1,68	.54	.006
SOL * †	control	23	9.11 (10.36)	8.39 (8.33)	.027	1,69	.87	<.001
(minutes)	YI	49	9.46 (14.97)	14.07 (18.13)	2.33	1,69	.13	.033
WASO†	control	22	50.73 (18.14)	57.14 (19.79)	1.46	1,66	.23	.022
(minutes)	YI	46	59.80 (22.12)	53.74 (15.72)	2.76	1,66	.10	.040
sleep	control	22	.87 (.040)	.87 (.030)	1.04	1,66	.31	.016
Efficiency (%)	YI	46	.86 (.004)	.87 (.037)	1.48	1,66	.23	.022
light sleep	control	23	176.83 (55.30)	184.69 (47.48)	.44	1,68	.51	.007
duration (min)	YI	47	192.59 (45.39)	183.82 (43.42)	1.13	1,68	.29	.016
SWS †	control	23	106.74 (24.80)	107.44 (20.70)	.009	1,68	.92	<.001
Duration (min)	YI	47	106.49 (28.00)	112.34 (30.92)	1.28	1,68	.26	.019
SWS † **	control	23	18.61 (20.40)	15.39 (16.27)	.19	1,68	.66	.003
Latency (min)	YI	47	20.92 (34.46)	24.44 (30.34)	. 47	1,68	.50	.007
SWS latency.	control	23			.45	1,68	.50	.007
(transformed)	YI	47			2.13	1,68	.15	.030
REM †	control	23	70.43 (28.12)	69.39 (28.53)	.023	1,68	.88	<.001
Duration (min)	YI	47	71.04 (25.74)	66.72 (34.80)	. 80	1,68	.37	.012
REM †	control	23	98.30 (44.25)	113.70 (48.49)	2.16	1,67	.15	.032
Latency (min)	YI	46	104.61 (47.80)	99.72 (45.66)	.44	1,67	.51	.007
SPO2 † (%)	control	23	97.13 (4.20)	96.87 (4.21)	.057	1,69	.81	.001
	YI	48	97.12 (4.82)	96.71 (2.95)	.31	1,69	.58	.004
RDI † **	control	23	10.90 (6.45)	10.24 (5.55)	.25	1,68	.62	.004
Events/hour	YI	47	14.92 (10.99)	15.32 (12.96)	.19	1,68	.66	.003
RDI †	control	23			.06	1,68	.81	.001
(transformed)	YI	47			2.11	1,68	.15	.030
ODI †	control	23	7.83 (6.70)	7.74 (5.94)	.005	1,69	.94	<.001
Events/hour	YI	48	11.34 (10.45)	11.70 (11.30)	.19	1,69	.66	.003

Notes:

* Homogeneity assumption not satisfied for this variable. No suitable transformation found

** Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable

† TST – total sleep time, TTB – total time in bed, SOL – sleep onset latency, WASO – wake after sleep onset, SWS – slow wave sleep stage, REM – rapid eye movement stage, SE – sleep efficiency, SPO2 – blood oxygen level, RDI – respiratory disturbance index, ODI –oxygen desaturation index

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Table 4.19

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for objective sleep study variables

Variable	group	N	M (SD) - pre	M (SD) - post	F	df	p	η2
TTB †	YLC	20	431.70 (71.09)	393.30 (77.36)	3.27	1,68	.045	.046
(minutes)	YHC	28	419.96 (70.68)	428.86 (76.93)	.25	1,68	.62	.004
	control	23	402.3 (91.43)	416.30 (73.98)	.51	1,68	.48	.008
TST †	YLC	20	375.70 (68.72)	347.40 (72.75)	2.30	1,67	.13	.033
(minutes)	YHC	27	375.04 (48.46)	382.81 (73.59)	.23	1,67	.63	.003
	control	23	355.91 (80.92)	363.78 (62.06)	.20	1,67	.65	.003
SOL† *	YLC	20	7.10 (9.78)	12.80 (16.70)	1.46	1,68	.23	.02
(minutes)	YHC	28	11.14 (17.77)	14.98 (19.33)	.93	1,68	.34	.01
	control	23	9.11 (10.36)	8.39 (8.33)	.027	1,68	.87	<.001
WASO†	YLC	19	63.37 (23.91)	53.63 (16.03)	2.60	1,65	.092	.043
(minutes)	YHC	27	57.30 (20.86)	53.81 (15.68)	.69	1,65	.47	.008
	control	22	50.73 (18.14)	57.14 (19.79)	1.46	1,65	.23	.022
sleep	YLC	19	.85 (.05)	.87 (.04)	1.69	1,65	.20	.025
efficiency	YHC	27	.87 (.04)	.87 (.05)	.25	1,65	.62	.004
(%)	control	22	.87 (.04)	.87 (.03)	1.04	1,65	.31	.016
light sleep	YLC	20	189.70 (41.57)	176.45 (41.44)	.93	1,67	.39	.014
duration	YHC	27	195.44 (48.03)	189.30 (45.43)	.32	1,67	.57	.005
(minutes)	control	23	176.83 (55.30)	184.69 (47.48)	.44	1,67	.51	.007
SWS †	YLC	20	106.25 (24.59)	101.25 (23.64)	.80	1,67	.52	.006
duration	YHC	27	106.67 (30.75)	120.56 (33.45)	4.30	1,67	.042	.060
(minutes)	control	23	106.74 (24.80)	107.44 (20.70)	.009	1,67	.92	<.001
SWS †,**	YLC	20	24.05(46.89)	31.27 (39.12)	.52	1,67	.43	.009
latency	YHC	27	18.61 (21.92)	20.11 (21.56)	.049	1,67	.83	.001
(minutes)	control	23	18.61 (20.40)	15.39 (16.27)	.19	1,67	.66	.003

Table	4.19		continued	from previous	Page:			
Variable	**group**	**N**	**M (SD) - pre**	**M (SD) - post**	**F**	**df**	**p**	**η2**
SWS.	YLC	20			2.31	1,67	.13	.033
latency	YHC	27			.37	1,67	.54	.006
(transf.)	control	23			.45	1,67	.50	.007
REM †	YLC	20	73.40 (31.35)	64.75 (34.66)	2.31	1,67	.13	.03
duration	YHC	27	69.29 (21.17)	68.18 (35.49)	.008	1,67	.93	<.001
(minutes)	control	23	70.43 (28.12)	69.39 (28.53)	.023	1,67	.88	<.001
REM	YLC	20	99.32 (44.89)	88.39 (42.47)	.90	1,66	.35	.019
latency	YHC	27	108.33 (50.22)	107.68 (46.90)	.004	1,66	.95	<.001
(minutes) †	control	23	98.30 (44.25)	113.70 (48.49)	2.16	1,66	.15	.032
SPO2 †	YLC	20	96.55(5.07)	96.95 (2.52)	.12	1,68	.73	.002
(%)	YHC	28	97.53 (4.69)	96.54 (3.25)	1.02	1,68	.31	.015
	control	23	97.13 (4.20)	96.87 (4.21)	.057	1,68	.81	.001
RDI † **	YLC	20	15.53 (11.32)	17.22 (14.97)	1.41	1,67	.24	.021
Events/hour	YHC	27	14.47 (10.93)	13.92 (11.33)	.20	1,67	.66	.003
	control	23	10.90 (6.45)	10.24 (5.55)	.25	1,67	.62	.004
RDI †	YLC	20			.29	1,67	.62	.004
(transf.)	YHC	27			2.20	1,67	.14	.032
	control	23			.06	1,67	.81	.001
ODI †	YLC	20	13.17 (12.05)	13.28 (12.84)	.007	1,68	.93	<.001
Events/hour	YHC	28	10.03 (9.15)	10.58 (10.22)	.25	1,68	.62	.004
	control	23	7.82 (6.70)	7.74 (5.94)	.005	1,68	.94	<.001

Notes:

* Homogeneity assumption not satisfied for this variable. No suitable transformation found

** Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable

† TST – total sleep time, TTB – total time in bed, SOL – sleep onset latency, WASO – wake after sleep onset, SWS – slow wave sleep stage, REM – rapid eye movement stage, SE – sleep efficiency, SPO2 – blood oxygen level, RDI – respiratory disturbance index, ODI –oxygen desaturation index

4.5.3.2 Objective sleep quality measures -MANOVA analyses

Four different multivariates were examined. The Insomnia multivariate dependent variable was made up of SOL, WASO, TST and sleep efficiency (SE). The SWS multivariate dependent variable was made up of SWS duration and SWS latency (transformed) measures. The REM multivariate dependent variable was made up of REM duration and REM latency measures. The OSA multivariate dependent variable was made up of RDI (transformed), ODI, and SPO2 measures The data for these multivariates were analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 multivariate analysis of variance (MANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 multivariate analysis of variance (MANOVA) was used. The between-subjects factor was group - low compliance yoga participants (YLC), high compliance yoga participants (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). ITT analyses of the insomnia multivariate simple main effects for time within group revealed a significant pre - to post-intervention change for the intervention group only. OT analyses of the SWS multivariate simple main effects for time within group revealed a significant pre - to post-intervention changes for the YHC subset group only. MANOVA results summary are shown in table 4.20 and table 4.21 below followed by detailed results of the analyses in which significant changes were noted.

Table 4.20

Pre- to post-intervention change by group (control, YI) results for 2X2 mixed MANOVAs of objective sleep study variables

Multivariate	**group**	**N**	**Λ**	**F**	**df**	**p**	**η2**
Insomnia	control	22	.97	.40	4,63	.81	.025
multivariate	YI	46	.84	3.02	4,63	.024	.16
OSA	control	23	1.00	.077	3,66	.97	.004
multivariate	YI	47	.93	1.63	3,66	.19	.069
SWS	control	23	.99	.23	2,66	.80	.007
multivariate	YI	46	.94	1.99	2,66	.14	.057
REM	control	23	.97	1.06	2,65	.35	.032
multivariate	YI	45	.99	.25	2,65	.78	.008

Table 4.21

Pre- to post-intervention change by group (YLC, YHC, control) results for 2 X 3 mixed MANOVAs based on sleep study variables

Multivariate	**group**	**N**	**Λ**	**F**	**df**	**p**	**η2**
Insomnia	YLC	19	.92	1.29	4,62	.28	.077
multivariate	YHC	27	.89	1.99	4,62	.11	.11
	control	22	.97	.39	4,62	.81	.025
OSA	YLC	20	.094	.094	3,65	.96	.004
multivariate	YHC	27	.91	2.16	3,65	.10	.091
	control	23	1.00	.078	3,65	.97	.004
SWS	YLC	20	.96	1.50	2,65	.23	.044
multivariate	YHC	26	.89	4.05	2,65	.022	.11
	control	23	.99	.23	2,65	.80	.007
REM	YLC	19	.98	.67	2,64	.51	.021
multivariate	YHC	26	1.00	.012	2,64	.99	<.001
	control	23	.97	1.05	2,64	.35	.032

4.5.3.3 Comparing WLC matched to themselves as YI

As discussed in section 4.2, the first 31 participants recruited to the study were assignd to WLC. Five participants dropped and the remaining 26 completed 12 weeks control phase. Subjective and objective measures were taken pre- and post- control phase. The 26 WLC *completers* were then offered to participate in a 12 week yoga intervention (YI) with 16 accepting and 10 declining. This WLC subset group although relatively small served as a well matched control, to themselves as YI subset group (see section 3.5.1). Since no significant pre- to post- control changes were seen in this group, the pre-intervention values were taken as the mean of the pre- and post- control phase scores. Paired samples t tests were then conducted comparing the mean WLC measure scores to the post- YI phase measure scores. No significant changes were seen in any of the objective sleep quality measures with this analysis. The Results are shown in table 4.22.

Table 4.22
Objective sleep quality measures scores of WLC subset group – matched to themselves as YI subset group. Paired samples t test.

Measure	N	M (SD) - pre	M (SD) - post	t	df	p
Total time in bed (TTB) (min)	14	421.36 (65.25)	449.14 (84.16)	-1.40	13	.18
Total sleep time (TST) (min)	13	377.88 (51.00)	398.08 (89.67)	-.99	12	.34
SOL (min)	14	7.59 (6.17)	12.86 (12.88)	-1.24	13	.24
WASO (min)	13	53.86 (16.00)	54.14 (19.88)	-.067	12	.95
Sleep efficiency (%)	13	.88 (.069)	.87 (.047)	.65	12	.53
light sleep duration (min)	13	188.077 (40.24)	202.077 (47.74)	-.96	12	.36
SWS duration (min)	13	113.23 (17.30)	126.85 (32.64)	-1.51	12	.16
SWS latency (min)	14	17.54 (17.43)	20.07 (19.70)	-.30	13	.77
REM sleep duration (min)	13	74.19 (23.99)	66.46 (39.26)	.65	12	.52
REM sleep latency (min)	14	117.30 (29.22)	102.54 (54.48)	1.08	13	.30
blood oxygen saturation(%)	14	93.29 (1.85)	93.11 (1.28)	.38	13	.71
RDI (events/hr)	13	9.47 (5.73)	9.99 (5.73)	-.49	12	.63
ODI (events/hr)	14	6.47 (3.93)	6.92 (3.20)	-.50	13	.63

4.6 Presence of OSA and its effect on study measures

As mentioned above, the present study incorporated a multi stage screening process designed to include participants presenting with complaints of insomnia and exclude conditions such as OSA or other medical and/or psychiatric conditions which may affect sleep (see section 3.2.2). The screening process followed current diagnostic recommendations for patients presenting with insomnia complaints, which do not include an overnight sleep study (see section 1.5.6). Nevertheless, a significant proportion of participants were diagnosed post sleep studies, as having some level of co-morbid obstructive sleep apnea (OSA). OSA diagnosis results reported below were made based on all available data, including a detailed post intervention medical examination by a sleep physician and data from pre- and post-intervention sleep studies. Table 4.23 below shows apnea frequency in study population, with 69% of participants diagnosed with *some* level OSA (very light to severe) that had not been detected by the pre-study multistage screening procedure.

Table 4.23
Apnea severity levels frequency in study population, diagnosed post study

	total		**control**		**yoga**		**Follow-up required?**
OSA diagnosis	number	percent	number	percent	number	percent	
No OSA	14	18.9%	4	12.9%	13	22.0%	no
Very light or Light	18	24.3%	11	35.5%	16	27.1%	no
moderate OSA	23	31%	6	19.4%	21	35.6%	yes
Severe OSA	10	13.5%	2	6.5%	8	13.6%	yes
Insufficient data	9	12%	8	25.8%	1	1.7%	no
total	74	100%	31	100%	59	100%	

The MAP apnea probability scores were calculated by means of the MAP algorithm using MAP items in conjunction with BMI, age and gender data (see section 3.4.2.4). Pearson's Chi Square tests revealed no statistically significant relationship between diagnosed OSA degree, (none, mild, moderate, severe) as reported above, and the MAP pre intervention apnea probability scores, $\chi^2 2$ (192, N = 65) = 195.0, p= .43 and also no statistically significant relationship was seen between diagnosed OSA degree and the post-intervention MAP apnea probability score, $\chi^2 2$ (183, N = 62) = 186.0, p = .42. These analyses demonstrate that the MAP apnea probability score was not useful at predicting OSA in the study population.

MAP apnea probability scores were further analysed using 'intention to treat' (ITT) and 'on treatment' (OT) analyses. For the former, a 2 x 2 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group (YI, control). The within-subjects factor was time (pre- versus post-intervention). For the latter, a 2 x 3 mixed analysis of variance (ANOVA) was used. The between-subjects factor was group - low compliance yoga participants subset group (YLC), high compliance yoga participants subset group (YHC) and control. The within-subjects factor was time (pre- versus post-intervention). Means and standard deviations and ANOVA results of the two analyses are shown in table 4.24 and table 4.25 below. No significant pre- to post-intervention change in MAP apnoea probability scores was seen in any of the groups. However, since Pearson's Chi Square tests analysis reported above revealed this measure had not been reliable in predicting OSA in the present study's population, the results in tables 4.24 and 4.25 below are probably of limited value.

Table 4.24

Pre- and post-intervention means and standard deviations and 2 x 2 mixed ANOVA pre to post intervention change by group (control, yoga) results for MAP apnea probability score

Variable	**group**	**N**	**M (SD)- pre**	**M (SD)- post**	**F**	**df**	**p**	**η2**
Apnea Probability score (%)	control	22	.37 (.29)	.36 (.29)	.06	1,66	.80	.001
	yoga	46	.42 (.31)	.42 (.33)	.32	1,66	.91	.005

Table 4.25

Pre- and post-intervention means and standard deviations and 2 x 3 mixed ANOVA pre to post intervention change by group (YLC, YHC, control) results for MAP apnea probability score

Variable	**group**	**N**	**M (SD)- pre**	**M (SD)- post**	**F**	**df**	**p**	**η2**
Apnea probability Score (%)	YLC	20	.42 (.31)	.37 (.29)	.44	1,65	.51	.007
	YHC	26	.42 (.32)	.46 (.35)	.32	1,65	.57	.005
	control	22	.37 (.29)	.36 (.29)	.06	1,65	.80	.001

In order to examine whether the presence of co-morbid OSA had been a confounding factor with the measures used in the present study, Pearson's correlation tests were conducted to determine the relationship between OSA degree (none, mild, moderate, severe), and between the change pre- to post-intervention in measure scores in both the control and intervention groups. Detailed results of the analyses are shown in tables 4.26 to 4.31. For the intervention group a statistically significant positive correlation was found between OSA degree and pre to post change in the PSQI global score (r=.34, p=.023, N=45), PSQI sleep efficiency subscale score (r=.52, p=.001,

N=41) and POMS tension subscale score (r=.29, p=.043, N=49). A statistically significant negative correlation was found between OSA degree and pre to post change in the SF36 global score (r= -.33, p=.032, N=42), SF36 role limitation due to physical factors subscale score (r= -.38, p=.007, N=50), SF36 role limitation due to emotional factors subscale score (r= -.46, p=.001, N=48) and SF36 social function subscale score (r= -.29, p=.030, N=56). For the control group a statistically significant positive correlation was found between OSA degree and pre to post change in the MAP apnea probability score (r=.43, p=.052, N=21) and objective sleep latency score (r=.48, p=.028, N=21). Other measures were not affected by the presence or degree of OSA. With PSQI and POMS scales a higher post-intervention mean score indicates a deterioration, while with SF36 scales a higher post-intervention mean score indicates an improvement. Therefore a positive correlation between OSA and PSQI and POMS subscale scores and a negative correlation between OSA and SF36 subscale scores indicates that in all of these measures OSA had a negative impact on improvement. Nevertheless, in these measures, pre- to post-intervention improvement had been seen in the YI group but not in the control group despite the presence of co-morbid OSA. These findings indicate that yoga can improve QoL and sleep quality even in the presence of a co-morbid OSA component. This is discussed in more detail in the discussion chapter.

Since compliance levels proved to have a significant effect on pre- to post-intervention changes in many measures, and since the presence of OSA was detected in a significant proportion of study participants, Pearson's Chi Square test was conducted to examine the relationship between presence or degree of OSA and between compliance levels. Pearson's Chi Square test revealed no statistically significant relationship between OSA degree, (none, mild, moderate, severe) and between compliance level, $\chi^2(3,N=58)=1.94$. This finding indicates that OSA did not affect practice compliance level

and therefore the presence or degree of OSA did not have a fundamentally confounding affect on OT analysis

Table 4.26

Results of Pearson's correlation test between OSA degree (none, mild, moderate, severe) and pre- to post- change in **subjective sleep quality measure** scores in the **yoga** (YI) group

Pre to Post change	**r**	**p**	**N**	**Note**
KSS score change	.15	.28	54	
ESS score change	.13	.35	56	
MAP score change	.16	.27	46	
MAP apnea probability score change	.22	.093	58	
PSQI global score change	.34	.023	45	#1
PSQI subjective subscale score change	.037	.79	55	
PSQI latency subscale score change	.16	.30	42	
PSQI duration subscale score change	.31	.026	52	
PSQI efficiency subscale score change	.52	.001	41	#1
PSQI disturbance subscale score change	.006	.97	50	
PSQI medication subscale score change	.089	.52	55	
PSQI dysfunction subscale score change	.009	.95	50	

Notes:

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in this measure

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Table 4.27

Results of Pearson's correlation test between OSA degree (none, mild, moderate, severe) and pre- to post change in **objective sleep quality** measure scores in the **yoga** (YI) group

Pre to Post change	**r**	**p**	**N**	**Note**
Mean blood oxygen saturation level (SPO2)	-.22	.13	47	
Respiratory disturbance index (RDI)	-.22	.13	46	
Oxygen desaturation index (ODI)	.018	.90	47	
Sleep onset latency (SOL)	-.172	.25	47	
REM latency	.019	.90	46	
SWS latency	-.062	.68	47	
Total time in bed (TTB)	.092	.54	47	
Total sleep time (TST)	.093	.54	46	
Total wake time after sleep onset (WASO)	.17	.26	45	
Total light sleep duration	-.040	.79	46	
Total REM duration	.00	1.00	46	
Total SWS duration	.19	.19	46	
Sleep efficiency (SE)	-.14	.33	47	

Notes:

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in this measure

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Table 4.28

Results of Pearson's correlation test between OSA degree (none, mild, moderate, severe) and between change pre to post in **subjective sleep quality** measure scores in the **control**

Pre to Post change	**r**	**p**	**N**	**Note**
KSS score change	.18	.44	20	
ESS score change	.06	.81	21	
MAP score change	-.01	.96	19	
MAP apnea probability score change	.43	.052	21	#2
PSQI global score change	-.25	.33	17	
PSQI subjective subscale score change	-.28	.23	20	
PSQI latency subscale score change	.05	.83	18	
PSQI duration subscale score change	-.14	.59	18	
PSQI efficiency subscale score change	-.50	.11	11	
PSQI disturbance subscale score change	.25	.31	19	
PSQI medication subscale score change	-.25	.27	21	
PSQI dysfunction subscale score change	.28	.23	20	

Notes:

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in this measures

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Table 4.29

Results of Pearson's correlation test between OSA degree (none, mild, moderate, severe) and change pre to post in **objective sleep quality** measure scores in the **control** group.

Pre to Post change	r	p	N	Note
Mean blood oxygen saturation level	-.037	.87	21	
Respiratory disturbance index (RDI)	-.26	.26	21	
Oxygen Desaturation index (ODI)	.024	.92	21	
Total de-saturations	-.01	.96	21	
Sleep latency	.48	.028	21	#2
REM latency	.053	.82	20	
SWS latency	.028	.91	20	
Total time in bed	-.18	.44	21	
Total net sleep time	-.09	.70	21	
Total wake time after sleep onset	-.28	.21	21	
Total light sleep duration	.18	.42	21	
Total REM duration	-.33	.15	21	
Total SWS duration	-.31	.18	21	
Sleep efficiency (SE)	-.27	.23	21	

Notes:

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in this measures

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Table 4.30

Results of Pearson's correlation test between OSA degree (none, mild, moderate, severe) and between change pre **quality of life** measure scores in the **yoga** (YI) group

Pre to Post change	**r**	**p**	**N**	**Note**
DASS global score change	.14	.31	52	
DASS stress subscale score change	.10	.46	53	
DASS anxiety subscale score change	.16	.23	56	
DASS depression subscale score change	.13	.35	53	
POMS global score change	.012	.94	49	
POMS tension subscale score change	.29	.043	49	#2
POMS depression subscale score change	.24	.10	49	
POMS anger subscale score change	.19	.19	49	
POMS fatigue subscale score change	.24	.088	52	
POMS confusion subscale score change	.23	.10	50	
POMS vigour subscale score change	.16	.26	50	
SF36 physical function subscale score change	-.009	.95	55	
SF36 physical role limitations score change	-.38	.007	50	#1
SF36 body pain subscale score change	.17	.22	55	
SF36 general health subscale score change	-.21	.13	53	
SF36 vitality subscale score change	-.026	.86	52	
SF36 social function subscale score change	-.29	.03	56	#1
SF36 emotional role limitation score change	-.46	.001	48	#1
SF36 mental health subscale score change	-.19	.18	52	
SF36 global score change	-.33	.032	42	#1

Notes:
N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)
#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure
#2 Significant correlation found however ANOVA revealed no significant pre to post change in measure

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Table 4.31

Results of Pearson's correlation test between OSA degree (none, mild, moderate, severe) and pre to post change in subjective **quality of life** measure scores in the **control** group

Pre to Post change	**r**	**p**	**N**	**Note**
DASS global score change	.07	.80	17	
DASS stress subscale score change	-.05	.84	18	
DASS anxiety subscale score change	.10	.69	18	
DASS depression subscale score change	.12	.63	17	
POMS global score change	-.30	.22	18	
POMS tension subscale score change	-.003	.99	18	
POMS depression subscale score change	-.07	.79	17	
POMS anger subscale score change	-.09	.75	16	
POMS fatigue subscale score change	-.06	.80	18	
POMS confusion subscale score change	.03	.91	18	
POMS vigour subscale score change	-.22	.39	17	
SF36 physical function subscale score change	.18	.45	19	
SF36 role limitations (physical) score change	-.09	.70	19	
SF36 body pain subscale score change	-.40	.09	19	
SF36 general health subscale score change	-.13	.64	16	
SF36 vitality subscale score change	.05	.85	16	
SF36 social function subscale score change	-.03	.91	19	
SF36 role limitation (emotional) subscale score change	-.18	.46	18	
SF36 mental health subscale score change	-.008	.98	16	
SF36 global score change	-.43	.11	15	

Note: N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

Chapter 5. Discussion

5.1 Overview

The present study was a pragmatic study that examined a typical geriatric population, presenting with insomnia symptoms. The screening process incorporated a standard diagnostic procedure which was based on prevailing clinical diagnostic guidelines that currently do not recommend prescribing sleep studies for patients presenting with insomnia symptoms (see section 1.5.6). In other words, a 'real life' situation was investigated and the results are therefore widely applicable to this population. The yoga protocol used in this study is widely applicable throughout the western world as it is non-sectarian and not specific to a particular yoga method or school while still being representative of mainstream yoga practices.

The present study both supports and expands on findings of other studies on yoga for improving geriatric sleep quality and QoL. A single study conducted in India (Manjunath & Telles, 2005) and two studies conducted in Taiwan (Chen et al., 2008; Chen et al., 2009; Chen et al., 2010) have shown yoga practice resulted in significant improvements in some subjective sleep quality and quality of life measures (QoL) in older people. These studies have been conducted in non-western cultural settings (Manjunath & Telles, 2005; Chen et al., 2008; Chen et al., 2009; Chen et al., 2010) and the intervention periods were twice as long in comparison to the present study (six months versus 12 weeks). The yoga interventions in these studies relied on three (Chen et al., 2008; Chen et al., 2009; Chen et al., 2010) or six (Manjunath & Telles, 2005) yoga classes each week, while the present study incorporated two yoga classes per week in conjunction with daily home-based self-practice of yogic meditation and relaxation techniques using an audio CD. The shorter overall intervention period, the fewer number of weekly classes and the addition of home-

based self-practice were deemed more suitable for a western cultural setting, although a longer intervention period may have possibly resulted in more significant changes in additional measures. As the present study incorporated a significant home-based self-practice component, it was decided to also examine practice compliance and its impact on the efficacy of yoga intervention. The present study's results on practice compliance may help improve future home-based self-practice yoga programs as discussed in detail below.

In comparison to other studies on yoga for geriatric QoL and sleep quality (Manjunath & Telles, 2005; Chen et al., 2008; Chen et al., 2009; Chen et al., 2010), the present study has examined a wider range of sleep quality and QoL measures in order to get a more comprehensive view of changes in psychological and sleep status. In contrast to previous studies, the present study has also examined a range of objective sleep quality measures. These were conducted in the natural home environment and not in a clinic, making the measurement conditions closer to a 'real life' situation. Using objective measures was considered essential in view of previous findings of significant discrepancies between subjective and objective sleep quality results (Haimov, 2005) discussed in detail below. The results of the objective measures both support and expand on findings of previous studies on the effect of yoga on the deep sleep stage (Patra &. Telles, 2010; Patra & Telles, 2009; Telles et al., 2000) as discussed in detail below. The results of the objective measures used in the present study have also helped show how yoga affects sleep quality and QoL of older people with co-morbid insomnia and OSA as discussed in detail below.

5.2 Study limitations

The present study's modest budget put limitations on available resources and timelines which impacted on the experimental design and overall sample size. Total

available resource dictated a mixed experimental design that lacked double-blinding and randomisation (see section 3.1). These weaknesses were mitigated to some extent by the recruitment process method, the use of a supplementary analysis for WLC, and the use of single blinding as follows:

1. The study's advertisement campaign targeted a wide population base throughout the Jerusalem metropolitan area and applicants were processed on a 'first come first serve basis' (see section 3.2). The wide target population base and the non-preferential 'first come first serve' process introduced an element of quasi randomness into the recruitment and treatment assignment process with the first 31 accepted to the study being assignd to WLC and all subsequent subjects admitted to the study assignd to YI. Nevertheless, applicants' response time to the advertisement campaign, which determined their processing placement, may have possibly been influenced to some extent by various extraneous and potentially confounding psychological and socio-economical factors.
2. All WLC completers were contacted post control phase and offered assignment to YI and 16 accepted the offer (see section 4.2). A supplementary statistical analysis was conducted, with 16 WLC subjects matched to themselves as YI. This analysis had a stronger control design, than in the main analyses which used WLC as control for the entire YI group. This supplementary analysis supported the findings of the main analyses by revealing similar trends and some similar results (see sections 3.51, and 3.5.2.).
3. Participant identity code numbers were used to blind administrative staff and sleep scientists to participants' identity and treatment assignment (see section 5.2).

Nevertheless, since the mixed study design utilised in the present study lacked double-blinding and randomisation (see section 3.1), all significant findings on both subjective and objective measures should be confirmed by a future larger scale randomised controlled trial.

The subjective instruments used in the present study have been translated from English into the Hebrew Language. Studies had been conducted to validate Hebrew versions of the SF-36 (Lewin-Epstein et al., 1998), the PSQI (Shochat et al., 2007) and the POMS (Netz et al., 2005) but there is still a need for validation studies of the Hebrew versions of DASS, MAPS, KSS and ESS for diverse Hebrew speaking populations as the validity of the translated versions cannot be automatically assumed and this may present a limitation in interpreting the results of these instruments. Nevertheless, in the present study it was decided to use the Hebrew versions of KSS, ESS and MAPS due to the simple language and concepts used in these questionnaires. The significant overlap between DASS and POMS (see above) enabled comparing the results of overlapping subscales.

5.3 The OSA factor

The present study employed a screening process intended to exclude any disorders and conditions which may affect sleep, including OSA (see section 3.2.2). The screening process followed current medical guidelines for patients presenting with insomnia complaints and included a systematic medical examination and review of medical and psychiatric history and if OSA was suspected, the patient was referred to further medical investigation, including PSG and not accepted to the study unless negative findings were reported (see section 3.2.2 – 3.2.3). However, limited resources did not allow using objective sleep studies to screen applicants for OSA during the

recruitment phase of the study (see section 3.2.2). A second systematic medical examination and review of all data obtained during the study, including results of all sleep studies, was conducted post-intervention. This resulted in close to 69 percent of study participants being diagnosed with some level OSA, with 24.3 percent -very light or light OSA, 31 percent moderate OSA and 13.5 percent severe OSA (see section 4.6).

Several studies have shown a high prevalence of insomnia complaints in patients with OSA (Krakow et al., 2001; Smith et al., 2004; Krell & Kapur, 2005; Chung, 2005) and one study has shown a high prevalence of undiagnosed OSA (29 % with AHI>15 and 43 % with AHI>5) amongst elderly with insomnia complaints (Lichstein et al., 1999), yet, until recently the association between OSA and insomnia has received relatively little attention in both research and clinical practice (Beneto et al., 2009). Nevertheless, in view of the systematic screening procedure used in the present study, although without the use of sleep studies for screening for OSA (see section 3.2.2), the prevalence of undiagnosed co-morbid OSA in study participants was higher than expected. These findings raised the following questions:

1. Why was such a high percentage of OSA/morbid OSA cases not detected by study's multi stage screening process?
2. Was co-morbid OSA a confounding factor and to what extent? What are the implications for data analysis and interpretation of the results?
3. Was co-morbid OSA affected by the intervention?
4. What are the implications of these findings on diagnostic and treatment guidelines for older adults presenting with insomnia symptoms?

Previous studies have shown a higher prevalence of OSA in geriatric populations than in the general population (Ancoli-Israel et al., 1991; Delbarton et al., 1996; Wolkove

et al., 2007) (see section 1.6.4). Ancoli-Israel et al. (1991) reported that in a survey of sleep-disordered breathing of 427 randomly selected older American adults, aged 65 years and over, 24 percent were found with AI≥5 and 62 percent with RDI≥10. The findings reported by Ancoli-Israel et al. (1991) are similar to those revealed in the present study. However, in the former study, participants were selected randomly, where as in the present study an elaborate screening process described above was utilised. Therefore, such a high prevalence of previously undetected OSA suggests that the screening procedures used were inadequate.

In the present study the apnea probability score, which is derived from several MAPS questionnaire items in conjunction with body mass index (BMI), age and gender, using a special algorithm, was unreliable at predicting OSA. Furthermore, no statistically significant relationship was found between OSA degree (none, mild, moderate, severe), as diagnosed post intervention, based on all available data collected during the study, and between both pre- and post-intervention apnea probability scores derived from the MAPS (see section 4.6).The MAPS has previously been reported in several studies to be a useful tool for discriminating between patients with and patients without sleep apnoea in both general and older populations (Maislin et al., 1995; Maislin et al., 1996) (see section 3.4.2.4), yet the results of the present study strongly support the findings of a more recent meta-analysis of clinical screening tests for OSA (Ramachandran & Josephs, 2009) that suggest that both the MAP and systematic clinical reviews may yield a significant proportion of false negative predictions for OSA.

In the present study the significant proportion of OSA prediction false negatives may be related to the fact that the MAP apnea prediction algorithm uses several MAP items including frequency of snoring, snorting and gasping for air in conjunction with BMI, age, and gender. In the present study, many respondents ticked the option “I do not know”

(scored zero – same as for the option "never") in response to the first three items. This may be related to the fact that 55 percent of the study population reported living on their own (see table 4.1) and therefore lacked external feedback on their sleep disturbances. Several participants who were living with a partner also responded "I do not know" to these items. When queried later about it they explained that their insomnia symptoms (e.g. having difficulty falling asleep or waking up frequently during the night) had a disruptive influence on their partner's sleep and that forced them to sleep in separate rooms. Consequently their partner could not give them reliable feedback on their sleep disruptions.

Failure of the MAP to predict apnea in the study population, demonstrates the difficulty of excluding apnea in a geriatric population presenting with complaints of insomnia, as the standard medical examination process also relies on questions similar in content to MAP items. The physician also conducts a physical examination and reviews detailed medical history/records. But if a patient presents with insomnia complaints and the examination and questioning yield negative findings, current medical diagnosis would most likely be 'insomnia' and treatment regimen prescribed accordingly. In their large scale (n=5615) study on the predictors of sleep-disordered breathing (SDB) in community-dwelling adults (aged 40 to 98) Young et al. (2002b) found that as age increased, the magnitude of associations between SDB and snoring, breathing pauses and physical attributes such as body mass index and neck girth, decreased. They concluded that breathing pauses and obesity may be particularly insensitive indicators for identifying SDB in older adults. They also concluded that there is a need to attain a better understanding of predictive factors for SDB, particularly in older people. The results of the present study support Young et al.'s conclusions and suggest one possible reason why self reported snoring and breathing pauses are unreliable for prediction of SDB in older adults. Overall, it seems that diagnosis based on physical examination, medical history and instruments

such as MAP are not reliable in detecting OSA in an elderly population presenting with insomnia complaints, while in- home sleep studies using portable monitoring in conjunction with a computerised diagnosis system used in the present study, has helped detect and diagnose previously undiagnosed OSA in an elderly population at similar rates to those reported by previous large scale studies that had utilised PSG (Ancoli-Israel et al., 1991; Young et al., 2002b).

The findings of the present study support previous studies' findings on the high prevalence of undiagnosed OSA in the elderly (Lichstein et al., 1999; Ancoli-Israel et al., 1991) and suggest the possible benefit of prescribing objective sleep studies for all elderly presenting with complaints of insomnia, despite of the relatively high costs involved, before prescribing pharmacotherapy for their insomnia symptoms. The present study's findings also support Lichstein et al.'s (1999) conclusions on the need to use objective sleep studies when screening participants for research on geriatric insomnia.

