

Psychological Factors Influencing Adherence to
Nasal Continuous Positive Airway Pressure in
Obstructive Sleep Apnoea Patients

Simon Roger Mamone

(3088364)

Bachelor of Science (Honours) (Psychology)

Master of Psychology (Organisational)

Submitted in fulfilment of the requirements for the degree of
Doctor of Philosophy

Department of Psychology

Victoria University

June 2014

Keywords

List of key words:

Adherence

Continuous Positive Airway Pressure

Health Belief

Health Locus of Control

Mood

Non-adherence

Obstructive Sleep Apnoea

Personality

Psychological predictors

Self-efficacy

Abstract

Obstructive sleep apnoea (OSA) is a chronic sleep-related breathing disorder that if left untreated leads to serious adverse health consequences, poor quality of life, and also impacts negatively on society. Continuous positive airway pressure (CPAP) is widely acknowledged as the best available treatment for moderate to severe OSA. CPAP treatment has been linked to reduced co-morbidities as well as improved quality of life. However, adherence to CPAP therapy is a major obstacle to effective long-term treatment. The aim of this study was to explore and identify predictors of CPAP adherence in a sample of patients with moderate to severe OSA. Specifically the study explored; 1) the combination of psychological factors—mood, personality self-efficacy, health locus of control, and health belief—that best predicted adherence and non-adherence to CPAP use; 2) the impact of adherent CPAP use on mood following the implementation phase; and 3) the impact of adherent CPAP use on sleep-related variables collected from polysomnography at the diagnostic phase. Traditionally, much of the research on OSA and treatment adherence has focussed on sleep-related variables that are likely to predict CPAP adherence. In contrast, the current study explored the predictive efficacy of psychological factors. A total of 156 sleep study patients were invited to participate in the present study with 69 adherent patients participating in both the diagnostic and implementation phase and 87 non-adherent patients only participating in the diagnostic phase. The sample comprised mainly of men (65%) diagnosed with moderate to severe OSA, with a mean age of 49 years, and a mean body mass index of 32. Predictor variables included mood, self-efficacy, personality, health locus of control, and health beliefs. Results from a discriminant function analysis revealed that anger/hostility, vigour/activity and depression/dejection on the mood measure and self-efficacy, internal health locus of control, and perceived susceptibility and perceived benefits on the health belief measure were significant predictors accounting for 59% of the variance of CPAP

adherence. Cross-validated classification showed that the overall predictive accuracy was 88%. The results also showed a positive and strong statistically significant reduction in the Apnoea-Hypopnoea Index as well as a positive and strong statistically significant increase in O₂ saturation at implementation of CPAP use that demonstrated that CPAP treatment continues to remain an effective treatment option for OSA sufferers. While more research is still needed to exploring the predictive value of a range of psychological factors in relation to CPAP non-adherence in moderate to severe OSA patients the present study provides initial useful information for predicting adherence and non-adherence. This information is likely to be vital to the development and design of intervention strategies based on the health belief model to increase adherence given the prevalence of OSA and non-adherence to CPAP treatment.

Doctor of Philosophy Declaration

“I, Simon Roger Mamone, declare that the PhD thesis titled ‘Psychological Factors Influencing Adherence to Nasal Continuous Positive Airway Pressure in Obstructive Sleep Apnoea Patients’ is no more than 100,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references, and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.”

Signature:

Date:

Acknowledgments

It was not without trials and tribulations that I submit this thesis and as such my profound gratitude goes to my PhD supervisor, Associate Professor Gerard Kennedy, who has mentored me in research, critical thinking, and scientific writing. He has had a profound influence on my education, not to mention my professional growth as a psychologist and as an individual. I am indebted to his invaluable insights, suggestions for improvements, motivation, and most of all friendship.

This research was supported by Victoria University (VU) and the respiratory departments at Southern Health and Austin Health and as such I am indebted to the people within these institutions for the opportunities they provided me. Specifically, I would like to extend a big thank you to the School of Social Science and Psychology at VU and in particular the university's staff and committee members within the Office for Postgraduate Research for the perseverance and patience. I would also like to extend a big thank you to the staff at the respiratory departments at Southern Health and Austin Health, in particular Dr Michael Ho, Dr Maree Barnes and most of all Lisa Vessey. Without their ongoing support for this project and attending to my countless needs, completion of this thesis may not have been possible.

I would also like to express my gratitude to peers and friends Paul Agius and Jason Ferris for their statistical guidance throughout this research project. These scholars have provided wonderful support. It is an honour to be friends with Paul and Jason and to have worked with them both. I am also indebted to Dr Kristin Argall, from Express Editing Writing and Research, who assisted me with editing and proofing according to the Institute for Professional Editors' Guidelines for Editing Research Theses.'

I am also grateful to my family members for standing by me throughout the completion of this thesis. I have benefitted from the never-ending support of my parents Jose and Elsa Mamone, and many thanks to my sister Joanne, who also supported me throughout the program and assisted me with follow-up phone calls to patients during the data collection phase. Also, I am thankful for the support and encouragement of my partner, Jane Goller; I could not have completed this thesis without you and our beautiful baby son Harris. You are both an inspiration to me, and I believe I would not have been successful without your support and assistance in data collection and proof reading this dissertation.

Finally, I appreciate the support and participation from the patients who gave up their time for me, in uncertain and troubling periods, to make this project viable. I believe that the completion of this study has contributed to the growing body of literature surrounding the issues faced by most OSA patients relating to adherence to CPAP use. Together we have achieved much and gained ground in this area – THANK YOU.

TABLE OF CONTENTS

Keywords	i
Abstract	ii
Doctor of Philosophy Declaration	iv
Acknowledgments.....	v
LIST OF FIGURES	xii
LIST OF TABLES	xiii
LIST OF ABBREVIATIONS.....	xv
LIST OF APPENDICES.....	xx
CHAPTER 1	1
1.1 Introduction.....	1
1.2 Sleep Apnoea	7
1.2.1 Definition	7
1.2.2 Types of sleep apnoea.....	8
1.3 Diagnosis of OSA	10
1.4 Clinical Features of OSA	13
1.4.1 Pathophysiology.....	14
1.4.2 Risk factors	16
1.4.2.1 Obesity.	16
1.4.2.2 Age.	17
1.4.2.3 Sex.	18
1.4.2.4 Anatomy.	19

1.4.2.5 Genetics.	21
1.4.2.6 Other risk factors.	21
1.4.3 Epidemiology and prevalence.....	23
1.5 Quality of Life.....	27
1.6 CPAP Treatment for OSA	28
1.6.1 Issues surrounding CPAP treatment for OSA.....	31
1.6.2 Determinants of CPAP adherence	34
1.7 Adherence	38
1.7.1 Terminology–compliance or adherence.....	38
1.7.2 Adherence research	40
1.7.3 CPAP adherence rates.....	43
1.8 Psychological Determinants of CPAP Adherence.....	46
1.8.1 Mood	47
1.8.2 Personality.....	64
1.8.3 Health Belief Model.....	69
1.8.4 Locus of Control	75
1.8.5 Self-Efficacy	78
1.8.6 Daytime sleepiness.....	82
1.9 Statement of Significance and Contribution to Knowledge	83
1.10 Aims	86
1.11 Exploratory Question	87
CHAPTER 2	88