Although portable monitoring is still not recommended for screening of OSA in *asymptomatic* populations (Collop et al., 2007) (see section 1.3.3), it may however, be a useful tool for *initial* OSA screening of elderly patients presenting with insomnia symptoms prior to prescribing sedative-hypnotics, in particular benzodiazepines, due to their respiratory depressant effects that may worsen sleep-related breathing disorders (SRDB) that may even cause complete obstructive sleep apnea in heavy snorers with long term use (Guilleminault, 1990; Wagner et al., 1998) (see section 1.5.12.2). When used chronically benzodiazepines may also lead to physiologic and psychologic depence (Longo & Johnson, 2000). The respiratory depressant effects in conjunction with the potentially addictive characteristics of benzodiazepines may cause a vicious circle in case of failure to diagnose the presence of SRDB, or a co-morbid SRDB, as the symptoms may persist or even deteriorate, which in turn may lead to chronic use and forming of an addiction, which

may lead to further aggravation of the SRDB and its symptoms, including excessive daytime sleepiness (EDS). This vicious circle may be avoided by detecting SRDB earlier. This vicious circle may have also occurred in a proportion of the participants in the present study.

The high proportion of co-morbid OSA diagnosed post-intervention in the present study was a possible confounding factor since both OSA and insomnia are associated with reduced sleep quality, physical health, mental health and quality of life in general (Young et al., 2002a; Neubauer, 2001; Pepperell, et al. 2002; Bédard et al., 1991). While co-morbid OSA appeared to be a factor working against improvement in overall subjective sleep quality and sleep efficiency measures, a significant ($p<.05$) improvement was nonetheless found in these measures, as well as in most other subjective sleep quality measures in the intervention group but not in control group suggesting that yoga improved sleep quality despite the presence of comorbid OSA. Furthermore, the results of pre- to post-intervention changes in OSA measures suggest that yoga does not result in significant change in OSA status.

In an ideal geriatric insomnia study, all participants would be totally free of OSA, as well as other conditions which may affect sleep quality, such as physical pain. However, it seems that the present study's population better reflects the 'real life' situation of older adults presenting with insomnia complaints with a high proportion also having undiagnosed co-morbid OSA, and perhaps other undiagnosed health conditions. The significant improvements seen in the intervention but not in the control group in many quality of life health measures and subjective sleep quality measures indicate that yoga significantly improved subjective sleep quality, physical, mental and social quality of life in the study population despite the influence of co-morbid OSA working to reduce these improvements.

Future studies on yoga intervention for improving QoL and sleep quality of the elderly, may take advantage of the findings of the present study and apply a modified experimental design as follows:

1. A substantially larger budget would be assignd to enable recruiting a much larger number of subjects with a randomised controlled design and screening of all applicants using portable sleep monitoring.

2. A precise OSA diagnosis would be made pre-study and both intervention and control groups would be stratified according to participants' OSA status as follows: no OSA, light OSA, moderate OSA and severe OSA.

3. A much larger number of participants would be assignd to both control and yoga intervention groups to ensure that each stratified subset would be adequately powered.

4. In the present study a much higher proportion of women applied to join the study and consequently the study included 81 percent woman. An ideal study would include an equal number of men and women. This may require screening a larger number of applicants still and significantly extending the recruitment phase

5. The statistical analysis should examine closely the relationships between levels of OSA and pre- to post-intervention changes in measures, in each stratified subset group as well as for the entire intervention and control groups.

5.4 Slow wave sleep (SWS)

A significant pre- to post-intervention improvement was seen in the YHC subset group but not in other groups in the deep sleep (SWS) duration which increased

significantly but no pre to post changes were seen with other sleep stages (i.e. light sleep and REM) in any of the groups. Previous short studies have shown that Cyclic Meditation (CM) yogic practice was associated with increased proportion of SWS duration and decreased proportion of REM sleep on the night that followed practice (Patra &. Telles, 2010; Patra & Telles, 2009; Telles et al., 2000) (see section 1.7.13.2.2). The SWS is believed to contribute to the restorative physiological processes that occur during sleep. An association has been found between SWS and secretion of Growth Hormone and increased insulin sensitivity (in humans) (Van Cauter et al., 1998; Van Cauter et al., 1997; Gronfier et al., 1996; Holl, 1991) and higher rates of brain protein synthesis (in rats) (Ramm & Smith, 1990) (see section 1.3.2). Arguably, the significant improvement in subjective measures of sleep quality, fatigue and vitality in the YHC intervention subset group only, may also be related to the relation between SWS and restorative physiological processes which result in participants feeling more refreshed in the morning. Further research is required to determine the underlying mechanisms by which yoga affects sleep staging and how yoga intervention may be used to achieve better sleep architecture.

5.5 Discrepancy between subjective and objective measures.

The results reveal a marked discrepancy between subjective and objective insomnia related measures. Several previous studies have also reported similar discrepancies and have suggested that subjective perception of sleep may be significantly affected by psychological factors. Haimov (2005) reports a substantial discrepancy between subjective and objective sleep quality measures of 98 healthy elderly subjects with the subjective sleep quality measures significantly associated with self esteem and a sense of coherence while Klein et al. (2003) report lower perception of sleep quality among patients with post traumatic stress disorder (PTSD) caused by motor vehicle crash (MVC) compared to MVC survivors with no PTSD or other non

PTSD patients while no significant differences were found amongst the three groups on objective actigraphy measures. Edinger et al. (2000) have shown that psychological factors may mediate subjective self reported sleep satisfaction but at the same time may also predict objective sleep difficulties. The discrepancy between objective and subjective sleep measures may also result from a 'Sleep State Misperception' disorder defined by the ICSD as a sleep disorder '*in which a complaint of insomnia or excessive sleepiness occurs without objective evidence of sleep disturbance'* (AASM, 2010, pp. 32-33). This disorder may appear as a convincing and genuine sleep complaint by patients with no psychological disorders and has been suggested to result from excessive mentation during sleep which causes a perception of being awake or from actual changes in sleep physiology that are too subtle to be detected by PSG (AASM, 2010, p.33). It has also been suggested that this disorder may be a sleep equivalent of hypochondriasis (AASM, 2010, p.33). Furthermore, a previous study has shown that polysomnography findings did not correlate well with all PSQI component scores and suggested that the discrepancy may be related to the fact that PSQI elicits self estimate of usual sleep quality over a one-month period, thus decreasing its' sensitivity to daily variations (Buysse et al., 1989).

Similar factors may have been involved in the discrepancy seen in the present study between subjective and objective measures. The present study has shown that the yoga intervention significantly improved a range of psychological factors (see section 4.4) which may have improved subjective sleep satisfaction and not objective sleep quality. On the other hand, the significant improvement (p=.024) seen in the yoga group but not in the control group with the insomnia objective measures multivariate (made up of SOL, WASO, TST and SE), while no change was seen with any of these variables independently, may indicate an objective subtle change which had taken place in sleep

physiology that was only detected by multivariate analysis. A longer intervention period (e.g. six months) may possibly have resulted in significant changes in several independent insomnia related variables. Also, the subjective feeling of having slept better may also be related to the significant (p=.042) objective increase of 11.5% in the duration of SWS (deep sleep) stage seen in the YHC group discussed above. In other words, the sleep had become significantly 'deeper' and therefore more refreshing despite the fact that there was no significant change in other objective insomnia related measures (i.e. SOL, WASO, TST and SE).

It can also be speculated that being connected to the portable sleep monitoring equipment may have had some effect on participants' sleep patterns. For example, the discomfort associated with walking around connected to the equipment may have resulted in some participants retiring to bed earlier than they would normally do. It may have also been a factor affecting participants to remain in bed or spend shorter time out of bed in case of an awakening during the night. This in turn may have resulted in a more rapid sleep onset, an overall shorter WASO and higher sleep efficiency.

Since diminished subjective sleep quality is one of the most frequent health complaints in the elderly (Prinz, 1995), the significant improvement in most aspects of subjective sleep status in the yoga group is an important finding despite the discrepancy seen between the subjective and corresponding objective sleep quality measures.

5.6 Use of sedatives/hypnotics and relaxants.

Despite significant improvements (p<.05) in subjective sleep quality measures in the YI group in general and in the YHC subset group in particular, there was no corresponding reduction in use of sedatives/hypnotics and relaxants, with close to half of participants still reporting regular usage (see section 4.5.2.5). This may be partially explained by the

potentially addictive characteristics of these medications (Longo & Johnson, 2000). Perhaps a longer intervention period may be needed to reduce dependency on these medications.

Daytime impairment was measured *directly* by KSS and ESS sleepiness scores and the PSQI daytime dysfunction subscale score and indirectly by the POMS fatigue and the SF36 vitality subscales scores. No significant change was seen in ESS sleepiness scores or in PSQI daytime dysfunction subscale scores with any group. However, a significant (p=.010) deterioration was seen in the control group in the KSS sleepiness score, a significant (p=.010) improvement in the POMS fatigue subscale score and a significant (p=.053) improvement in the SF36 vitality subscale score were seen in the intervention group and not in the control group. The lack of significant change in sleepiness and daytime dysfunction measures may possibly be related to persistent consumption of sedatives/hypnotics and relaxants, as regular use of these medications has been shown to be associated with daytime drowsiness (Nowell et al., 1997) (see section 1.5.12.2). The lack of significant change in sleepiness and daytime dysfunction measures may also be related to the high prevalence of co-morbid OSA among participants see section 4.6) as OSA has also been shown to be associated with daytime drowsiness, sleepiness and dysfunction (Young et al., 2002b; Berg, 2008) (see section 1.6.4) and no significant change was seen in direct OSA related measures in any of the groups (see results section 4.6)

5.7 Association between quality of life factors and insomnia

As discussed above, insomnia is often associated with anxiety, stress and depression (Schneider, 2002; Buysse et al., 2005; NIH, 2003; Lichstein et al., 2001) and geriatric insomnia specifically has been strongly associated with anxiety, neuroticism and depression (Beullens, 1999). The present study has demonstrated that yoga results in

significant improvements ($p<.05$) in many subjective measures for insomnia and also for anxiety, stress and depression. Additional studies are required to further examine the psycho-physiological mechanisms involved in the strong association between anxiety, stress, depression and insomnia and how yoga affects those mechanisms. This will enable developing more effective yoga interventions for these conditions. Overall, the present study's findings support prescribing yoga to older adults presenting with primary insomnia symptoms. Furthermore, the study's findings support prescribing yoga to older adults presenting with co-morbid insomnia and OSA symptoms but do not support prescribing yoga intervention for primary OSA without a significant co-morbid insomnia component.

5.8 Safety, acceptance and compliance of yoga intervention

It appears that the yoga intervention used in this study was well accepted and safe with compliance levels being variable. The acceptability of yoga is evidenced by the rapid and wide-ranging response to the recruitment advertising campaign and the low dropout rate, while the safety is indicated by the lack of adverse events. However, compliance in general and home practice compliance in particular was below recommended level. Overall, only 10 percent of participants achieved a practice compliance level equal or above the *recommended* level of three daily practice units. The median compliance level was close to *the requested minimum* of one daily practice unit (20 – 25 minutes per day) (see section 4.3). On treatment (OT) subset analysis revealed that the high compliance yoga participants subset group (YHC) (with compliance equal or above median) improved significantly in a wide range of sleep quality and QoL measures compared to the low compliance subset group (YLC) (below median practice level) and control (see section 4.4 – 4.5), thus demonstrating the importance of achieving high practice compliance level for improving sleep quality and quality of life.

Compliance with any therapy is always an important consideration. The results of the daily logs revealed that for the present study compliance level did not vary much over the 12 weeks intervention period with most participants maintaining a constant individual compliance level and changes in compliance being related to transient events (i.e., catching a cold, social/ family engagements etc.).

Overall, study findings revealed that achieving an adequate practice compliance level was essential for effective application of the yoga protocol, especially for improving insomnia status. It seems that at least 25 minutes of daily practice is required to achieve these benefits. This is significantly less than the level of practice used in previous studies (Manjunath and Telles, 2005) and roughly 50 percent of the intervention group was able to sustain this level of practice compliance indicating that present study's yoga protocol may be suitable for urban geriatric population in western settings. The use an audio CD incorporated in the present study may have been a factor in keeping compliance at reasonable levels and use of audio visual aids should be considered in future studies.

Chapter 6. Conclusions

1. Yoga practice, as applied in the present study, appears to be a safe, easy to implement, and well accepted non-drug intervention for the elderly in a western urban cultural setting.
2. In a geriatric population with primary insomnia, or insomnia with comorbid obstructive sleep apnea (OSA), practicing yoga for at least 25 minutes a day for twelve weeks improved most aspects of subjective sleep status. Specifically, improvements were seen in subjective measures of sleep latency, sleep duration, self assessed sleep quality, fatigue and vitality. Improvements were also found in duration of the deep sleep (SWS) stage.
3. In a geriatric population with primary insomnia or insomnia with comorbid obstructive sleep apnea (OSA), practicing yoga for at least 25 minutes a day (roughly one 'practice unit' as discussed in sections 3.3.6, 3.5.3 and 4.3) for twelve weeks also improved many aspects of psychological and emotional well being. Specifically, improvements were seen in levels of depression, anxiety, stress, tension and anger. Improvements were also seen in vitality and daily function in physical, emotional and social roles.
4. Twelve weeks of regular yoga practice did not affect use of sedative-hypnotic medications in study's population.
5. Twelve weeks of regular yoga practice did not improve OSA status in the study's population. While the presence of OSA partially reduced the efficacy of yoga intervention in improving overall sleep quality and sleep efficiency and in improving some aspects of life quality, specifically carrying out daily roles and social function, the presence of OSA did not reduce the efficacy of yoga practice in improving other aspects of sleep quality including subjective measures of self

assessed sleep quality, sleep latency and sleep duration, as well as the duration of the deep sleep stage (SWS). Furthermore, the presence of OSA did not reduce the efficacy of yoga in improving most aspects of psychological well being.

6. While yoga was readily accepted by an elderly population, maintaining practice levels at a minimum of 25 minutes per day appears to be crucial for improving sleep quality and quality of life in this population. Factors that may increase participation include providing materials to support home practice, maintaining a yoga class component with one or two weekly classes to introduce, revise and reinforce the exercises prescribed for home practice, as well as providing a choice of exercises that can be graded according to different abilities.
7. Obstructive sleep apnea (OSA) appears to be present to varying degrees in the majority of elderly people presenting with insomnia symptoms. The MAP questionnaire, detailed doctor examination and full medical history appear inadequate to diagnose OSA reliably in this population. Although portable monitoring is not recommended for screening of OSA in *asymptomatic* populations, it may however, be a useful tool for *initial* OSA screening of elderly patients presenting with insomnia symptoms prior to prescribing sedative-hypnotics and for screening for OSA in research settings.

Chapter 7. Implications for future research

Due to the high prevalence of co-morbid insomnia and OSA in the geriatric population, inclusion/exclusion criteria and screening procedures for similar future studies should enable screening all applicants for OSA and stratification of subjects according to their OSA status (no OSA, light OSA, moderate OSA and severe OSA) in order to examine the precise relationship between OSA level and changes in all measures.

The present study has shown that yoga can be applied effectively to improve sleep and life quality in elderly presenting with complaints of insomnia. However, this study has also revealed factors that reduce intervention efficacy, including low compliance and presence of undetected and untreated co-morbid obstructive sleep apnea (OSA).

Further research is now needed to:

1. Examine if short and medium duration yoga programs can result in long term persistence of self home-based practice
2. Identify yoga protocols that yield better compliance levels.
3. Develop recommendations for optimal treatment strategies for co-morbid insomnia and OSA including treatment priorities and sequence. For example, at what stage of treatment should yoga be introduced to treat the co-morbid insomnia component in co-morbid insomnia & OSA disorder?
4. Examine long term effects of short and medium duration yoga programs on sleep quality and quality of life

Yoga encompasses a diversity of practices and techniques. Additional research is also needed to:

1. Examine various physical and meditative yoga techniques to determine most effective for improving sleep quality.
2. Establish optimal ratio between home and class practice.
3. Establish optimal ratio between physical and meditative practices
4. Establish optimal dose – effect ratios

An Audio CD was used successfully in the present study to support home based self practice of meditative exercises. Future studies may benefit from making use of additional audio-visual aids such as video recordings of yoga sessions as well as software for personal computers with a menu system and links to detailed instructions, supporting material, detailed daily sleep and practice logs and questionnaires. Future studies may also benefit from making use of the internet to allow study participants to communicate with their yoga teachers and participate in online yoga classes. This would allow future studies to increase the number of classes and offer daily classes at convenient times. Applying such measures, may allow increasing the sample size and improving home practice compliance level. Introduction of online research systems may also be used for long term observation and follow up of practice outcomes and compliance levels.

Using portable monitoring, in conjunction with a computer assisted FDA and CE approved sleep diagnostic system has been used successfully in present study. Future research in the area of geriatric insomnia would benefit from making use of such tools both as screening and diagnostic tools. The robustness of the ECG and pulse oximetry signals in conjunction with the reduced number of required channels may enable conducting diverse large scale studies on the effect of mind-body interventions on sleep, autonomic nervous system balance, the relaxation response etc. The measures could easily be taken in participants' home environments, during meditation and relaxation sessions and even during gentle exercise.

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Appendixes

List of appendixes

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Appendix 1. Ethics committee approval

Phone: 9925 2251

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peter.burke@rmit.edu.au

24 April 2008

Dear Jonathan,

Project No 36/07: A randomised clinical trial of integrated yoga style practice intervention for improving sleep and quality of life in the elderly

I am pleased to advise that this project is now approved by the Human Research Ethics Committee for the period specified in the application, that is, until **31 March 2009**. The project has been classified as **level 3** as it involves higher risks to the participants than discomfort or inconvenience.

Responsibilities of primary investigator

It is important to emphasise that primary investigators are responsible for ensuring that the project proceeds according to the proposal approved by the Human Research Ethics Committee. The Committee's approval of the project is not absolute. New and unforeseen ethical issues may arise. A researcher should continue to consider the ethical dimensions of the research as the project progresses.

Adverse events or unexpected outcomes

As the primary investigator you have a significant responsibility to monitor the research and to take prompt steps to deal with any unexpected outcomes. You must notify the Committee immediately of any serious or unexpected adverse effects on participants, or unforeseen events, which may affect the ethical acceptability of your project. Any complaints about the project received by the researcher must be referred immediately to the Ethics Executive Officer.

Reporting

Approval to continue a project is conditional on the submission of annual reports (see attached sample form). A final report should also be provided at the conclusion of the project. If your work is completed within twelve months a final report only is required. Report forms are available from the Human Research Ethics Committee web site: (http://www.rmit.edu.au/rd/hrec_apply).

Please note that failure to submit reports will mean that a project is no longer approved, and/or that approval will be withheld from future projects.

Conditions of approval

The Human Research Ethics Committee may apply additional conditions of approval beyond the submission of annual/final reports.

Conflicts of interest

When reporting the research, the researcher should again disclose any actual or potential conflicts of interest, including any financial or other interest or affiliation that bears on the research. Conflicts of interest can arise after a project has been approved, and where they do they must be reported as soon as possible.

Amendments

If, as you proceed with your investigation you find reason to amend your research method, you should advise the Human Research Ethics Committee and seek approval for the proposed changes. If you decide to discontinue your research before its planned completion you must also advise the Committee of this and of the circumstances. Depending on the type of amendment — whether it is minor or major — will determine how long the review process for an amendment will take.

Storage of Data

All data should *normally* be stored on University Network systems. These systems provide high levels of manageable security and data integrity, can provide secure remote access, are backed on a regular basis and can provide Disaster Recover processes should a large scale incident occur. The use of portable devices such as CDs and memory sticks is valid for archiving, data transport where necessary and some works in progress. The authoritative copy of all current data should reside on appropriate network systems; and the principal investigator is responsible for the retention and storage of the original data pertaining to the project for a minimum period of five years.

If you anticipate any problems in meeting this requirement please contact me to discuss an alternative secure data storage arrangement.

All reports or communication regarding this project is to be forwarded to the Ethics Executive Officer.

National Ethics Application Form (NEAF)

As you may be aware during 2008 RMIT University is introducing a number of reforms to human research ethics arrangements including the format of applications. Currently RMIT accepts applications on a variety of forms. However, it is intended that by the middle of the year all RMIT applications would be lodged on the online-based National Ethics Application Form. Therefore you are requested to use this format when you next make an application. For further information and to access NEAF see:

https://www.neaf.gov.au/

On behalf of the Human Research Ethics Committee I wish you well with your research.

Yours sincerely,

Peter Burke

Ethic Executive Officer -RMIT Human Research Ethics Committee

cc: Prof Marc Cohen

Appendix 2. Clinical trial registration

ClinicalTrials.gov
Protocol Registration System

Protocol Registration Preview

A Study of Yoga for Treating Geriatric Insomnia

This study has been completed.

Sponsor:	Shaare Zedek Medical Center
Collaborators:	Royal Melbourne Institute of Technology University The Australia-Israel Scientific Exchange Foundation (AISEF) Israel Yoga Teachers Association
Information provided by:	Shaare Zedek Medical Center
ClinicalTrials.gov Identifier:	NCT00661843

Purpose

Insomnia is common in the elderly population and is associated with increased health problems, reduced quality of life and greater use of sleep inducing drugs. This research aims to examine the effectiveness of Yoga practice to treat insomnia in elderly people, determine the ability to enhance their quality of life and determine if it is suitable to western culture and conditions.

Condition	Intervention	Phase
Primary Geriatric Insomnia Sleep Initiation and Maintenance Disorders	Integrated Yoga style practice	N/A

Study Type: Interventional
Study Design: Supportive Care, Parallel Assignment, Open Label, Non-Randomized, Safety/Efficacy Study

Official Title: A Mixed Design, With Wait List Control Crossover to Intervention, Clinical Trial of Integrated Yoga Style Practice Intervention for Improving Sleep and Quality of Life in the Elderly Population

Further study details as provided by Shaare Zedek Medical Centre:
Primary Outcome Measure:

- Subjective sleep and life quality assessment using standard questionnaires and sleep logs

  All measures taken at baseline and post intervention for all participants participant who crossed over measured pre and post control phase and post intervention phase

- Objective home based sleep studies using Embletta mobile sleep recording system in conjunction with the HPC1000 sleep analysis system.

  All measures taken pre and post intervention. Participants who crossed over measured pre control, post control and post intervention phases

Enrolment: 74
Study Start Date: May 2008
Study Completion Date: February 2009
Primary Completion Date: January 2009

Arms	Assigned Interventions
Experimental: 1 Intervention group: Intervention constitutes of 2 supervised yoga classes per week incorporating gentle Yoga postures, relaxation and meditation sequences In addition: daily home based sessions of yogic relaxation and meditation using a pre-recorded audio CD	Integrated Yoga style practice Intervention constitutes of 2 supervised yoga style classes per week incorporating gentle postures, relaxation and meditation sequences. In addition daily home based sessions of yogic relaxation, and meditation a an audio CD
No Intervention: 2 Wait List Control to be crossed over after control phase completed. Participants of this group studied using same objective and subjective outcome measures.	

Introduction: Geriatric insomnia is prevalent, reducing life quality, diminishing cognition and increasing risk of accidents and mortality. Treatment with sedative-hypnotic drugs has limited effectiveness and further increases the risk of accidents and falls. Yoga has been shown to increase well-being in the elderly.

Hypotheses

1. Integrated yoga style practice can improve sleep quality/quantity
2. Integrated yoga style practice can improve quality of life.

Objectives:

1. Examine effectiveness of yoga for insomnia and reduction in use of hypnotics/relaxants in the elderly;

2. Determine whether yoga enhances quality of life in the elderly; and 3. Determine whether yoga is suitable for elderly in western culture(s).

Methods:

A mixed design crossover controlled trial (n =74, age range 60-87, M = 74.4, SD = 7.1) with 2 weekly classes incorporating physical and meditative yoga, and daily home practice of meditative yoga for 12 weeks. Measures included self-reported assessment of sleep quality (Sleep Logs, KSS, ESS, PSQI, and MAP), mood states (DASS, POMS), general health (SF-36) and mobile objective home sleep studies.

Eligibility

Ages Eligible for Study: 60 Years and older

Genders Eligible for Study: Both

Inclusion Criteria:

- Age 60 years or older
- Independent, self mobile
- Suffering from light to severe primary insomnia

Exclusion Criteria:

- Age less than 60 years
- Currently or in the past engaged in regular yoga practice
- Suffering from any physical or mental health condition or disability which may affect sleep, or for which Yoga practice is counter indicated or may lead to health risks or complications.
- Suffering from co-morbid insomnia in conjunction with another condition clearly salient to sleep disturbance.

Contacts and Locations

Locations

Israel, Israel

Shaare Zedek Medical Center, Sleep Lab

Jerusalem, Israel, Israel, 91031

Investigators

Study Chair:	Anda Baharav, MD	Shaare Zedek Medical Center
Study Chair:	Jonathan Halpern, M.Sc.	Royal Melbourne Institute of Technology University
Principal Investigator:	Clement Cahan, MD	Shaare Zedek Medical Center

More Information

Responsible Party:	Shaare Zedek Medical Center (Dr. Clement Cahan, head of the Sleep Lab, Shaare Zedek Medical Center)
Study ID Numbers:	SZMCSL0001
Health Authority:	Israel: Ministry of Health

Appendix 3. Medical questionnaires and interview formats

Appendix 3.1 Initial Telephone Screening Interview Format (English Version)

Hello. My name is [interviewer's name]. I am part of a research team at the Shaare Zedek Medical Centre, Jerusalem, and conducting research on the effect of Yoga practice on Insomnia in the elderly aged 60 years and over. It is a joint effort of Shaare Zedek medical Centre and R.M.I.T. University. I am working with Dr. Clement Cahan Director of the sleep lab at Shaare Zedek Medical centre. The information we collect will help us develop better methods to help elderly people suffering from insomnia.

First, let me give you some general information about this study and what it will require from you as a participant: The study will be conducted over 12 weeks. Participants may be assigned to a Yoga practice group or to a waiting group. The assignment may be random so there is no way of knowing in advance which group you will be assigned to. A condition for joining the study is to agree to join whatever group you are assigned to. The waiting group will wait for 3 to 4 months before being assigned to a Yoga practice group.

Participants in a yoga group will be required to attend two gentle yoga classes per week and also practice special relaxation and meditation exercises at home. The yoga classes have been designed to suit elderly people. Each class will be 1 hour in duration. You will need to be able to come to classes by private or public transport. Classes and will be held at a convenient venue close to the Shaare Zedek medical centre, with lots of parking space and convenient access to public transport. The practice rooms will be clean and well ventilated. We will

provide chairs and yoga mats. Participants will need to come in comfortable loose cloths. The yoga teachers are all experienced, qualified and certified by the Yoga Teachers Association.

The daily home practice includes relaxation and meditation exercises which are done while lying in bed or sitting in a chair following pre-recorded instructions on an audio CD. You need an audio CD player in order to listen to the CD. We can provide audio cassettes to people who only have a cassette player. If you have an MP3 player, we can provide an internet website link where you can download the entire audio CD in MP3 format.

Scientific research requires collecting information. Your privacy will be respected. We will only collect information about your sleep, health and well being. All data we collect will be kept strictly confidential. No personal information will be disclosed to anyone outside the research team and will never be published anywhere. In order to be a candidate to join the study, you will need to provide us with a letter from your doctor, including full medical information and any other information relating to your sleep disturbances. For the purpose of collecting data you will also be invited to the medical centre several times for approximately one hour to fill questionnaires and attend medical interviews by doctors of our team. You will have an opportunity to discuss any concerns you may have related to your health and sleep with our medical team.

We will also monitor your sleep overnight two or three times. This will be done using a mobile sleep lab which will enable you to sleep at home in your own bed. The mobile sleep lab consists of a small recording unit you wear with a

strap around your waist. The recorder has several wires which are connected to sensors that are applied with stickers to your finger and several places on your body. These sensors are external - non invasive and harmless. During the 12 weeks of yoga program you will be asked to fill daily logs with information related your sleep patterns and daily routine.

Do you think you may be interested in joining the study? If so, I would like to ask you a few questions, just to make sure you fit the joining criteria of this particular study.

1) This research studies people aged 60 and over. Is your age 60 or over?
2) All forms and questionaries are in Hebrew (or English if you prefer) do you think you will be able to understand and reply to these questionnaires?
3) Are you currently suffering from any serious medical condition?
4) Are you currently suffering from any psychiatric condition?
5) Do you currently have any other serious personal problem at present? If so:
    1. How does it affect you life and sleep?
    2. Are you currently seeking professional help for this?
6) Are you currently getting professional help for any condition or problem?
7) Have you ever attended a sleep lab? Seen a sleep specialist? If so- can you remember the diagnosis?

<u>If candidate fits criteria so far:</u>

Thank you. I think you may be a suitable candidate. In order to continue to the next screening stage we need some information from your doctor. I will mail

you a special form for your doctor. Please mail back the forms and attach the information that your doctor provides. The form has our address and phone number. You or your doctor can contact us if you have further questions.

Can you please give me your full name, mailing address and phone number?

<u>If candidate does not fit criteria:</u>

I am sorry but from the information you have provided it seems that you would not be able to attend this particular study.

Thank you very much for your time,

Appendix 3.2 Medical Approval Form by Personal Physician (Hebrew Version)

טופס אישור רפואי

לכל המעוניין,

א.ג.נ.,

אני הח"מ מאשר/ת בזה

כי גב' / מר _____

כשיר/ה לפעילות גופנית במסגרת סדנת יוגה (התרגילים מותאמים לגיל המבוגר).

נתוני דופק ולחץ דם של הנ"ל:

דופק: _____

לחץ דם: _____

בכבוד רב

ד"ר _____

מס הסמכה: _____

טלפון: _____

תאריך: _____

<u>נא לצרף תדפיס רפואי</u>

<u>נא להחזיר את הטופס בהקדם ל</u>

הלפריות מעבדת שינה עבור מחקר שינה שערי צדק
מרכז רפואי שערי צדק, ירושלים 96222
02-6666319

Appendix 3.3 Approval of patient's participation in study by personal physician (English version)

Medical approval form

To whomever it may concern,

I hereby confirm that

Mr./Mrs./Ms._____

Is physically fit to participate in gentle yoga training

I have checked heart rate and blood pressure and they are:

Heart rate:_____

Blood pressure_____/_____

_____

(Physician signature and stamp)

Regards,

Dr._____

Licence no._____ :

Phone no._____ :

Date:_____

Please attach full medical transcript to this form

Please return the form to

J. Halpern, Sleep lab, Shaare Zedek Medical centre, Jerusalem02-6666319

Appendix 3.4 General demographic and medical form (Hebrew version)

מחקר יוגה לנדודי שינה – טופס רפואי כללי

תאריך:_____

שם המשתתף:_____

גיל:_____משקל:_____גובה:_____

מצב בריאות כללי:

<u>מחלות רקע:</u>

לב: _____יתר לחץ דם: _____

סכרת: _____כליות: _____

נשימה: _____ אלרגיות: _____

סרטן: _____כאבים: _____

אחר:

בעיות נפשיות: מתח\חרדה_____

סיפור משפחתי של הפרעות שינה\נדודי שינה

<u>רשימת תרופות</u> :

1._____2._____3._____

4._____5._____6._____

תרופות שינה:_____ תרופות הרגעה:_____

שאלות:

תלונות:

<u>למילוי על ידי הצוות הרפואי שערי צדק</u>:

הערות:

המלצות:

רופא בודק:

חתימה:

Appendix 3.5 General Demographic and Medical form (English version)

Yoga for geriatric insomnia – general medical form

Date:_____:

Participant name (first/middle/last) _____:

Age_____weight_____height_____:

General health

Physiological Medical history

cardio_____hypertension_____

Diabetes_____kidneys_____

Breathing_____ :allergies_____

Cancer_____ pains_____ :

Mental conditions, stress/anxiety/depression_____

Family history of sleep disorders_____

Current medications

_____.3_____.2_____.1

_____.6_____.5_____.4

Hypnotics _____relaxants_____ :

Questions:

Complaints:

Fields below to be filled by the medical staff at SZMC

Comments:

Recommendation:

Physician's name:

Signature:

Appendix 4. Consent and invitation forms

Appendix 4.1 Prescribed Consent Form for Persons Participating In Research Projects Involving Tests and/or Medical Procedures (RMIT English versions)

Portfolio Science, Engineering & Technology

School of Health Sciences

Name of participant: _____

Project Title: A clinical trial to assess the effectiveness of Yoga style practice for improving sleep and quality of life in an elderly population

Name(s) of investigators: (1) Jonathan Halpern (RMIT) Phone 03 9925 7440

(2) Prof. Marc Cohen (RMIT) Phone 03 9925 7440

(3) Dr. Gerard Kennedy (Victoria Uni.) Phone 03 991 92481

1. I have received a statement explaining the tests/procedures and interview/questionnaire involved in this project.
2. I consent to participate in the above project, the particulars of which - including details of tests or procedures or interviews or questionnaires - have been explained to me.
3. I authorise the investigator or his or her assistant to use with me the tests or procedures or to interview me or administer a questionnaire referred to in 1 above.
4. I acknowledge that:
   (a) The possible effects of the tests or procedures have been explained to me to my satisfaction.

(b) I have been informed that I am free to withdraw from the project at any time and to withdraw any unprocessed data previously supplied (unless follow-up is needed for safety).

(c) The project is for the purpose of research and/or teaching. It may not be of direct benefit to me.

(d) (d) The privacy of the personal information I provide will be safeguarded and only disclosed where I have consented to the disclosure or as required by law.

(e) (e) The security of the research data is assured during and after completion of the study. The data collected during the study may be published, and a report of the project outcomes will be provided to all participants and scientific publications. Any information which will identify me will not be used.

Particpnt's Consent

Participant Date:

_____ _____

(Signature)

Witness: Date

_____ _____

(Signature)

Participants should be given a photocopy of this consent form after it has been signed.

Any complaints about your participation in this project may be directed to the Executive Officer, RMIT Human Research Ethics Committee, Research & Innovation, RMIT, GPO Box 2476V, Melbourne, 3001. The telephone number is (03) 9925 2251.

Details of the complaints procedure are available from the above address.

Prescribed Consent statement Version no. 1, 10 Nov. 07

Appendix .4.2 Prescribed consent form for persons participating in research projects involving tests and/or medical procedures (SZMC English version)

Hospital	Shaare Zedek Medical Centre
Department	Sleep research centre
Name of participant:	
Project Title:	A clinical trial to assess the effectiveness of Yoga style practice for improving sleep and quality of life in an elderly population
Name(s) of investigators:	

(1) Dr. Clement Cahan (SZMC) Phone: 02 666 6319

(2) Jonathan Halpern (at SZMC) Phone: 02 666 6319

5. I have received a statement explaining the tests/procedures and interview/questionnaire involved in this project.
6. I consent to participate in the above project, the particulars of which - including details of tests or procedures or interviews or questionnaires - have been explained to me.
7. I authorise the investigator or his or her assistant to use with me the tests or procedures or to interview me or administer a questionnaire referred to in 1 above.
8. I acknowledge that:

(f) The possible effects of the tests or procedures have been explained to me to my satisfaction.

(g) I have been informed that I am free to withdraw from the project at any time and to withdraw any unprocessed data previously supplied (unless follow-up is needed for safety).

(h) The project is for the purpose of research and/or teaching. It may not be of direct benefit to me.

(d) The privacy of the personal information I provide will be safeguarded and only disclosed where I have consented to the disclosure or as required by law.

(e) The security of the research data is assured during and after completion of the study. The data collected during the study may be published, and a report of the project outcomes will be provided to all participants and scientific publications. Any information which will identify me will not be used.

Participnt's Consent

Participant: Date:

_____ _____

(Signature)

Witness: Date

_____ _____

(Signature)

Participants should be given a photocopy of this consent form after it has been signed.

Any complaints about your participation in this project may be directed to the Executive Officer, Shaare Zedek Research Ethics Committee, Shaare Zedek Medical Centre, P. O. Box 3235 Jerusalem 91031 Israel

Plain language statement Version no. 1, 10 Nov. 07

Appendix 4.3 Consent form - Israel ministry of health – 'Helsinki' committee-

(Hebrew version)

_____מספר הבקשה בוועדת הלסינקי (למילוי על-ידי מזכירות הוועדה):

אני החתום מטה:

	שם פרטי ומשפחה:
	מספר תעודת זהות:
מיקוד:	כתובת:

א) מצהיר/ה בזה כי אני מסכים/ה להשתתף במחקר רפואי[1], כמפורט במסמך זה.

ב) מצהיר/ה בזה, כי אני משתתף/לא משתתף[2] בזמן חתימת מסמך זה, בניסוי רפואי אחר במשך כל תקופת מחקר זה.

ג) מצהיר/ה בזה כי הוסבר לי על-ידי:

שם החוקר/חוקר המשנה המסביר:

1. כי החוקר הראשי (שם הרופא): ד"ר קלמנט כהאן קיבל ממנהל המוסד הרפואי, בו ייערך הניסוי, אישור לביצוע המחקר הרפואי בבני-אדם, כמשמעותו בתקנות בריאות העם (ניסויים רפואיים בבני-אדם) תשמ"א-1980 (להלן הניסוי הרפואי).
2. כי לחוקר הראשי ולחוקרי המשנה יש/אין[1] זיקה ליוזם המחקר[3].
3.

פרט: כי המחקר הרפואי נערך בנושא: **תרגול בסגנון יוגה אינטגרטיבית לשיפור** –אם יש **איכות השינה ואיכות החיים של האוכלוסיה המזדקנת**

4. כי אני חופשי/ה לבחור שלא להשתתף במחקר הרפואי, וכי אני חופשי/ה להפסיק בכל עת את השתתפותי במחקר, כל זאת מבלי לפגוע בזכותי לקבל את הטיפול המקובל.
5. כי במקרה של מילוי שאלון – אני רשאי/ת שלא לענות על כל השאלות שבשאלון או על חלק מהן.
6. כי מובטח לי שזהותי האישית תשמר סודית על-ידי כל העוסקים והמעורבים במחקר ולא תפורסם בכל פרסום, כולל בפרסומים מדעיים.

[1] מחקר רפואי, כולל: לקיחת דמים, שאלונים, מחקר אפידמיולוגי, מחקר בדגימות רקמה וכו', פרט למחקר גנטי.
[2] מחק את המיותר
[3] אם החוקר הראשי הוא גם יוזם המחקר, יש לציין זאת במפורש.

7. כי המוסד הרפואי פעל להסדרת כיסוי ביטוחי הולם של החוקרים, הרופאים והצוות הרפואי העוסקים בניסוי הקליני מפני תביעות שיוגשו ע"י משתתפים בניסוי הקליני ו/או תביעות צד ג' הקשורות עם הניסוי הקליני בין בתקופת ביצוע הניסוי ובין לאחריו. אין באמור כדי לפגוע בזכויותיי על פי כל דין.
8. כי מובטחת לי נכונות לענות לשאלות שיועלו על-ידי וכן האפשרות להיוועץ בגורם נוסף (לדוגמא רופא-משפחה, בני משפחה וכו'), באשר לקבלת החלטה להשתתף בניסוי הרפואי ו/או להמשיך בו.
9. כי בכל בעיה הקשורה לניסוי הרפואי אוכל לפנות לפרופ'/ד"ר_____

, בכל שעות היממה._____מספר טלפון/משיבון:

הנני מצהיר/ה כי נמסר/ה לי מידע מפורט על המחקר הרפואי, על פי הנושאים המפורטים להלן:

1. מטרות המחקר;
2. התקופה הצפויה למשך ההשתתפות במחקר והמספר בקירוב של המשתתפים במחקר;
3. תיאור ההליכים השונים במשך תקופת המחקר, תוך הבחנה ברורה בין ההליכים המחקריים לבין ההליכים המקובלים ברפואה;
4. היתרונות הצפויים למשתתף או לאחרים, כתוצאה מהמחקר;
5. הסיכונים הידועים ו/או אי-הנוחות שניתן לחזותם למשתתף במחקר;
6. מידע רלוונטי אחר:

ד) הנני מצהיר/ה בזה, כי את הסכמתי הנ"ל נתתי מרצוני החופשי וכי הבנתי את כל האמור לעיל. כמו-כן, קיבלתי עותק של טופס הסכמה מדעת זה, נושא תאריך וחתום כדין.

ה) עם חתימתי על טופס הסכמה זה, הנני מתיר ליוזם המחקר הרפואי, לוועדת הלסינקי המוסדית, לגוף המבקר במוסד הרפואי ולמשרד הבריאות גישה ישירה לתיקי הרפואי, לשם אימות שיטות המחקר הרפואי והנתונים הקליניים. גישה זו למידע הרפואי שלי תבוצע תוך שמירת סודיות, בהתאם לחוקים ולנהלים של שמירת סודיות.

תאריך	חתימת המשתתף/ת במחקר	שם המשתתף/ת במחקר הרפואי

במקרה הצורך[4]

תאריך	חתימת העד	מספר תעודת זהות	שם העד הבלתי תלוי

<u>הצהרת החוקר/חוקר המשנה</u>:

ההסכמה הנ"ל נתקבלה על-ידי, וזאת לאחר שהסברתי למשתתף/ת בניסוי הרפואי כל האמור לעיל וכן וידאתי שכל הסבריי הובנו על-ידו/ידה.

תאריך	חתימתו	שם החוקר/חוקר המשנה המסביר

[4] תאריך החתימה.

Appendix 4.4 Invitation to participate in a research project (English version)

PROJECT INFORMATION STATEMENT

Project Title*:*

Integrated yoga style practice for improving sleep and quality of life in the elderly population

Investigators*:*

1. Dr. Clement Cahan, Director of Sleep Lab at Shaare Zedek Medical Centre
2. Dr. Anda Baharav, Sleep specialist, Sleep Lab at Shaare Zedek Medical Centre
3. Mr Jonathan Halpern (PhD degree student, School of Health Sciences, RMIT University, jonthan.halpern@student.rmit.edu.au, 02-6666319)
4. Prof. Marc Cohen (Senior Supervisor, School of Health Sciences, RMIT University, marc.cohen@rmit.edu.au, 03- 99257440)
5. Dr. Gerard Kennedy (Supervisor: Victoria University, School of Psychology, Gerard.kennedy@vu.edu.au, 03- 9919 2481)

Dear Sir / Madam,

You are invited to participate in a research project being conducted by Shaare Zedek Medical Centre and RMIT University in the Jerusalem area. This information sheet describes the project in straightforward language, or 'plain English'. Please read this sheet carefully and be confident that you understand its contents before deciding whether to participate. If you have any questions about the project, please ask one of the investigators.

Who is involved in this research project? Why is it being conducted?

My name is Jonathan Halpern. I am a PhD student at RMIT school of Health Sciences. My supervisors are Dr. Clement Cahan, Director of Sleep Lab at Shaare Zedek Medical Centre (SZMC), Dr. Anda Baharav, Sleep specialist, Sleep Lab at SZMC, Professor Marc Cohen PhD MBBS from RMIT School of Health Sciences, currently President of the Australasian Integrative Medicine Association and Dr. Gerard Kennedy, Victoria University – Dept. of Psychology, a medical psychologist and sleep and insomnia expert. This study is conducted as part of my research towards a PhD degree. It has been approved by the Human Research Ethics Committees at RMIT and at SZMC.

Why have you been approached?

You have been approached at random because you may fit the criteria (healthy adult, 60 years or older, suffering from disturbed sleep). If you fit these criteria then you may apply to participate in the trial.

What is the project about? What are the questions being addressed?

Insomnia is common in the elderly population and is associated with increased health problems, reduced quality of life and greater use of sleep drugs. This research aims to examine the effectiveness of Integrated Yoga style practice for treating insomnia in elderly people, and enhancing their quality of life. We hope to find out whether:

1. Integrated yoga style practice can improve sleep quality/quantity
2. Integrated yoga style practice can improve quality of life.

We expect to have at least two groups. One group will undergo yoga training. The other group will serve as Wait List Control group and will join a yoga group later.

If I agree to participate, what will I be required to do?

Participants in a Yoga group will be required to attend - free of charge - Yoga style training, two times per week for three months. Each session will be one hour long. Yoga training will include gentle Yoga poses, relaxation and meditation exercises. Our Yoga teachers are experienced and certified. Yoga group participants will also be required to perform relaxation, and meditation exercises daily using an audio CD supplied free of charge. Participants will be required to fill out sleep logs daily and various forms and questionnaires before and after the study including, sleep quality and life quality questionnaires. Participants will also be required to spend up to 3 nights connected to a mobile sleep measurement device (at home) to monitor the quality and quantity of their sleep. These measurements are very safe and non invasive and measure electrical activity of the heart, movement, position, oxygen saturation etc.

We extend an invitation to anyone interested in participating to come to our office, view the questionnaire forms and view a folder which contains additional information about the intervention

What are the risks or disadvantages associated with participation?

The risk of injury or adverse physical or emotional response resulting from gentle yoga or relaxation practice during the trial is low. However, as in any kind of physical exercise there is always a risk of discomfort, pain, or unexpected injury. The staff will do their utmost to minimise these risks. Any complaint or concern you report will be dealt with promptly. Participation in this research is voluntary and you may withdraw at anytime. If you are unduly concerned about your responses to any of the questionnaire items or if you find participation in the project emotionally or physically distressing or difficult in any way, you should contact us to discuss your concerns with confidentiality and suggest appropriate follow-up, if necessary.

If any of the medical or sleep examinations, during the trial, reveal any data which is in your interest to know, such as any serious health risk, you may be contacted and referred to someone who can be of assistance.

What are the benefits associated with participation?

All Participants in the study will undergo free sleep assessment as well as some other free non invasive medical examinations. If any of these reveal any data which is in your interest to know, you may be contacted and referred to someone who can be of assistance. Participants in the Yoga group (s) will also gain the benefit of undergoing free yoga and relaxation training for three months. Similar trials in the past have reported some improvement in various aspects of participants' physical and emotional well being, and sleep quality as well. Participants will gain basic knowledge of yoga practice, meditation and relaxation techniques, which they may choose to continue applying in their lives beyond the scope of this trial.

What will happen to the information I provide?

Any information that you provide can be disclosed only if (1) to protect you or others from harm, or (2) a court order is produced, or (3) you provide the researchers with written permission.

Participants need to be identified in the research records only, for health and safety reasons, i.e. to enable us to contact a participant if necessary in case any of the medical or sleep data, during the trial, reveal anything which is in your interest to know, such as any serious health risk. The data collected will be analysed by me, my supervisors, and by sleep lab staff. Data will be used for research and the results may be disseminated in reports, scientific papers, academic conferences etc. The results will be aggregated and reported in a manner, which maintains participant's total anonymity. The research data will be kept securely at SZMC for a period of 5 years before being destroyed.

What are my rights as a participant?

Participants' rights, include:

1. The right to withdraw their participation at any time, without prejudice.
2. The right to have any unprocessed data withdrawn and destroyed, provided it can be reliably identified, and provided that so doing does not increase the risk for the participant.
3. The right to have any questions answered at any time.
4. The right not to perform any exercise which they feel may be harmful or stressful to them in any way.

Whom should I contact if I have any questions?

Please contact the following people, if you have any further questions:

1. Prof. Marc Cohen, School of Health Sciences, RMIT University, marc.cohen@rmit.edu.au, 03- 99257440
2. Dr. Gerard Kennedy (Victoria University, School of Psychology, Gerard.kennedy@vu.edu.au, 03- 9919 2481
3. Dr. Clement Cahan, Director of Sleep Lab at Shaare Zedek Medical Centre , Jerusalem, 02-6666319

What other issues should I be aware of before deciding whether to participate?

We require that you provide us with accurate and full information before during and after the trial in the questionnaires and others examinations. This is essential to help us ensure your health and safety. Also, please report any problem immediately to your Yoga instructor or to the investigators. We also require that participants fully accept in advance the selection and assignment process. We also require that all participants attend all scheduled examinations, fill out fully and submit all questionnaires on time, attend all yoga practice sessions and perform the relaxation exercises as prescribed at home (for those in the yoga group). The success of the trial as well as your well being depends on your full cooperation. We ask that you join only if you are fully committed to comply with the requirements and believe that you will be able to do so. Yours sincerely

Name: *Jonathan Halpern* 15 Nov. 07

(Signature of Principal Investigator)

Mr Jonathan Halpern (PhD degree student, School of Health Sciences, RMIT University)

Name: 15 Nov. 07

(Signature of senior supervisor if applicable)

Prof. Marc Cohen (PhD, MBBS, Senior Supervisor, School of Health Sciences, RMIT University)

Name: 15 Nov. 07

(Signature of Investigator)

Dr. Gerard Kennedy (PhD, Medical Psychologist, Senior lecturer Victoria University, School of Psychology)

Any complaints about your participation in this project may be directed to the Executive Officer, RMIT Human Research Ethics Committee, Research & Innovation, RMIT, GPO Box 2476V, Melbourne, 3001.

Details of the complaints procedure are available at: http://www.rmit.edu.au/rd/hrec_complaints

Plain language statement Version no. 1, 10 Nov. 07

Appendix 5. Subjective measures questionnaires (English versions)

Appendix 5.1 Karolinska Sleepiness Scale (KSS)

The following is a nine point scale to describe your sleepiness level. Tick the box next to the point that describes how SLEEPY you feel RIGHT NOW.

1.	☐	**extremely alert**
2.	☐	
3.	☐	**alert**
4.	☐	
5.	☐	**neither alert nor sleepy**
6.	☐	
7.	☐	**sleepy-but no difficulty remaining awake**
8.	☐	
9.	☐	**extremely sleepy-fighting sleep**

Appendix 5.2 Epworth Sleepiness Scale (ESS)

The following questions refer to sleepiness or the tendency to doze off when relaxed. How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in recent times. Even if you haven't done some of these things recently, try to work out how they would have affected you. Choose the most appropriate number for each situation by putting an X in one box for each question.

	0	1	2	3
Situation ↓	**never**	**slight chance**	**Moderate chance**	**High chance**
1) Sitting and reading				
2) Watching TV				
3) Sitting, inactive in a public place (e.g. theatre or a meeting)				
4) As a passenger in a car for an hour without a break				
5) Lying down to rest in the afternoon when circumstances permit				
6) Sitting and talking to someone				
7) Sitting quietly after a lunch without alcohol				
8) In a car, while stopped for a few minutes in traffic				

Appendix 5.3 Pittsburgh Sleep Quality Questionnaire (PSQI)

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

(1) During the past month, what time have you usually gone to bed at night?

BED TIME: _____

(2) During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

NUMBER OF MINUTES: _____

(3) During the past month, what time have you usually gotten up in the morning?

GETTING UP TIME: _____

(4) During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed)

HOURS OF SLEEP PER NIGHT: _____

(5) For each of the following questions, tick X in the box [ ] of the one response that best describes your sleeping patterns. Please answer all questions.

During the past month, how often have you had trouble sleeping because you:

	Not during the past month	**Less than once a week**	**Once or twice a week**	**Three or more times a week**
a. Cannot get to sleep within 30 minutes	[ ]	[ ]	[ ]	[ ]
b. Wake up in the middle of the night or early morning	[ ]	[ ]	[ ]	[ ]
c. Have to get up to use the bathroom	[ ]	[ ]	[ ]	[ ]
d. Cannot breath comfortably	[ ]	[ ]	[ ]	[ ]
e. Cough or snore loudly	[ ]	[ ]	[ ]	[ ]
f. Feel too cold	[ ]	[ ]	[ ]	[ ]

g. Feel too hot	[ ]	[ ]	[ ]	[ ]
h. Had bad dreams	[ ]	[ ]	[ ]	[ ]
i. Have pain	[ ]	[ ]	[ ]	[ ]

j. Other reason(s), please describe:

k. How often during the past have you had trouble sleeping because of the above reasons

Not during the past month	**Less than once a week**	**Once or twice a week**	**Three or more times a week**
[ ]	[ ]	[ ]	[ ]

(6) During the past month, how would you rate your sleep quality overall?

- Very good [ ]
- Fairly good [ ]
- Fairly bad [ ]
- Very bad [ ]

(7) During the past month, how often have you taken medicine to help you sleep (Prescribed or "over the counter")?

- Not during the past month [ ]
- Less than once a week [ ]
- Once or twice a week [ ]
- Three or more times a week [ ]

(8) During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

- Not during the past month [ ]
- Less than once a week [ ]
- Once or twice a week [ ]
- Three or more times a week [ ]

(9) During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

- No problem at all [ ]
- Only a very slight problem [ ]
- Somewhat of a problem [ ]
- A very big problem [ ]

(10) Do you have a bed partner or roommate?

- No bed partner or roommate [ ]
- Partner/roommate in other room [ ]
- Partner in same room, but not same bed [ ]
- Partner in same bed [ ]

(11) If you have a roommate or bed partner, ask him/her how often in the past month you have had:

	Not during the past month	**Less than once a week**	**Once or twice a week**	**Three or more times a week**
a. Loud snoring	[ ]	[ ]	[ ]	[ ]
b. Loud pauses between breaths while asleep	[ ]	[ ]	[ ]	[ ]
c. Legs twitching or jerking while you sleep	[ ]	[ ]	[ ]	[ ]
d. Episode of disorientation or confusion during sleep	[ ]	[ ]	[ ]	[ ]

e. Other restlessness while you sleep; please describe:

_____

_____

f. How often during the past have you had trouble sleeping because of the above reason?

Not during the past month	**Less than once a week**	**Once or twice a week**	**Three or more times a week**
[ ]	**[ ]**	**[ ]**	**[ ]**

Appendix 5.4 Multivariate Apnea Prediction index (MAP) questionnaire

The following questions are about your sleep

During the last month, have you had, or have you been told about the following symptoms: (show the frequency by putting a cross in one box)

During the last month, have you had, or have you been told about the following symptoms: (show the frequency by putting a cross in one box)

	0	1	2	3	4	0
Frequency ➔ **Symptoms ⬇**	never	rarely	1-2 times p/week	3-4 times p/week	5-7 times p/week	Don't know
1. snorting or gasping						
2. loud snoring						
3. breathing stops, choke or struggle for breath						
4. frequent awakenings						
5. tossing, turning or thrashing						
6. difficulty falling asleep						
7. legs feel jumpy or jerky						
8. morning headaches						
9. falling asleep when at work or school						
10. falling asleep when driving						
11. excessive sleepiness during the day						
12. awaken feeling paralysed, unable to move for short period's						
13. find yourself in a vivid dreamlike state when falling asleep or awakening even though you know you're awake						

Please continue to the next page ➔

Appendix 5.5 Profile of Mood States- Short Form (POMS-SF) Questionnaire

Below is a list of words that describe feelings people have. Please read each one carefully. Then, **mark an X** in the box that best describes how you have been feeling during the past week including today

	Not at all	A little	moderately	Quite a bit	Extremely			Not at all	A little	Moderately	Quite a bit	Extremely
1. Tense							**20. Discouraged**					
2. Angry							**21. Resentful**					
3. Worn out							**22. Nervous**					
4. Unhappy							**23. Miserable**					
5. Lively							**24. Cheerful**					
6. Confused							**25. Bitter**					
7. Peeved							**26. Exhausted**					
8. Sad							**27. Anxious**					
9. Active							**28. Helpless**					
10. On edge							**29. Weary**					
11. Grouchy							**30. Bewildered**					
12. Blue							**31. Furious**					
13. Energetic							**32. Full of pep**					
14. Hopeless							**33. Worthless**					
15. Uneasy							**34. Forgetful**					
16. Restless							**35. Vigorous**					
17. Unable to							**36. Uncertain**					
18. Fatigued							**37. Bushed**					
19. Annoyed												

Appendix 5.6 Depression Anxiety Stress Scale (DASS – 42) Questionnaire

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found myself getting upset by quite trivial things	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I just couldn't seem to get going	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I had a feeling of shakiness (e.g., legs going to give way)	0	1	2	3
8	I found it difficult to relax	0	1	2	3
9	I found myself in situations that made me so anxious I was most relieved when they ended	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting upset rather easily	0	1	2	3
12	I felt that I was using a lot of nervous energy	0	1	2	3
13	I felt sad and depressed	0	1	2	3
14	I found myself getting impatient when I was delayed in any way (e.g., lifts, traffic lights, being kept waiting)	0	1	2	3
15	I had a feeling of faintness	0	1	2	3
16	I felt that I had lost interest in just about everything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I perspired noticeably (e.g., hands sweaty) in the absence of high temperatures or physical exertion	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life wasn't worthwhile	0	1	2	3

Please continue on next page ➔

Reminder of rating scale:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

22	I found it hard to wind down	0	1	2	3
23	I had difficulty in swallowing	0	1	2	3
24	I couldn't seem to get any enjoyment out of the things I did	0	1	2	3
25	I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)	0	1	2	3
26	I felt down-hearted and blue	0	1	2	3
27	I found that I was very irritable	0	1	2	3
28	I felt I was close to panic	0	1	2	3
29	I found it hard to calm down after something upset me	0	1	2	3
30	I feared that I would be "thrown" by some trivial but unfamiliar task	0	1	2	3
31	I was unable to become enthusiastic about anything	0	1	2	3
32	I found it difficult to tolerate interruptions to what I was doing	0	1	2	3
33	I was in a state of nervous tension	0	1	2	3
34	I felt I was pretty worthless	0	1	2	3
35	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
36	I felt terrified	0	1	2	3
37	I could see nothing in the future to be hopeful about	0	1	2	3
38	I felt that life was meaningless	0	1	2	3
39	I found myself getting agitated	0	1	2	3
40	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
41	I experienced trembling (e.g., in the hands)	0	1	2	3
42	I found it difficult to work up the initiative to do things	0	1	2	3

Appendix 5.7 Health Survey (SF36) questionnaire

This questionnaire asks for your views about your general health, how you feel and how well you are able to do various activities. Please answer every question by checking one choice only for each. Thank you.

1. In general, would you say your health is?

□ 1. Excellent □ 2. Very good □ 3. Good □ 4. Fair □ 5. Poor

2. Compared to one year ago, how would you rate your health in general now?

□ 1. MUCH BETTER than one year ago.

□ 2. Somewhat BETTER now than one year ago.

□ 3. About the SAME as one year ago.

□ 4. Somewhat WORSE now than one year ago.

□ 5. MUCH WORSE now than one year ago.

3. The following items are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?

Activities	**1. Yes, limited a lot**	**2. Yes, limited a Little**	**3. Not limited at all**
a) Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports?			
b) Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf?			
c) Lifting or carrying groceries?			
d) Climbing several flights of stairs?			
e) Climbing one flight of stairs?			

f) Bending, kneeing or stooping?			
g) Walking more than a mile?			
h) Walking several blocks?			
i) Walking one block?			
j) Bathing or dressing yourself?			

4. During the past 4 weeks, have you had any of the following problems with your work or other regular activities *as a result of your physical health*?

	Yes	No
a) Cut down on the **amount of time** you spent on work or other activities?		
b) **Accomplished less** than you would like?		
c) Were limited in the **kind** of work or other activities?		
d) Had **difficulty** performing the work or other activities (for example it took extra effort)?		

5. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

	Yes	No
a) Cut down on the **amount of time** you spent on work or other activities?		
b) **Accomplished less** than you would like?		
c) Didn't do work or other activities as **carefully** as usual?		

6. **During the <u>past 4 weeks</u>, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbours, or groups?**

□ 1. Not at all □ 2. Slightly □ 3. Moderately □ 4. Quite a bit □ 5. Extremely

7. How much bodily pain have you had during the past 4 weeks?

□ 1. None □ 2. Very mild □ 3. Mild □ 4. Moderate □ 5. Severe □ 6. Very severe

8. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

□ 1. Not at all □ 2. A little bit □ 3. Moderately □ 4. Quite a bit □ 5. Extremely

9. These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the past 4 week …

	1. All of the time	2. Most of the time	3. A good bit of the time	4. Some of the time	5. A little of the time	6. None of the time
a) Did you feel full of pep?						
b) Have you been a very nervous person?						
c) Have you felt so down in the dumps that nothing could cheer you up?						
d) Have you felt calm and peaceful?						

e) Did you have a lot of energy?						
f) Have you felt downhearted and blue?						
g) Do you feel worn out?						
h) Have you been a happy person?						
i) Did you feel tired?						

10. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives, etc.)?

□ 1. All of the time

□ 2. Most of the time.

□ 3. Some of the time

□ 4. A little of the time.

□ 5. None of the time.

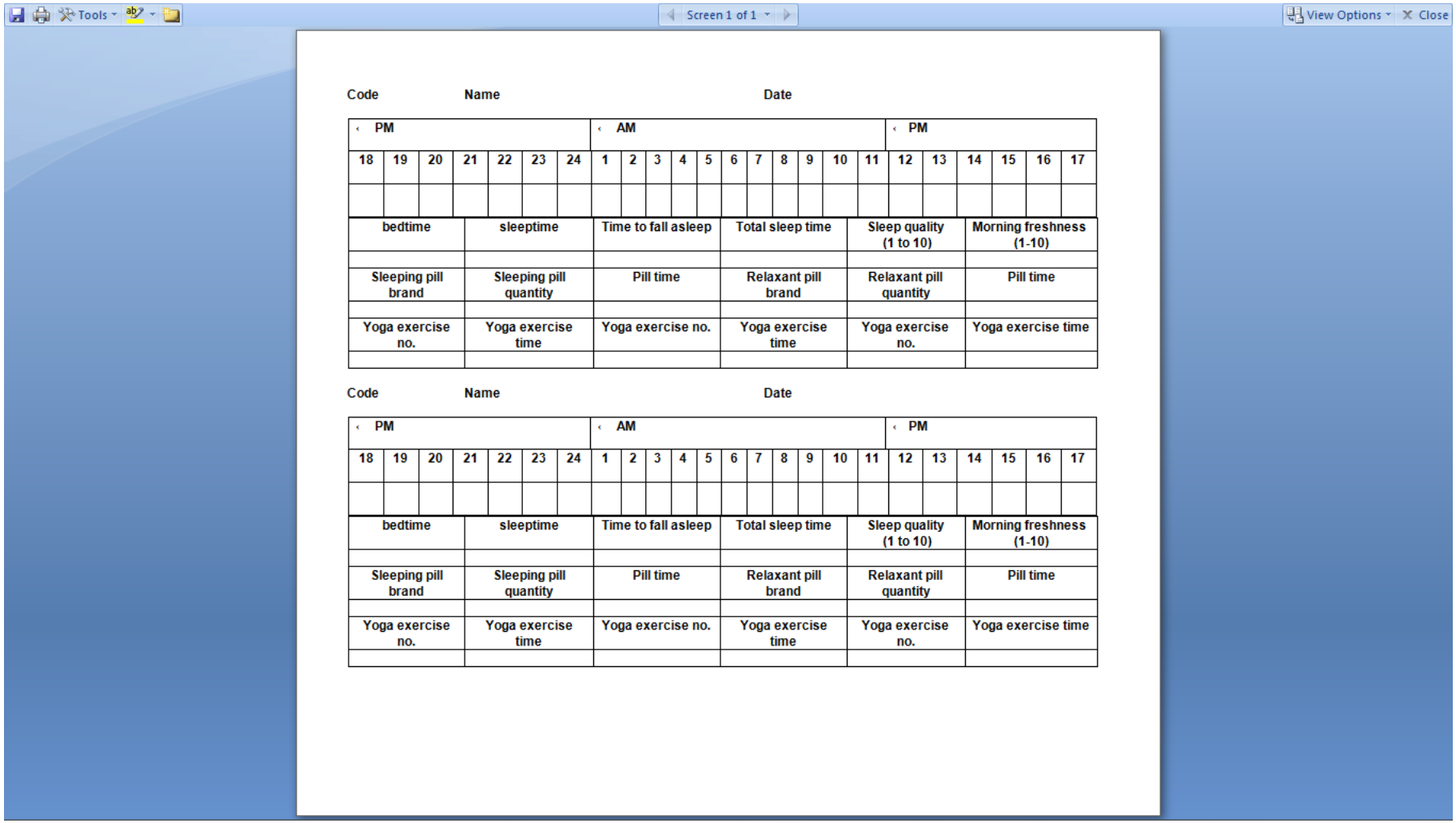
11. How TRUE or FALSE is each of the following statements for you?

	1. Definitely true	2. Mostly true	3. Don't know	4. Mostly false	5. Definitely false
a) I seem to get sick a little easier than other people?					

b) I am as healthy as anybody I know?					
c) I expect my health to get worse?					
d) My health is excellent?					

➔

Appendix 5.8–Daily Sleep and practice log (DSPL) English version



Code Name Date

‹ PM							‹ AM											‹ PM						
18	19	20	21	22	23	24	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17

bedtime	sleeptime	Time to fall asleep	Total sleep time	Sleep quality (1 to 10)	Morning freshness (1-10)
Sleeping pill brand	Sleeping pill quantity	Pill time	Relaxant pill brand	Relaxant pill quantity	Pill time
Yoga exercise no.	Yoga exercise time	Yoga exercise no.	Yoga exercise time	Yoga exercise no.	Yoga exercise time

Code Name Date

‹ PM							‹ AM											‹ PM						
18	19	20	21	22	23	24	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17

bedtime	sleeptime	Time to fall asleep	Total sleep time	Sleep quality (1 to 10)	Morning freshness (1-10)
Sleeping pill brand	Sleeping pill quantity	Pill time	Relaxant pill brand	Relaxant pill quantity	Pill time
Yoga exercise no.	Yoga exercise time	Yoga exercise no.	Yoga exercise time	Yoga exercise no.	Yoga exercise time

Comments:

1. The method for filling out the table at the top of the sleep diary:
2. A period of continuous sleep is indicated with a horizontal line
3. Getting out of bed is indicated with an upward pointing arrow
4. Getting into bed is indicated with a downward pointing arrow.

The researcher is then able to determine the number and duration of mid sleep awakenings as well as early morning awakenings , bed time, sleep onset, and final arising.

Appendix 6. Subjective measures – questionnaires (Hebrew Versions)

Appendix 6.1 Karolinska Sleepiness Scale (KSS) – Hebrew version

המרכז הרפואי שערי צדק – מעבדת שינה

<u>שאלונים בנושא איכות שינה , מצב רוח ובריאות</u>

בסדרת השאלונים הבאה שאלות כיצד את/ה <u>מרגיש/ה או מעריך/ה</u> <u>את מצבך</u>. אין צורך להתלבט יותר מדי כי מדובר בהרגשה / הערכה סובייקטיבית של כל אדם לגבי עצמו <u>כרגע</u> . אין תשובות נכונות יותר או פחות. כל משתתף ימלא את שמו ואת התאריך . לאחר מכן אנו ניתן לכל שאלון קוד סודי <u>והאינפורמציה תישמר בעילום שם עם הקוד הסודי בלבד באופן שפרטיותכם תשמר לחלוטין.</u>

שם משתתף: _____
קוד משתתף:_____ תאריך:_____

נא הקף בעיגול את הספרה בשורה המתארת בצורה להלן סולם לדרוג הנטייה להירדם. המדויקת ביותר <u>כיצד אתה מרגיש ברגע זה</u>. נא לבחור באפשרות אחת בלבד. **תודה.**

1	**מאד ערני**
2	**משהו בין אפשרות 1 לאפשרות 3**
3	**ערני**
4	**משהו בין אפשרות 3 לאפשרות 5**
5	**לא ערני ולא רדום**
6	**משהו בין אפשרות 5 לאפשרות 7**
7	**רדום אך מסוגל להישאר ער בקלות**
8	**משהו בין אפשרות 7 לאפשרות 9**
9	**מאד רדום. נדרש מאמץ רב כדי להישאר ער ולא להירדם**

Appendix 6.2 EPWORTH SLEEPINESS SCALE (ESS) – Hebrew version

שם משתתף: _____
קוד משתתף:_____ תאריך:_____

עד כמה יש לך נטייה לחטוף תנומה או להירדם ממש במצבים הבאים, בניגוד לסתם הרגשת עייפות? השאלה מתייחסת לחיי היום-יום הנוכחיים שלך. אפילו אם בזמן האחרון לא עשית את אחת הפעולות ולא שהית במצבים המפורטים, נסה לשער כיצד אלה היו משפיעים עליך. השתמש בדירוג הבא (מ- 0 עד 3) על מנת לבחור את המספר המתאים ביותר לכל מצב/פעולה:

<u>נא בחר מסולם האפשרויות הבא למתן תשובה:</u>

(0) לעולם לא הייתי מנמנם במצב זה
(1) סיכוי נמוך שהייתי מנמנם במצב זה
(2) סיכוי בינוני שהייתי מנמנם במצב זה
(3) סיכוי גבוה שהייתי מנמנם במצב זה

נא בחר באפשרות אחת בלבד בכל ,נא סמן עיגול סביב הספרה של האפשרות המתאימה ביותר בשורה

<u>המצב</u>	לעולם לא הייתי מנמנם במצב זה	סיכוי נמוך שהייתי מנמנם במצב זה	סיכוי בינוני שהייתי מנמנם במצב זה	סיכוי גבוה שהייתי מנמנם
קריאה בישיבה	0	1	2	3
צפייה בטלוויזיה	0	1	2	3
ישיבה פסיבית במקום ציבורי (תיאטרון, סרט, קונצרט)	0	1	2	3
כנוסע במכונית (לא כנהג) במשך שעה רצופה	0	1	2	3
שכיבה למנוחה בשעות אחר הצהריים, כאשר הדבר מתאפשר	0	1	2	3
שיחה עם מישהו בישיבה	0	1	2	3
ישיבה רגועה לאחר ארוחת צהריים (שבה לא שתית שתייה אלכוהולית)	0	1	2	3
ישיבה במכונית (כנהג), בזמן עצירה קצרה (רמזור, פקק)	0	1	2	3

Appendix 6.3 Pittsburgh Sleep Quality Index (PSQI). – Hebrew version

שם משתתף: _____
קוד משתתף:_____ תאריך:_____

הוראות: השאלות הבאות עוסקות בהרגלי השינה שלך במהלך החודש האחרון בלבד. התשובות צריכות לשקף את מרבית הימים והלילות בחודש האחרון.
נא סמן איקס מתחת לתשובה המתאימה ביותר לאותה שאלה
נא לענות על כל השאלות תשובה אחת בלבד בכל שורה.

במהלך החודש האחרון:

1. מתי בדרך כלל הלכת למיטה? _____
2. כמה זמן (בדקות) לקח לך להירדם בכל לילה? _____
3. מתי בדרך כלל קמת בבוקר? _____
4. כמה שעות **ישנת** בלילה? (רק שעות שינה – לא שעות במטה)? _____
5. נא מלא בטבלא תדירות בעיות שינה שסבלת מהן במהלך החודש האחרון

במהלך החודש האחרון באיזה תדירות סבלת מבעיות בשינה עקב הסיבות הבאות:	**לא סבלתי בחודש האחרון**	**פחות מפעם בשבוע**	**פעם או פעמיים בשבוע**	**שלוש פעמים בשבוע או יותר**
א. לא יכול/ה להירדם במשך 30 דקות				
ב. מתעורר/ת באמצע הלילה או מוקדם בבוקר				
ג. חייב/ת לקום בלילה לשירותים				
ד. נושם/ת לא בנוח				
ה. משתעל/ת או נוחרת בחוזקה				
ו. מרגיש/ה מאד קר				
ז. מרגיש/ה מאד חם				
ח. יש לך חלומות רעים				
ט. סובל/ת מכאבים				
י. סיבות אחרות: תאר/י אותן במילים בשורה זו:				
	לא סבלתי בחודש האחרון	**פחות מפעם בשבוע**	**פעם או פעמיים בשבוע**	**שלוש פעמים בשבוע או יותר**
יא. באיזה תדירות הפריעו לך הסיבות הנ"ל לישון:				

6. במהלך החודש האחרון, כיצד את/ה מעריך/ה את איכות שנתך בסה"כ?

טובה מאד	טובה	רעה	רעה מאד

7. במהלך החודש האחרון, באיזה תדירות לקחת תרופה לשינה (במרשם או ללא מרשם)?
נא לסמן איקס תחת התשובה המתאימה ביותר. נא לבחור בתשובה אחת בלבד.

לא לקחתי כלל בחודש האחרון	לקחתי פחות מפעם בשבוע	לקחתי פעם או פעמיים בשבוע	לקחתי שלוש פעמים בשבוע או יותר

8. במהלך החודש האחרון, באיזו תדירות הייתה לך בעיה להישאר ערה בנהיגה, בארוחות, או בפגישות חברתיות?