2.1 METHOD	88
2.1.1 Patients	88
2.1.2 Materials	89
2.1.2.1 Explanatory statement.	89
2.1.2.2 Demographics questionnaire.	89
2.1.2.4 Eysenck Personality Questionnaire-Revised Short Form.	91
2.1.2.6 Multidimensional Health Locus of Control-Form C.	93
2.1.2.7 Epworth Sleepiness Scale.	95
2.1.3 Procedure	97
2.1.3.1 Diagnostic phase.	98
2.1.3.2 Implementation phase.	99
2.1.3.3 Data analysis phase.	99
2.1.4 Preparation	100
2.1.4.1 Polysomnograph procedure.	100
2.1.4.2 Monitoring parameters.	102
CHAPTER 3	108
3.1 Results.....	108
3.1.1 Treatment of Missing Values and Outliers	109
3.1.1.1 Missing values.	109
3.1.1.2 Outliers.	112
3.2 Alpha.....	113

3.3 Test of Assumptions	113
3.4 Reliability Analyses	115
3.5 Statistical Analysis.....	119
3.5.1 Patient characteristics.....	119
3.5.2 Examination of Sleep-Related Variables	122
3.5.3 Exploration of Psychological Factors	125
3.5.3.1 Psychological predictors of CPAP adherence.....	130
4.1 Discussion	135
4.2 Review of Exploratory Questions and Explanation of Findings	137
4.2.1 Characteristics of the sample	137
4.2.2 Exploratory Question One	140
4.2.2.1 Mood.....	141
4.2.3 Social Cognitive Factors	144
4.2.3.1 Health Locus of Control.....	146
4.2.3.2 Self-Efficacy.....	148
4.2.3.3 Health Belief.....	150
4.2.3.4 Personality.....	152
4.2.4 Exploratory Question Two.....	154
4.2.5 Exploratory Question Three.....	158
4.2.5.1 Sleepiness.....	161
4.3 Clinical Implications	165
4.3.1 Intervention	166

4.3.2 Intervention Options to Increase CPAP Adherence.....	167
4.3.2.1 Supportive interventions.	167
4.3.2.2 Educational interventions.	168
4.3.2.3 Cognitive behavioural interventions.	168
4.3.2.4 Mixed strategy interventions.	169
4.4 Limitations, Strengths and Future Direction.....	169
4.5 Conclusion	172
REFERENCES	175

LIST OF FIGURES

Figure 1	Dickens' "Fat Boy".....	2
Figure 2	An integrated map of the pathogenesis of the cyclical nature of OSA.....	15
Figure 3	Summary of risk factors grouped as modifiable and non-modifiable.....	16
Figure 4	Diagram of normal and apneic an upper airway.....	20
Figure 5	CPAP device.....	28
Figure 6	Patient connected to a polysomnography.....	104
Figure 7	Polysomnography results.....	106
Figure 8	Parameters monitored during a polysomnography.....	107
Figure 9	Educational level of the study participants.....	119
Figure 10	ROC curve differentiating clinically significant psychological predictors....	133
Figure 11	Profile of a CPAP-adherent patient compared to a non-adherent patient.....	141

LIST OF TABLES

Table 1	Landmark Sleep Studies of OSA During the 1970s and 1980s.....	4
Table 2	Medical Consequences of Sleep Apnoea.....	7
Table 3	Common Symptoms of Obstructive Sleep Apnoea.....	10
Table 4	AHI and Oxygen Saturation Values Representing Different Severity Levels.....	12
Table 5	Risk Factors for Obstructive Sleep Apnoea.....	22
Table 6	Large Sample Studies on the Prevalence of OSA.....	24
Table 7	Estimated Prevalence of Moderate to Severe OSA in Australia, 2010.....	26
Table 8	Reports of Minor Adverse Events from CPAP.....	30
Table 9	Intervention Studies to Improve CPAP Adherence.....	35
Table 10	Summary of the Findings from the Literature on Mood and Sleep Apnoea.....	50
Table 11	Treatment Options for Obstructive Sleep Apnoea.....	85
Table 12	Stages of Sleep.....	108
Table 13	Reliability Coefficients for the POMS, EPQ-RS, MHLC-C, GSE, and Health Belief Measure at the Diagnostic and Implementation Phases (n = 69).....	116
Table 14	Perceptions of OSA and CPAP Use at Diagnostic and Implementation Phases (n = 69).....	120
Table 15	Means, Standard Deviations and Percentage for the Sleep-Related Variables (Sleep Efficiency, Sleep Latency, O ₂ saturation, AHI, BMI, and Sleepiness) at the Diagnostic and Implementation Phases.....	123

Table 16	Means, Standard Deviations and Percentages Associated with Psychological Factors (Mood, Personality, General Self-Efficacy, Health Locus of Control, Health Belief) at the Diagnostic Phase and Six Months following the Implementation Phase.....	126
Table 17	Stepwise Statistics Loadings Ranked According to Relative Importance of the Predictors for CPAP Adherence and Non-Adherence.....	131
Table 18	Means and Standard Deviations for Identified Predictor Variables for the Adherent and Non-Adherent Groups.....	134

LIST OF ABBREVIATIONS

%	Percentage
<	Less than
>	Greater than
<i>A</i>	Alpha
ADHD	Attention Deficit Hyperactivity Disorder
AHI	Apnoea-Hypopnoea Index
AIDS	Acquired Immunodeficiency Syndrome
ANOVA	Analysis of Variances
APA	American Pharmacist Association
ASP	Average Sleep Propensity
Auto-PAP	Auto-titrating Positive Airway Pressure
BAS	Behavioural Activation System
BDI	Beck Depression Inventory
Bi-level PAP	Bi-level Positive Airflow Pressure
BIS	Behavioral Inhibition System
BMI	Body Mass Index
BSI	Brief Symptoms Inventory
CAD	Coronary Artery Disease
CES-D	Center for Epidemiologic Studies
CES-D Scale	Center for Epidemiological Studies Depression Scale
CI	Confidence Interval
CNS	Central Nervous System

CO ₂	Carbon Dioxide
CPAP	Continuous Positive Airway Pressure
CSA	Central Sleep Apnoea
CVA	Cerebrovascular Accidents
DA	Discriminant Functional Analysis
DS-14	Type D Scale 14
ECG	Electrocardiograph
ECG or EKG	Electrocardiogram
EDS	Excessive Daytime Sleepiness
EEG	Electroencephalogram
EM	Expectation-maximization
EMG	Electromyogram
EOG	Electrooculogram
EPQ-RS	Eysenck Personality Questionnaire-Revised Short
ESS	Epworth Sleepiness Scale
ESS	Epworth Sleepiness Scale
FOSQ	Functional Outcomes of Sleep Questionnaire
FPI	Freiberger Personality Inventory
GG	Genioglossus
GSE	General Self Efficacy Scale
HADS	Hospital Anxiety and Depression Scale
HAM-D	Hamilton Rating Scale for Depression;
HBM	Health Belief Model
HDRS	The Hamilton Depression Rating Scale;

HIV	Human Immunodeficiency Virus
HLC	Health Locus of Control
ICSD	International Classification of Sleep Disorders
IPIP	International Personality Item Pool-Five-Factor Model measure
IQ	Intelligence Quotient
<i>M</i>	Mean
MANOVA	Multiple Analysis of Variances
MCAR	Missing Completely at Random
MET	Motivational Enhancement Therapy
MHLC	Multidimensional Health Locus of Control
MHLC-C	Multidimensional Health Locus of Control – Form C
MI	Multiple Imputation
MMPI	Minnesota Multiphasic Personality Inventory
MOOD-SR	MOOD Questionnaire
MSLT	Multiple Sleep Latency Test
MSQ	Mini Sleep Questionnaire
MVA	Missing Value Analysis
MWT	Maintenance of Wakefulness Test
<i>N or n</i>	Sample Size
NA	Negative Affectivity
NCPAP	Nasal Continuous Airway Pressure
NIH	National Institutes of Health
NREM AHI	Non-Rapid Eye Movement Apnoea-Hypopnoea Index
NREM O ₂	Non-Rapid Eye Movement Oxygen Saturation

O ₂	Oxygen
OR	Odds Ratio
OSA	Obstructive Sleep Apnoea
<i>p</i>	Statistical Significance
PAP	Positive Airway Pressure
PAQ	Patient Assessment Questionnaire
POMS	Profile of Mood States;
POMS-SF	Profile of Mood States - Short form
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
RDI	Respiratory Disturbance Index
REM	REM - Rapid Eye Movement
REM AHI	Rapid Eye Movement Apnoea-Hypopnoea Index
REM O ₂	Rapid Eye Movement oxygen saturation
<i>Rho</i>	Spearman's Rank Correlation Coefficient
SADS	Schedule for Affective Disorders and Schizophrenia;
SAQLI	Sleep Apnoea Quality of Life Index
SAS	Sleep Apnoea Symptoms
SCL	Symptom Checklist
SCT	Social Cognitive Theory
<i>SD</i>	Standard Deviation
SDB	Sleep-Disordered Breathing
SDS	Zung Self-Rating Depression Scale
SE	Standard Error

SECI	Side Effects of CPAP Inventory
SEMSA	Self-Efficacy Measure for Sleep Apnoea
SI	Social Inhibition
SIDS	Sudden Infant Death Syndrome
SPSS	Statistical Package for Social Science
STAI	The State-Trait Anxiety Inventory
TM	Transtheoretical Model
TMAS	Taylor Manifest Anxiety Scale
TMD	Total Mood Disturbance
UK	United Kingdom
U.S.	United States
VU	Victoria University
WHO	World Health Organization
WHR	Waist-Hip Ratio
WSCS	Wisconsin Sleep Cohort Study

LIST OF APPENDICES

Appendix A	Austin Health consent form.....	216
Appendix B	Victoria University consent form.....	218
Appendix C	Demographic questions.....	221
Appendix D	The Profile of Mood States-Short Form.....	224
Appendix E	General Self Efficacy Scale.....	226
Appendix F	The Eysenck Personality Questionnaire.....	228
Appendix G	Multidimensional Health Locus of Control–Form C.....	231
Appendix H	Health Belief Scale.....	233
Appendix I	Epworth Sleepiness Scale.....	235

CHAPTER 1

1.1 Introduction

It was not until the 1970s that researchers began to study and understand sleep apnoea, although medical literature suggests that it was noted in the early 19th century. While it is highly likely that common symptoms of sleep apnoea—loud snoring and daytime sleepiness—have been noted since the “dawn of time”, it was not until the 1870s that British physicians began to reporting on several cases of obstructive apnoeas, describing them as “fruitless contractions of the inspiratory and expiratory muscles against glottic obstruction with accompanying cyanosis during sleep” (Lavie, 2003, p. 24). Such findings led to early research pioneered by physicians Hunter, Cheyne and Stoke in the 19th century on daytime sleepiness and periodic breathing in patients with heart failure (Lavie, 2003). It was not until after the mid-19th century that physicians began to observe initial links between obese individuals and daytime sleepiness, describing them as having “Pickwickian syndrome” after Charles Dickens’ “Fat Boy” Joe from the 1837 *Pickwick Papers* (Dickens, 1837) (see Figure 1). In mid-1950s, over 100 years later, researchers began to draw firm conclusions regarding the link between obesity and the control of breathing, although the association with a sleep disorder was not initially considered by respiratory physiologists and neurophysiologists (Lavie, 2003). Obese patients with daytime sleepiness were reported to experience daytime “CO₂ retention” or experience “CO₂ poisoning”, a description that failed to consider the impact of the upper airway in the respiratory process as well as the effects of sleep on ventilation and ventilatory stability (Bickelmann, Burwell, Robined, & Whaley, 1956, cited in Lavie, 2003).

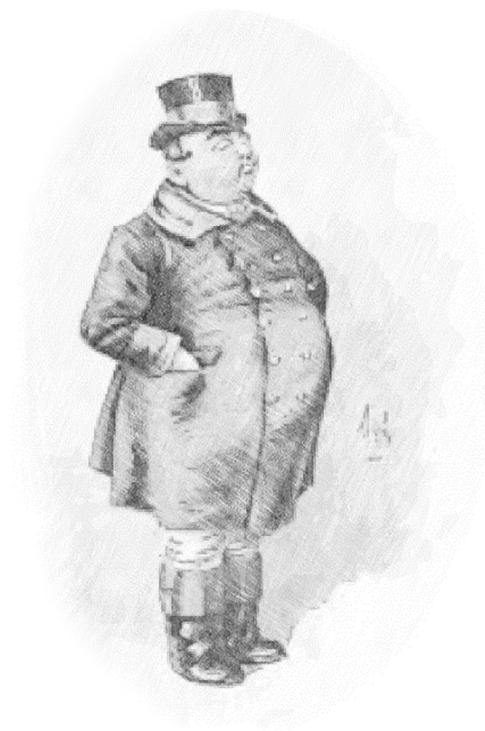


Figure 1. Dickens' "Fat Boy". Described as being upright with his eyes closed as if in sleep and having an expression of "calmness and repose". Such an image depicts symptoms of modern-day sleep apnoea, as it is recognised that many people with OSA fall asleep at inappropriate times and inappropriate situations (e.g., in a theatre, at the dinner table and even while driving)

Adapted from, *Dickens' "Fat Boy": A Classic Case of Sleep Apnoea*, by K. White, 2000.

Retrieved from http://www.talkaboutsleap.com/sleep-disorders/archives/Snoring_apnoea_dickens.htm.

It was not until Bulow's study in the early 1960s that a more accurate description of obstructive sleep apnoea (OSA) was reported and by the mid-1960s, Gastaut, Tassinari, and Duron reported the first comprehensive account of OSA in obese individuals as intermittent airway obstruction with frequent arousals (cited in Lavie, 2003). Gastaut et al.'s study specifically drew links between obesity, sleep-induced airway obstruction, sleep fragmentation,

and daytime sleepiness (Lavie, 2003). However, regardless of the importance of Gastaut et al.'s findings, OSA research continued to progress very slowly, and it was not until mid-1970s through to the early 1980s that physiological research really got underway. During this period, a number of reports paved the way for sleep and breathing research and the introduction of continuous positive airway pressure (CPAP) as the non-invasive treatment option for OSA sufferers—a move away from the use of tracheostomy in the early 1970s (see Table 1).