כלל לא בחודש האחרון	פחות מפעם בשבוע	פעם או פעמיים בשבוע	שלוש פעמים בשבוע או יותר

9. במהלך החודש האחרון, עד כמה הייתה לך בעיה לשמור על התלהבות על מנת לעשות דברים?

כלל לא בעיה בחודש האחרון	בעיה במידה מועטה	בעיה במידה בינונית	בעיה במידה גדולה

10. במהלך החודש האחרון, כיצד את/ה מעריך/ה את איכות שנתך בסה"כ?

טובה מאד	טובה	רעה	רעה מאד

11. באם יש לך שותף/ה לחדר ו/או למיטה נא שאל אותו/ה באיזו תדירות היו לך התופעות הבאות בזמן השינה ? נא לסמן איקס תחת התשובה המתאימה ביותר. נא לבחור בתשובה אחת בלבד.

	כלל לא בחודש האחרון	פחות מפעם בשבוע	פעם או פעמיים בשבוע	שלוש פעמים בשבוע או יותר
א. נחירות קולניות				
ב. הפסקות ארוכות ביו נשימות				
ג. תנועות וקפיצות ברגליים				
ד. תופעות בלבול , איבוד כוון				
ה. תופעות אחרות של חוסר שקט – נא לתאר במילים				
	כלל לא בחודש האחרון	פחות מפעם בשבוע	פעם או פעמיים בשבוע	שלוש פעמים בשבוע או יותר
ו. באיזה תדירות הפרעה שנתך על ידי התופעות המתוארות בשאלה זו ?				

נא לעבור לדף הבא←

Appendix 6.4 Multivariable Apnea Prediction Index (MAP) – Hebrew

שם משתתף: _____ קוד משתתף:_____
תאריך:_____

במהלך השינה עשויות להופיע תופעות שונות בתדירות שונה מאדם לאדם. נא ציין אם במהלך החודש האחרון שמת לב (או נאמר לך ע"י אחרים) לתופעות הבאות. נא סמן בטור של האפשרות המתאימה ביותר. נא בחר באפשרות אחת בלבד בכל שורה.

	<u>תאור התופעה</u>	**לעולם לא**	**פחות מפעם בשבוע**	**1 – 2 פעמים בשבוע**	**3 – 4 פעמים בשבוע**	**5 – 7 פעמים בשבוע**	**לא יודע**
1	חרחורים או שאיפות אויר פתאומיות וחדות						
2	נחירות חזקות						
3	הפסקות נשימה, מחנק, קשיי נשימה / נשימה מאומצת						
4	מתעורר תכופות משינה						
6	קשיים להירדם						
7	רגליים קופצות או בועטות						
8	כאבי ראש בבוקר						
9	נרדם בעבודה או בלימודים						
10	נרדם תוך כדי נהיגה						
11	עייפות יתר במשך היום						
12	מתעורר בהרגשה שאתה משותק / חוסר יכולת תנועה לפרק זמן קצר						
13	תוך כדי ההרדמות או ההתעוררות משינה מוצא עצמך במצב כמו חלום אך בו זמנית מודע לכך שבעצם ער						

Appendix 6.5 Depression Anxiety Stress Scale (DASS) – Hebrew version

שם משתתף: _____ קוד משתתף:_____
תאריך:_____

נא קרא ובכל סעיף וסמן עגול אחד סביב המספר 0, 1, 2, או 3 בהתאם למידת ההתאמה להרגשתך בשבוע שחלף. נא לבחר אפשרות אחת בלבד. להלן הסולם לדירוג מידת הנכונות / התאמה של הנאמר למצבך בשבוע החולף:

0 לגמרי לא נכון
1 נכון במידה מסוימת או נכון חלק מן הזמן בלבד
2 נכון במידה רבה או נכון חלק ניכר מן הזמן
3 נכון במידה רבה מאד או נכון רב הזמן

1	אבדתי את שלוות הנפש בגלל דברים פעוטים	0	1	2	3
2	הייתי מודע ליובש בפה	0	1	2	3
3	לא הייתי מסוגל לחוות רגשות חיוביים כלשהם	0	1	2	3
4	חשתי קושי בנשימה (לדוגמה נשימות מהירות או קוצר נשימה שלא בעת מאמץ גופני)	0	1	2	3
5	התקשיתי להיכנס לפעולה / להתחיל דברים	0	1	2	3
6	נטיתי לתגובות מוגזמות במצבים שונים	0	1	2	3
7	הרגשתי חוסר יציבות (למשל חולשה ברגליים)	0	1	2	3
8	היה לי קשה להירגע	0	1	2	3
9	מצאתי עצמי במצבים שעוררו בי חרדה והרגשתי הקלה מרובה כשחלפו	0	1	2	3
10	הרגשתי חוסר תקווה	0	1	2	3
11	אבדתי את שלוות הנפש בקלות	0	1	2	3
12	הרגשתי שאני חי על 'עצבים' / מוציא אנרגית עצבים רבה	0	1	2	3
13	הייתי עצוב ומדוכא	0	1	2	3
14	אבדתי את סבלנותי כשנאלצתי להתעכב בדרך כלשהיא (למשל במעלית, ברמזור, בהמתנה בתור)	0	1	2	3
15	חשתי חולשה	0	1	2	3
16	הרגשתי שאני מאבד עניין בכל דבר שהוא	0	1	2	3
17	הרגשתי חסר ערך	0	1	2	3
18	פתחתי רגישות יתר (נפשית)	0	1	2	3
19	הזעתי בצורה מוגברת (למשל בכפות הידיים) בהעדר מאמץ גופני ולמרות שלא היה חם	0	1	2	3

20	חשתי פחד ללא סיבה הגיונית	0	1	2	3
21	הרגשתי שאין ערך לחיי	0	1	2	3
22	התקשיתי להוריד קצב ולהירגע	0	1	2	3
23	היו לי קשיים בבליעה	0	1	2	3
24	התקשיתי ליהנות ממה שעשיתי	0	1	2	3
25	הייתי מודע לפעימות ליבי (קצב לב מוגבר או לא סדיר) ללא מאמץ גופני נלווה	0	1	2	3
26	הרגשתי שפוף ומדוכדך	0	1	2	3
27	התעצבנתי בקלות	0	1	2	3
28	הייתי קרוב למצב אבדן עשתונות / פאניקה	0	1	2	3
29	היה לי קשה להירגע לאחר שמשהוא הוציא אותי משלוותי	0	1	2	3
30	חששתי שלא אצליח להתמודד עם משימה פשוטה אך חדשה	0	1	2	3
31	התקשיתי להתלהב מכל דבר שהוא	0	1	2	3
32	התקשיתי לגלות סבלנות כשהפריעו לי כשהייתי עסוק במשהו	0	1	2	3
33	הייתי מתוח ועצבני	0	1	2	3
34	הרגשתי חסר ערך	0	1	2	3
35	גיליתי חוסר סובלנות כלפי כל מה שעיכב אותי במהלך פעילות כלשהיא	0	1	2	3
36	חשתי פחד נורא	0	1	2	3
37	חשתי חוסר תקווה מוחלט	0	1	2	3
38	הרגשתי שהחיים חסרי משמעות	0	1	2	3
39	הייתי עצבני	0	1	2	3
40	חששתי שאקלע לפאניקה ואראה מגוחך בעיני אחרים	0	1	2	3
41	הרגשתי רעד / רעידות (למשל בידיים)	0	1	2	3
42	התקשיתי לתפוש יזמה ולבצע דברים	0	1	2	3

נא לעבור לדף הבא←

Appendix 6.6 PROFILE OF MOOD STATES (POMS) – Hebrew version

בשאלון זה רשימת מילים המתארות מצבי רוח שונים שבני אדם עשויים לחוות. בכל סעיף / שורה נא בחר את האפשרות המתאימה ביותר למצב רוחך בשבוע האחרון.נא סמן המתאימה לאפשרות הנכונה.

		בכלל לא	במידה מועטה	במידה בינונית	במידה רבה	במידה רבה מאד
1	מתוח					
2	כועס					
3	שחוק					
4	ללא שמחה					
5	מלא חיים					
6	מבולבל					
7	מעוצבן וחסר סבלנות					
8	עצוב					
9	פעיל					
10	עצבים מתוחים עד					
11	במצב רוח רע					
12	מדוכא / שפוף					
13	מלא אנרגיה					
14	חסר תקווה					
15	חסר שקט נפשי					
16	חסר מנוחה					
17	לא מסוגל להתרכז					
18	עייף					
19	מעוצבן					
20	חוסר תקווה / אמונה					
21	נרגן על יחס גרוע					
22	עצבני					
23	אומלל					
24	עליז					
25	חש מרירות					
26	נטול כוחות					
27	חרד					
28	חסר תמיכה / אונים					
29	יגע					
30	מרגיש אבוד					
31	זועם					
32	תוסס					
33	חסר ערך					
34	שכחן					
35	מלא עצמה וכוח					
36	חוסר ודאות לגבי דברים					
37	תשוש לחלוטין					

Appendix 6.7 Health Survey (SF-36) – Hebrew version

תאריך: _____ שם משתתף: _____קוד משתתף: _____

(1) נא הביע את דעתך מה מצב הבריאות שלך באופן כללי ?
נא לסמן עגול סביב הספרה מעל האפשרות המתאימה/קרובה ביותר. (נא לבחור אפשרות אחת בלבד.)

1	2	3	4	5
מצוין	טוב מאוד	טוב	בינוני	רע

(2) בהשוואה <u>לשנה אחת אחורה</u> כיצד היית מדרג/ת את מצבך הבריאותי כפי שהוא <u>עכשיו</u>.
נא לסמן עגול סביב הספרה מעל האפשרות המתאימה/קרובה ביותר. (נא לבחור אפשרות אחת בלבד.)

1	2	3	4	5
הרבה יותר טוב מאשר לפני שנה	יותר טוב מאשר לפני שנה	בערך כמו לפני שנה	יותר גרוע מאשר לפני שנה	הרבה יותר גרוע מאשר לפני שנה

(3) השאלות הבאות נוגעות לפעילותך ביום רגיל. נא ציין האם מצבך הבריאותי מגביל אותך מלבצע את הפעילויות הבאות ואם כן באיזו מידה. נא לבחור אפשרות אחת בלבד.

בכל שורה נא לסמן עגול סביב הספרה של האפשרות המתאימה/קרובה ביותר. (נא לבחור אפשרות אחת בלבד.)

<u>הפעילות</u>	מוגבל מאד (1)	מוגבל חלקית (2)	לא מוגבל כלל (3)
(א) פעילות נמרצת כריצה, הרמת חפצים כבדים, השתתפות בפעילות ספורטיבית מאומצת.	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל

(ב) פעילויות מתונות כהזזת שולחן, דחיפת שואב אבק, משחקים קלים	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ג) נשיאת סל מצרכים	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ד) עליה במדרגות - מספר קומות	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ה) עליה במדרגות - קומה אחת בלבד	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ו) להתכופף, לכופף ברכיים, לכרוע	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ז) הליכה – למעלה מקילומטר וחצי	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ח) הליכה – 750 מטר	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(ט) הליכה –100 מטר	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל
(י) להתרחץ ולהתלבש	1 מוגבל מאד	2 מוגבל חלקית	3 לא מוגבל כלל

4) <u>בארבעת השבועות</u> האחרונים האם מצבך הבריאותי או הגופני גרם לך לבעיות הבאות בעבודתך או פעילויות השגרתיות. בכל שורה נא לסמן עגול סביב הספרה של האפשרות המתאימה/קרובה ביותר? (נא לבחור אפשרות אחת בלבד.)

	כן (1)	לא (2)
(א) נאלצתי לקצר את כמות הזמן שאני נמצא בעבודה או בפעילויות אחרות	1 כן	2 לא
(ב) הספקתי לעשות פחות ממה שרציתי	1 כן	2 לא
(ג) הייתי מוגבל/ת בסוג העבודה או הפעילות שיכולתי לעשות	1 כן	2 לא
(ד) התקשיתי בביצוע עבודה או פעילות אחרת (למשל – נדרש מאמץ יתר)	1 כן	2 לא

(5) <u>בארבעת השבועות</u> האחרונים האם מצבים נפשיים (כגון חרדה או דיכאון) גרמו לך לבעיות הבאות בעבודתך או פעילויות השגרתיות? (נא לבחור אפשרות אחת בלבד.)

	כן (1)	לא (2)
(א) נאלצתי לקצר את כמות הזמן שאני נמצא בעבודה או בפעילויות אחרות	1 כן	2 לא
(ב) הספקתי לעשות פחות ממה שרציתי	1 כן	2 לא
(ג) לא בצעתי את העבודה או פעילויות אחרות בתשומת הלב הרגילה	1 כן	2 לא

(6) <u>בארבעת השבועות</u> האחרונים באיזו מידה השפיעו בעיות בריאות גופנית או בעיות נפשיות (כגון חרדה או דיכאון) על פעילויות החברתיות השגרתיות וקשריך עם משפחה, חברים, מכרים, שכנים או קבוצות חברתיות אחרות (נא לבחור אפשרות אחת בלבד.)

1	2	3	4	5
כלל לא	מעט מאד	במידה בינונית	הרבה	הרבה מאד

(7) <u>בארבעת השבועות</u> האחרונים מה הייתה מידה הכאב הגופני שחשת (אם בכלל)?

1	2	3	4	5	6
אף כאב	כאב קל מאד	כאב קל	כאב בינוני	כאב חזק	כאב חזק מאד

(8) <u>בארבעת השבועות</u> האחרונים באיזו מידה השפיעו הכאבים גופניים מהם סבלת על יכולתך לעבוד (גם מחוץ לבית וגם בעבודות בית)? (נא לבחור אפשרות אחת בלבד.)

1	2	3	4	5
כלל לא	מעט מאד	במידה בינונית	הרבה	הרבה מאד

(9) השאלות הבאות הם לגבי הרגשתך ומצבך הכללי <u>בארבעת השבועות האחרונים.</u>

נא בחר/י את התשובה הקרובה ביותר. נא לבחור אפשרות אחת בלבד)

איזה חלק מן הזמן בארבעת השבועות האחרונים –

	כל הזמן (1)	רב הזמן (2)	חלק ניכר מן הזמן (3)	חלק מן הזמן (4)	מעט מן הזמן (5)	כלל לא (6)
(א) האם הרגשת מלא חיים ?	1	2	3	4	5	6
(ב) האם היית מאד עצבני?	1	2	3	4	5	6
(ג) האם הרגשת כה מדוכדך/ת עד שדבר כלשהוא לא יכול לשמח אותך?	1	2	3	4	5	6
(ד) האם הרגשת רגוע ושלוו?	1	2	3	4	5	6
(ה) האם הייתה לך הרבה אנרגיה?	1	2	3	4	5	6
(ו) האם הרגשת מדוכדך ועצוב?	1	2	3	4	5	6
(ז) האם הרגשת תשוש?	1	2	3	4	5	6
(ח) האם היית שמח?	1	2	3	4	5	6
(ט) האם חשת עייפות?	1	2	3	4	5	6

(10) <u>בארבעת השבועות</u> האחרונים באיזו מידה השפיעו בעיות בריאות גופנית או בעיות נפשיות (כגון חרדה או דיכאון) על פעילויות החברתיות (כגון ביקור אצל משפחה, חברים)?

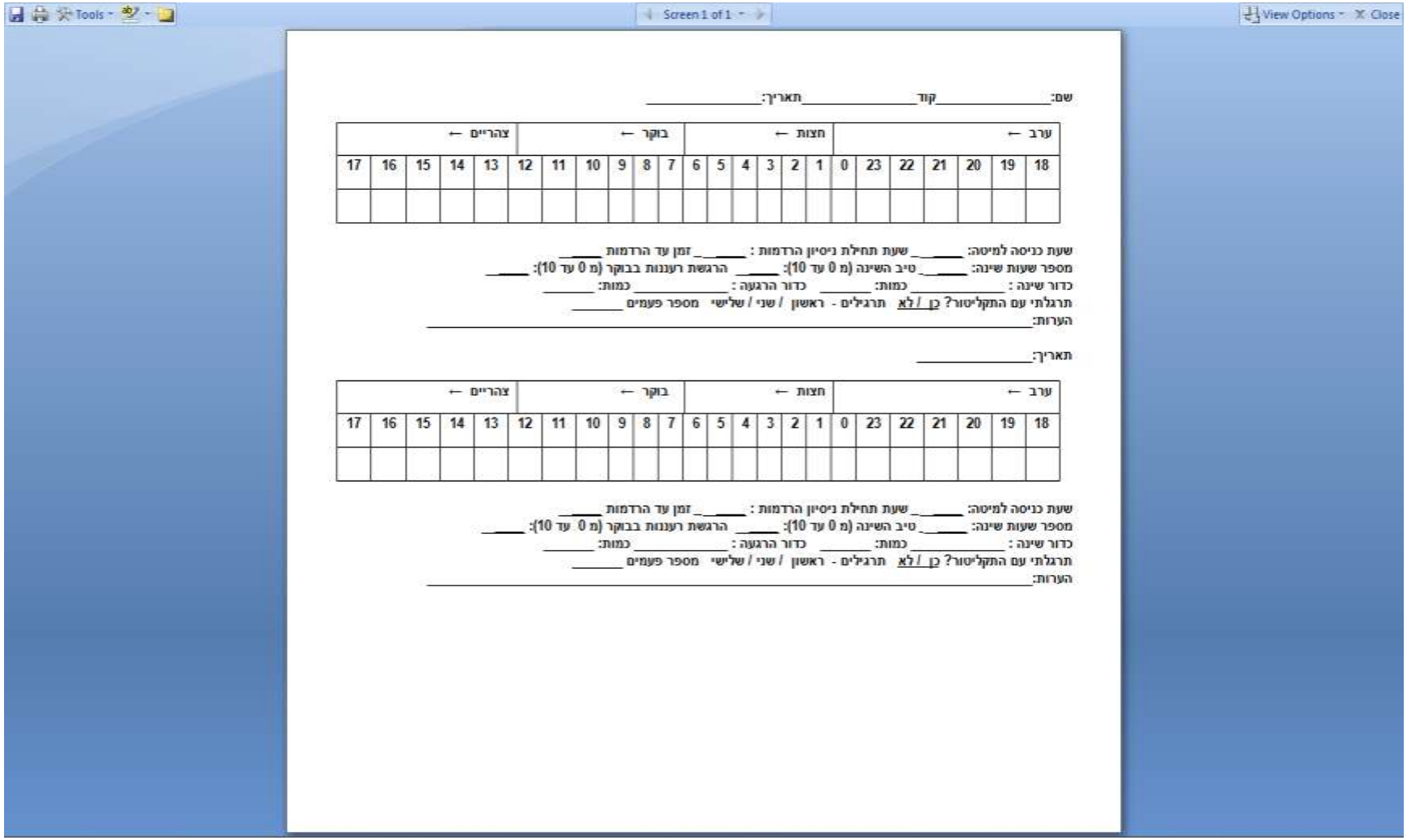
1	2	3	4	5
כל הזמן	רב הזמן	חלק מן הזמן	מעט מהזמן	בכלל לא

(11) באיזו מידה הקביעות הבאות נכונות או לא נכונות (במקרה שלך)?

	(1) נכון מאד	(2) נכון בדרך כלל	(3) לא יודע	(4) בדרך כלל לא נכון	(5) בכלל לא נכון
(א) אני נוטה לחלות יותר בקלות מרב האנשים	1	2	3	4	5
(ב) אני בריא לפחות כמו כל מי שאני מכיר	1	2	3	4	5
(ג) אני צופה שבריאותי תלך ותהיה גרועה יותר	1	2	3	4	5
(ד) מצב בריאותי מצוין	1	2	3	4	5

נא הקדישו רגע לוודא שעניתם על כל הסעיפים. כלומר <u>שבכל</u> סעיף מוקף בעיגול מספר <u>אחד בלבד</u>

Appendix 6.8 Daily Sleep and Practice Log (DSPL) – Hebrew version



שם:_____קוד_____תאריך:_____

ערב ←												חצות ←							בוקר ←						צהריים ←						
18	19	20	21	22	23	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17

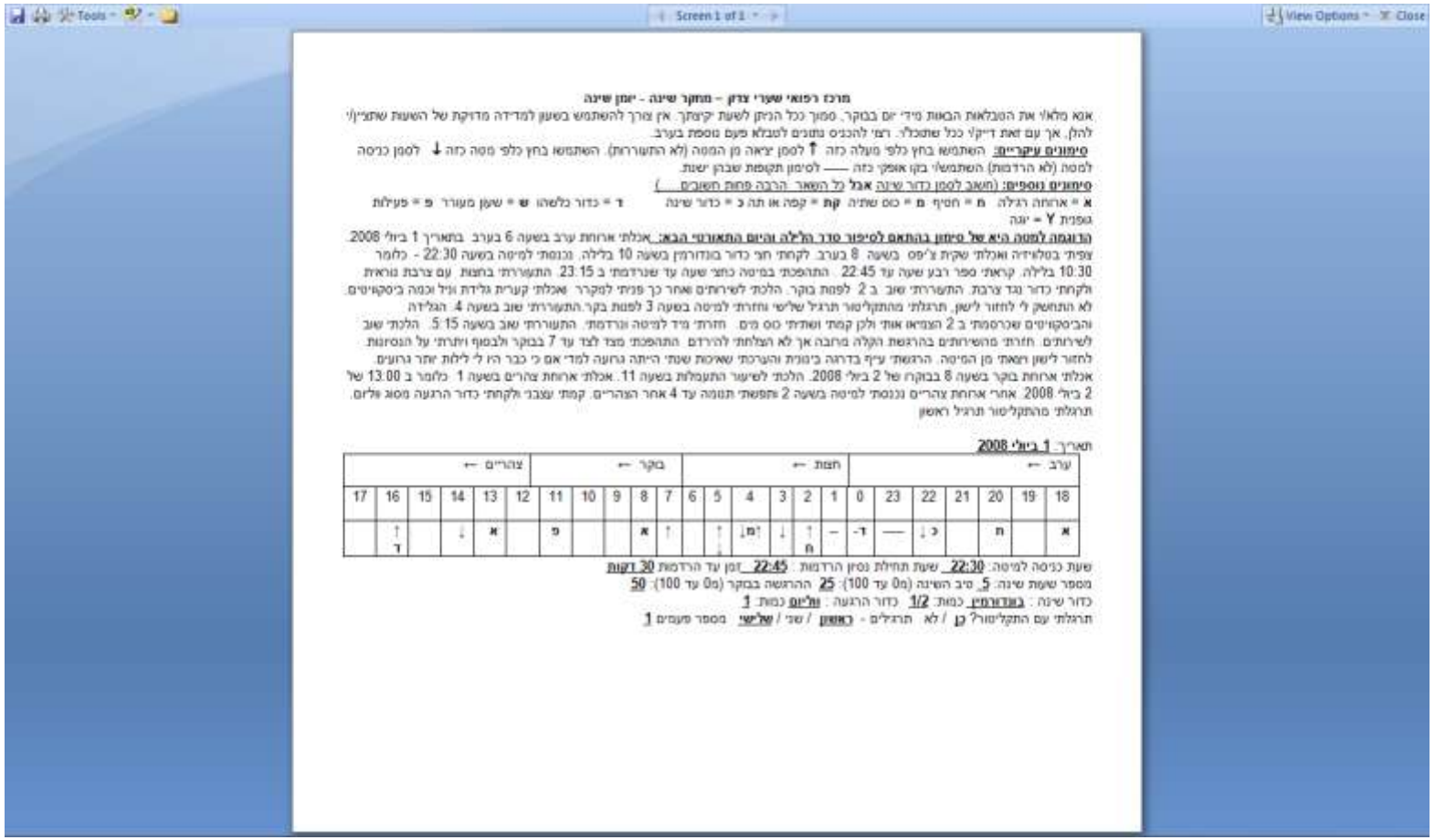
שעת כניסה למיטה: _____ שעת תחילת ניסיון הרדמות : _____ זמן עד הרדמות _____
מספר שעות שינה: _____ טיב השינה (מ 0 עד 10): _____ הרגשת רעננות בבוקר (מ 0 עד 10): _____
כדור שינה : _____ כמות: _____ כדור הרגעה : _____ כמות: _____
תרגלתי עם התקליטור? כן / לא תרגילים - ראשון / שני / שלישי מספר פעמים _____
הערות:_____

תאריך:_____

ערב ←												חצות ←							בוקר ←						צהריים ←						
18	19	20	21	22	23	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17

שעת כניסה למיטה: _____ שעת תחילת ניסיון הרדמות : _____ זמן עד הרדמות _____
מספר שעות שינה: _____ טיב השינה (מ 0 עד 10): _____ הרגשת רעננות בבוקר (מ 0 עד 10): _____
כדור שינה : _____ כמות: _____ כדור הרגעה : _____ כמות: _____
תרגלתי עם התקליטור? כן / לא תרגילים - ראשון / שני / שלישי מספר פעמים _____
הערות:_____

App.6.9 Daily Sleep and practice log - Hebrew version (DSPL) - Instructions and filling out example



מרכז רפואי שערי צדק – מחקר שינה - יומן שינה

אנא מלא/י את הטבלאות הבאות מידי יום בבוקר, סמוך ככל הניתן לשעת יקיצתך. אין צורך להשתמש בשעון למדידה מדויקת של השעות שתציין/י להלן, אך עם זאת דייק/י ככל שתוכל/י. רצוי להכניס נתונים למבלא פעם נוספת בערב.

סימונים עיקריים: השתמש בחץ כלפי מעלה כזה ↑ לסמן יציאה מן המיטה (לא התעוררות). השתמש בחץ כלפי מטה כזה ↓ לסמן כניסה למטה (לא הרדמות) השתמש/י בקו אופקי כזה —— לסימון תקופות שבהן ישנת.

סימונים נוספים: (חשוב לסמן כדור שינה **אבל** כל השאר הרבה פחות חשובים)

א = אחוזת הגילה **מ** = חסיף **ם** = כוס שתיה **קת** = קפה או תה **כ** = כדור שינה **ד** = כדור כלשהו **ש** = שעון מעורר **פ** = פעילות גופנית **ץ** = יוגה

הדוגמה למטה היא של סימון בהתאם לסיפור סדר הלילה והיום התאורטי הבא: אכלתי ארוחת ערב בשעה 6 בערב. בתאריך 1 ביולי 2008. צפיתי בסלויזיה ואכלתי שקית צ'יפס בשעה 8 בערב. לקחתי חצי כדור בנדורמין בשעה 10 בלילה. נכנסתי למיטה בשעה 22:30 - כלומר 10:30 בלילה. קראתי ספר רבע שעה עד 22:45. התהפכתי במיטה כחצי שעה עד שנרדמתי ב 23:15. התעוררתי בחצות עם צרבת נוראית ולקחתי כדור נגד צרבת. התעוררתי שוב ב 2 לפנות בוקר. הלכתי לשירותים ואחר כך פניתי למקרר. אכלתי קערית גלידת וניל וכמה ביסקוויטים. לא התחשק לי לחזור לישון, תרגלתי מהתקליטור תרגיל שלישי וחזרתי למיטה בשעה 3 לפנות בקר. התעוררתי שוב בשעה 4. הגלידה והביסקוויטים שכרסמתי ב 2 הצמיאו אותי ולכן קמתי ושתיתי כוס מים. חזרתי מיד למיטה ונרדמתי. התעוררתי שוב בשעה 5:15. הלכתי שוב לשירותים. חזרתי מהשירותים בהרגשת הקלה מרובה אך לא הצלחתי להירדם. התהפכתי מצד לצד עד 7 בבוקר ולבסוף ויתרתי על הנסיונות לחזור לישון וקמתי מן המיטה. הרגשתי עייף בדרכה ביומית והערכתי שאיכות שנתי הייתה גרועה למדי אם כי כבר היו לי לילות יותר גרועים. אכלתי ארוחת בוקר בשעה 8 בבוקר1 של 2 ביולי 2008. הלכתי לשיעור התעמלות בשעה 11. אכלתי ארוחת צהריים בשעה 1 כלומר ב 13:00 של 2 ביולי 2008. אחרי ארוחת צהריים נכנסתי למיטה בשעה 2 נפשתי תנומה עד 4 אחר הצהריים. קמתי שבעי ולקחתי כדור הרגעה מסוג וליום. תרגלתי מהתקליטור תרגיל ראשון

תאריך: **1 ביולי 2008**

ערב ←							חצות ←						בקר ←						צהריים ←						
18	19	20	21	22	23	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
א		ח		כ ↓	——	ד-	–	↑ ם	↓	↑ם↓	↑		↑	א			פ		א	↓		↑ ר	

שעת כניסה למיטה: **22:30** שעת תחילת נסיון הרדמות : **22:45** זמן עד הרדמות **30 דקות**

מספר שעות שינה: **5** , טיב השינה (0a עד 100): **25** ההרגשה בבקר (0a עד 100): **50**

כדור שינה : **בנדורמין** כמות: **1/2** כדור הרגעה : **וליום** כמות **1**

תרגלתי עם התקליטור? **כן** / לא תרגילים - **ראשון** / שני / **שלישי** מספר פעמים **1**

Appendix 7. Embletta ® mobile sleep lab

Appendix 7.1 Embletta ® data recording unit – technical specifications

Table App.7.1

Embletta ® data acquisition and recording unit – technical specifications

Dimensions (H x W x D)	0.8"x2.5"x4□9" / 2□x65x12□ mm
Weight	Embletta 0.24lb/110g, batteries: 0.1lb/46g
Batteries	2 AA batteries (12 hours duration)
Housing construction	Injection moulded plastic, covered with aluminium shields.
Number of channels	7 sensors, 14 derived signals
Recording capacity	Up to 12 hours (16MB memory)
Maximum patient leakage current	10μA, complying with EN6060-1
Operating temperature	40°F to 120°F (+5°C to +50°C)
Storage temperature	0°F to 120°F (-20°C to +50°C)
Humidity	90% non-condensing
IEC 601-1 Classifications	Class IIa, Type BF

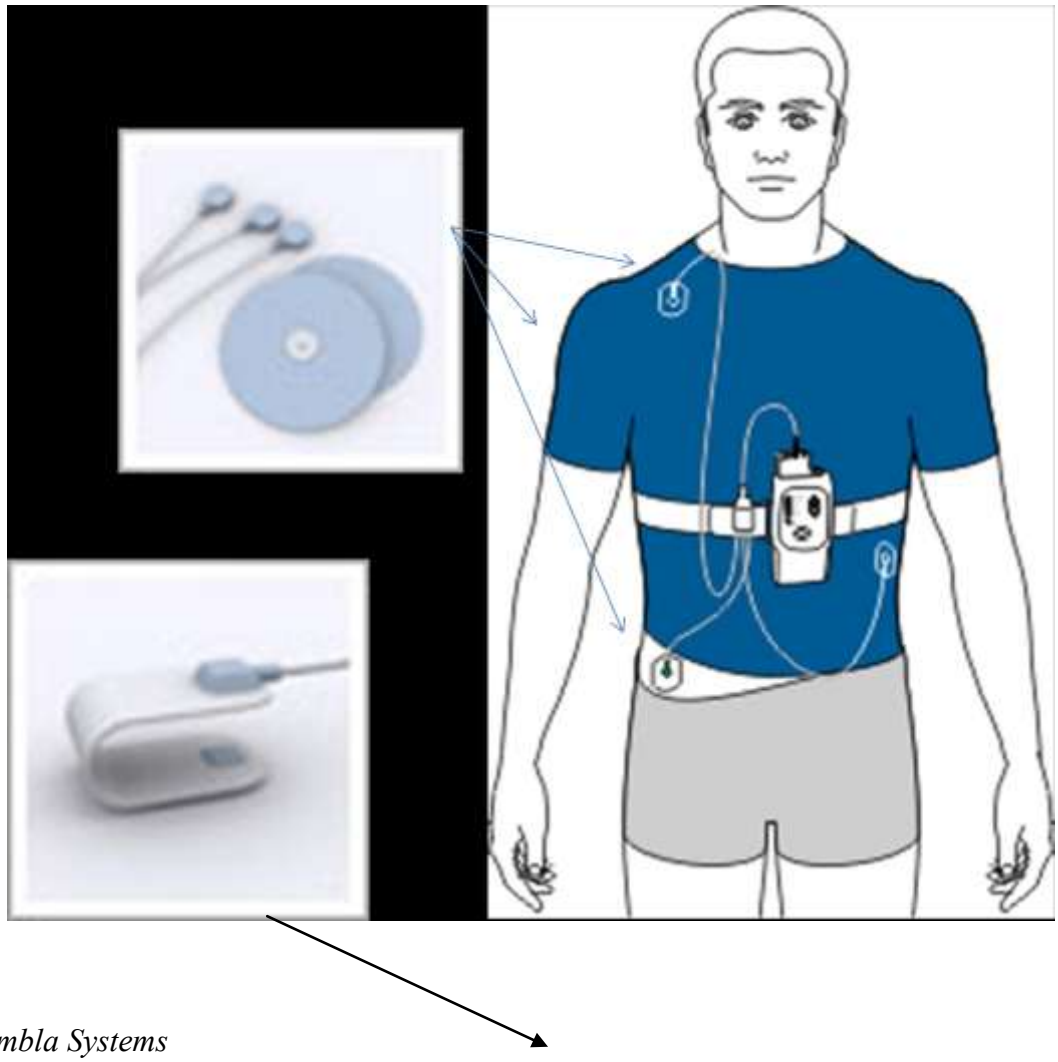
Appendix 7.2 Embletta ® recording unit features

Table App.7.2

Embletta ® available and used features

Measure	Available Feature	**used**
ECG	X30: x10 Proxy + (1) EKG	
EEG	X50: x10 Proxy + (1) EEG	
combination	X100: X10 Proxy + (4) ExG	✓
Flow/Pressure	pressure transducer	
Nasal/Oral	flow (thermistor)	
Snore	by pressure transducer or by neck sensor	
Abdominal Movement	respiratory effort sensor	
Chest Wall Movement	respiratory effort sensor	
Oximetry	SpO2 averaged and not averaged	✓
Pulse Rate	oximeter	✓
Body Position	built-in position/activity sensor	✓
Activity	built-in position/activity sensor	✓
Flow Limitation	pressure transducer	
Event	event button	