Instrumental during this time was the landmark paper produced by Sullivan, Berthon-Jones, Issa, and Eves (1981), who applied procedures used on canines to develop the first nasal CPAP mask for humans. Sullivan et al. used a plaster cast of their patient's nose and constructed a fibreglass nasal mask fitted with air inlets and outlets that was attached to the patient's face each night with a silicone adhesive to deliver positive air pressure. Sullivan et al. reported on observations of five patients with symptoms of noisy snoring and excessive daytime sleepiness to the point where their lives were seriously impacted (two were unemployed as a result of falling asleep at work, and one, a 13-year-old boy, was unable to stay awake at school and had consequently been categorised as 'mentally retarded'). Sullivan et al. conducted three all-night sleep studies on each patient. On the third night CPAP was implemented and reported to completely prevent upper airway occlusion in each of the five patients, thus eliminating apnoea. Sullivan et al. described the application of CPAP as 'a pneumatic splint for the nasopharyngeal airway' and apnoea was shown to return when air pressure was dropped. However, acceptance of this treatment did not come immediately, and application of their findings to a wider audience was even slower and initially accompanied with scepticism.

Table 1

Landmark Sleep Studies of OSA During the 1970s and 1980s

	Year	Author	Title	Description
1.	1974	Orem, Montplaisir, Dement	Changes in the activity of respiratory neurons during sleep	Impact of sleep on the brain stem and respiratory neuronal activity in unanesthetised cats.
2.	1976	Brouillette, Thach	Characterisation of the in vitro effects of 5- hydroxytryptamine (5- HT) on identified neurones of the rat dorsal motor nucleus of the vagus.	Neuromuscular reflex mechanism maintaining extrathoracic airway patency in rabbits.
3.	1976	Phillipson, Murphy, Kozar	Regulation of respiration in sleeping dogs.	Sleep effects on reflex control of breathing in the dog and identification of a sensitive CO ₂ - induced apnoeic threshold in sleeping humans
4.	1978	Remmers, deGroot, Sauerland, Anch	Pathogenesis of upper airway occlusion during sleep.	Description of anatomical and neurophysiological determinants of upper airway occlusion in the sleeping human, which provided a unifying “balance of forces” concept of OSA pathogenesis

5.	1981	Sullivan, Issa, Berthon-Jones, Eves	Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares.	The landmark introduction of CPAP application as the noninvasive treatment for obstructive sleep apnoea.
----	------	--	--	---

Source: Dempsey, Sigrid, Veasey, Barbara, Morgan, & O'Donnell (2010).

In the 1990s, Fletcher, Lesske, Qian, Miller, and Unger (1992) paved the way for studies exploring long-term cardiovascular consequences of OSA by inducing sleep apnoea in rats. Using 12-second infusions of nitrogen gas into daytime sleeping chambers, four groups of male rats (250–375 g) were subjected to cyclic hypoxia (3–5% nadir ambient oxygen) every 30 seconds, seven hours per day for up to 35 days, to create daytime hypertension. Fletcher and colleagues were one of the first to study an association between chronic high blood pressure and OSA and hypothesised that repetitive episodic hypoxia patterned after the hypoxia seen in OSA could contribute to the elevation of blood pressure.

At the same time, Mezzanotte, Tangel, and White (1992) studied the waking genioglossus (GG) electromyogram activity in 11 OSA patients and 14 age-matched control patients to determine if GG activity was higher in an awake state in patients diagnosed with OSA. Mezzanotte et al. concluded that neuromuscular compensation presented during wakefulness in OSA patients may be lost during sleep, leading to airway collapse. This suggested that airway collapse could be reduced with CPAP use.

In 1993, Young et al. published the first population-based study conducted using laboratory sleep studies. Young et al. utilised the data from the Wisconsin Sleep Cohort Study, a longitudinal study of the natural history of cardiopulmonary disorders of sleep, to estimate the prevalence of undiagnosed sleep-disordered breathing among adults. Using a random sample, Young et al. selected 602 employed men and women aged 30 to 60 years old to determine the frequency of episodes of apnoea and hypopnoea per hour of sleep. The estimated prevalence of sleep-disordered breathing—defined as an apnoea-hypopnoea score of five or greater—was 9% for women and 24% for men. Young et al. estimated that 2% of women and 4% of men in the middle-aged workforce met the minimal diagnostic criteria for sleep apnoea syndrome (an apnoea-hypopnoea score of five and greater and daytime sleepiness). Again, men and obesity were strongly associated with the presence of sleep-disordered breathing. In their study, Young et al. also reported that habitual snorers, both men and women, tended to have a higher prevalence of apnoea-hypopnoea scores of 15 and greater. Most importantly, Young et al.'s findings provided evidence to suggest that the prevalence of undiagnosed sleep-disordered breathing was high among men and much higher than suspected among women. This study also drew a strong association between undiagnosed sleep-disordered breathing and daytime sleepiness, demonstrating the potential importance and impact that sleep apnoea has on public health. It is also probable that the prevalence of sleep-disordered breathing is actually higher than reported by Young and colleagues (Popescu, Latham, Allgar, & Elliott, 2001).

Since Young et al.'s landmark study in the 1990s, research on sleep apnoea has gained much momentum. Literature in this domain has historically focused on clinical and population-based research with attention placed on prevalence, causes, consequences, and treatment of OSA.

As such, sleep apnoea has attracted many researchers from varying disciplines, psychology being one of the more recent specialities to contribute to the growth of research in this area.

1.2 Sleep Apnoea

1.2.1 Definition

Derived from the Greek word “apnoea” literally meaning “without breath,” sleep apnoea is characterised by the recurrent cessations or interruptions of breathing during sleep due to the functional collapse of the upper airway. Cessation can be partial (hypopnoea), contributing to decreased tidal volume, or complete (apnoea), contributing to the loss of ventilatory effort. As a result, an individual’s capacity to take in oxygen is decreased, contributing to a lowered blood oxygen level that triggers the brain to prompt the individual to breathe again via gasping, which in turn “jump starts” the breathing process until the next cessation. Many medical consequences have been associated with sleep apnoea and they are strongly related to pathophysiological findings linked to sleep fragmentation (leading to complications arising from sleep deprivation) and blood oxygen desaturation in severe cases (see Table 2).

Table 2

Medical Consequences of Sleep Apnoea

Cardiovascular consequences	Other consequences	Other associated conditions
Hypertension (high blood pressure)	Trauma (traffic accidents)	Obesity
Heart failure	Glaucoma	Obesity syndromes, such as

		Prader-Willi syndrome
Atherosclerosis (heart attacks, angina)	Snoring spouse syndrome	Polycystic ovary disease
Atherosclerosis (stroke)	Diminished libido	Renal failure
Atrial fibrillation	In children: illness like attention deficit hyperactivity disorder (ADHD)	Hypothyroidism
Ventricular arrhythmias	In children: slowed growth	Marfan syndrome
Pulmonary hypertension		Charcot-Marie-Tooth disease
		Post-polio syndrome
		Gastro-esophageal reflux
		Worsening of epilepsy

1.2.2 Types of sleep apnoea

There are three different types of sleep apnoea:

1. Obstructive sleep apnoea. OSA is a chronic condition characterised by repetitive episodes of upper airway collapse during sleep. It may contribute to a range of cardiovascular complications and other serious effects (see Table 2). While OSA was once believed to be uncommon, today it accounts for 75–80% of diagnoses made in diagnostic sleep study laboratories worldwide. OSA remains a substantial health problem in society, with up to 2% of women and 4% of men suffering from this condition (Young et al., 1993; Peppard & Gottlieb, 2002; Kjelsberg, Ruud, & Stavem, 2005; Olsen, Amith, Oei, & Douglas, 2008; Moran, Everhart, Davis,

Wuensch, Lee, & Demaree, 2011). It is believed that the actual prevalence of OSA is in fact double that currently reported and diagnosed (Popescu et al., 2001). OSA is usually associated with the following commonly identifiable symptoms: daytime sleepiness, loud or chronic snoring, choking or snorting, and long pauses in breathing (see Table 3) (Popescu et al., 2001).

2. Central sleep apnoea (CSA). CSA is a relatively rare form of sleep apnoea where the airway remains open, but the thoracic and abdominal muscles (i.e., diaphragm and chest muscles) temporarily fail to process instructions from the brain to continue moving air to and from the lungs. Because the airway is typically open, a CSA sufferer does not snore loudly but does experience daytime sleepiness. The prevalence of CSA is asymptomatic given the very low number of reported cases in the general population (less than 1%) (Bixler, Vgontzas, Ten Have, Tyson, & Kales, 1998). CSA is more common among people over 60 years of age, and it is often associated with other medical conditions such as, sleep disorders, insomnia, neurological disorders, and heart failure where the prevalence of CSA is said to be as high 40% to 60% (Javaheri et al., 1998; Lanfranchi et al., 1999; Lavie, Pillar, & Malhotra, 2002).
3. Mixed sleep apnoea – This type is characterised by the signs and symptoms of both CSA and OSA. It often begins as CSA and develops into the obstructive form (Lavie et al., 2002).

Table 3

Common Symptoms of Obstructive Sleep Apnoea

Daytime symptoms	Symptoms during sleep
Daytime sleepiness or fatigue	Restlessness during sleep
Headaches in the morning	Dry mouth or sore throat
Trouble concentrating, forgetfulness, depression, or irritability	Sudden awakenings with a sensation of gasping or choking
Sexual dysfunction	Snoring
Difficulty getting up in the mornings	Night sweats

1.3 Diagnosis of OSA

The International Classification of Sleep Disorders (ICSD), Revised Diagnostic and Coding Manual states that “obstructive sleep apnoea syndrome is characterised by repetitive episodes of upper airway obstruction that occur during sleep, usually associated with a reduction in blood oxygen saturation” (American Academy of Sleep Medicine, 2001). The American Academy of Sleep Medicine recommends the following diagnostic criteria for determining OSA:

- A. The patient has a complaint of excessive sleepiness or insomnia. Occasionally, the patient may be unaware of clinical features that are observed by others.
- B. Frequent episodes of obstructed breathing occur during sleep.
- C. Associated features include:
 - 1. loud snoring
 - 2. morning headaches

3. a dry mouth upon awakening
4. chest retraction during sleep in young children

D. Polysomnographic monitoring demonstrates:

1. more than five obstructive apnoeas, greater than 10 seconds in duration, per hour of sleep and one or more of the following:
 - a. frequent arousals from sleep associated with the apnoeas
 - b. bradycardia; and
 - c. arterial oxygen desaturation in association with the apnoeic episodes
2. A mean sleep latency of less than 10 minutes as measured by the Multiple Sleep Latency Test (MSLT).

E. The symptoms can be associated with other medical disorders (e.g., tonsil enlargement).

F. Other sleep disorders can be present (e.g., periodic limb movement disorder or narcolepsy).

For a diagnosis, a full night's sleep study should be undertaken in a sleep laboratory where ideally six hours of sleep is recorded. The sleep study generally consists of continuous arterial oxygen and electrocardiograph (ECG) monitoring and polysomnography (PSG), as well as measurements of airflow through the nose and/or mouth and thoracic cage movement during all stages of sleep. OSA severity is assessed by the Respiratory Disturbance Index (RDI) and/or the Apnoea-Hypopnoea Index (AHI). The AHI is the most commonly reported index in sleep disorder research as well as sleep studies and it is calculated by dividing the number of respiratory disturbances by the total number of sleep hours (see Table 4). It must be noted that

the AHI cut-off points have not been adequately determined and are mainly based on epidemiological data and consensus (Young et al. 1993; American Academy of Sleep, 1999).

Table 4

AHI and Oxygen Saturation Values Representing Different Severity Levels

	AHI	Minimum O ₂ saturation (%)
Normal	<5	>95
Mild	5–15	>85
Moderate	15–30	>65
Severe	>30	<65

A detailed medical history is taken, and a thorough otolaryngological examination is conducted to provide information about airway morphology. Furthermore, a general physical examination is also commonly conducted to detect any co-existing cardiopulmonary abnormalities. Blood samples may be taken to assess for polycythaemia resulting from hypoxic bone marrow stimulation. A chest radiograph and ECG may also be conducted to evaluate cardiopulmonary functioning and identify any co-existing abnormality, as well as a spirometry test where a saw toothed patterned result may be indicative of possible airway obstruction during sleep (Lavie et al., 2002).

One method used to localise the site of airway obstruction is a fibre-optic pharyngoscopy with or without Mueller's manoeuvre, which is carried out in the sitting and supine positions. Mueller's manoeuvre involves vigorous inspiration with the nose and mouth closed. The movements of the pharyngeal wall during this manoeuvre reportedly replicate obstructive events

occurring during sleep apnoea. It is considered that fibre-optic pharyngoscopy would yield better information if it could be performed during sleep, but this method is generally viewed as too invasive and impractical (Lavie et al., 2002).

The aforementioned investigations are commonly used for diagnosis and enable the necessary distinction to be made between type, frequency, and severity of apnoeic episodes, as well as their incidence during rapid eye movement (REM) sleep and non-REM sleep. For a diagnosis of CSA, at least 80% of the events are required to be of central origin. The RDI and/or AHI is used to assess the severity of OSA and CSA by means of measuring of the rate of sleep-disordered breathing events per hour of sleep and the magnitude of associated oxygen desaturation (low partial pressure of oxygen in the arterial blood) (Lavie et al., 2002; Somers, et al., 2008).

1.4 Clinical Features of OSA

The greatest risk factors associated with a diagnosis of OSA include obesity, an age greater than 65 years old, and being male (Kripke, Ancoli-Israel, Klauber, Wingard, Mason, & Mullaney, 1997). A history of loud, intermittent snoring is present in almost all patients with OSA, and most only seek medical advice at the request of their partner or other family members. Loud snoring is a sign of partial upper airway obstruction and is caused by vibration of the soft palate due to airflow through the narrowed airway. Snoring ceases when the obstruction becomes complete, causing apnoea (Kripke et al., 1997).

Daytime sleepiness due to repeated nocturnal waking is a major incapacitating symptom of OSA, which can have serious social, economic (job loss, lost work time), and safety consequences (work and road accidents). Decreased quality of life, fatigue, neuro-cognitive impairments, increased risk of mood disorders, morning headaches, decreased libido, and impotence are also common symptoms of OSA (see Table 2) (Young, Blustein, Finn, & Palta, 1997; Masa, Rubio, & Findley, 2000; Aloia, Arnedt, Stepnowsky, Hecht, & Borrelli, 2005).