Appendix 7.3 Embletta ® recording unit connection to sensors



© Embla Systems

Figure App.7.3

Embletta® unit connection scheme with 3 ECG sensors and an oximeter sensor

** Curtesy of Embla systems*

Appendix 7.4 Embletta ® recording unit image

© Embla Systems

Figure App.7.4
Embletta® mobile recording unit graphical image

** Curtesy of Embla systems*

Appendix 8. HC1000P Sleep analysis system graphic displays

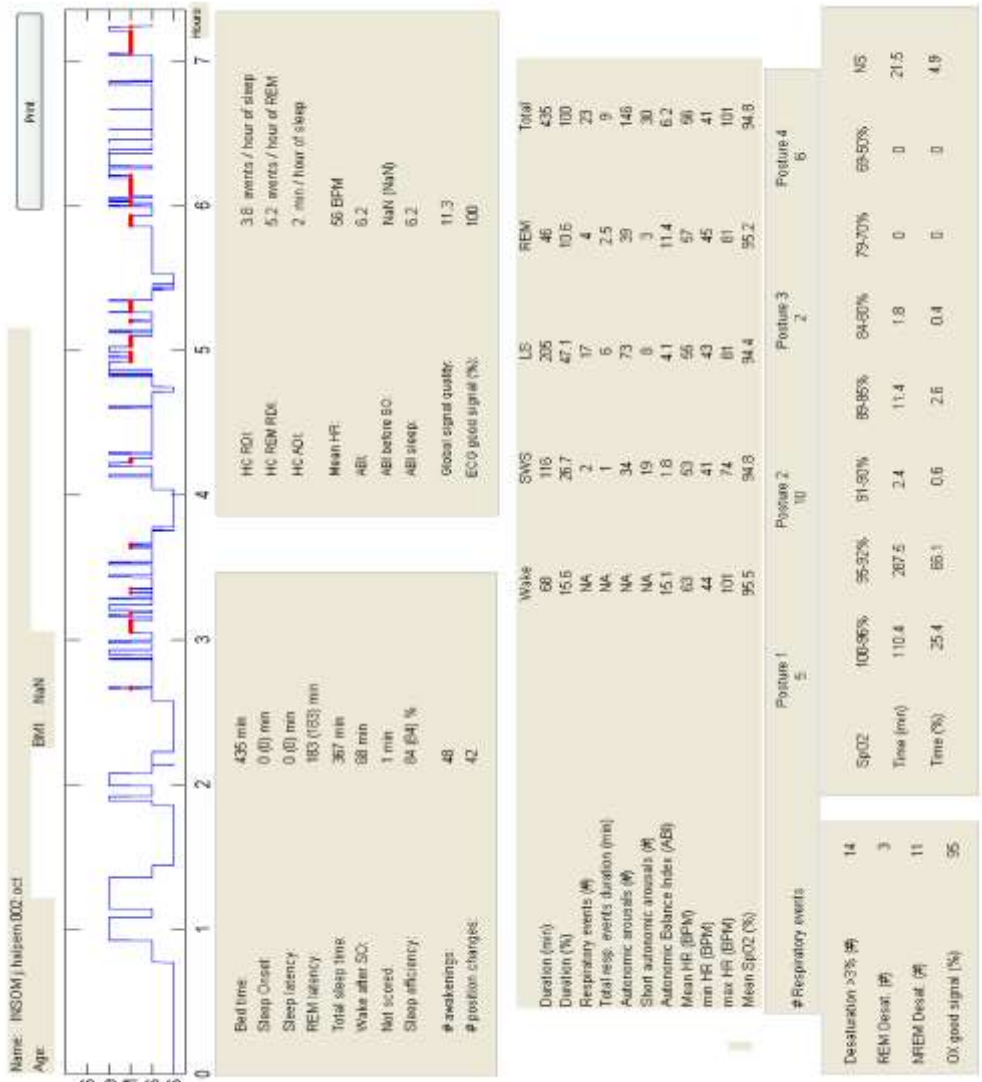


Figure App.8.1

HC1000P report summary screen shot

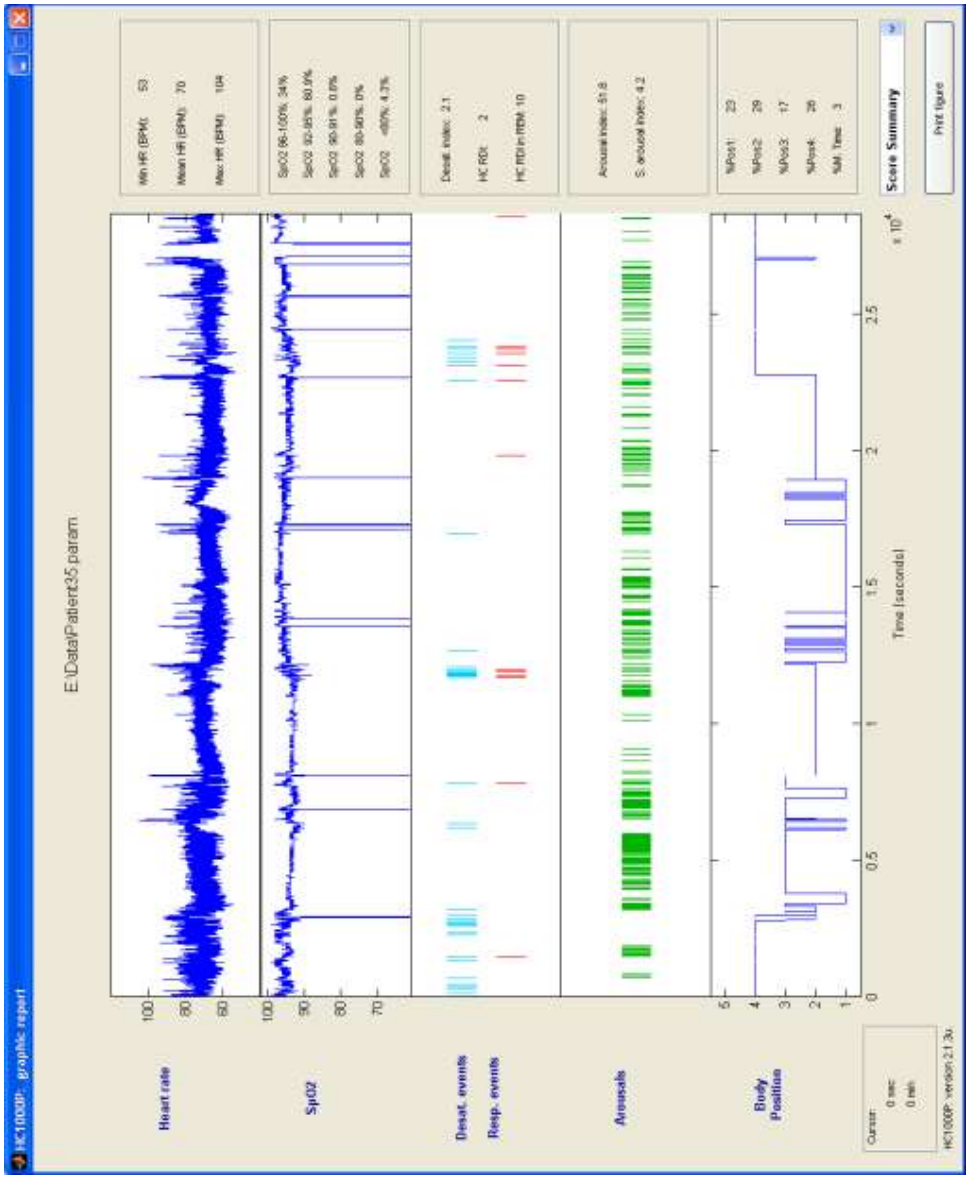


Figure App.8.2

HC1000P graphic report screen shot

Appendix 9. Yoga poses used in practice protocol

Appendix 9.1 Yoga poses images

The following table includes images of the full Asana sequence including alternative postures (ALT) and modified poses (MOD). For example, pose No. 1 is the first pose in the sequence; pose no.1-ALT is an alternative pose to pose no. 1. Pose no. 2 is the second pose in the sequence etc. MOD indicates a modified traditional pose

Table App.9.1
Yoga poses sequence used in the study

No.	Sanskrit name	English name	Modification.	image
1	Tadasana	Mountain pose		
1 - ALT	Tadasana	Mountain pose	MOD # 1	
2	Tadasana	Mountain pose	MOD # 1A	

3	Tadasana	Mountain pose	MOD # 1B	
4	Virabhadrasana II	Hero pose II		
4-ALT	Virabhadrasana II	Hero pose II	MOD # 1	
5	Bikram style Ardha-Chandrasana	Bikram style half moon pose		
5-ALT	Ardha-Chandrasana	modified half moon pose	MOD # 2	
6	Adho Mukha Svanasana	downward facing dog pose		

6-ALT	Adho Mukha Svanasana	downward facing dog pose	MOD # 3	
7	Manibandha Chakra	Wrist rotations		
8	Goolf Chakra	Ankle rotations		
9	Skandh Chakra	shoulder rotations		
10	Garurasana	Eagle pose	MOD # 4	
11	Savasana	Corpse pose		

11-ALT		seated relax	MOD #11	
12	Ardha Pavana muktasana	Half Wind removing pose		
12-ALT	Ardha Pavana muktasana	Half Wind removing pose	MOD # 5	
13	Bhujangasana (easy version)	"Baby" Cobra Pose	MOD # 6	
13-ALT	Bhujangasana (ALT)	standing baby cobra	MOD # 7	
14	Ardha shalbhasana	Half Locust Pose		

14-ALT	Ardha shalbhasana (ALT)	standing half Locust	MOD # 7A	
15	Marjaryasana	cat pose		
15-ALT	Marjaryasana	cat pose	MOD # 3	
16	Bitilasana	Cow pose		
16-ALT	Bitilasana	Cow pose	MOD # 3	
17	Marichyasana (easy version)	Spinal twist		

17-ALT	Chair twist	Spinal twist	MOD # 8	
18	Ardha-Kurmasana	Half Tortoise Pose	MOD # 9	
18-ALT		chair - Tortoise	MOD # 10	
19	Balasana	Child's pose		
19-ALT	Balasana	Child's pose	MOD # 13	
20	Paschimotta nasana	Seated forward bend	MOD # 12	

20-ALT	Paschimotta nasana	Chair –forward bend	MOD # 12A	
21	Savasana	Corpse pose		
21-ALT		Chair - relax	MOD #13	
21-ALT		Chair - relax	MOD #11	

Comment: [n-ALT]= alternative pose to pose number [#n]

Appendix 9.2 Yoga poses sequence details

Table App.9.2 Details of yoga pose sequence

No.	Sanskrit name	English name	type	base	Rep	Modified	Note
1	Tadasana	Mountain pose	STA		1		
1-ALT	Tadasana	Mountain pose	STA		1	MOD # 1	
2	Tadasana	Mountain pose	STA		1 - 2	MOD # 1A	
3	Tadasana	Mountain pose	STA		1 - 2	MOD # 1B	
4	Virabhadrasana II	Hero pose II	STA		1 - 2		RS, LS
4-ALT	Virabhadrasana II	Hero pose II	STA		1 - 2	MOD # 1	RS, LS
5	Bikram style Ardha-Chandrasana	Bikram style half moon pose	STA		1 - 2	MOD # 2	RS, LS
5-ALT	Ardha-Chandrasana	modified half moon pose	STA		1 - 2	MOD # 2A	
6	Adho Mukha Svanasana	downward facing dog pose	STA		1 - 2		
6-ALT	Adho Mukha Svanasana	downward facing dog pose	STA	CHR	1 - 2	MOD # 3	
7	Manibandha Chakra	Wrist rotations	SIT	CHR	3 -9		CW, CCW
8	Goolf Chakra	Ankle rotations	SIT	CHR	3 -9		CW, CCW
9	Skandh Chakra	shoulder rotations	SIT	CHR	3 -9		CW, CCW
10	Garurasana	Eagle pose	SIT	CHR	1 - 2	MOD # 4	RS, LS
11	Savasana	Corpse pose	SUP	MAT	1		
11-ALT		seated relax	SIT	CHR	1	MOD #11	
12	Ardha Pavana muktasana	Half Wind removing pose	SUP	MAT	1 - 2		RS, LS
12-ALT	Ardha Pavana muktasana	Half Wind removing pose	SUP	CHR	1 - 2	MOD # 5	RS, LS
13	Bhujangasana (easy version)	“Baby” Cobra Pose	PRN	MAT	1 - 2	MOD # 6	
13-ALT		standing baby cobra	SIT	WAL	1 - 2	MOD # 7	
14	Ardha shalbhasana	Half Locust Pose	PRN	MAT	1 - 2		
14-ALT		standing half Locust	STA	WAL	1 - 2	MOD # 7	
15	Marjaryasana	cat pose		MAT	2-4		
15-ALT	Marjaryasana	cat pose		CHR	2-4	MOD # 3	
16	Bitilasana	Cow pose		MAT	2-4		
16-ALT	Bitilasana	Cow pose		CHR	2-4	MOD # 3	
17	Marichyasana (easy version)	Spinal twist	SIT	MAT	1 - 2		RS, LS
17-ALT	Chair twist	Spinal twist	SIT	CHR	1 - 2	MOD # 8	RS, LS
18	Ardha-Kurmasana	Half Tortoise Pose	SIT	MAT	1 - 2	MOD # 9	
18-ALT	Ardha-Kurmasana	chair - Tortoise	STA	CHR	1 - 2	MOD # 10	
19	Balasana	Child’s pose	SIT	MAT	1 - 2	MOD # 9	
19-ALT		Chair child pose	STA	CHR	1 - 2	MOD # 10	
20	Paschimottanasana	Seated forward bend	SIT	MAT	1 - 2	MOD # 12	
20-ALT		Chair –forward bend	SIT	CHR	1 - 2	MOD # 12	
21	Savasana	Corpse pose	SUP	MAT	1		
21-ALT		Chair – relax 1	SIT	CHR	1	MOD #11	
21-ALT		Chair – relax 2	SIT	CHR	1	MOD #11a	

Notes:

Abbreviations used in table App.9.2

The following abbreviations are used: RS – right side, LS- left side, CW – clockwise, CCW – counter clockwise, STA- standing pose. SIT – sitting pose, PRN – prone pose, SUP- supine pose, MAT – mat based pose, CHR – chair base pose, WAL – using wall, MOD –modified pose, ALT - alternative

Modifications indicated in table App.9.2

MOD # 1 - Holding onto back of chair for balance

MOD # 1A –shifting weight to right, left, front, back while engaging abdominal muscles gently

COM #1B – raising hands while breathing in. Lowering hands while breathing out

MOD # 2 – when bending to right – right hand on hip and left hand over head and vice versa

MOD # 2A – same as MOD # 2 but with right hand on back of chair for support

MOD # 3 – holding seat of chair or top of back of chair and bending forward

MOD # 4 – in chair sitting position arms interlaced with left elbow below right and legs – right knee over left knee and vice versa on the other side

MOD # 5 – in chair sitting position raise knee towards trunk and interlace hands over or below knee

MOD # 6 – gentle upper back raise with neck in line with the spine, facing down , palms on floor next to armpits facing down

MOD # 7 – facing wall, hands on wall, weight forward slightly, back arched looking up

MOD # 7A – facing wall, hands on wall, weight forward slightly, back arched looking up. One leg extended back, off the floor

MOD # 8 when twisting to the right – left hand over right tight grabbing chair sit and right hand grabbing back of chair and vice versa

MOD # 9 – in case of knee restriction pose is executed with increased angle between calf and thigh (up to 90 degrees if needed)

MOD # 10 –standing and bending forward placing forehead and palms of hands on chair's sit.

MOD # 11 – sit upright back to chair's back support, palms resting on thighs, feet flat on ground, close eyes and relax

MOD # 11a – sit upright back to chair's back support, palms resting on thighs, with legs extended on another chair, close eyes and relax

MOD # 12 – bending forward with palms resting on thighs for support. Legs together

MOD # 12a – bending forward sitting on a chair with legs extended on another chair

MOD # 13 – sitting leaning forward with palms and head resting on back of another chair for support. Folded towel may be used under forehead

Appendix 10. Transcripts of meditation and relaxation exercises

Appendix 10.1 Breath counting meditation – transcript

Preparation: Please prepare yourself for the practice of breath counting meditation. Please sit on an arm chair, or in a reclining chair. Or a regular chair. Rest your hands on your lap. Relax them completely. You may also lie down in your bed if possible lie on your back with your feet slightly apart and the palms of your hands facing upwards. However, the sitting position is preferred for this exercise. If this is not possible please find a position which suits your physical condition. Gently close your eyes. Keep your lips touching lightly, relax your jaws. Take some time to make yourself even more comfortable. Try to breathe in deeply and then breathe out with a big sigh. As you sigh, try to feel you are letting go of stress and tension. Repeat it two more times.

Instructions: In this exercise you will pay close attention to your breath. You may focus your attention on your lower abdomen. Watch what happens to the abdomen as you inhale and exhale. When you inhale, your lower abdomen goes out, and when you exhale it goes back in. You may also focus your attention on your ribcage Watch what happens to the ribcage as you inhale and exhale. When you inhale, your ribcage expands sideway and when you exhale it goes back. Just choose one point of focus to observe the breath. The one that you can really feel is moving in and out when you inhale and exhale. Do not try to alter your breath. Do not try to control your breath in any way. Just observe the natural breath as it is. Observe your breath carefully. It may be deep. Or it may be shallow. It may be long or it may be short. There may be a short pause following the inhalation. There may be a short pause following the exhalation. Whatever your breath is at the moment – just observe it. Do not change it. Just be aware

of it. You will now start counting the breaths silently. Synchronize the count with the last part of the exhalation. As the air is being exhaled and you are getting close to the end of the exhalation count silently. You may count by whispering or just moving your lips silently. Start from one, two, three etc. If you forget the present count, start again from one. If a thought arises, do not fight it. It is natural for thoughts to arise in the mind. Just pay more attention to the counting. If you are very distracted by thoughts, utter the count a little louder till your mind becomes more concentrated on the counting. Keep counting till you I tell you to stop.

A few minutes later: keep observing your breath very attentively. Observe the inhalation. Observe the exhalation. Towards the end of the exhalation count silently. If you lost the count, that is OK. Just start again from one. From day to day your concentration will improve and you will be able to count continuously without losing the count for a longer period of time. If thoughts come, let them come and let them pass away, just like clouds in the sky or bubbles in a glass of soda water. Just give more attention to the counting, and eventually fewer thoughts will come to distract you.

At the end of twenty minutes: Conclusion: You may stop counting now. Become aware of your surroundings. Become aware of the sounds in the room. Become aware of sounds outside the room. Slowly open your eyes. Slowly stretch your arms and legs. Move your fingers. Move your toes. Breath counting practice has now come to its conclusion. Make sure you are completely awake and ready to return to daily activity.

Appendix 10.2 Muscle relaxation – transcript

Preparation: Please prepare yourself for the practice of muscle relaxation. Please lie down on a mat, or a bed, or in a reclining chair. If possible lie on your back with your feet slightly apart and the palms of your hands facing upwards. But if this is not possible please find a position which suits your physical condition. You may use a pillow or cushion or a folded blanket to keep your neck aligned with your spine. Cover yourself with a blanket to stay comfortably warm during the practice. Gently close your eyes. Keep your lips touching lightly, smile gently and relax your jaws as if you have experienced something pleasant, let the back of your neck relax. It is important that you be as comfortable as possible. So take some time to make yourself even more comfortable. Try to breathe in deeply and then breathe out with a big sigh. As you sigh, feel you are letting go of stress and tension. Repeat it two more times. It is important that you maintain this position without movement throughout the practice. Say to yourself in your mind: "I will not move throughout the practice. I will be aware of my body but I will not move". In this muscle relaxation exercise you need to stay awake and alert. Do not fall asleep. All you have to do is to listen to the teacher's voice. Do not analyse the words you hear or intellectualize. Do not try to concentrate too hard. Simply listen to the instructions and let your mind follow. From time to time a thought may come. That is OK. Do not try to stop the thoughts forcefully but do not react to them. Simply stay calm and alert and pay more attention to the instructions being called out in the moment.

Instructions: Throughout life we accumulate stress and tension. Some of the tension may manifest as muscular tension which may become chronic. Tense muscles may cause stiffness, reduced blood flow, musculoskeletal pains and poor posture. In this exercise you will direct your attention sequentially to muscle groups throughout the

body from top to bottom starting from the feet, then calves, thighs, lower back, middle back, upper back, lower abdomen, chest, shoulders, upper arms, lower arms, palms and fingers, scalp, temples, back of the head, neck, forehead, eyebrows, cheeks, nose, mouth, chin, and throat. Whenever a muscle group or body part is called out, direct your awareness there. Once your awareness is focused on a particular body part, you will be asked to quietly tell that body part "relax". This can be done by whispering or silently moving the lips. You can also try to breathe out, and visualise the breath flowing through that particular muscle group, helping it to relax more. This is repeated several times for each muscle group. If you find it very difficult to relax a particular muscle group, you can try to consciously contract that muscle group while inhaling fully and then consciously relax it while exhaling. But if you find it easy to relax a muscle just by focusing on it and telling it to relax, then there is no need to contract and release.

Now let us begin. Direct your awareness to your feet. Throughout life tension may accumulate in the muscles of the feet. Inhale fully, and as you exhale tell the muscles in your feet: "relax!" Feel the tension in your feet dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the feet caressing and relaxing them. Repeat this several times. With every repetition more and more tension dissolves, and the feet muscles are becoming softer and more relaxed.

Now move your awareness to your calves. Throughout life tension may accumulate in the muscles of the calves. Inhale fully, and as you exhale tell the muscles in your calves: "relax!" Feel the tension in your calves dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the calves caressing and relaxing them.

Repeat this several times. With every repetition more and more tension dissolves, and the calves' muscles are becoming softer and more relaxed.

Direct your awareness to your thighs. Throughout life tension may accumulate in the muscles of the thighs. Inhale fully, and as you exhale tell the muscles in your thighs: "relax!" Feel the tension in your thighs dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the thighs caressing and relaxing them. Repeat this several times. With every repetition more and more tension dissolves, and the thigh muscles are becoming softer and more relaxed.

Direct your awareness to your buttocks. Throughout life tension may accumulate in the muscles of the buttocks. Inhale fully, and as you exhale tell the muscles in your buttocks: "relax!" Feel the tension in your buttocks dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the buttocks caressing and relaxing them. Repeat this several times. With every repetition more and more tension dissolves, and the buttocks muscles are becoming softer and more relaxed.

Direct your awareness to your lower back. Throughout life tension may accumulate in the muscles of the lower back. Inhale fully, and as you exhale tell the muscles in your lower back: "relax!" Feel the tension in your lower back dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the lower back muscles caressing and relaxing them. Repeat this several times. With every repetition more and more tension dissolves, and the lower back muscles are becoming softer and more relaxed.

Direct your awareness to your lower abdomen. Throughout life tension may accumulate in the muscles of the lower abdomen. Inhale fully, and as you exhale tell the muscles in your lower abdomen: “relax!” Feel the tension in your lower abdomen dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the lower abdomen caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the lower abdomen muscles are becoming softer and more relaxed.

Direct your awareness to your upper back and scapula. Throughout life tension may accumulate in the muscles of the upper back and scapula. Inhale fully, and as you exhale tell the muscles in your upper back and scapula: “relax!” Feel the tension in your upper back and scapula dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the upper back and scapula muscles caressing and relaxing them. Repeat this several times. With every repetition more and more tension dissolves, and the upper back and scapula muscles are becoming softer and more relaxed.

Direct your awareness to your chest. Throughout life tension may accumulate in the muscles of the chest. Inhale fully, and as you exhale tell the muscles in your chest: “relax!” Feel the tension in your chest dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the chest caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the chest muscles are becoming softer and more relaxed.

Direct your awareness to your shoulders. Throughout life tension may accumulate in the muscles of the shoulders. Inhale fully, and as you exhale tell the muscles in your shoulders: “relax!” Feel the tension in your shoulders dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the shoulders caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the shoulders muscles are becoming softer and more relaxed.

Direct your awareness to your upper arms. Throughout life tension may accumulate in the muscles of the upper arms. Inhale fully, and as you exhale tell the muscles in your upper arms: “relax!” Feel the tension in your upper arms dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the upper arms caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the upper arms muscles are becoming softer and more relaxed.

Direct your awareness to your lower arms. Throughout life tension may accumulate in the muscles of the lower arms. Inhale fully, and as you exhale tell the muscles in your lower arms: “relax!” Feel the tension in your lower arms dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the lower arms caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the lower arms muscles are becoming softer and more relaxed.

Direct your awareness to your hands and fingers. Throughout life tension may accumulate in the muscles of the hands and fingers. Inhale fully, and as you exhale tell the muscles in your hands and fingers: “relax!” Feel the tension in your hands and

fingers dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the hands and fingers caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the hands and fingers are becoming softer and more relaxed

Direct your awareness to your neck. Throughout life tension may accumulate in the muscles of the neck. Inhale fully, and as you exhale tell the muscles in your neck: "relax!" Feel the tension in your neck dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the neck caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the neck muscles are becoming softer and more relaxed.

Direct your awareness to your scalp. Throughout life tension may accumulate in the muscles of the scalp. Inhale fully, and as you exhale tell the muscles in your scalp: "relax!" Feel the tension in your scalp dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the scalp caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the scalp muscles are becoming softer and more relaxed.

Direct your awareness to your forehead. Throughout life tension may accumulate in the muscles of the forehead. Inhale fully, and as you exhale tell the muscles in your forehead: "relax!" Feel the tension in your forehead dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the forehead caressing and relaxing it.

Repeat this several times. With every repetition more and more tension dissolves, and the forehead muscles are becoming softer and more relaxed.

Direct your awareness to your temples. Throughout life tension may accumulate in the muscles of the temples. Inhale fully, and as you exhale tell the muscles in your temples: “relax!” Feel the tension in your temples dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the temples caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the temples muscles are becoming softer and more relaxed.

Direct your awareness to your cheeks. Throughout life tension may accumulate in the muscles of the cheeks. Inhale fully, and as you exhale tell the muscles in your cheeks: “relax!” Feel the tension in your cheeks dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the cheeks caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the cheeks muscles are becoming softer and more relaxed.

Direct your awareness to your eye sockets. Throughout life tension may accumulate in the muscles of the eye socket. Inhale fully, and as you exhale tell the muscles in your eye socket: “relax!” Feel the tension in your eye socket dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the eye socket caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the eye sockets muscles are becoming softer and more relaxed.

Direct your awareness to your jaw. Throughout life tension may accumulate in the muscles of the jaw. Inhale fully, and as you exhale tell the muscles in your jaw: "relax!" Feel the tension in your jaw dissolving away. You may do this by whispering or simply by moving your lips. You may also just breathe out and visualise that the breath is flowing through the jaw caressing and relaxing it. Repeat this several times. With every repetition more and more tension dissolves, and the jaw muscles are becoming softer and more relaxed.

Now let your whole body relax. Your whole body is becoming more and more relaxed. All your muscles are soft, limp, heavy. You are sinking down through the mat.

Conclusion: Say to yourself: "I am practicing muscle relaxation. I am lying down on a mat in this room". Take some time now to scan your body. How does your body feel now compared to how it felt in the beginning of the exercise? Is there still tension anywhere in your body? Where? Try to use the same technique to dissolve any remaining tension which you may have.

Slowly open your eyes. Slowly stretch your arms and legs. Move your fingers. Move your toes. Slowly support your weight and move to a sitting position. Muscle relaxation practice has now come to its conclusion. Make sure you are completely awake and ready to return to daily activity.

Appendix 10.3 Yoga Nidra transcript

Adapted from several Yoga Nidra protocols by Swami Satyananda Swaraswati, of the Bihar School of Yoga (Satyananda, 1976, pp. 81-150)

Preparation: Please prepare yourself for the practice of Yoga Nidra. Please lie down on a mat, or a bed, or in a reclining chair. If possible lie on your back with your feet slightly apart and the palms of your hands facing upwards. But if this is not possible please find a position which suits your physical condition. You may use a pillow or cushion or a folded blanket to keep your neck aligned with your spine. Cover yourself with a blanket to stay comfortably warm during the practice. Gently close your eyes. Keep your lips touching lightly, relax your jaws, and let the back of your neck relax. It is important that you be as comfortable as possible. So take some time to make yourself even more comfortable. Try to breathe in deeply and then breathe out with a big sigh. As you sigh, feel you are letting go of stress and tension. Repeat it two more times.

Instructions: It is important that you maintain this position without movement throughout the practice. Say to yourself in your mind: "I will not move throughout the practice. I will be aware of my body but I will not move". In Yoga Nidra you need to stay awake and alert. Do not fall asleep. All you have to do in Yoga Nidra is to listen to the teacher's voice. Do not analyse the words you hear or intellectualize. Do not try to concentrate too hard. Simply listen to the instructions and let your mind follow. From time to time a thought may come. That is OK. Do not try to stop the thoughts forcefully but do not react to them. Simply stay calm and alert and pay more attention to the instructions being called out in the moment.

Relaxation: Say to yourself mentally: "I am becoming more and more relaxed". With every breath you breathe out, feel the tension in your muscles dissolving more and

more. Relax your feet, toes, ankles, calves, knees, thighs, hamstrings, buttocks, lower middle and upper back muscles, shoulders, chest, arms, elbows, hands, fingers, head and neck. Relax the whole body. Your whole body is soft. It is melting into the mat. It is dissolving into the mat.

Resolution to stay awake and aware: Say to yourself mentally with full intention: "I will now practice Yoga Nidra. I will be relaxed. I will stay wide awake. I will remain alert and attentive. I will be fully aware. I will not fall asleep."

Positive affirmation (The 'Resolve'): It is now time to make a positive affirmation. It can be a life goal or it can be a wish for any positive change in any area of your life, such as health, relationships, work. Say it inside your mind with full conviction and passion. Say it from your heart. Feel as if what you ask for has already been granted to you. Repeat it three times.

Rotation of awareness: We will now use our awareness to scan our body. We will rotate our awareness quickly from one body part to the next. Just listen to the instructions. Relax your mind and let it follow the instructions. Do not try to concentrate too hard. Repeat the body part which is mentioned mentally in your mind. Direct your mind to that body part and try to be aware of it and feel it with your senses. Just be aware of it do not try to concentrate too hard. If you cannot feel anything that is all right. Just direct your mind to that body part. If you miss an instruction or lose concentration for a second that is all right. Just stay calm and focus again on the instructions and direct your awareness there.

Maintain awareness. Maintain alertness. Stay awake.

Right side: Be aware of the right side of your body. You will now rotate your awareness through the right side of the body, part by part. Start with right thumb, be aware of your right thumb and repeat mentally right thumb, sense your right thumb. Rotate your awareness to the second finger, third finger, fourth finger, fifth finger. Sense all five fingers together. Right palm of the right hand, back of the right hand, right wrist, right forearm, feel the right elbow, upper right arm, right shoulder, right armpit, right chest, right ribcage, right waist, right hip, right buttock, right thigh, right hamstring, right knee, right calf, right ankle, right heel, sole of right foot, top of the right foot, right big toe, second toe, third toe, fourth toe, fifth toe, feel all five toes together.

Left side: Now shift your awareness to the left side of your body. You will now rotate your awareness through the left side of the body part by part. Start with left thumb, sense your left thumb and repeat mentally left thumb. Rotate your awareness to the second finger, third finger, fourth finger, fifth finger. Sense all five fingers together, palm of the left hand, back of the left hand, left wrist, left forearm, feel the left elbow, upper left arm, left shoulder, left armpit, left chest, left ribcage, left waist, left hip, left buttock, left thigh, left hamstring, left knee, left calf, left ankle, left heel, left sole, left top of the foot, left big toe, second toe, third toe, fourth toe, fifth toe, feel all five toes together.

Maintain awareness. Maintain alertness. Stay awake.

Right to left: You will now direct your awareness skipping from the right side of the body to the left side and moving from top to bottom. Right heel, left heel, both heels together. Right ankle, left ankle, both ankles together. Right calf, left calf, both calves together. Right knee, left knee, both knees together. Right thigh, left thigh, both thighs

together. Right hamstring, left hamstring, both hamstrings together. Right buttock, left buttock, both buttocks together, right hip, left hip, both hips together. Waist, lower abdomen, upper abdomen, whole of the abdomen. Right side of the chest, left side of the chest, whole chest. Right collarbone, left collarbone, centre of the collarbones, throat. Right shoulder, left shoulder, right arm, left arm, right elbow, left elbow, right hand, left hand. Right thumb, second finger, third finger, fourth finger, fifth finger, all five fingers together, Palm of the right hand, back of the right hand. Left thumb, second finger, third finger, fourth finger, fifth finger, all five fingers together, palm of the left hand, back of the left hand. Right side of the back, left side of the back, lower back middle back, upper back. Back of the neck, front of the neck, the whole neck. Whole back. Be aware of the whole back. Sense the whole back. Chin, lower lip, upper lip, both lips together, teeth, tongue. Right cheek, left cheek, both cheeks. Right nostril, left nostril, both nostrils, tip of the nose, whole nose. Right eyelid, left eyelid, both eyelids, right eyeball, left eyeball, both eyeballs together. Right eyebrow, left eyebrow, both eyebrows. Right temple, left temple, forehead, back of the head, crown of the head. Whole face, be aware of whole head. Sense your whole head.

Right and left together: You will now direct your awareness symmetrically to both sides of the body at the same time moving quickly from bottom to top, starting from your toes up to the crown of the head. All the ten toes together. Both soles together, both heels, both ankles, both calves, both knees, both thighs, both buttocks, lower back, middle back, upper back, both shoulders, armpits, chest, ribcage. Sense all ten fingers together, both palms, back of the hands, both wrists, forearms, both elbows, both arms, shoulders, neck, back of head, scalp, forehead, eye sockets, temples, nose, upper lip, lower lip, inside the mouth, tongue, chin.

Large body parts: You will now direct your awareness to larger body parts. Whole right arm, whole left arm, whole right leg, whole left leg, whole front of the body, whole back of the body, whole right side of the body, whole left side of the body. Whole body together, whole body together, whole body together.

Maintain awareness. Maintain alertness. Do not fall asleep.

Hot and cold Sensations: You will now direct your awareness to sensations of heat and cold in your body: First, try to feel the body becoming very hot. Try to feel heat sensation throughout the body. Recall a time when you felt very hot. Try to recreate the same sensation of heat now just as you have experienced it in the past. Now try to feel the opposite sensation: Try to feel the body becoming very cold. Try to feel cold sensation throughout the body. Recall a time when you felt very cold. Try to recreate the same sensation of cold now just as you have experienced it in the past.

Heavy and light Sensations: You will now direct your awareness to sensations of heaviness and lightness in your body: First, try to feel the body becoming very heavy. Your whole body is so heavy you can't move. It is so heavy it is pressing down hard and sinking into the mat. Your muscles are heavy. Your whole body is heavy like lead. Now try to feel the opposite sensation: Try to feel the body becoming very light. Your whole body is so light you feel you are floating upwards. It is so light that it is floating off the mat. Your muscles are very light and loose. Your whole body is light like a helium balloon floating up and drifting in the wind.

Pain and Pleasure Sensations: You will now direct your awareness to sensations of pain and pleasure in your body: First, try to feel pain somewhere in your body. Try to remember a time when you had pain somewhere in your body. Recreate that feeling of pain in your body.

Now try to feel the opposite sensation: Try to pleasure somewhere in your body. Try to remember a time when you had a pleasant sensation in your body. Recreate that feeling of pleasure in your body.

Maintain awareness. Maintain alertness. Do not fall asleep.

Breath Awareness: You will now direct your attention to your breath. We inhale and exhale all the time but most of the time we are not aware of it. You will focus your awareness on the breath now.

Focus on abdomen: Now focus you attention on your lower belly. Notice what happens to it when you inhale and when you exhale. When you inhale, your belly rises and when you exhale it comes back down. Inhale and notice your belly rise. Exhale and notice it go down. As the belly is coming down count inwardly "18". Then watch the belly going up again and when it comes back down count inwardly "17". Keep counting down towards the end of each exhalation till you reach "1". If you lose your count start again from "18".

Focus on chest and rib cage: Now focus your attention on your chest and rib cage. Notice what happens to it when you inhale and when you exhale. When you inhale, the ribcage expands sideways and the chest expands up when you exhale the ribcage contracts towards the centre and the chest contracts down. As you exhale count inwardly "18". Next exhalation say "17". Keep counting down this way till you reach "1". If you lose your count start again from "18".

Focus on throat: Now focus you attention on your throat. Notice what happens to it when you inhale and when you exhale. Notice the sensation and listen to the sound of air going through the throat as you inhale. Notice the sensation in the throat and listen to

the sound of air going through the throat as you inhale and exhale. As you exhale count inwardly “18”. Next exhalation say “17”. Keep counting down this way till you reach “1”. If you lose your count start again from “18”.

Focus on nostrils: Now focus you attention on your nostrils. Notice what happens when you inhale and when you exhale. When you inhale, cooler air goes in through the nostrils and when you exhale warmer air goes out. As you exhale count inwardly “18”. Next exhalation say “17”. Keep counting down this way till you reach “1”. If you lose your count start again from “18”.

Maintain awareness. Maintain alertness. Do not fall asleep.

Focus on the inner space: Now you may stop counting. You may stop observing your breath. Just relax. Relax your whole body. Whole body is very relaxed. Direct your attention to the inner space just above the bridge of your nose and between your eyebrows. Just observe the inner space. Relax your eyes do not strain. Do not try to visualise anything. Just observe. Whatever you see there or don’t see there is fine. Just watch your inner space. You may see colours. Your may see light. You may recall things that happened to you today or in the distant path. It does not matter. Just observe. Just relax.

Maintain awareness. Maintain alertness. Do not fall asleep.

Rapid sequence of visualisations: Please maintain your focus on your inner space between your eyebrows. Relax your eyes do not strain. We will now go through a sequence of rapid visualisations. Listen to my voice. When I say a name of an object try to see the object in your inner space. Each object or scene will be called out three times.

Do not strain. Whether you see it clearly or vaguely or not at all – it does not matter. Just relax, listen to my voice and let your mind follow.