1.4.1 Pathophysiology

OSA occurs as a result of the collapse of the upper airway during sleep that limits airflow and in more severe cases reduces blood oxygen levels during sleep. Physiologically, structural narrowing, generally in the velopharynx and/or tongue base hypopharyngeal region, contributes to airway occlusion as the tongue comes into contact with the soft palate and posterior pharyngeal wall, leading to the progressive collapse of the lower pharyngeal airway and the lateral oropharyngeal wall. This results in higher negative inspiratory pressure to maintain ventilation, subsequently contributing to hypoxia (decreased blood oxygen levels), hypercapnia and arousal, as well as forcing expiration as airway patency is restored (see Figure 2) (Hussain, 1996; Lavie et al., 2002).

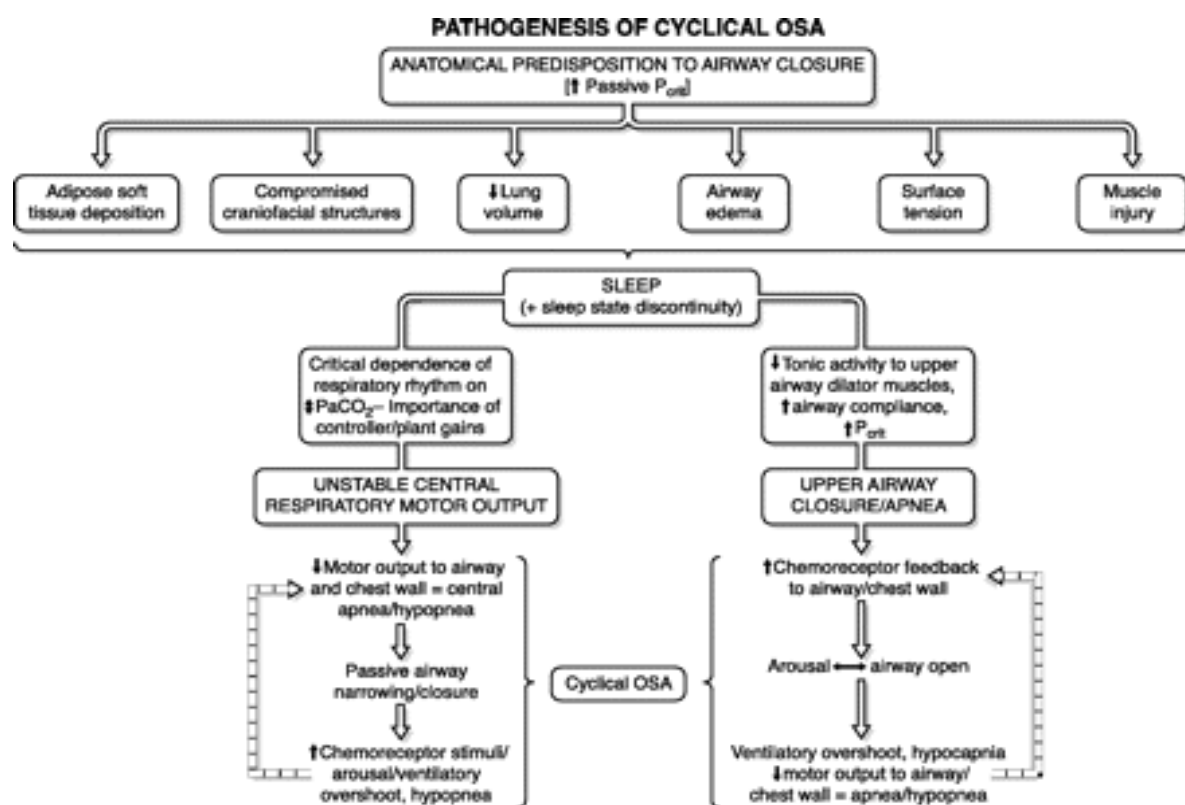


Figure 2. An integrated map of the pathogenesis of the cyclical nature of OSA. This map highlights various structural and functional determinants of an anatomical predisposition for airway closure and the essential component of sleep. From Pathophysiology of Sleep Apnoea, by J. Dempsey, S. Veasey, B. Morgan, & C. O'Donnell, 2010, *Physiological Reviews* 1(90), 47-112. Copyright 2010 by the American Physiological Society. Reprinted with permission.

1.4.2 Risk factors

Several risk factors are responsible for the development and progression of OSA and can be categorised into modifiable and non-modifiable risk factors (see Figure 3).

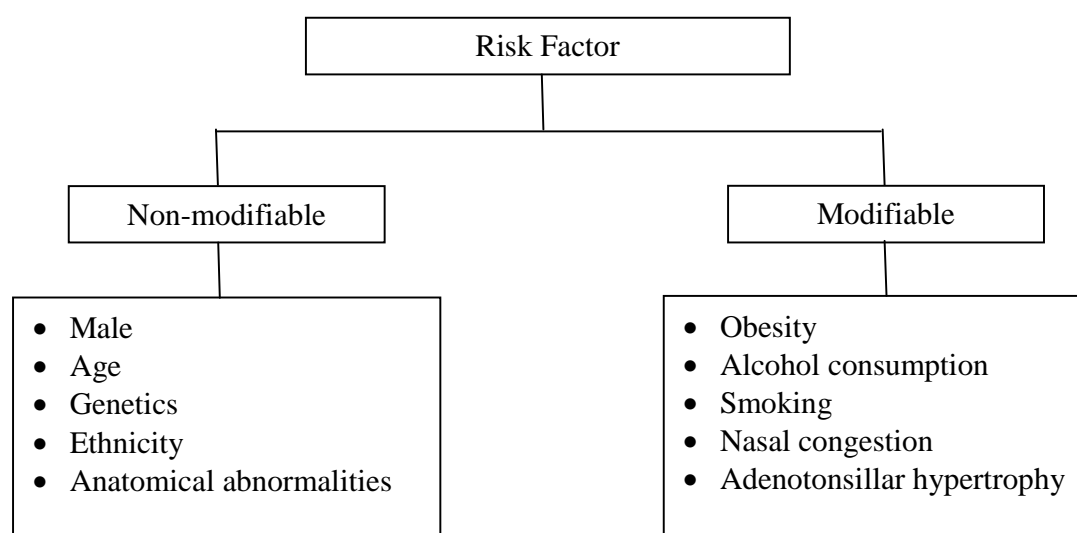


Figure 3. Summary of risk factors grouped as modifiable and non-modifiable. Adapted from Sleep Disordered Breathing Disorders, S. Sharma, A. Khanna, & A. Sharma, A. n.d., *The Association of Physicians of India*. Retrieved from http://www.apiindia.org/medicine_update_2013/chap104.pdf.

1.4.2.1 Obesity.

Obesity has been identified as the most likely single predictive risk factor for the development of OSA. Numerous studies have reported a high prevalence of OSA in obese patients, with up to 70% of those classified as morbidly obese (body mass index/BMI of 40

kg/m₂ or more) suffering from OSA (Davies & Stradling, 1990; Davies, Ali, & Stradling, 1992; Hoffstein & Mateika, 1992; Katz, Stradling, Slutsky, Zamel, & Hoffstein, 1990; Levinson et al., 1993; Millman, Carlisle, McGarvey, Eveloff, & Levinson, 1995; Shelton, Woodson, Gay, & Suratt, 1993; Shinohara, et al., 1997 all cited in Young, Peppard. & Gottlieb, 2002).