- Pink Rose; Pink Rose; Pink Rose.
- A golden egg; a golden egg; a golden egg.
- A beach with golden sand; a beach with golden sand; a beach with golden sand.
- Ocean waves breaking on the shore; Ocean waves breaking on the shore; Ocean waves breaking on the shore.
- A sail ship on the horizon; a sail ship on the horizon; a sail ship on the horizon.
- Golden sunset on the beach; Golden sunset on the beach; Golden sunset on the beach.
- Evening sky with purple clouds; Evening sky with purple clouds; Evening sky with purple clouds;
- Birds flying through evening skies; Birds flying through evening skies; Birds flying through evening skies.
- Clear night skies filled with sparkling stars; clear night skies filled with sparkling stars; clear night skies filled with sparkling stars
- Full moon; full moon; full moon;
- Rain storm at night; rain storm at night; rain storm at night.
- High mountains with snowy peaks; high mountains with snowy peaks; high mountains with snowy peaks.
- A thick green forest; a thick green forest; a thick green forest.

- People gathering round a campfire; people gathering round a campfire; people gathering round a campfire.
- People singing and laughing; People singing and laughing; People singing and laughing;
- Sun rising; sun rising; sun rising.
- White clouds in a blue sky; white clouds in a blue sky; white clouds in a blue sky;
- Snow covered mountain peaks; Snow covered mountain peaks; Snow covered mountain peaks;
- a cool breeze in the forest; a cool breeze in the forest; a cool breeze in the forest
- A herd of horses galloping; a herd of horses galloping; a herd of horses galloping.
- Clear mountain stream; clear mountain stream; clear mountain stream.
- A waterfall; a waterfall; a waterfall.
- Lonely house on top of a hill; lonely house on top of a hill; lonely house on top of a hill.
- A beautiful rose garden; a beautiful rose garden; a beautiful rose garden.
- A pink rose; A pink rose; A pink rose
- A golden egg; A golden egg; A golden egg;
- A beautiful diamond; A beautiful diamond; A beautiful diamond;
- You sitting in meditation with friends; You sitting in meditation with friends; You sitting in meditation with friends;

Maintain awareness. Maintain alertness. Do not fall asleep.

Repeating the auto-suggestion: It is now time to repeat the positive affirmation you made at the beginning of the practice. Your life goal, a wish for a positive change in your life, health, relationship etc. Say it silently inside your mind, with full conviction and passion. Say it from your heart. Feel as if what you ask for has already been granted to you. Repeat it three times.

Conclusion: Say to yourself: "I am practicing yoga Nidra. I am lying down on a mat in this room". Visualize yourself lying on the mat. Feel your whole body lying on the mat. Quickly scan your body from head to toe, and feel your whole body on the mat. Visualize the room around you. Become aware of your surroundings. Become aware of the sounds in the room. Become aware of sounds outside the room. Slowly open your eyes. Slowly stretch your arms and legs. Move your fingers. Move your toes. Slowly support your weight and move to a sitting position. Yoga Nidra practice has now come to its conclusion. Make sure you are completely awake and ready to return to daily activity.

Appendix 11. Detailed statistical analysis

Appendix 11.1 Subjective measures, Intention to Treat (ITT) analysis (Intervention versus control), Analysis of variance (ANOVA)

Appendix 11.1.1 Karolinska Sleepiness Scale (KSS) (*)

Scores on the Karolinska Sleepiness Scale (KSS) were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.94, F(1,76)=5.21, p=.025, η^2=.064

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,76)=.40, p=.79, η^2=.006 but a significant post-intervention group difference, F(1,76)=6.47, p=.013, η^2=.078

Further, the only significant pre- to post intervention change was seen in the control group which deteriorated significantly, Λ =.92, F(1,76)=6.88, p=.01, η^2=.083

There was a significant main effect for time, Λ =.95, F(1, 76)=4.39, p=.041, η^2=.054

There was no significant group main effect, F(1, 76)=2.31, p=.13, η^2=.029

Table App.11.1.1

KSS scores - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	2.79 (1.35)	4.00 (1.93)	7.04	1,76	.01	.083
intervention	3.02 (1.51)	2.96 (1.53)	.033	1,76	.86	<.001

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

Appendix 11.1.2 Epworth Sleepiness Scale (ESS)

Scores on the Epworth Sleepiness Scale (ESS) were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,80)=.20, p=.65, η^2=.003

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 80)=.047, p=.83, η^2=.001 and no significant post-intervention group difference, F(1, 80)=.30, p=.37, η^2=.004

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =.96, F(1, 80)=3.55, p=.063, η^2=.042 nor was there a significant group main effect, F(1, 80)=.19, p=.67, η^2=.002

Table App.11.1.2

ESS scores - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	7.00 (3.67)	7.85 (3.47)	1.97	1,80	.16	.024
intervention	6.80 (3.88)	7.49 (3.83)	1.61	1,80	.21	.020

Appendix 11.1.3 Pittsburgh Sleep Quality Index (PSQI) - global score

Scores on the Pittsburgh Sleep Quality Index (PSQI) global score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.97, F(1,64)=1.67, p=.20, η^2=.025

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,64)=.13, p=.72, η^2=.002 and no significant post-intervention group difference, F(1,64)=2.12, p=.15, η^2=.032

Further, the only significant pre- to post intervention change was seen in the intervention group which improved significantly, Λ =.90, F(1,64)=6.84, p=.011,η^2=.097

There was no significant main effect for time, Λ =.96, F(1,64)=2.75, p=.10, η^2=.041 nor was there a significant group main effect, F(1,64)=1.02, p=.32, η^2=.016

Table App.11.1.3

PSQI global scores - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	10.14 (3.21)	10.00 (3.08)	.057	1,64	.81	.001
intervention	9.82 (3.49)	8.67 (3.62)	6.84	1,64	.011	.097

Appendix 11.1.4 PSQI - subjective sleep quality sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - subjective sleep quality sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(1,78)=1.23, p=.27, η^2=.016 Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,78)=2.37, p=.13, η^2=.030 but a significant post-intervention group difference, F(1,78)=12.22, p=.001 η^2=.13

Further, the only significant pre- to post intervention change was seen in the intervention group which improved significantly, Λ =.89, F(1,78)=9.83, p=.002, η^2=.11

There was a significant main effect for time, Λ =.93, F(1,78)=5.74, p=.019,η^2=.069

There was also a significant group main effect, F(1,78)=1.02, p=.002, η^2=.11

Table App.11.1.4

PSQI subjective sleep quality sub-scores -2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	1.84 (.62)	1.72 (.54)	.59	1,78	.44	.008
intervention	1.60 (.65)	1.27 (.52)	9.83	1,78	.002	.11

Appendix 11.1.5 Pittsburgh Sleep Quality Index (PSQI) - sleep latency sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - sleep latency sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,62)=1.52, p=.11, η^2=.002

Follow up analysis of simple main effects for this interaction revealed a significant difference among the groups at pre-intervention, F(1,62)=6.57, p=.013, η^2=.096 and a significant post-intervention group difference, F(1,62)=4.06, p=.048, η^2=.061

A significant pre- to post intervention change was seen both with the intervention group, Λ =.80, F(1,62)=8.72, p=.004, η^2=.16 and with the control, Λ =.90, F(1,62)=6.50, p=.013, η^2=.095, both improving significantly

There was a significant main effect for time, Λ =.81, F(1,62)=14.41, p<.001,η^2=.19

There was a significant group main effect, F(1,62)=6.44, p=.014, η^2=.094

Table App.11.1.5

PSQI sleep latency sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	2.45 (.67)	2.00 (.93)	6.70	1,62	.012	. 099
intervention	1.86 (.98)	1.48 (1.02)	8.72	1,62	.004	.12

Appendix 11.1.6 Pittsburgh Sleep Quality Index (PSQI) - sleep duration sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - sleep duration sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.97, F(1,73)=4.96, p=.029,η^2=.064

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,73)=.034, p=.85, η^2<.001

but a significant post-intervention group difference, F(1,73)=5.66, p=.020, η^2=.072

Further, the only significant pre- to post intervention change was seen in the intervention group which improved significantly, Λ =.94, F(1,73)=13.38, p=.042, η^2=.055

There was no significant main effect for time, Λ =1.00, F(1,73)=.004, p=.95, η^2=.006

There was a significant group main effect, F(1,73)=4.96, p=.029, η^2=.06

Table App.11.1.6

PSQI sleep duration sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	2.04 (.82)	2.26 (.75)	1.87	1,73	.17	.025
intervention	2.00 (.99)	1.77 (.85)	4.29	1,73	.042	.055

Appendix 11.1.7 Pittsburgh Sleep Quality Index (PSQI) - sleep efficiency sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) sleep efficiency sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.93, F(1,53)=4.21, p=.045, η^2=.074

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,53)=.049, p=.82, η^2=.001

but a significant post-intervention group difference, F(1,53)=5.68, p=.021,η^2=.097

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.93, F(1,53)=4.23, p=.045, η^2=.074

There was a significant main effect for time, Λ =1.00, F(1,53)=.001, p=.98,η^2<.001

There was no significant group main effect, F(1,53)=1.99, p=.16, η^2=.036

Table App.11.1.7

PSQI sleep efficiency sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	1.50 (1.29)	1.86 (1.10)	1.41	1,53	.24	.026
intervention	1.41 (1.22)	1.05 (1.09)	4.23	1,53	.045	.074

App.11.1.8 Pittsburgh Sleep Quality Index (PSQI) - sleep disturbance sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - sleep disturbance sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,70)=.27, p=.61, η^2=.004

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,70)=.25, p=.62, η^2=.004

and no significant post-intervention group difference, F(1, 70)=.009, p=.93,η^2<.001

There was no significant pre to post intervention change in any of the other groups.

There was no significant main effect for time, Λ =1.01, F(1,70)=.27, p=.61 ,η^2=.004

nor was there a significant group main effect, F(1,70)=.060, p=.81, η^2=.001

Table App.11.1.8

PSQI sleep disturbance sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	1.27 (.46)	1.27 (.63)	.00	1,70	1.00	<.001
intervention	1.34 (.56)	1.26 (.49)	.87	1,70	.35	.012

App.11.1.9 Pittsburgh Sleep Quality Index (PSQI) sleep medication sub score (*)

Scores on the Pittsburgh Sleep Quality Index (PSQI) sleep medication sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,79)=.006, p=.94, η^2<.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,79)=3.70, p=.058, η^2=.045

and no significant post-intervention group difference, F(1,79)=3.51, p=.065, η^2=.043

Further, no significant pre to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =1.00, F(1,79)=.20, p=.65, η^2=.003

There a significant group main effect, F(1,79)=4.03, p=.048, η^2=.049

Table App.11.1.9

PSQI sleep medication sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	1.77 (1.18)	1.73 (1.15)	.050	1,79	.82	.001
intervention	1.38 (1.41)	1.33(1.41)	.22	1,79	.64	.003

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.1.10 Pittsburgh Sleep Quality Index (PSQI) dysfunction sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) dysfunction sub score were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,72)=.009, p=.93, η^2<.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,72)=.44, p=.51, η^2=.006

but a significant post-intervention group difference, F(1,72)=.70, p=.040, η^2=.010

Further, no significant pre to post intervention change was seen in any of the groups.

There was no significant main effect for time, Λ =1.0, F(1,72)=.26, p=.61, η^2=.004

nor was there a significant group main effect, F(1,72)=.86, p=.36, η^2=.012

Table App.11.1.10

PSQI dysfunction sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	.92 (.58)	.88 (.68)	.06	1,72	.80	.001
intervention	.80 (.76)	.74 (.63)	.28	1,72	.60	.004

App.11.1.11 Apnea probability score derived from MAP

Apnea probability scores derived from MAP using an algorithm which incorporates age, gender, body mass index (BMI) and several MAP components, were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,66)=.039, p=.84, η^2=.001 Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,66)=.40, p=.53, η^2=.006 nor a significant post-intervention group difference, F(1,66)=.62, p=.43, η^2=.009

Further, no pre to post intervention change was seen in any of the groups.

There was no significant main effect for time, Λ =1.00, F(1,66)=. 045, p=.83, η^2=.001 nor was there a significant group main effect, F(1,66)=.64, p=.42, η^2=.010

Table App.11.1.11

MAP derived apnea probability scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	.37 (.29)	.36 (.29)	.06	1,66	.80	.001
intervention	.42 (.31)	.42 (.33)	.32	1,66	.91	.005

App.11.1.12 Depression Anxiety Stress Scale (DASS) global score

Depression Anxiety Stress Scale (DASS) global scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,71)=.12, p=.73, η^2=.002

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,71)=.10, p=.75, η^2=.001

nor a significant post-intervention group difference, F(1,71)=.54, p=.46, η^2=.008

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.91, F(1,71)=7.03, p=.010, η^2=.09

There was a significant main effect for time, Λ =.92, F(1,71)=6.21, p=.015,η^2=.080

There was no significant group main effect, F(1,71)=.29, p=.59, η^2=.004

Table App.11.1.12

DASS global - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) -post	F	df	p	η^2
control	23.48 (23.30)	19.86 (16.28)	1.61	1,71	.21	.022
interven.	21.87 (17.32)	17.06 (14.03)	7.03	1,71	.010	.090

App.11.1.13 Depression Anxiety Stress Scale (DASS) - stress sub-score

Depression Anxiety Stress Scale (DASS) stress sub-scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,73)=..99, p=.76 η^2=.07

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,73)=.18, p=.67, η^2=.003 nor a significant post-intervention group difference, F(1,73)=.62, p=.43, η^2=.008

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.93, F(1,73)=5.64, p=.020, η^2=.072

There was a significant main effect for time, Λ =.93, F(1,73)=5.12, p=.027,η^2=.66

There was no significant group main effect, F(1,73)=.41, p=.52, η^2=.006

Table App.11.1.13

DASS stress sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	10.50 (11.08)	8.91 (7.65)	1.35	1,73	.25	.018
intervention	9.53 (7.90)	7.43 (7.27)	5.64	1,73	.020	.072

App.11.1.14 Depression Anxiety Stress Scale (DASS) anxiety sub-score

Depression Anxiety Stress Scale (DASS) anxiety sub-scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,76)=.52, p=.47, η^2=.007

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,76)=.002, p=.96, η^2<.001

nor a significant post-intervention group difference, F(1,76)=.30, p=.58, η^2=.004

Further, the intervention group improved pre to post intervention but not significantly, Λ =.95, F(1,76)=3.66, p=.060, η^2=.046

There was no significant main effect for time, Λ =.98, F(1,76)=1.71, p=.19,η^2=.022

There was no significant group main effect, F(1,76)=.062, p=.80, η^2=.001

Table App.11.1.14

DASS anxiety sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	7.00 (6.60)	6.68 (5.80)	.12	1,76	.73	.002
intervention	7.07 (5.72)	5.96 (4.96)	3.66	1,76	.060	.046

App.11.1.15 Depression Anxiety Stress Scale (DASS) depression sub-score

Depression Anxiety Stress Scale (DASS) depression sub-scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,72)=.62, p=.43, η^2=.008

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,72)=.16, p=.69, η^2=.002

nor a significant post-intervention group difference, F(1,72)=1.66, p=.20, η^2=.023

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.93, F(1,72)=5.77, p=.019, η^2=.074

There was no significant main effect for time, Λ =.96, F(1,72)=3.14, p=.080, η^2=.042

There was no significant group main effect, F(1,72)=.74, p=.40, η^2=.010

Table App.11.1.15

DASS depression sub-scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	6.00 (6.86)	5.38 (5.43)	.34	1,72	.56	.005
intervention	5.34 (6.23)	3.74 (4.75)	5.77	1,72	.019	.074

App.11.1.16 Profile of Mood States (POMS) global score (*)

Profile of Mood States (POMS) global scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,64)=.093, p=.76,η^2=.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,64)=.53, p=.47, η^2=.008

nor a significant post-intervention group difference, F(1,64)=1.62, p=.21, η^2=.025

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.90, F(1,64)=7.21, p=.009, η^2=.10

There was a significant main effect for time, Λ =.91, F(1,64)=6.64, p=.012, η^2=.094

There was no significant group main effect, F(1,64)=1.12, p=.29, η^2=.017

Table App.11.1.16

POMS global scores - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	16.16 (25.65)	11.21 (20.86)	1.81	1,64	.18	.028
intervention	11.96 (19.10)	5.68 (13.58)	7.21	1,64	.009	.10

* Homogeneity assumption not satisfied for this variable. No suitable transformation found

App.11.1.17 Profile of Mood States (POMS) depression sub - score (*)

Profile of Mood States (POMS) depression sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,68)=.33, p=.57, η^2=.005

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,68)=.88, p=.35, η^2=.013

nor a significant post-intervention group difference, F(1,68)=3.22, p=.077, η^2=.045

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.94, F(1,68)=4.54, p=.037, η^2=.063

There was no significant main effect for time, Λ =.94, F(1,68)=3.10, p=.083,η^2=.044

There was no significant group main effect, F(1,68)=2.07, p=.16, η^2=.029

Table App.11.1.17

POMS depression sub scores -2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	4.67 (6.37)	4.10 (4.71)	.50	1,68	.48	.007
intervention	3.47 (4.11)	2.35(3.24)	4.54	1,68	.037	.063

* Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable. Results as follows

App.11.1.18 POMS depression sub score (-2 power transformation)

(-2.0) power transformation of (POMS depression sub score +100) was performed. Homogeneity obtained on pre and post measures. Transformed scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,68)=.62, p=.43, η^2=.009

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,68)=.74, p=.34, η^2=.011

nor a significant post-intervention group difference, F(1,63)=3.30, p=.074, η^2=.046

There was no significant main effect for time, Λ =.99, F(1,68)=.62, p=.43, η^2=.009

There was no significant group main effect, F(1,68)=1.99, p=.16, η^2=.028

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly:

Table App.11.1.18

POMS transformed depression sub scores -2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	Λ	F	df	p	η^2
control	1.00	.30	1,68	.59	.004
intervention	.93	5.14	1,68	.027	.070

App.11.1.19 Profile of Mood States (POMS) tension sub - score

Profile of Mood States (POMS) tension sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,69)=.73, p=.79, η^2=.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,69)=.35, p=.56, η^2=.005

nor a significant post-intervention group difference, F(1,69)=1.15, p=.29, η^2=.016

Further, no significant pre to post intervention change was seen with any group

There was no significant main effect for time, Λ =.91, F(1,69)=2.33, p=.13,η^2=.033

nor was there a significant group main effect, F(1,69)=.79, p=.38, η^2=.011

Table App.11.1.19

POMS tension sub scores - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	6.14 (7.03)	5.41 (4.50)	.58	1,69	.45	.008
intervention	5.29 (4.86)	4.24 (4.09)	2.61	1,69	.11	.036

App.11.1.20 Profile of Mood States (POMS) anger sub - score

Profile of Mood States (POMS) anger sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.95, F(1,67)=3.31, p=.073, η^2=.047

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,67)=1.01, p=.75, η^2=.002

but a significant post-intervention group difference, F(1,67)= 4.53, p=.037, η^2=.063

No significant pre to post intervention change was seen with any group

There was no significant main effect for time, Λ =.97, F(1,67)=.56, p=.46,η^2=.008

nor was there a significant group main effect, F(1,67)=1.42, p=.24, η^2=.021

Table App.11.1.20

POMS anger sub scores - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	4.70 (4.93)	5.15 (4.16)	.41	1,67	.52	.006
intervention	4.33 (4.20)	3.24 (3.00)	3.31	1,67	.073	.047

App.11.1.21 Profile of Mood States (POMS) fatigue sub - score

Profile of Mood States (POMS) fatigue sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(1,72)=2.60, p=.11, η^2=.035

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 72)=.20, p=.66, η^2=.003

nor a significant post-intervention group difference, F(1, 72)= 1.90, p=.17, η^2=.026

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.91, F(1,72)=7.08 p=.010, η^2=.089

There was no significant main effect for time, Λ =.98, F(1, 72)=1.64, p=.20,η^2=.022

There was no significant group main effect, F(1, 72)=.20, p=.66, η^2=.003

Table App.11.1.21

POMS – fatigue sub scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	4.59 (3.71)	4.73 (2.80)	.04	1,72	.84	.001
intervention	4.96 (3.06)	3.79 (2.62)	7.08	1,72	.010	.089

App.11.1.22 Profile of Mood States (POMS) confusion sub - score

Profile of Mood States (POMS) confusion sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(1,70)=2.57, p=.11, η^2=.035

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 70)=.42, p=.52, η^2=.006

nor a significant post-intervention group difference, F(1, 70)=.97, p=.33, η^2=.014

No significant pre to post intervention change was seen with any of the groups.

There was a significant main effect for time, Λ =1.00, F(1, 70)=.049, p=.82,η^2=.001

There was no significant group main effect, F(1, 70)=.007, p=.93, η^2<.001

Table App.11.1.22

POMS – confusion sub scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	2.95 (3.00)	3.45 (2.63)	.69	1,70	.41	.010
intervention	3.45 (3.24)	2.82 (2.47)	2.72	1,70	.10	.037

App.11.1.23 Profile of Mood States (POMS) vigour sub - score

Profile of Mood States (POMS) vigour sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,69)=.28, p=.60, η^2=.004

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 69)=.92, p=.34, η^2=.013

nor a significant post-intervention group difference, F(1, 69)=.52, p=.47, η^2=.007

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =.99, F(1, 69)=.38, p=.54, η^2=.006

There was no significant group main effect, F(1, 69)=.90, p=.35, η^2=.013

Table App.11.1.23

POMS – vigour sub scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	8.33 (3.84)	10.52(8.86)	2.20	1,69	.14	.031
intervention	9.94 (7.24)	10.00 (5.20)	.007	1,69	.93	<.001

App.11.1.24 SF36 (Health Survey) global score

SF36 (Health Survey) global scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(1,61)= 2.71, p=.10, η^2=.043

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 61)=.37, p=.55, η^2=.088

and no significant post-intervention group difference, F(1, 61)=.36, p=.55, η^2=.096

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.89, F(1,61)=7.54, p=.008, η^2=.11

There was a significant main effect for time, Λ =.97, F(1, 61)=1.87, p=.18, η^2=.030

There was a significant group main effect, F(1, 61)<.001, p=.98, η^2=.030

Table App.11.1.24

SF36 – global scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	68.16 (11.25)	67.82 (11.69)	.03	1,61	.87	<.001
intervention	66.21 (11.87)	69.90 (12.73)	7.54	1,61	.008	.11

App.11.1.25 SF36 (Health Survey) Physical functioning sub - score (*)

SF36 (Health Survey) Physical functioning sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(1,77)=1.43, p=.89, η^2<001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 77)=2.54, p=.11, η^2=.032

nor a significant post-intervention group difference, F(1, 77)=.47, p=.50, η^2=.006

No significant pre to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =1.00, F(1, 77)=.20, p=.89,η^2<.001

There was no significant group main effect, F(1, 77)=1.42, p=.24, η^2=.018

Table App.11.1.25

SF36 - Physical functioning sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	81.81 (11.22)	79.79 (14.41)	.63	1,77	.43	.008
intervention	75.05 (19.40)	76.64 (20.40)	.91	1,77	.34	.012

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.1.26 SF36 (Health Survey) Role limitations due to physical health sub - score

SF36 (Health Survey) Role limitations due to physical health sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.94, F(1,71)=4.60, p=.035, η^2=.061

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 71)=1.31, p=.26, η^2=.018

nor a significant post-intervention group difference, F(1, 71)=.50, p=.48, η^2=.007

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.94, F(1,71)=4.64, p=.035, η^2=.061

There was no significant main effect for time, Λ =1.00, F(1, 71)=.75, p=.78,η^2=.001

There was no significant group main effect, F(1, 71)=.65, p=.79, η^2=.001

Table App.11.1.26

SF36 – Physical Role limitations sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	65.22 (37.49)	57.61 (39.48)	1.26	1,71	.26	.08
intervention	54.33 (37.95)	64.17 (35.60)	4.64	1,71	.035	.061

App.11.1.27 SF36 (Health Survey) body pain sub - score

SF36 (Health Survey) body pain sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =1.00, F(1,76)=.013, p=.91, η^2<.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 76)=.65, p=.80, η^2=.001

nor a significant post-intervention group difference, F(1, 76)=.32, p=.72, η^2=.002

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =1.00, F(1, 76)=.038, p=.85, η^2<.001

There was no significant group main effect, F(1, 76)=.12, p=.73, η^2=.002

Table App.11.1.27

SF36 – body pain sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	65.78 (22.77)	64.96 (25.23)	.033	1,76	.86	<.001
intervention	67.25 (23.46)	67.04 (22.05)	.006	1,76	.94	<.001

App.11.1.28 SF36 (Health Survey) general health sub - score

SF36 (Health Survey) general health sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,71)=.19, p=.66, η^2=.003

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 71)=0.94, p=.76, η^2=.001

nor a significant post-intervention group difference, F(1, 71)=.39, p=.53, η^2=.006

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =1.0, F(1,71)=.01, p=.75, η^2=.001

There was no significant group main effect, F(1, 71)=.22, p=.62, η^2=.003

Table App.11.1.28

SF36 – general health sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	59.99 (19.45)	59.80 (14.05)	.005	1,71	.94	<.001
intervention	61.41 (16.85)	62.57 (17.70)	.51	1,71	.47	.007

App.11.1.29 SF36 (Health Survey) vitality/energy sub - score

SF36 (Health Survey) vitality/energy sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,70)=.14, p=.87, η^2=.004

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 70)=1.29, p=.28, η^2=.036

nor a significant post-intervention group difference, F(1, 70)=2.09, p=.13, η^2=.057

A significant pre to post intervention change was seen in the intervention group Λ =.94, F(1, 70)=3.86, p=.053, η^2=.052 which improved significantly.

There was a significant main effect for time, Λ =.94, F(1, 70)=4.25, p=.043, η^2=.058

There was no significant group main effect, F(1, 70)=2.11, p=.13, η^2=.058

Table App.11.1.29

SF36 – vitality/energy sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	58.25 (17.50)	61.50 (18.36)	.83	1, 70	.37	.012
intervention	59.33 (17.35)	63.65 (17.84)	3 86	1, 70	.053	.052

App.11.1.30 SF36 (Health Survey) social functioning sub – score (*)

SF36 (Health Survey) social functioning sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.93, F(1,77)= 5.96, p=.017, η^2=.072

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1,77)=.63, p=.43, η^2=.008

and no significant post-intervention group difference, F(1, 77)=2.58, p=.11, η^2=.032

Further, the only significant pre to post intervention change was seen in the intervention group which improved significantly, Λ =.94, F(1, 77)=4.91, p=.030, η^2=.060

There was no significant main effect for time, Λ =1.00, F(1, 77)=.003, p=.96,η^2=.001

nor was there a significant group main effect, F(1, 77)=.26, p=.61, η^2=.003

Table App.11.1.30

SF36 – social functioning sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	81.52 (21.93)	75.00 (22.61)	2.36	1,77	.13	.03
intervention	77.45 (21.93)	83.70 (21.57)	4.91	1,77	.030	.060

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.1.31 SF36 Role limitations due to emotional function sub – score (*)

SF36 (Health Survey) Role limitations due to emotional function sub – scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.88, F(1,68)= 8.61, p=.005, η^2=.11

Follow up analysis of simple main effects for this interaction revealed a significant difference among the groups at pre-intervention, F(1, 68)=7.10, p=.009, η^2=.11

but no significant post-intervention group difference, F(1, 68)=.26, p=.61, η^2=.004

Further, a significant pre to post intervention change was seen both with the control group which deteriorated significantly, Λ =.94, F(1, 68)=4.61, p=.035, η^2=.064 and the intervention group which improved significantly Λ =.94, F(1, 68)=4.25, p=.043, η^2=.059

There was no significant main effect for time, Λ =.99, F(1, 68)=.39, p=.53, η^2=.006

There was a significant group main effect, F(1, 68)=1.73, p=.14, η^2=.09

Table App.11.1.31

SF36 – emotional role limitations sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
control	86.36 (26.54)	68.18 (39.14)	4.61	1,68	.035	.064
intervention	61.11 (40.29)	72.92 (35.57)	4.25	1,68	.043	.059

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.1.32 SF36 (Health Survey) emotional well being/mental health subscore

SF36 (Health Survey) emotional well being/mental health sub - scores were analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,70)= .057, p=.81, η^2=.010

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 70)=.83, p=.37, η^2=.012

but a significant post-intervention group difference, F(1, 70)=.37, p=.54, η^2=.025

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =.99, F(1, 70)=.74, p=.39, η^2=.010

There was a significant group main effect, F(1, 70)=.81, p=.37, η^2=.011

Table App.11.1.32

SF36 – emotional well being sub - scores 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	67.00 (17.60)	69.65 (15.75)	.42	1,70	.52	.006
intervention	70.92 (15.92)	71.65 (17.22)	.35	1,70	.56	.005

Appendix 11.2 Objective measures, Intention to Treat (ITT) analysis (Intervention versus control), Analysis of variance (ANOVA)

App.11.2.1 Total time in bed (TTB)

'Total time in bed' was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(1,69)= 1.05, p=.31, η^2=.015

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 69)=1.33, p=.25, η^2=.019

nor a significant post-intervention group difference, F(1, 69)=.013, p=.91, η^2<.001

No significant pre - to post intervention change was seen with any group .

There was no significant main effect for time, Λ =1.00, F(1, 69)=.019, p=.89, η^2=.005

There was no significant group main effect, F(1, 69)=.44, p=.51, η^2=.006

Table App.11.2.1

Total time in bed - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	402.3 (91.43)	416.30 (73.98)	.50	1,69	.48	.008
YI	424.85 (70.33)	414.04 (78.31)	.61	1,69	.44	.009

App.11.2.2 Total sleep time (TST)

Total sleep time was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,68)= .52, p=.47, η^2=.008

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=1.34, p=.25, η^2=.019

nor a significant post-intervention group difference, F(1, 68)=.005, p=.83, η^2=.001

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.16, F(1, 68)=.08, p=.99, η^2<.001

There was no significant group main effect, F(1, 68)=.73, p=.40, η^2=.011

Table App.11.2.2

Total sleep time - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η2
Control	355.91 (80.92)	363.78 (62.06)	.20	1,68	.65	.003
intervention	375.32 (57.24)	367.74 (74.57)	.38	1,68	.54	.006

App.11.2.3 Sleep onset latency (SOL) [*]

Sleep latency was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,69)=1.00, p=.32, η^2=.014

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 69)=.010, p=.92, η^2<.001

nor a significant post-intervention group difference, F(1, 69)=2.04, p=.16, η^2=.029

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.99, F(1, 69)=.54, p=.47, η^2=.008

There was no significant group main effect, F(1, 69)=1.33, p=.25, η^2=.019

Table App.11.2.3

Sleep latency - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	9.11 (10.36)	8.39 (8.33)	.027	1,69	.87	<.001
intervention	9.46 (14.97)	14.07 (18.13)	2.33	1,69	.13	.033

* Normality assumption not satisfied for this variable. No suitable transformation found.

App.11.2.4 Wake After Sleep Onset (WASO)

Total wake time duration was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(1,66)=3.78, p=.056, η^2=.054

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 66)=2.80, p=.10, η^2=.041

nor a significant post-intervention group difference, F(1, 66)=.59, p=.45, η^2=.009

Further, no significant pre - to post intervention change was seen with any of the groups, although both intervention groups improved and the control deteriorated but not significantly.

There was no significant main effect for time, Λ =1.00, F(1, 66)=.003, p=.96, η^2<.001

There was no significant group main effect, F(1, 66)=.56, p=.45, η^2=.008

Table App.11.2.4

Total WASO - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, and control)

group	M (SD) - pre	M (SD) - post	F	df	p	η2
Control	50.73 (18.14)	57.14 (19.79)	1.46	1,66	.23	.022
intervention	59.80 (22.12)	53.74 (15.72)	2.76	1,66	.10	.040

App.11.2.5 Sleep Efficiency (SE)

Sleep efficiency was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.97, F(1,66)=2.36, p=.13, η^2=.034

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 66)=1.71, p=.31, η^2=.025

nor a significant post-intervention group difference, F(1, 66)=.040, p=.23, η^2=.012

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =1.0, F(1, 66)=.23, p=.88, η^2<.001

There was no significant group main effect, F(1, 66)=.21, p=.64, η^2=.003

Table App.11.2.5

Sleep efficiency - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, and control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	.87 (.040)	.87 (.030)	1.04	1,66	.31	.016
intervention	.86 (.004)	.87 (.037)	1.48	1,66	.23	.022

App.11.2.6 Total light sleep duration

Total Light Sleep duration was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(1,68)=1.34, p=.25, η^2=.019

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=1.61, p=.21, η^2=.023

nor a significant post-intervention group difference, F(1, 68)=.006, p=.74, η^2<.001

Further, no significant pre - to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =1.00, F(1, 68)=.004, p=.95, η^2<.001

There was no significant group main effect, F(1, 68)=.61, p=.44, η^2=.009

Table App.11.2.6

Total light sleep duration - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	176.83 (55.30)	184.69 (47.48)	.44	1,68	.51	.007
intervention	192.59 (45.39)	183.82 (43.42)	1.13	1,68	.29	.016

App.11.2.7 Total Slow Wave stage (SWS) duration

Total Slow Wave Sleep (deep sleep) duration was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,68)=.33, p=.57, η^2=.005

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=.001, p=.97, η^2<.001

but a significant post-intervention group difference, F(1, 68)=.47, p=.49, η^2=.007

No significant pre - to post intervention change was seen with any group.