To date, clinical research has provided little explanation of the exact mechanism(s) by which obesity causes OSA. However, it is widely suggested that the relationship between obesity and OSA relates to a combination of several factors. These factors include upper airway narrowing (as stated above), weakening of the upper airway muscles, and decreased lung volumes. Central obesity, where fat distributions are localised to the abdomen and upper part of the body, contribute more to the risk for OSA than general obesity. Some researchers consider this to be the best explanation for why a greater proportion of men suffer from OSA. Neck circumference index and waist-hip ratio (WHR) have also been identified as predictors of OSA (Lavie et al., 2002). The predictive association between obesity and OSA is supported by research showing that weight reduction in morbidly obese patients often either eliminated or more commonly reduced the severity of OSA (Young et al., 2002; Lavie et al., 2002). Physical activity and an exercise training regime have been shown to have positive moderate treatment efficacy for the reduction of AHI in obese adults, which suggests that exercise may be beneficial for the management of OSA beyond weight reduction (Kline et al., 2011).

1.4.2.2 Age.

Like obesity, it is unclear what the exact mechanism(s) are in aging that are risk factors for OSA. There are no clear anatomical or physiological factors associated with ageing that

predispose individuals to an increased risk of apnoea. However, the most likely explanation is that with increasing age many people gain weight. In addition, there may be deterioration in the integrity of the muscles in the neck and throat area. Historically, a number of longitudinal and cross-sectional studies have documented that OSA is age-dependant (Ancoli-Israel et al., 1991; Bixler, Vgontzas, Ten Have, Tyson, & Kales, 1998; Durán, Esnaola, Rubio, & Iztueta, 2001; Budhiraja et al., 2007). In children, reports of OSA (in individuals three to five years of age) are generally associated with narrow airways, large tonsils, and adenoids (Lavie, et al. 2002). Studies have shown that OSA prevalence is low during adolescence and early adulthood and then dramatically increases during middle age and old age (i.e., 60 to 70 years of age) independently of BMI (Ancoli-Israel et al., 1991; Budhiraja et al., 2007).

1.4.2.3 Sex.

Population-based studies have commonly reported a strong link between being male and OSA (Young et al., 1993; Young, Evans, Finn, & Palta, 1997; Young et al., 2002). Men have been found to have between a two- and five-fold increased risk of OSA compared with weight-matched women. This ratio is increased in sleep clinic populations where in comparison to women, about 10 times more men present with sleep problems (Young et al., 1993; Young et al., 2002). Although no conclusive evidence is provided, a number of studies have reported four explanations to account for the prevalence of OSA within men compared to women (Strohl & Redline, 1996). The first pertains to sex-related differences in anatomy and physiology of the upper airway and respiratory control system (Strohl & Redline, 1996; Lavie et al., 2002). The second addresses the possibility that androgens may inhibit upper airway muscle activity in men, while in women oestrogen and progesterone may stimulate respiration (Strohl & Redline, 1996;

Lavie et al., 2002). The third suggests that central obesity, which is more common in men because of the distribution of fat on the body, is the main cause of OSA (Strohl & Redline, 1996; Lavie et al., 2002). The fourth suggests that sex-related, genetic, anatomical differences in the upper airway (inclusive of hormone-mediated explanations) may cause OSA (Strohl & Redline, 1996; Lavie et al., 2002). Reports of increased OSA prevalence in post-menopausal women support the possibility that sex hormones play a role in OSA (Krystal, Edinger, Wohlgenuth, & Marsh, 1998 cited in Young et al., 2002). Other studies have reported that hormone replacement therapy in post-menopausal women results in increased upper airway muscle, thus decreasing OSA (Lavie et al., 2002). However, the replication of such studies where men were treated with hormone therapy has yielded opposite findings, which suggests that hormonal differences alone may not explain the sex-related difference in OSA (Young et al., 2002; Lavie et al., 2002).

1.4.2.4 Anatomy.

Anatomical narrowing of the upper airway has been identified as a risk factor for OSA. Using imaging technology, clinical studies have shown that having a small pharyngeal airway increases the possibility of OSA. Other anatomical factors include upper airway narrowing as a result of obesity or from specific craniofacial abnormalities, mid-face hypoplasia, mandibular hypoplasia, increased soft tissue size, hypertrophy of the tonsils and adenoids (which are especially important in children), and increased uvula size or long soft palate (Lavie, et al., 2002).

Commonly an obstruction occurs in the upper airway, but the nasal anatomy also plays a role in OSA (see Figures 4). Nasal polyps, deflected nasal septum, or chronic nasal congestion

contribute to increased nasal resistance that can result in increased negative pressure by the diaphragm and an increased tendency of the upper airway to collapse (Lavie, et al., 2002).

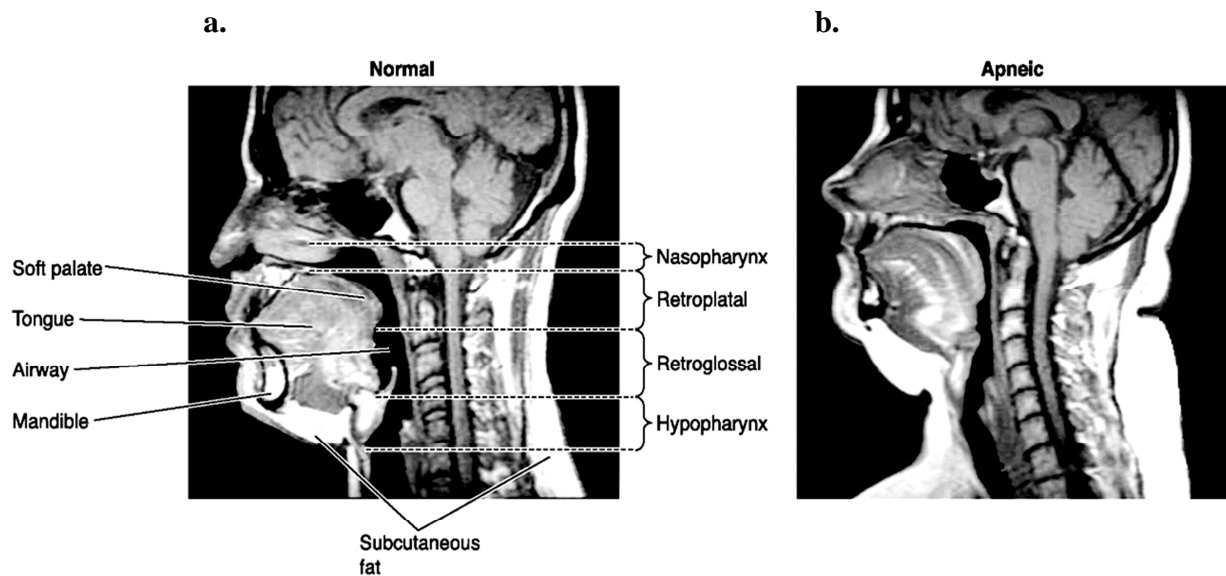


Figure 4. a. Diagram of the upper airway showing the soft palate is normal in length and total size. The tongue is normal in size and is angled forward. The upper airway at the level of the nasopharynx and hypopharynx is normal in size and contour. B. In contrast to the apneic where there is an elongated and enlarged soft palate impinges on the posterior airway at the level of the nasopharynx. In addition, a retruding jaw pushes an enlarged tongue posteriorly to impinge on the hypopharyngeal. Adapted from Pathophysiology of Sleep Apnoea, by J. Dempsey, S. Veasey, B. Morgan, & C. O'Donnell, 2010, *Physiological Reviews* 1(90), 47-112. Copyright 2010 by the American Physiological Society. Adapted with permission.