There was no significant main effect for time, Λ =.99, F(1, 68)=.53, p=.47, η^2=.008

There was no significant group main effect, F(1, 68)=.19, p=.67, η^2=.003

Table App.11.2.7

Total SWS duration - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	106.74 (24.80)	107.44 (20.70)	.009	1,68	.92	<.001
intervention	106.49 (28.00)	112.34 (30.92)	1.28	1,68	.26	.019

App.11.2.8 Slow Wave stage (SWS) latency [*]

Slow Wave stage (SWS) latency was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,68)=.57, p=.45, η^2=.008

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=0.088, p=.80, η^2=.001

nor a significant post-intervention group difference, F(1, 68)=1.78, p=.18, η^2=.026

Further, no significant pre - to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =1.00, F(1, 68)=.001, p=.97, η^2<.001

There was no significant group main effect, F(1, 68)=.97, p=.33, η^2=.014

Table App.11.2.8

Total SWS latency - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	18.61 (20.40)	15.39 (16.27)	.19	1,68	.66	.003
intervention	20.92 (34.46)	24.44 (30.34)	. 47	1,68	.50	.007

* Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable. Results as follows:

App.11.2.9 Slow Wave stage (SWS) latency (-1 power transformation)

Slow Wave stage (SWS) latency was transformed by using (-1) power transformation on (SWS latency +1) and then analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,68)=.082, p=.77, η^2=.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=.15, p=.70, η^2=.002

nor a significant post-intervention group difference, F(1, 68)<.001, p=.99, η^2<.001

There was no significant main effect for time, Λ =.97, F(1, 68)= 1.92, p=.17, η^2=.028

There was no significant group main effect, F(1, 68)=.11, p=.74, η^2=.002

Further, no significant pre - to post intervention change was seen with any of the groups:

Table App.11.2.9

Transformed SWS latency - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	Λ	F	df	p	η^2
Control	.99	.45	1,68	.50	.007
intervention	.97	2.13	1,68	.15	.030

Appendix 11.2.10 Total Rapid Eye Movement stage (REM) duration

Total Rapid Eye Movement stage (dream stage) duration was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,68)=.15, p=.70, η^2=.002

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=.008, p=.93, η^2<.001

nor a significant post-intervention group difference, F(1, 68)=.10, p=.75, η^2=.001

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.99, F(1, 68)=.40, p=.53, η^2=.006

There was no significant group main effect, F(1, 68)=.027, p=.87, η^2<.001

Table App.11.2.10

Total REM duration- 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	70.43 (28.12)	69.39 (28.53)	.023	1,68	.88	<.001
intervention	71.04 (25.74)	66.72 (34.80)	. 80	1,68	.37	.012

App.11.2.11 Rapid Eye Movement stage (REM) latency

Rapid Eye Movement stage (dream stage) latency was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(1,67)=2.52, p=.12, η^2=.036

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 67)=.28, p=.60, η^2=.004

nor a significant post-intervention group difference, F(1, 67)=1.38, p=.24, η^2=.020

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.99, F(1, 67)=.68, p=.41, η^2=010

There was no significant group main effect, F(1, 67)=.15, p=.70, η^2=.002

Table App.11.2.11

REM latency- 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	98.30 (44.25)	113.70 (48.49)	2.16	1,67	.15	.032
intervention	104.61 (47.80)	99.72 (45.66)	.44	1,67	.51	.007

App.11.2.12 Mean blood oxygen saturation (SPO2) during night

Mean blood oxygen saturation level was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(1,69)= .014, p=.61, η^2=.004

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 69)<.001, p=.99, η^2<.001nor a significant post-intervention group difference, F(1, 69)=.35, p=.85, η^2=.001

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =1.00, F(1, 69)=.26, p=.61, η^2<.001

There was no significant group main effect, F(1, 69)=.011, p=.92, η^2<.001

Table App.11.2.12

Mean blood oxygen saturation level- 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	97.13 (4.20)	96.87 (4.21)	.057	1,69	.81	.001
intervention	97.12 (4.82)	96.71 (2.95)	.31	1,69	.58	.004

App.11.2.13 Respiratory Disturbance Index (RDI) [*]

Respiratory Disturbance Index (RDI) was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,68)= .43, p=.51, η^2=.006

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=2.62, p=.10, η^2=.037

nor a significant post-intervention group difference, F(1, 68)=3.23, p=.077, η^2=.045

Further, no significant change was seen with any of the groups pre to post intervention

There was no significant main effect for time, Λ =1.00, F(1, 68)=.025, p=.87, η^2<.001

There was no significant group main effect, F(1, 68)=3.22, p=.077, η^2=.045

Table App.11.2.13

RDI - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	10.90 (6.45)	10.24 (5.55)	.25	1,68	.62	.004
intervention	14.92 (10.99)	15.32 (12.96)	.19	1,68	.66	.003

* Homogeneity assumption not satisfied for this variable. Homogeneity corrected using suitable power transformation (-1.939). ANOVA performed on transformed variable. Results as follows:

App.11.2.14 Respiratory Disturbance Index (RDI) (-1.939 Power Transformation)

Transformed Respiratory Disturbance Index (RDI) was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(1,68)=1 .07, p=.30, η^2=.015

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 68)=.48, p=.49, η^2=.007

nor a significant post-intervention group difference, F(1, 68)=.47, p=.49, η^2=.007

There was no significant main effect for time, Λ =.99, F(1, 68)=.40, p=.53, η^2=.006

There was no significant group main effect, F(1, 68)=.14, p=.71, η^2=.002

Further, no significant pre to post intervention change was seen with any of the groups:

Table App.11.2.14

Transformed RDI - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	Λ	F	df	p	η^2
Control	.99	.06	1,68	.81	.001
intervention	.97	2.11	1,68	.15	.030

App.11.2.15 Oxygen Desaturation Index (ODI)

Apnea Duration Index (ODI) was analysed using a 2 x 2 mixed analysis of variance (ANOVA). The between-subjects factor was group (intervention, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.91, F(1,69)= .096, p=.76, η^2=.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(1, 69)=2.16, p=.15, η^2=.030

and no significant post-intervention group difference, F(1, 69)=2.47, p=.12, η^2=.035

Further, no significant change was seen with any of the groups pre to post intervention

There was no significant main effect for time, Λ =1.00, F(1, 69)=.036, p=.85, η^2=.001

There was no significant group main effect, F(1, 69)=2.55, p=.11, η^2=.036

Table App.11.2.15

ODI - 2 x 2 mixed ANOVA - Pre to Post intervention change by group (intervention, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
Control	7.82 (6.70)	7.74 (5.94)	.005	1,69	.94	<.001
YI	11.34 (10.45)	11.70 (11.33)	.19	1,69	.66	.003

Appendix 11.3 Objective measures, Intention to Treat (ITT) analysis (Intervention versus control), Multivariate analysis of variance (MANOVA)

App.11.3.1 Insomnia related multivariate analysis of variance (MANOVA)

The data for the insomnia multivariate was analysed using a 2 x 2 mixed multivariate analysis of variance (MANOVA). The two-level between-subjects factor was group (intervention, and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of four measures related to insomnia namely: sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST) and sleep efficiency (SE).

Multivariate tests revealed no significant main effect for phase, $\Lambda = .95$, $F(4, 63) = .78$, $p = .54$, $\eta^2 = .047$, no significant multivariate main effect for group, $\Lambda = .93$, $F(4, 63) = 1.17$, $p = .33$, $\eta^2 = .069$, and no significant multivariate interaction, $\Lambda = .90$, $F(4, 63) = 1.71$, $p = .16$, $\eta^2 = .098$.

When the sub-scales for the multivariate main effect for phase were considered separately, there was no significant univariate phase main effect, no significant univariate group main effect, and no significant univariate interaction.

Examination of the marginal means showed that across all groups, for WASO and SE (sleep efficiency) remained without change, for TST improved, and for SOL deteriorated slightly from baseline to post-intervention.

Analysis of the multivariate simple main effects for time/phase within group revealed a significant pre - to post-intervention change for the intervention group only:

Table App.11.3.1

Insomnia related multivariate analysis of variance (MANOVA) - Pre to Post intervention change by group (intervention, control)

group	Λ	F	df	p	η^2
control	.97	. 40	4, 63	. 81	. 025
intervention	.84	3.02	4, 63	.024	.16

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App.11.3.2 OSA related multivariate analysis of variance (MANOVA)

The data for OSA was analysed using a 2 x 2 mixed multivariate analysis of variance (MANOVA). The two-level between-subjects factor was group (YI, control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of three measures related to OSA namely, RDI (transformed), ODI, and mean SPO2. Multivariate tests revealed no significant main effect for phase, $\Lambda = .99$, $F(3, 66) = .26$, $p = .85$, $\eta^2 = .012$, no significant multivariate main effect for group, $\Lambda = .95$, $F(3, 66) = 1.18$, $p = .33$, $\eta^2 = .051$, and no significant multivariate interaction $\Lambda = .96$, $F(3, 66) = .91$, $p = .44$, $\eta^2 = .040$. When the sub-scales for the multivariate main effect for phase were considered separately, there was no significant univariate phase main effect, no significant univariate group main effect, and no significant univariate interaction. Examination of the marginal means showed a slight deterioration in SPO2 and ODI measures and slight improvement in RDI measure - across all groups from baseline to post-intervention. Analysis of the multivariate simple main effects for time/phase within group revealed no significant pre- to post-intervention change for any of the groups:

Table App.11.3.2
OSA related multivariate analysis of variance (MANOVA) - Pre to Post intervention change by group (YI, control)

group	Λ	F	df	p	η^2
control	.97	.077	3,66	.97	.004
YI	.93	1.63	3,66	.19	.069

App.11.3.3 Slow Wave Sleep (SWS) - multivariate analysis of variance (MANOVA)

The data for the SWS was analysed using a 2 x 2 mixed multivariate analysis of variance (MANOVA). The two-level between-subjects factor was group (intervention, and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of two measures related to SWS, namely, SWS duration and SWS latency (transformed). Multivariate tests revealed no significant multivariate main effect for phase, $\Lambda = .96$, $F(2,66) = 1.25$, $p = .29$, $\eta^2 = .037$, no significant multivariate main effect for group, $\Lambda = .99$, $F(2, 66) = .27$, $p = .76$, $\eta^2 = .008$, and no significant multivariate interaction, $\Lambda = .99$, $F(2, 66) = .38$, $p = .69$, $\eta^2 = .011$.

When the sub-scales for the multivariate main effect for phase were considered separately, there was neither a significant univariate phase main effect, nor a significant univariate group main effect, nor a significant univariate phase by group. Examination of the marginal means showed that, for SWS duration, participants across all groups improved from baseline to post-intervention, but for SWS latency - remained unchanged from baseline to post-intervention. Analysis of the multivariate simple main effects for time/phase within group revealed no significant pre- to post-intervention change for any group:

Table App.11.3.3
SWS related multivariate analysis of variance (MANOVA) - Pre to Post intervention change by group (intervention, control)

group	Λ	F	df	p	η^2
control	.99	.23	2,66	.80	.007
intervention	.94	1.99	2,66	.14	.057

App.11.3.4 REM sleep - multivariate analysis of variance (MANOVA)

The data for the REM sleep was analysed using a 2 x 2 mixed multivariate analysis of variance (MANOVA). The two-level between-subjects factor was group (intervention, and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of two measures related to REM, namely, REM duration and REM latency (transformed).

Multivariate tests revealed no significant multivariate main effect for phase, $\Lambda = .99$, $F(2,65)=.36$, $p = .70$, $\eta^2 = .011$, no significant multivariate main effect for group, $\Lambda = 1.00$, $F(2, 65) =.053$, $p = .95$, $\eta^2 = .002$, and no significant multivariate interaction, $\Lambda = .96$, $F(2, 65) =1.20$, $p = .31$, $\eta^2 = .036$.

When the sub-scales for the multivariate main effect for phase were considered separately, there was neither a significant univariate phase main effect, nor a significant univariate group main effect, nor a significant univariate phase by group.

Examination of the marginal means showed that, for REM duration, participants across all groups improved from baseline to post-intervention, but for REM latency deteriorated from baseline to post-intervention

Analysis of the multivariate simple main effects for time/phase within group revealed no significant pre- to post-intervention change for any group.

Table App.11.3.4
REM related multivariate analysis of variance (MANOVA) - Pre to Post intervention change by group (intervention, control)

group	Λ	F	df	p	η^2
Control	.97	1.06	2,65	.35	.032
intervention	.99	.25	2,65	.78	.008

App.11.4 Subjective measures, 'On treatment' (OT) subset (low compliance versus high compliance versus control)– analysis of variance (ANOVA)

App.11.4.1 Karolinska Sleepiness Scale (KSS) (*)

Scores on the Karolinska Sleepiness Scale (KSS) were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.90, F(2,75)=4.00, p=.022, η^2=.096

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,75)=.23, p=.79, η^2=.006 but a significant post-intervention group difference, F(2,75)=5.36, p=.007, η^2=.12

Further, the only significant pre- to post intervention change was seen in the control group which deteriorated significantly, Λ =.91, F(1,75)=7.04, p=.01, η^2=.086

There was no significant main effect for time, Λ =.97, F(1, 75)=2.51, p=.12, η^2=.032 nor was there a significant group main effect, F(2, 75)=2.04, p=.14, η^2=.052

Table App.11.4.1

KSS scores - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	2.96 (1.46)	3.46 (1.93)	1.20	1,75	.28	.016
high comp.	3.07 (1.57)	2.57 (.97)	1.51	1,75	.22	.020
control	2.79 (1.35)	4.00 (1.93)	7.04	1,75	.01	.086

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.4.2 Epworth Sleepiness Scale (ESS)

Scores on the Epworth Sleepiness Scale (ESS) were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(2,79)=.13, p=.88, η^2=.003

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 79)=.91, p=.41, η^2=.023 and no significant post-intervention group difference, F(2,79)=.82, p=.44, η^2=.020

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =.96, F(1,79)=3.45, p=.067, η^2=.042 nor was there a significant group main effect, F(2, 79)=1.01, p=.37, η^2=.025

Table App.11.4.2

ESS scores - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	6.08 (4.23)	6.69 (4.42)	1.04	1,79	.31	.013
high comp.	7.43 (3.49)	7.78 (3.58)	.60	1,79	.44	.008
control	7.00 (3.67)	7.85 (3.47)	1.97	1,79	.16	.024

App.11.4.3 Pittsburgh Sleep Quality Index (PSQI) - global score

Scores on the Pittsburgh Sleep Quality Index (PSQI) global score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.82, F(2,63)=6.80, p=.002, η^2=.18

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,63)=.62, p=.54, η^2=.019 and no significant post-intervention group difference, F(2,63)=2.53, p=.088, η^2=.074

Further, the only significant pre- to post intervention change was seen in the high compliance group which improved significantly, Λ =.77, F(1,63)=18.90, p<.001,η^2=.23

There was no significant main effect for time, Λ =.95, F(1,63)=3.36, p=.072, η^2=.051 nor was there a significant group main effect, F(2,63)=.56, p=.57, η^2=.018

Table App.11.4.3

PSQI global scores - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	9.17 (3.94)	9.72 (4.03)	.74	1,63	.39	.012
high comp.	10.26 (3.14)	7.96 (3.22)	18.90	1,63	<.001	.23
control	10.14 (3.21)	10.00 (3.08)	.057	1,63	.81	.001

App.11.4.4 Pittsburgh Sleep Quality Index (PSQI) - subjective sleep quality sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - subjective sleep quality sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(2,77)=.69, p=.50, η^2=.018 Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,77)=1.26, p=.29, η^2=.032 but a significant post-intervention group difference, F(2,77)=6.04, p=.004, η^2=.136

Further, the only significant pre- to post intervention change was seen in the high compliance group which improved significantly, Λ =.92, F(1,77)=6.66, p=.012, η^2=.08

There was a significant main effect for time, Λ =.90, F(1,77)=8.56, p=.005,η^2=.1

There was also a significant group main effect, F(2,77)=5.05, p=.009, η^2=.12

Table App.11.4.4

PSQI subjective sleep quality sub-scores -2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	1.56 (.71)	1.28 (.46)	3.23	1,77	.076	.04
high comp.	1.63 (.61)	1.27 (.58)	6.66	1,77	.012	.08
control	1.84 (.62)	1.72 (.54)	.59	1,77	.44	.008

App.11.4.5 Pittsburgh Sleep Quality Index (PSQI) - sleep latency sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - sleep latency sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,61)=1.52, p=.23, η^2=.047

Follow up analysis of simple main effects for this interaction revealed a significant difference among the groups at pre-intervention, F(2,61)=3.37, p=.041, η^2=.10 and a significant post-intervention group difference, F(2,61)=3.96, p=.024, η^2=.11

A significant pre- to post intervention change was seen in the high compliance group, Λ =.84, F(1,61)=11.56, p=.001, η^2=.16 and also with the control, Λ =.90, F(1,61)=6.70, p=.012, η^2=.099, both improving significantly

There was a significant main effect for time, Λ =.82, F(1,61)=13.10, p=.001,η^2=.177

There was a significant group main effect, F(2,61)=4.22, p=.019, η^2=.12

Table App.11.4.5

PSQI sleep latency sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	1.94 (1.03)	1.82 (1.07)	.35	1,61	.56	.006
high comp.	1.80 (.96)	1.24 (.93)	11.56	1,61	.001	.166
control	2.45 (.67)	2.00 (93)	6.70	1,61	.012	. 099

App.11.4.6 Pittsburgh Sleep Quality Index (PSQI) - sleep duration sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - sleep duration sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.83, F(2,72)=7.40, p=.001,η^2=.17

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,72)=1.64, p=.20, η^2=.044

but a significant post-intervention group difference, F(2,72)=3.12, p=.050, η^2=.080

Further, the only significant pre- to post intervention change was seen in the high compliance group which improved significantly, Λ =.84, F(1,72)=13.38, p<.001,η^2=.15

There was no significant main effect for time, Λ =.99, F(1,72)=.41, p=.53, η^2=.006

nor was there a significant group main effect, F(2,72)=1.12, p=.33, η^2=.03

Table App.11.4.6

PSQI sleep duration sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	1.74 (1.10)	1.87 (.87)	.67	1,72	.41	.009
high comp.	2.21(.86)	1.69 (.85)	13.38	1,72	<.001	.16
control	2.04 (.82)	2.26 (.75)	1.87	1,72	.17	.025

App.11.4.7 Pittsburgh Sleep Quality Index (PSQI) - sleep efficiency sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) sleep efficiency sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.88, F(2,52)=3.38, p=.042, η^2=.11

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,52)=.22, p=.80, η^2=.008

but a significant post-intervention group difference, F(2,52)=3.23, p=.048,η^2=.11

Further, the only significant pre to post intervention change was seen in the hi compliance group which improved significantly, Λ =.89, F(1,52)=6.75, p=.012, η^2=.115

There was no significant main effect for time, Λ =.99, F(1,52)=.44, p=.51,η^2=.008

nor was there a significant group main effect, F(2,52)=.98, p=.38, η^2=.036

Table App.11.4.7

PSQI sleep efficiency sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	1.28 (1.23)	1.22 (1.00)	.044	1,52	.83	.001
high comp.	1.52 (1.24)	.91 (1.16)	6.75	1,52	.012	.11
control	1.50 (1.29)	1.86 (1.10)	1.41	1,52	.24	.026

App.11.4.8 Pittsburgh Sleep Quality Index (PSQI) - sleep disturbance sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) - sleep disturbance sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,69)=1.70, p=.19, η^2=.047

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,69)=.80, p=.45, η^2=.023

and no significant post-intervention group difference, F(2, 69)=.34, p=.71,η^2=.01

The only improvement pre to post intervention was seen with the hi compliance group although not significantly, Λ =.95, F(1,69)=3.49, p=.066, η^2=.048 . There was no significant pre to post intervention change in any of the other groups as well.

There was no significant main effect for time, Λ =1.01, F(1,69)=.28, p=.60 ,η^2=.004

nor was there a significant group main effect, F(2,69)=.048, p=.95, η^2=.001

Table App.11.4.8

PSQI sleep disturbance sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	1.24 (.54)	1.33 (.48)	5.3	1,69	.47	.008
high comp.	1.41 (.57)	1.21 (.49)	3.49	1,69	.066	.048
control	1.27 (.46)	1.27 (.63)	.00	1,69	1.00	<.001

App.11.4.9 Pittsburgh Sleep Quality Index (PSQI) sleep medication sub score (*)

Scores on the Pittsburgh Sleep Quality Index (PSQI) sleep medication sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.0, F(2,78)=.009, p=.99, η^2<.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,78)=1.87, p=.16, η^2=.046

and no significant post-intervention group difference, F(2,78)=1.80, p=.17, η^2=.044

Further, no significant pre to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =1.00, F(1,78)=.25, p=.62, η^2=.003

nor was there a significant group main effect, F(2,78)=2.05, p=.13, η^2=.05

Table App.11.4.9

PSQI sleep medication sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	1.44 (1.39)	1.40 (1.41)	.052	1,78	.82	.001
high comp.	1.33 (1.45)	1.27 (1.48)	.174	1,78	.68	.002
control	1.77 (1.18)	1.73 (1.15)	.050	1,78	.82	.001

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.4.10 Pittsburgh Sleep Quality Index (PSQI) dysfunction sub score

Scores on the Pittsburgh Sleep Quality Index (PSQI) dysfunction sub score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,71)=.35, p=.71, η^2=.01

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,71)=.69, p=.50, η^2=.019

nor a significant post-intervention group difference, F(2,71)=2.63, p=.079, η^2=.069

Further, no significant pre to post intervention change was seen in any of the groups.

There was no significant main effect for time, Λ =1.0, F(1,71)=.25, p=.62, η^2=.003

nor was there a significant group main effect, F(2,71)= 2.22, p=.12, η^2=.059

Table App.11.4.10

PSQI dysfunction sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	.91 (.81)	.95 (.65)	.71	1,71	.79	.001
high comp.	.71 (.71)	.57 (.57)	.89	1,71	.35	.012
control	.92 (.58)	.88 (.68)	.06	1,71	.80	.001

App.11.4.11 Apnea probability score derived from MAP

Apnea probability scores derived from MAP using an algorithm which incorporates age, gender, body mass ratio (BMR) and several MAP components, were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,65)=.40, p=.67, η^2=.012 Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,65)=.20, p=.82, η^2=.006 nor a significant post-intervention group difference, F(2,65)=.69, p=.50, η^2=.021

Further, no pre to post intervention change was seen in any of the groups.

There was no significant main effect for time, Λ =1.00, F(1,65)=. 061, p=.81, η^2=.001 nor was there a significant group main effect, F(2,65)=.40, p=.67, η^2=.012

Table App.11.4.11

MAP derived apnea probability scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	.42 (.31)	.37 (.29)	.44	1,65	.51	.007
high comp.	.42 (.32)	.46 (.35)	.32	1,65	.57	.005
control	.37 (.29)	.36 (.29)	.06	1,65	.80	.001

App.11.4.12 Depression Anxiety Stress Scale (DASS) global score

Depression Anxiety Stress Scale (DASS) global score were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,70)=1.88, p=.16, η^2=.051

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,70)=.24, p=.79, η^2=.007

nor a significant post-intervention group difference, F(2,70)=.65, p=.53, η^2=.018

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.87, F(1,70)=10.78, p=.002, η^2=.13

There was a significant main effect for time, Λ =.90, F(1,70)=7.48, p=.008,η^2=.097

There was no significant group main effect, F(2,70)=.14, p=.87, η^2=.004

Table App.11.4.12

DASS global - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	20.04 (19.21)	19.04 (13.08)	.14	1,70	.71	.002
high comp.	23.31 (15.86)	15.48 (14.77)	10.78	1,70	.002	.13
control	23.48 (23.30)	19.86 (16.28)	1.67	1,70	.20	.023

App.11.4.13 Depression Anxiety Stress Scale (DASS) - stress sub-score

Depression Anxiety Stress Scale (DASS) stress sub-scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.97, F(2,72)=1.09, p=.34, η^2=.03

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,72)=.27, p=.76, η^2=.008

nor a significant post-intervention group difference, F(2,72)=.43, p=.65, η^2=.012

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.91, F(1,72)=7.50, p=.008, η^2=.094

There was a significant main effect for time, Λ =.92, F(1,72)=6.27, p=.015,η^2=.079

There was no significant group main effect, F(2,72)=.21, p=.81, η^2=.006

Table App.11.4.13

DASS stress sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	8.71 (7.86)	8.00 (7.13)	.30	1,72	.59	.004
high comp.	10.21 (8.01)	6.97 (7.48)	7.50	1,72	.008	.094
control	10.50 (11.08)	8.91 (7.65)	1.37	1,72	.24	.019

App.11.4.14 Depression Anxiety Stress Scale (DASS) anxiety sub-score

Depression Anxiety Stress Scale (DASS) anxiety sub-scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,75)=1.78, p=.17, η^2=.045

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,75)=.53, p=.59, η^2=.014

nor a significant post-intervention group difference, F(2,75)=.18, p=.84, η^2=.005

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.92, F(1,75)=6.8, p=.011, η^2=.083

There was no significant main effect for time, Λ =.97, F(1,75)=2.67, p=.11,η^2=.034

There was no significant group main effect, F(2,75)=.14, p=.87, η^2=.004

Table App.11.4.14

DASS anxiety sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	6.19 (5.64)	6.15 (4.10)	.002	1,75	.96	<.001
high comp.	7.83 (5.78)	5.80 (5.67)	6.79	1,75	.011	.083
control	7.00 (6.60)	6.68 (5.80)	.12	1,75	.73	.002

App.11.4.15 Depression Anxiety Stress Scale (DASS) depression sub-score

Depression Anxiety Stress Scale (DASS) depression sub-scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.94, F(2,71)=2.2, p=.12, η^2=.058

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,71)=.11, p=.89, η^2=.003

nor a significant post-intervention group difference, F(2,71)=2.03, p=.14, η^2=.054

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.88, F(1,71)=9.67, p=.003, η^2=.12

There was a significant main effect for time, Λ =.94, F(1,71)=4.56, p=.034, η^2=.060

There was no significant group main effect, F(2,71)=.53, p=.59, η^2=.015

Table App.11.4.15

DASS stress sub-scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	5.08 (7.41)	4.88 (5.91)	.05	1,71	.83	.001
high comp.	5.55 (5.18)	2.79 (3.34)	9.69	1,71	.003	.12
control	6.00 (6.86)	5.38 (5.43)	.35	1,71	.55	.005

App.11.4.16 Profile of Mood States (POMS) global score (*)

Profile of Mood States (POMS) global scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(1,63)=.38, p=.68,η^2=.012

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,63)=.42, p=.66, η^2=.013

nor a significant post-intervention group difference, F(2,63)=.80, p=.46, η^2=.025

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.91, F(1,63)=6.45, p=.014, η^2=.028

There was a significant main effect for time, Λ =.88, F(2,63)=8.16, p=.006, η^2=.11

There was no significant group main effect, F(2,63)=.60, p=.55, η^2=.019

Table App.11.4.16

POMS global scores - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	10.05(17.27)	5.90 (12.46)	1.40	1,63	.24	.022
high comp.	13.50 (20.67)	5.50 (14.65)	6.45	1,63	.014	.093
control	24.89 (24.45)	11.21 (20.85)	1.80	1,68	.18	.028

* Homogeneity assumption not satisfied for this variable. No suitable Power transformation found

App.11.4.17 Profile of Mood States (POMS) depression sub - score (*)

Profile of Mood States (POMS) depression sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,67)=.18, p=.83, η^2=.005

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,67)=.50, p=.61, η^2=.015

nor a significant post-intervention group difference, F(2,67)=1.63, p=.20, η^2=.046

No significant pre to post intervention change was seen with any group .

There was a significant main effect for time, Λ =.94, F(1,67)=4.35, p=.041,η^2=.061

There was no significant group main effect, F(2,67)=1.08, p=.35, η^2=.031

Table App.11.4.17

POMS depression sub scores -2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	3.18 (4.33)	2.18 (3.98)	1.60	1,67	.21	.023
high comp.	3.70 (4.00)	2.48 (2.56)	2.92	1,67	.09	.042
control	4.67 (6.37)	4.10 (4.71)	.50	1,67	.48	.007

* Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable. Results as follows

App.11.4.18 Profile of Mood States (POMS) depression sub score (-2.0) power transformation

(-2.0) power transformation of (POMS depression sub score +100) was performed. Homogeneity obtained on pre and post measures. Transformed scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,67)=.31, p=.73, η^2=.009

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,67)=.47, p=.63, η^2=.014

nor a significant post-intervention group difference, F(2,63)=1.72, p=.19, η^2=.049

There was a significant main effect for time, Λ =.94, F(1,67)=4.40, p=.040, η^2=.062

There was no significant group main effect, F(2,67)=1.10, p=.34, η^2=.032

Further, no significant pre to post intervention change was seen with any group:

Table App.11.4.18

Transformed POMS depression sub scores -2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
low comp.	.97	1.96	1,67	.17	.028
high comp.	.96	3.12	1,67	.082	.044
control	1.00	.29	1,67	.59	.004

App.11.4.19 Profile of Mood States (POMS) tension sub - score

Profile of Mood States (POMS) tension sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(2,68)=.84, p=.43, η^2=.024

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,68)=.57, p=.57, η^2=.017

nor a significant post-intervention group difference, F(2,68)=.58, p=.56, η^2=.017

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.94, F(1,68)=4.21, p=.04, η^2=.09

There was no significant main effect for time, Λ =.96, F(1,68)=.62, p=.96,η^2=.040

Table App.11.4.19

POMS tension sub scores - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	4.52 (4.73)	4.35 (3.20)	.034	1,68	.85	.001
high comp.	5.96 (4.97)	4.15 (4.81)	4.21	1,68	.044	.058
control	6.14 (7.03)	5.41 (4.50)	.58	1,68	.45	.008

App.11.4.20 Profile of Mood States (POMS) anger sub - score

Profile of Mood States (POMS) anger sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.92, F(2,66)=3.03, p=.05, η^2=.084

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,66)=.86, p=.43, η^2=.025

nor a significant post-intervention group difference, F(2,66)= 2.24, p=.11, η^2=.064

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.89, F(1,66)=8.28, p=.005, η^2=.11

There was no significant main effect for time, Λ =.97, F(1,66)=2.03, p=.16,η^2=.03

nor was there a significant group main effect, F(2,66)=1.06, p=.35, η^2=.031

Table App.11.4.20

POMS anger sub scores - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	3.48 (3.62)	3.17 (2.62)	.22	1,66	.64	.003
high comp.	5.08 (4.59)	3.31 (3.35)	8.28	1,66	.005	.11
control	4.70 (4.93)	5.15 (4.16)	.41	1,66	.52	.006

App.11.4.21 Profile of Mood States (POMS) fatigue sub - score

Profile of Mood States (POMS) fatigue sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(2,71)=.39, p=.28, η^2=.035

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 71)=.39, p=..68, η^2=.011

nor a significant post-intervention group difference, F(2, 71)= 1.46, p=.24, η^2=.040

Only the intervention groups showed improvement pre to post intervention but not significantly. Low compliance: Λ =.95, F(1, 71)=3.51, p=.065,η^2=.047, high compliance: Λ =.95, F(1, 71)=3.47, p=.067,η^2=.047

There was a significant main effect for time, Λ =.95, F(1, 71)=3.89, p=.02,η^2=.052

There was no significant group main effect, F(2, 71)=.63, p=.54, η^2=.017

Table App.11.4.21

POMS – fatigue sub scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	4.60 (3.19)	3.40 (2.36)	3.51	1,71	.065	.047
high comp.	5.30 (2.96)	4.15 (2.84)	3.47	1,71	.067	.047
control	4.59 (3.71)	4.73 (2.80)	.04	1,71	.84	.001

App.11.4.22 Profile of Mood States (POMS) confusion sub - score

Profile of Mood States (POMS) confusion sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,69)=1.76, p=.18, η^2=.049

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 69)=.29, p=.75, η^2=.008

nor a significant post-intervention group difference, F(2, 69)=.66, p=.52, η^2=.019

Only the high compliance group improved but not significantly, Λ =.95, F(1,69)=3.50, p=.066, η^2=.05

There was a significant main effect for time, Λ =.99, F(1, 69)=.62, p=.43,η^2=.009

There was no significant group main effect, F(2, 69)=.005, p=.99, η^2<.001

Table App.11.4.22

POMS – confusion sub scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	3.29 (3.33)	3.04 (2.20)	.19	1,69	.67	.003
high comp.	3.65 (3.20)	2.62 (2.73)	3.50	1,69	.066	.048
control	2.95 (3.00)	3.45 (2.63)	.69	1,69	.41	.010

App.11.4.23 Profile of Mood States (POMS) vigour sub - score

Profile of Mood States (POMS) vigour sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.97, F(2,68)=1.10, p=.34, η^2=.031

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 68)= 2.58, p=.083, η^2=.070

nor a significant post-intervention group difference, F(2, 68)=.65, p=.53, η^2=.019

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =.99, F(1, 68)=.97, p=.33, η^2=.014

There was no significant group main effect, F(2, 68)=1.78, p=.18, η^2=.050

Table App.11.4.23

POMS – vigour sub scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	8.04 (5.19)	8.96 (5.85)	.44	1,68	.51	.006
high comp.	11.69 (8.45)	10.96 (4.40)	.30	1,68	.58	.004
control	8.33 (3.84)	10.52(8.86)	2.20	1,68	.14	.031

App.11.4.24 SF36 (Health Survey) global score

SF36 (Health Survey) global scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,60)= 1.40, p=.25, η^2=.045

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 60)=2.88, p=.064, η^2=.088

but a significant post-intervention group difference, F(2, 60)=3.20, p=.048, η^2=.096

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.92, F(1,60)=5.23, p=.03, η^2=.08

There was a significant main effect for time, Λ =.94, F(1, 60)=4.08, p=.05, η^2=.064

There was a significant group main effect, F(2, 60)=3.34, p=.042, η^2=.10

Table App.11.4.24

SF36 – global scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	61.68 (9.67)	64.81 (11.88)	2.33	1,60	.13	.04
high comp.	69.65 (12.39)	73.73 (12.19)	5.23	1,60	.030	.08
control	68.16 (11.25)	67.82 (11.69)	.03	1,60	.87	<.001

App.11.4.25 SF36 (Health Survey) Physical functioning sub - score (*)

SF36 (Health Survey) Physical functioning sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(2,76)=.71, p=.50, η^2=.018

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 76)=1.26, p=.29, η^2=.032

nor a significant post-intervention group difference, F(2, 76)=.23, p=.79, η^2=.006

No significant pre to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =1.00, F(1, 76)=.74, p=.79,η^2=.001

There was no significant group main effect, F(2, 76)=.70, p=.50, η^2=.018

Table App.11.4.25

SF36 - Physical functioning sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	75.31 (19.71)	76.82 (21.52)	.37	1,76	.55	.005
high comp.	74.83 (19.40)	76.50 (19.79)	.54	1,76	.47	.007
control	81.81 (11.22)	79.79 (14.41)	.63	1,76	.43	.008

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.4.26 SF36 (Health Survey) Role limitations due to physical health sub - score

SF36 (Health Survey) Role limitations due to physical health sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.94, F(2,70)=2.3, p=.11, η^2=.062

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 70)=.88, p=.42, η^2=.025

nor a significant post-intervention group difference, F(2, 70)=.37, p=.69, η^2=.010

No significant pre to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =.98, F(1, 70)=1.16, p=.28,η^2=.016

There was no significant group main effect, F(2, 70)=.25, p=.78, η^2=.007

Table App.11.4.26

SF36 – Physical Role limitations sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	50.00 (38.73)	61.11 (38.04)	2.45	1,70	.12	.034
high comp.	57.47 (37.75)	66.38 (34.25)	2.18	1,70	.14	.030
control	65.22 (37.49)	57.61 (39.48)	1.26	1,70	.26	.08

App.11.4.27 SF36 (Health Survey) body pain sub - score

SF36 (Health Survey) body pain sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(2,75)=.036, p=.96, η^2=.001

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 75)=.85, p=.43, η^2=.022

nor a significant post-intervention group difference, F(2, 75)=.63, p=.54, η^2=.016

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =1.00, F(1, 75)=.023, p=.88, η^2<.001

There was no significant group main effect, F(2, 75)=.94, p=.39, η^2=.024

Table App.11.4.27

SF36 – body pain sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	62.88 (25.23)	63.44 (21.76)	.017	1,75	.90	<.001
high comp.	70.90 (21.63)	70.03 (22.21)	.048	1,75	.83	.001
control	65.78 (22.77)	64.96 (25.23)	.033	1,75	.86	<.001

App.11.4.28 SF36 (Health Survey) general health sub - score

SF36 (Health Survey) general health sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(2,70)=.14, p=.87, η^2=.004

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 70)=1.62, p=.20, η^2=.044

nor a significant post-intervention group difference, F(2, 70)=2.39, p=.099, η^2=.064

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =1.0, F(1,70)=.24, p=.62, η^2=.003

There was no significant group main effect, F(2, 70)=2.25, p=.11, η^2=.060

Table App.11.4.28

SF36 – general health sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	56.94 (17.11)	57.58 (17.17)	.072	1,70	.79	.001
high comp.	65.39 (15.86)	67.02 (17.24)	.53	1,70	.47	.007
control	59.99 (19.45)	59.80 (14.05)	.005	1,70	.94	<.001

App.11.4.29 SF36 (Health Survey) vitality/energy sub - score

SF36 (Health Survey) vitality/energy sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(2,69)=.14, p=.87, η^2=.004

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 69)=1.29, p=.28, η^2=.036

nor a significant post-intervention group difference, F(2, 69)=2.09, p=.13, η^2=.057

No significant pre to post intervention change was seen with any of the groups.

There was a significant main effect for time, Λ =.94, F(1, 69)=4.25, p=.043, η^2=.058

There was no significant group main effect, F(2, 69)=2.11, p=.13, η^2=.058

Table App.11.4.29

SF36 – vitality/energy sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	55.21 (14.78)	58.40 (15.51)	.96	1, 69	.33	.014
high comp.	62.86 (18.89)	68.15 (18.73)	3.08	1, 69	.083	.043
control	58.25 (17.50)	61.50 (18.36)	.83	1, 69	.37	.012

App.11.4.30 SF36 (Health Survey) social functioning sub – score (*)

SF36 (Health Survey) social functioning sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.85, F(2,76)= 6.68, p=.002, η^2=.15

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2,76)=.31, p=.73, η^2=.008

but a significant post-intervention group difference, F(2, 76)=4.64, p=.013, η^2=.11

Further, the only significant pre to post intervention change was seen in the high compliance group which improved significantly, Λ =.86, F(1, 76)=12.09, p=.001, η^2=.137

There was no significant main effect for time, Λ =.99, F(1, 76)=.51, p=.48,η^2=.007

nor was there a significant group main effect, F(2, 76)=1.23, p=.30, η^2=.031

Table App.11.4.30

SF36 – social functioning sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	77.40 (19.69)	75.96 (26.20)	.13	1,76	.72	.002
high comp.	77.50 (20.86)	90.42 (13.80)	12.09	1,76	.001	.13
control	81.52 (21.93)	75.00 (22.61)	2.36	1,76	.13	.03

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.4.31 SF36 (Health Survey) Role limitations due to emotional function sub – score (*)

SF36 (Health Survey) Role limitations due to emotional function sub – scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

A significant time by group interaction was found, Λ =.88, F(2,67)= 4.42, p=.016, η^2=.12

Follow up analysis of simple main effects for this interaction revealed a significant difference among the groups at pre-intervention, F(2, 67)=6.12, p=.04, η^2=.15

but no significant post-intervention group difference, F(2, 67)=1.33, p=.27, η^2=.04

Further, the only significant pre to post intervention change was seen in the control group which deteriorated significantly, Λ =.94, F(1, 67)=4.57, p=.04, η^2=.06

There was no significant main effect for time, Λ =1.0, F(1, 67)=.22, p=.64, η^2=.003

There was a significant group main effect, F(2, 67)=3.43, p=.04, η^2=.09

Table App.11.4.31

SF36 – emotional role limitations sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	47.37 (38.99)	63.16 (41.41)	2.92	1,67	.09	.04
high comp.	70.11 (39.18)	79.31(27.33)	1.54	1,67	.22	.02
control	86.36 (26.54)	68.18 (39.14)	4.57	1,67	.035	.06

* Homogeneity assumption not satisfied for this variable. No suitable transformation found.

App.11.4.32 SF36 (Health Survey) emotional well being/mental health sub - score

SF36 (Health Survey) emotional well being/mental health sub - scores were analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,69)= .46, p=.64, η^2=.013

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 69)=1.61, p=.21, η^2=.045

but a significant post-intervention group difference, F(2, 69)=3.38, p=.04, η^2=.089

No significant pre to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =.99, F(1, 69)=.65, p=.42, η^2=.009

There was a significant group main effect, F(2, 69)=3.45, p=.037, η^2=.091

Table App.11.4.32

SF36 – emotional well being sub - scores 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	67.17 (14.67)	66.12 (16.41)	.08	1,69	.78	.001
high comp.	74.14 (16.49)	77.82 (17.50)	1.12	1,69	.29	.016
Control	67.00 (17.60)	69.65 (15.75)	.42	1,69	.52	.006

App.11.5 Objective measures, 'On treatment' (OT) subset (low compliance versus high compliance versus control) analysis of variance (ANOVA)

App.11.5.1 Total time in bed (TTB)

'Total time in bed' was analysed using a 2 x 3 mixed analysis of variance (ANOVA).

The between-subjects factor was group (low compliance, high compliance, control).

The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.94, F(2,68)= 1.98, p=.14, η^2=.055

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 68)=.79, p=.46, η^2=.023

nor a significant post-intervention group difference, F(2, 68)=1.28, p=.28, η^2=.036

Further, the only significant pre - to post intervention change was seen in the low compliance group which decreased significantly, Λ =.94, F(1,68)=4.31, p=.04, η^2=.06

There was no significant main effect for time, Λ =1.00, F(1, 68)=.20, p=.65, η^2=.005

There was no significant group main effect, F(2, 68)=.44, p=.64, η^2=.013

Table App.11.5.1

Total time in bed - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
YLC	431.70 (71.09)	393.30 (77.36)	3.27	1,68	.045	.046
YHC	419.96 (70.68)	428.86 (76.93)	.25	1,68	.62	.004
Control	402.3 (91.43)	416.30 (73.98)	.51	1,68	.47	.008

App.11.5.2 Total sleep time (TST)

Total sleep time was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(2,67)= 1.34, p=.27, η^2=.038

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.66, p=.52, η^2=.019

nor a significant post-intervention group difference, F(2, 67)=1.50, p=.23, η^2=.043

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.96, F(1, 67)=.18, p=.67, η^2=.003

There was no significant group main effect, F(2, 67)=1.53, p=.22, η^2=.044

Table App.11.5.2

Total sleep time - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
YLC	375.70 (68.72)	347.40 (72.75)	2.30	1,67	.13	.033
YHC	375.04 (48.46)	382.81 (73.59)	.23	1,67	.63	.003
Control	355.91 (80.92)	363.78 (62.06)	.20	1,67	.65	.003

App.11.5.3 Sleep onset latency (SOL) [*]

Sleep latency was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(2,68)=.54, p=.58, η^2=.016

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 68)=.51, p=.60, η^2=.015

nor a significant post-intervention group difference, F(2, 68)=1.12, p=.33, η^2=.032

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.98, F(1, 68)=1.35, p=.25, η^2=.020

There was no significant group main effect, F(2, 68)=.19, p=.31, η^2=.034

Table App.11.5.3

Sleep latency - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	7.10 (9.78)	12.80 (16.70)	1.46	1,68	.23	.02
high comp.	11.14 (17.77)	14.98 (19.33)	.93	1,68	.34	.01
Control	9.11 (10.36)	8.39 (8.33)	.027	1,68	.87	<.001

* Normality assumption not satisfied for this variable. No suitable transformation found.

App.11.5.4 Total wake after sleep onset duration (WASO)

Total wake time duration was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.94, F(2,65)=2.24, p=.11, η^2=.064

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 65)=1.87, p=.16, η^2=.054

nor a significant post-intervention group difference, F(2, 65)=.29, p=.75, η^2=.009

Further, no significant pre - to post intervention change was seen with any of the groups, although both intervention groups improved and the control deteriorated but not significantly.

There was no significant main effect for time, Λ =.99, F(1, 65)=.56, p=.46, η^2=.009

There was no significant group main effect, F(2, 65)=.51, p=.61, η^2=.015

Table App.11.5.4

WASO - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, and control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	63.37 (23.91)	53.63 (16.03)	2.60	1,65	.092	.043
high comp.	57.30 (20.86)	53.81 (15.68)	.69	1,65	.47	.008
Control	50.73 (18.14)	57.14 (19.79)	1.46	1,65	.23	.022

App.11.5.5 Sleep Efficiency (SE)

Sleep efficiency was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(2,65)=1.39, p=.26, η^2=.041

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 65)=1.71, p=.19, η^2=.025

nor a significant post-intervention group difference, F(2, 65)=.040, p=.67, η^2=.012

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =1.0, F(1, 65)=.22, p=.64, η^2=.003

There was no significant group main effect, F(2, 65)=.97, p=.37, η^2=.029

Table App.11.5.5

Sleep efficiency - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, and control)

group	M (SD) - pre	M (SD) - post	F	df	p	η2
low comp.	.85 (.05)	87 (.04)	1.69	1,65	.20	.025
high comp.	.87 (.04)	.87 (.05)	.25	1,65	.62	.004
Control	.87 (.04)	.87 (.03)	1.04	1,65	.31	.016

App.11.5.6 Total light sleep duration

Total Light Sleep duration was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(2,67)=.93, p=.49, η^2=.021

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.90, p=.41, η^2=.026

nor a significant post-intervention group difference, F(2, 67)=.47, p=.62, η^2=.014

Further, no significant pre - to post intervention change was seen with any of the groups.

There was no significant main effect for time, Λ =1.00, F(1, 67)=.26, p=.61, η^2=.004

There was no significant group main effect, F(2, 67)=.73, p=.49, η^2=.021

Table App.11.5.6

Total light sleep duration - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
YLC	189.70 (41.57)	176.45 (41.44)	.93	1,67	.39	.014
YHC	195.44 (48.03)	189.30 (45.43)	.32	1,67	.57	.005
Control	176.83 (55.30)	184.69 (47.48)	.44	1,67	.51	.007

App.11.5.7 Total Slow Wave stage (SWS) duration

Total Slow Wave Sleep (deep sleep) duration was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.95, F(2,67)=1.86, p=.16, η^2=.053

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.002, p=1.00, η^2<.001

but a significant post-intervention group difference, F(2, 67)=3.17, p=.048, η^2=.13

Further, a significant pre - to post intervention change was seen in the high compliance group which improved significantly, Λ =.94, F(2,67)=4.30, p=.042, η^2=.060

There was no significant main effect for time, Λ =.99, F(1, 67)=.58, p=.49, η^2=.009

There was no significant group main effect, F(2, 67)=1.38, p=.26, η^2=.040

Table App.11.5.7

Total SWS duration - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
YLC	106.25 (24.59)	101.25 (23.64)	.80	1,67	.52	.006
YHC	106.67 (30.75)	120.56 (33.45)	4.30	1,67	.042	.060
Control	106.74 (24.80)	107.44 (20.70)	.009	1,67	.92	<.001

App.11.5.8 Slow Wave stage (SWS) latency [*]

Slow Wave stage (SWS) latency was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,67)=.38, p=.68, η^2=.011

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.22, p=.80, η^2=.007

nor a significant post-intervention group difference, F(2, 67)=1.75, p=.18, η^2=.050

Further, no significant pre - to post intervention change was seen with any of the groups

There was no significant main effect for time, Λ =1.00, F(1, 67)=.12, p=.72, η^2=.002

There was no significant group main effect, F(2, 67)=1.17, p=.32, η^2=.034

Table App.11.5.8

Total SWS latency - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	24.05(46.89)	31.27 (39.12)	.52	1,67	.43	.009
high comp.	18.61 (21.92)	20.11 (21.56)	.049	1,67	.83	.001
Control	18.61 (20.40)	15.39 (16.27)	.19	1,67	.66	.003

* Homogeneity assumption not satisfied for this variable. Homogeneity corrected using power transformation and ANOVA performed on transformed variable. Results as follows:

App.11.5.9 Slow Wave stage (SWS) latency (-1.00 power transformation)

Slow Wave stage (SWS) latency was transformed by using (-1) power transformation on (SWS latency +1) and then analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,67)=.32, p=.72, η^2=.010

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.074, p=.93, η^2=.002

nor a significant post-intervention group difference, F(2, 67)=.96, p=.39, η^2=.028

There was no significant main effect for time, Λ =.96, F(1, 67)= 2.73, p=.10, η^2=.039

There was no significant group main effect, F(2, 67)=.52, p=.60, η^2=.015

Further, no significant pre - to post intervention change was seen with any of the groups:

Table App.11.5.9

Transformed SWS latency - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
low comp.	.97	2.31	1,67	.13	.033
high comp.	.99	.37	1,67	.54	.006
Control	.99	.45	1,67	.50	.007

App.11.5.10 Total Rapid Eye Movement stage (REM) duration

Total Rapid Eye Movement stage (dream stage) duration was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,67)=.37, p=.69, η^2=.011

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.10, p=.87, η^2=.004

nor a significant post-intervention group difference, F(2, 67)=.10, p=.89, η^2=.003

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =.99, F(1, 67)=.81, p=.37, η^2=.012

There was no significant group main effect, F(2, 67)=.14, p=.97, η^2<.001

Table App.11.5.10

Total REM duration- 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	73.40 (31.35)	64.75 (34.66)	2.31	1,67	.13	.03
high comp.	69.29 (21.17)	68.18 (35.49)	.008	1,67	.93	<.001
Control	70.43 (28.12)	69.39 (28.53)	.023	1,67	.88	<.001

App.11.5.11 Rapid Eye Movement stage (REM) latency

Rapid Eye Movement stage (dream stage) latency was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.96, F(2,66)=1.48, p=.23, η^2=.043

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 66)=.34, p=.71, η^2=.010

nor a significant post-intervention group difference, F(2, 66)=1.67, p=.20, η^2=.048

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =1.00, F(1, 66)=.044, p=.83, η^2=001

There was no significant group main effect, F(2, 66)=.19, p=.83, η^2=.006

Table App.11.5.11

REM latency- 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	99.32 (44.89)	88.39 (42.47)	.90	1,66	.35	.019
high comp.	108.33 (50.22)	107.68 (46.90)	.004	1,66	.95	<.001
Control	98.30 (44.25)	113.70 (48.49)	2.16	1,66	.15	.032

App.11.5.12 Mean blood oxygen saturation (SPO2) during night

Mean blood oxygen saturation level was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.99, F(2,68)= .42, p=.65, η^2=.012

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 68)=.26, p=.77, η^2=.008 nor a significant post-intervention group difference, F(2, 68)=.10, p=.90, η^2=.003

Further, no significant pre - to post intervention change was seen with any group

There was no significant main effect for time, Λ =1.00, F(1, 68)=.21, p=.65, η^2=.003

There was no significant group main effect, F(2, 68)=.054, p=.95, η^2=.002

Table App.11.5.12

Mean blood oxygen saturation level- 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	96.55(5.07)	96.95 (2.52)	.12	1,68	.73	.002
high comp.	97.53 (4.69)	96.54 (3.25)	1.02	1,68	.31	.015
Control	97.13 (4.20)	96.87 (4.21)	.057	1,68	.81	.001

App.11.5.13 Mean Respiratory Disturbance Index (RDI) [*]

Respiratory Disturbance Index (RDI) was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.97, F(2,67)= .93, p=.40, η^2=.027

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=1.36, p=.26, η^2=.039

but a significant post-intervention group difference, F(2, 67)=2.12, p=.13, η^2=.060

Further, no significant change was seen with any of the groups pre to post intervention

There was no significant main effect for time, Λ =1.00, F(1, 67)=.044, p=.83, η^2=.001

There was no significant group main effect, F(2, 67)=1.88, p=.16, η^2=.053

Table App.11.5.13

RDI - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
low comp.	15.53 (11.32)	17.22 (14.97)	1.41	1,67	.24	.021
high comp.	14.47 (10.93)	13.92 (11.33)	.20	1,67	.66	.003
Control	10.90 (6.45)	10.24 (5.55)	.25	1,67	.62	.004

* Homogeneity assumption not satisfied for this variable. Homogeneity corrected using suitable power transformation (-1.939). ANOVA performed on transformed variable. Results as follows:

App.11.5.14 Mean Respiratory Disturbance Index (RDI) (-1.939 Power Transformation)

Transformed Respiratory Disturbance Index (RDI) was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =.98, F(2,67)= .70, p=.50, η^2=.021

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 67)=.46, p=.63, η^2=.013

nor a significant post-intervention group difference, F(2, 67)=.52, p=.59, η^2=.015

There was no significant main effect for time, Λ =.99, F(1, 67)=.91, p=.34, η^2=.013

There was no significant group main effect, F(2, 67)=.41, p=.66, η^2=.012

Further, no significant change was seen with any of the groups pre to post intervention:

Table App.11.5.14

Transformed mean RDI - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
low comp.	1.00	.29	1,67	.62	.004
high comp.	.97	2.20	1,67	.14	.032
Control	1.00	.06	1,67	.81	.001

App.11.5.15 Mean Oxygen Desaturation Index (ODI)

Apnea Duration Index (ODI) was analysed using a 2 x 3 mixed analysis of variance (ANOVA). The between-subjects factor was group (low compliance, high compliance, control). The within-subjects factor was time/phase (pre versus post-intervention).

No significant time by group interaction was found, Λ =1.00, F(2,68)= 0.80, p=.92, η^2=.002

Follow up analysis of simple main effects for this interaction revealed no significant difference among the groups at pre-intervention, F(2, 68)=1.74, p=.18, η^2=.049

and no significant post-intervention group difference, F(2, 68)=1.66, p=.20, η^2=.047

Further, no significant change was seen with any of the groups pre to post intervention

There was no significant main effect for time, Λ =1.00, F(1, 68)=.075, p=.78, η^2.001

There was no significant group main effect, F(2, 68)=1.86, p=.16, η^2=.052

Table App.11.5.15

Mean ODI - 2 x 3 mixed ANOVA - Pre to Post intervention change by group (low compliance, high compliance, control)

group	M (SD) - pre	M (SD) - post	F	df	p	η^2
YLC	13.17 (12.05)	13.28 (12.84)	.91	1,68	.93	<.001
YHC	10.03 (9.15)	10.57 (10.22)	1.80	1,68	.62	.004
Control	7.82 (6.70)	7.74 (5.94)	<.001	1,68	.94	<.001

App.11.6 Objective measures, 'On treatment' (OT) subset (low compliance versus high compliance versus control) Multivariate Analysis of Variance (MANOVA)

App.11.6.1 Insomnia related objective multivariate analysis of variance (MANOVA)

The data for the insomnia multivariate was analysed using a 2 x 3 mixed multivariate analysis of variance (MANOVA). The three-level between-subjects factor was group (high compliance, low compliance and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of four measures related to insomnia namely: sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST) and sleep efficiency (SE)

Multivariate tests revealed no significant main effect for phase, $\Lambda = .91$, $F(4, 62) = 1.53$, $p = .20$, $\eta^2 = .090$, no significant multivariate main effect for group, $\Lambda = .85$, $F(8, 124) = 1.28$, $p = .26$, $\eta^2 = .076$, and no significant multivariate interaction, $\Lambda = .88$, $F(8, 124) = .98$, $p = .45$, $\eta^2 = .06$.

When the sub-scales for the multivariate main effect for phase were considered separately, there was no significant univariate phase main effect, no significant univariate group main effect, and no significant univariate interaction.

Examination of the marginal means showed that, for WASO and TST, participants across all groups improved, for SOL deteriorated and for sleep efficiency remained without change from baseline to post-intervention.

Analysis of the multivariate simple main effects for time/phase within group revealed no significant pre - to post-intervention change for any of the groups:

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Table App.11.6.1

Insomnia related objective 2 x 3 mixed - multivariate analysis of variance (MANOVA) Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
low comp.	.92	1.29	4,62	.28	.077
high comp.	.89	1.99	4,62	.11	.11
Control	.97	.39	4,62	.81	.025

App.11.6.2 Obstructive Sleep Apnea (OSA) - multivariate analysis of variance (MANOVA)

The data for the OSA was analysed using a 2 x 3 mixed multivariate analysis of variance (MANOVA). The three-level between-subjects factor was group (high compliance, low compliance and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of three measures related to OSA, including, RDI (transformed), ODI and SPO2.

Multivariate tests revealed no significant main effect for phase, $\Lambda = .97$, $F(3, 65) = .62$, $p = .60$, $\eta^2 = .028$, no significant multivariate main effect for group, $\Lambda = .93$, $F(6, 130) = .85$, $p = .53$, $\eta^2 = .038$, and no significant multivariate interaction $\Lambda = .93$, $F(6, 130) = .61$, $p = .22$, $\eta^2 = .034$.

When the sub-scales for the multivariate main effect for phase were considered separately, there was no significant univariate phase main effect, no significant univariate group main effect, and no significant univariate interaction.

Examination of the marginal means showed a slight deterioration of SPO2 measure and improvement in ODI and small improvement in RDI measures - across all groups from

baseline to post-intervention.

Analysis of the multivariate simple main effects for time/phase within group revealed a significant pre- to post-intervention change for the high compliance group but not for the other groups.

Table App.11.6.2

OSA related objective 2 x 3 mixed - multivariate analysis of variance (MANOVA) Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
YLC.	1.00	.094	3,65	.96	.004
YHC.	.90	2.16	3,65	.10	.091
Control	1. 00	.078	3,65	.97	.004

App.11.6.3 Slow Wave Sleep (SWS) - multivariate analysis of variance (MANOVA)

The data for the SWS was analysed using a 2 x 3 mixed multivariate analysis of variance (MANOVA). The three-level between-subjects factor was group (high compliance, low compliance and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of two measures related to SWS, namely, SWS duration and SWS latency (transformed).

Multivariate tests revealed no significant multivariate main effect for phase, $\Lambda = .95$, $F(2,65)= 1.65$, $p = .20$, $\eta^2 = .048$, no significant multivariate main effect for group, $\Lambda = .92$, $F(4, 130) = 1.36$, $p = . 25$, $\eta^2 = .040$, and no significant multivariate interaction, $\Lambda = .89$, $F(4, 130) = 1.92$, $p = .11$, $\eta^2 = .056$.

When the sub-scales for the multivariate main effect for phase were considered separately, there was neither a significant univariate phase main effect, nor a significant

univariate group main effect. Only the SWS duration univariate phase by group interaction test generated a significant outcome, $F(2, 66) = 3.3$, $p = .043$, $\eta^2 = .091$. Examination of the marginal means showed that, for SWS duration, participants across all groups improved from baseline to post-intervention, but for SWS latency - remained unchanged from baseline to post-intervention

Analysis of the multivariate simple main effects for time/phase within group revealed a significant pre- to post-intervention change for the high compliance group only:

Table App.11.6.3
SWS related objective 2 x 3 mixed - multivariate analysis of variance (MANOVA) Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
low comp.	.96	1.50	2,65	.23	.044
high comp.	.89	4.05	2,65	.022	.11
Control	.99	.23	2,65	.80	.007

App.11.6.4 Rapid Eye Movement sleep multivariate analysis of variance (MANOVA)

The data for the REM was analysed using a 2 x 3 mixed multivariate analysis of variance (MANOVA). The three-level between-subjects factor was group (high compliance, low compliance and control) and the two-level within-subjects factor was phase (baseline versus post-intervention). The multivariate dependent variable was made up of two measures related to REM, namely, REM duration and REM latency (transformed). Multivariate tests revealed no significant multivariate main effect for phase, $\Lambda = 1.00$, $F(2,64)=.096$, $p = .91$, $\eta^2 = .003$, no significant multivariate main effect for group, $\Lambda = .97$, $F(4, 128) =.47$, $p = .75$, $\eta^2 = .015$, and no significant

multivariate interaction, $\Lambda = .95$, $F(4, 128) = .81$, $p = .52$, $\eta^2 = .025$. When the sub-scales for the multivariate main effect for phase were considered separately, there was neither a significant univariate phase main effect, nor a significant univariate group main effect, nor a significant univariate time by group interaction, Examination of the marginal means showed that, for both measures, participants across all groups deteriorated very slightly from baseline to post-intervention. Analysis of the multivariate simple main effects for time/phase within group did not reveal a significant pre- to post-intervention change for any of the group:

Table App.11.6.4

REM related objective 2 x 3 mixed - multivariate analysis of variance (MANOVA) Pre to Post intervention change by group (low compliance, high compliance, control)

group	Λ	F	df	p	η^2
low comp.	.98	.67	2,64	.51	.021
high comp.	1.00	.012	2,64	.99	<.001
Control	.97	1.05	2,65	.36	.032

App.11.7 Testing for relationships between variables

App.11.7.1 Testing for Relationship between OSA degree and compliance level

Pearson's Chi Square test revealed no statistically significant relationship between OSA degree, (none, mild, moderate, severe) and between compliance level, χ^2 (3, N = 58) = 1.94, p = .58

App.11.7.2 Testing for Relationship between OSA degree, and MAP derived apnea probability score

Pearson's Chi Square test revealed no statistically significant relationship between OSA degree, (none, mild, moderate, severe) and between pre intervention apnea probability calculated from pre intervention MAP, $\chi^2 2$ (192, N = 65) = 195.0, p = .43

Pearson's Chi Square test revealed no statistically significant relationship between OSA degree, (none, mild, moderate, severe) and between post intervention apnea probability calculated from post intervention MAP, $\chi^2 2$ (183, N = 62) = 186.0, p = .42

App.11.7.3 Testing for Relationship between compliance level and age

Pearson's Chi Square test revealed no statistically significant relationship between compliance level and age, $\chi^2 2$ (23, N = 59) = 24.46, p = .38

App.11.7.4 Testing for Relationship between OSA degree and change pre to post intervention in subjective measure scores

Pearson's correlation test was used to determine whether a relationship exists between OSA degree (none, mild, moderate, severe), diagnosed post study, and between change pre to post in subjective and objective measure scores in control and intervention groups. Results are given in the following four tables

Table App.11.7.4.1

Pearson's correlation test between OSA degree and pre to post change in subjective measures in the intervention group

Pre to Post change	r	p	N	Note
KSS score change	.15	.28	54	
ESS score change	.13	.35	56	
MAP score change	.16	.27	46	
MAP apnea probability score change	.22	.093	58	
PSQI global score change	.34	.023	45	#1
PSQI subjective sub score change	.037	.79	55	
PSQI latency sub score change	.16	.30	42	
PSQI duration sub score change	.31	.026	52	
PSQI efficiency sub score change	.52	.001	41	#1
PSQI disturbance sub score change	.006	.97	50	
PSQI medication sub score change	.089	.52	55	
PSQI dysfunction sub score change	.009	.95	50	
DASS global score change	.14	.31	52	
DASS stress sub score change	.10	.46	53	
DASS anxiety sub score change	.16	.23	56	
DASS depression sub score change	.13	.35	53	
POMS global score change	.012	.94	49	
POMS tension sub score change	.29	.043	49	#2
POMS depression sub score change	.24	.10	49	
POMS anger sub score change	.19	.19	49	

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Pre to Post change	r	p	N	Note
POMS fatigue sub score change	.24	.088	52	
POMS confusion sub score change	.23	.10	50	
POMS vigour sub score change	.16	.26	50	
SF36 physical function sub score change	-.009	.95	55	
SF36 role limitations (physical) change	-.38	.007	50	#1
SF36 body pain sub score change	.17	.22	55	
SF36 general health sub score change	-.21	.13	53	
SF36 vitality sub score change	-.026	.86	52	
SF36 social function sub score change	-.29	.03	56	#1
SF36 role limitation (emotional) change	-.46	.001	48	#1
SF36 mental health sub score change	-.19	.18	52	
SF36 global score change	-.33	.032	42	#1

Notes:

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

Table App.11.7.4.2

Pearson's correlation test between OSA degree and pre to post change in subjective measures in the control group

Pre to Post change	r	p	N	Note
KSS score change	.18	.44	20	
ESS score change	.06	.81	21	
MAP score change	-.01	.96	19	
MAP apnea probability score change	.43	.052	21	#2
PSQI global score change	-.25	.33	17	
PSQI subjective sub score change	-.28	.23	20	
PSQI latency sub score change	.05	.83	18	
PSQI duration sub score change	-.14	.59	18	
PSQI efficiency sub score change	-.50	.11	11	
PSQI disturbance sub score change	.25	.31	19	
PSQI medication sub score change	-.25	.27	21	
PSQI dysfunction sub score change	.28	.23	20	
DASS global score change	.07	.80	17	
DASS stress sub score change	-.05	.84	18	
DASS anxiety sub score change	.10	.69	18	
DASS depression sub score change	.12	.63	17	
POMS global score change	-.30	.22	18	
POMS tension sub score change	-.003	.99	18	
POMS depression sub score change	-.07	.79	17	
POMS anger sub score change	-.09	.75	16	

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Pre to Post change	r	p	N	Note
POMS fatigue sub score change	-.06	.80	18	
POMS confusion sub score change	.03	.91	18	
POMS vigour sub score change	-.22	.39	17	
SF36 physical function sub score change	.18	.45	19	
SF36 role limitations (physical) change	-.09	.70	19	
SF36 body pain sub score change	-.40	.09	19	
SF36 general health sub score change	-.13	.64	16	
SF36 vitality sub score change	.05	.85	16	
SF36 social function sub score change	-.03	.91	19	
SF36 role limitation (emotional) change	-.18	.46	18	
SF36 mental health sub score change	-.008	.98	16	
SF36 global score change	-.43	.11	15	

Notes:

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

Table App.11.7.4.3

Pearson's correlation test between OSA degree and pre to post change in objective measures in the intervention group

Pre to Post change	r	p	N	Note
Mean blood oxygen saturation level (SPO2)	-.22	.13	47	
Respiratory disturbance index (RDI)	-.22	.13	46	
Total de-saturations	.091	.54	47	
Sleep onset latency (SOL)	-.172	.25	47	
REM latency	.019	.90	46	
SWS latency	-.062	.68	47	
Total time in bed (TTB)	.092	.54	47	
Total sleep time (TST)	.093	.54	46	
Total wake time after sleep onset (WASO)	.17	.26	45	
Total light sleep duration	-.040	.79	46	
Total REM duration	.00	1.00	46	
Total SWS duration	.19	.19	46	
Sleep efficiency (SE)	-.14	.33	47	

Notes:

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

Table App.11.7.4.4

Pearson's correlation test between OSA degree and pre to post change in objective measures in the control group

Pre to Post change	r	p	N	Note
Mean blood oxygen saturation level	-.037	.87	21	
Respiratory disturbance index (RDI)	-.26	.26	21	
Total de-saturations	-.01	.96	21	
Sleep latency	.48	.028	21	#2
REM latency	.053	.82	20	
SWS latency	.028	.91	20	
Total time in bed	-.18	.44	21	
Total net sleep time	-.09	.70	21	
Total wake time after sleep onset	-.28	.21	21	
Total light sleep duration	.18	.42	21	
Total REM duration	-.33	.15	21	
Total SWS duration	-.31	.18	21	
Sleep efficiency (SE)	-.27	.23	21	

Notes:

#1 Significant correlation found and ANOVA test revealed significant pre to post change in this measure

#2 Significant correlation found however ANOVA test revealed no significant pre to post change in measure

N- number of cases, r-Pearson's correlation, p - Significance (2 tailed)

11.8 Analyses of WLC subset matched to themselves as YI subset

Table 11.8.1 QoL scores of WLC subset– matched to themselves as YI subset. Paired samples t test.

Measure	M (SD) - pre	M (SD) - post	t	df	p
DASS global score	32.37 (21.30)	15.47 (8.85)	3.95	14	.001
DASS stress	14.10 (8.93)	7.13 (4.82)	4.09	14	.001
DASS anxiety	8.87 (5.12)	7.56 (3.44)	2.66	15	.018
DASS depression	8.59 (6.82)	3.94 (3.11)	3.28	15	.005
POMS global score	27.07 (18.80)	13.50 (10.34)	2.18	13	.048
POMS tension	17.036 (21.79)	5.28 (10.22)	2.45	14	.028
POMS depression	5.32 (5.67)	2.21 (2.66)	2.075	13	.058
POMS anger	5.13 (3.86)	3.07 (2.58)	2.79	14	.014
POMS fatigue	4.90 (2.89)	2.80 (2.18)	3.41	14	.004
POMS confusion	3.40 (2.53)	2.07 (1.79)	2.55	14	.023
POMS vigour	8.96 (3.34)	9.79 (4.06)	-.75	13	.47
SF36 physical function	81.26 (15.11)	85.11 (16.77)	-1.73	14	.10
SF36 physical role	64.06 (36.76)	70.31(40.02)	-1.52	15	.15
SF36 physical pain	65.87 (22.20)	71.50 (20.67)	-1.51	15	.15
SF36 general health	63.22 (13.60)	67.03 (15.24)	-1.41	14	.18
SF36 vitality	61.41 (16.10)	67.19 (17.41)	-1.17	15	.26
SF36 social function	76.17 (20.57)	90.62 (14.79)	-3.79	15	.002
SF36 emotional role	72.92 (29.11)	85.42 (24.25)	-1.69	15	.11
SF36 mental health	66.53 (12.16)	69.25 (22.89)	-.54	15	.60
SF36 global score	68.69 (10.00)	73.37 (10.37)	-1.74	14	.10

Table 11.8.2

Subjective Sleep quality measures scores of WLC subset group – matched to themselves as YI subset group. Paired samples t test.

Measure	**M (SD) - pre**	**M (SD) - post**	**t**	**df**	**p**
KSS	3.44 (.96)	2.94 (1.00)	1.62	15	.13
ESS	7.78 (2.83)	7.31 (3.38)	.61	15	.55
PSQI Global score	9.80 (2.53)	8.07 (3.77)	2.31	14	.037
PSQI subjective sleep quality	1.53 (.34)	1.37 (.62)	1.00	15	.33
PSQI sleep latency	2.00 (.71)	1.36 (.93)	3.63	13	.003
PSQI sleep duration	2.17 (.70)	1.73 (.88)	2.69	14	.017
PSQI sleep efficiency	1.18 (1.12)	1.07 (1.33)	.27	13	.79
PSQI sleep disturbances	1.34 (.51)	1.19 (.40)	1.32	15	.21
PSQI medication	.78 (1.12)	.75 (1.24)	1.74	15	.86
PSQI daytime dysfunction	1.03 (.72)	.69 (.70)	1.70	15	.11
MAP – apnoea probability	.35 (.26)	.44 (.34)	-1.09	14	.29

Table 11.8.3

Objective sleep quality measures scores of WLC subset group – matched to themselves as YI subset group. Paired samples t test.

Measure	M (SD) - pre	M (SD) - post	t	df	p
Total time in bed (TTB)	421.36 (65.25)	449.14 (84.16)	-1.40	13	.18
Total sleep time (TST)	377.88 (51.00)	398.08 (89.67)	-.99	12	.34
SOL	7.59 (6.17)	12.86 (12.88)	-1.24	13	.24
WASO	53.86 (16.00)	54.14 (19.88)	-.067	12	.95
Sleep efficiency	.88 (.069)	.87 (.047)	.65	12	.53
light sleep duration	188.077 (40.24)	202.077 (47.74)	-.96	12	.36
SWS duration	113.23 (17.30)	126.85 (32.64)	-1.51	12	.16
SWS latency	17.54 (17.43)	20.07 (19.70)	-.30	13	.77
REM sleep duration	74.19 (23.99)	66.46 (39.26)	.65	12	.52
REM sleep latency	117.30 (29.22)	102.54 (54.48)	1.08	13	.30
blood oxygen saturation	93.29 (1.85)	93.11 (1.28)	.38	13	.71
RDI	9.47 (5.73)	9.99 (5.73)	-.49	12	.63

Appendix 12. Advertisements

App.12.1 Advertisement (English version)

A study of yoga for improving sleep in the elderly

Sponsored by Shaare Zedek Medical Center, Israel Yoga Teachers Organization, Australia Israel Scientific Exchange Foundation, RMIT University – School of Health Sciences

- Insomnia (disturbed sleep, non restorative sleep), is very common in the elderly population
- Insomnia may cause tiredness, health problems and reduced quality of life, impaired cognitive ability, accidents and falls.
- sedative-hypnotic drugs can have serious negative side effects
- Yoga includes movement, stretches, relaxation, and meditation
- Yoga has been shown to improve conditions such as hypertension, stress, anxiety, chronic pain etc. Some research suggests that yoga also helps with insomnia.
- Our research program was designed to improve sleep and life quality.
- For 3 months participants will train with a yoga teacher twice a week) Jerusalem metropolitan area) and use a special relaxation CD at home. All FREE!
- If you are **60+** and feel your sleep quality is lacking you are invited to join the study.

- **For details please contact:**

  Jonathan on 02-6666319 or email sleepyoga@gmail.com

App.12.2 Advertisement (Hebrew version)

תכנית מחקר יוגה / הרפיה לשיפור איכות שינה וחיים לבני 60 ומעלה – <u>ללא תשלום</u>!

בחסות המרכז הרפואי שערי צדק ירושלים, ארגון מורי היוגה בישראל, הקרן לחילופי RMITמדע אוסטרליה – ישראל ואוניברסיטת

'אינסומניה' (הפרעות שינה, שינה לא מספקת) היא בעיית בריאות שכיחה בקרב 'גיל הזהב'.

'אינסומניה' גורמת לירידה בכושר המחשבה והתפקוד, לעייפות, תאונות ונפילות.

הטיפול התרופתי – כרוך בתופעות לוואי, כולל טשטוש, המהווה גורם סיכון לתאונות ונפילות.

יוגה כוללת תנועה, מתיחות, שחרור, הרפיה, תרגילי נשימה, רגיעה ומדיטציה.

מחקרים הראו שלתרגול יוגה השפעות חיוביות על לחץ דם, מחלות לב, אסטמה, סכרת, מתח, חרדה, וכו'

. תכנית המחקר נבנתה לשיפור איכות חיים בכלל והשינה בפרט.

המשתתפים יתרגלו יוגה 'רכה' מותאמת לגיל הזהב פעמיים בשבוע במשך שלשה חדשים באזור בית הכרם החל מחודש יוני וכן ילמדו לתרגל רגיעה והרפיה בבית בעזרת תקליטור מיוחד. <u>ללא תשלום</u>.

<u>בני 60 ומעלה מוזמנים להצטרף.</u>

<u>לקבלת פרטים / להרשמה נא צרו קשר עם: יהונתן בטלפון 02-6666319</u>

App.12.3 small add (Hebrew version)

דרושים/ות מתנדבים/ות למחקר רפואי הכולל טיפול/אבחון ניסיוני[5]

במחלקה: מעבדת שינה ..... **במוסד רפואי:** מרכז רפואי שערי צדק .....

מתקיים מחקר רפואי בנושא:

תרגול בסגנון יוגה אינטגרטיבית לשיפור איכות השינה ואיכות החיים של האוכלוסיה המזדקנת

.....

המעוניינים/ות להשתתף מתבקשים/ות לפנות בטלפון: 02-6666319

.....

בשעות: 5 -9 ..... **בימים:** א -ה.....

[5] מחק את המיותר