1.4.2.5 Genetics.

Several studies have reported an autosomal dominant pattern of inheritance of OSA, but the role that genetics play is not well understood (Strohl, Saunders, Feldman, & Hallett, 1978; Mathur & Douglas, 1995; Redline et al., 1995; Redline & Tishler, 2000). Nonetheless, it is widely acknowledged that the genetic link is multi-factorial and familial patterns are reported as potential indicators of OSA (Lavie et al., 2002). Research in this area has suggested a higher prevalence of OSA diagnoses in offspring whose parents were also previously diagnosed with OSA, with the relative risk of OSA being two- to four-fold greater in first-degree relatives. Some studies have reported that familial factors may account for as much as 40% of the variance in AHI and suggest that this variance remained significant after adjustment for BMI and cephalometric measurement (Redline et al. 1995; Redline & Tishler, 2000; Lavie, et al., 2002). While little is understood about the importance of genetics, Lavie and colleagues (2002) report an increased risk of OSA in lean relatives of OSA subjects along with a higher incidence of sudden infant death syndrome (SIDS) in these families.

1.4.2.6 Other risk factors.

Several additional unique, but less common risk factors are also known to contribute to OSA. These include diseases such as hypothyroidism (mainly caused by myxoedema and decreased upper airway muscle functions) and acromegaly (mainly resulting from macroglossia and potentially disordered ventilator control stability) (Lavie et al., 2002). Additionally, cerebrovascular accidents (CVA) and brainstem lesions have also been linked to OSA, although most commonly linked with CSA and not OSA (Lavie et al., 2002). Other predisposing factors

include neuromuscular diseases such as Duchenne and myotonic dystrophies, myopathies such as Nemaline-Rod myopathy or acid-maltase deficiency, and neuropathies such as Charcot-Marie-Tooth peripheral neuropathy or post-polio syndrome (Lavie et al., 2002). These muscular diseases increase the likelihood of hypoventilation and both central and obstructive types of sleep-disordered breathing events (Lavie et al., 2002). Quadriplegia also results in mild OSA as well as related neurological and cognitive deficits, sleepiness, and reduced quality of life (Berlowitz et al., 2012).

Chronic lung disease also contributes to events of oxygen desaturation, usually resulting from hypoventilation and low lung capacity. Whilst there is no real association between chronic lung disease and OSA, OSA does tend to be more severe in this population. In addition, the use of central nervous system (CNS) depressants such as hypnotics, opioids or alcohol increases the risk of apnoeas (Lavie, et al., 2002). Table 5 summarises the risk factors for OSA.

Table 5

Risk Factors for Obstructive Sleep Apnoea

Risk factors	Examples
Upper airway anatomy	Retrognathia, micrognathia, macroglossia, hypertrophy of tonsils or adenoids, increased uvula size, long soft palate, nasal narrowing
Obesity	Increases, especially with central obesity
Age and sex	OSA is more prevalent in men, and the

prevalence increases with age

Endocrine	Hypothyroidism, acromegaly
Genetic	OSA cluster in families, with relative risk of two - to fourfold in first-degree relatives
Neuromuscular	Myopathies, muscular dystrophies, neuropathies
Extrinsic	Alcohol, CNS depressants (hypnotics, opioids)

Source: Lavie et al. (2002).

1.4.3 Epidemiology and prevalence

The prevalence of OSA has been well documented in population-based studies, resulting in well-defined epidemiological characteristics of the condition (Chesson et al., 1997; Malhotra & White, 2002; ICSD-2, 2005). Population-based studies have estimated the prevalence of sleep-disordered breathing (AHI > five events per hour) to be approximately 9% in females and 24% in males and to be much higher (up to 80%) in the elderly. An AHI greater than five is insufficient alone for a diagnosis of OSA, and diagnosis is generally supported by characteristic complaints—most notably daytime sleepiness (Young, et al., 1993; Strohl & Redline, 1996). Using this definition, a prevalence of 2% in middle-aged females and 4% in middle-aged males has been commonly reported (see Table 6). This increases up to 30% in elderly people. It should

be noted however, that although subjective daytime sleepiness is currently required for the diagnosis of OSA, there is evidence to suggest that some people may be unaware of their sleepiness and that important complications of OSA such as hypertension may occur in the absence of sleepiness. Thus, the prevalence of OSA may be higher if cardiovascular complications are included in the definition (Popescu et al., 2001).

Table 6

Large Sample Studies on the Prevalence of OSA

Country	Study	Sample size	Ethnicity	Diagnostic method	Men	Women
US	Young et al. (1993)	602	White	Polysomnography	4%	2%
US	Bixler et al. (2001)	1741	White	Polysomnography	3.9%	1.2%
Australia	Bearprk et al. (1995)	485	White	MEASAM IV	3.1%	-
India	Udwadia et al. 2004)	250	Indian	Polysomnography	7.5%	4.5%
China	Ip et al. (2001)	258	Chinese	Polysomnography	4.1%	-
China	Ip et al. (2004)	457	Chinese	Polysomnography	-	2.1%
Korea	Kim et al. (2004)		Korean	Polysomnography	4.5%	3.2%

Source: Deloitte Access Economic (2011).

The most widely referenced population-based study on the prevalence OSA is the Wisconsin Sleep Cohort Study (American Academy of Sleep, 1999; Peppard et al., 2000; Young & Peppard, 2000; Malhotra & White, 2002; Young et al., 2002; Young, 2009). The primary goal of the Wisconsin Sleep Cohort Study (WSCS) was to provide an account of the natural history of sleep-disordered breathing (SDB) and other sleep disorders, with the longer-term goal of better understanding the total societal burden of SDB. The WSCS provided valuable and specific information describing the occurrence of SDB (including age- and sex-specific prevalence for mild, moderate, and severe SDB), an estimate (including longitudinal data) on the impact of SDB with regard to cardiovascular and morbidity and mortality, and identified risk factors for the development and progression of SDB (Young, 2009). The study design of the WSCS mirrored those of standard epidemiological cohort studies. The WSCS design utilised population-based sampling, recruitment of a probability sample, and collection of data at baseline and follow-up. Adding to the value of the design, the WSCS used laboratory PSG, which provided extensive biomedical data (sleep-related variables) and offered a means for the findings to be translated to the clinical setting (Young, 2009).

The WSCS study estimated that OSA affected approximately 2% of women and 4% of men in the north-central region of America (Peppard et al., 2000; Young & Peppard, 2000; Malhotra & White, 2002; Young et al., 2002; Young, 2009). However, up to 9% of women and 24% of men in this population had an AHI greater than five without the presence of coexisting OSA symptoms. These figures were comparable to those reported in Australia's most comprehensive reviews of sleep-related disorders: the 2004 Access Economics Study, "Wake up Australia: The value of healthy sleep" and the Deloitte Access Economic report "Re-awakening

Australia: The economic cost of sleep disorder in Australia, 2010.” These Australian reviews estimated the OSA prevalence in Australia to be between 3% and 5%.

The “Wake up Australia” study estimated that 1.2 million Australians (6% of Australia’s population) suffered from sleep-related disorders that were likely to impact on both the physiological and psychological functioning of an individual. The cost of poor sleep to the Australian population was estimated to be between \$3 billion and \$7 billion per annum—not inclusive of the social costs (Access Economics, 2004). The 2010 Deloitte Access Economic report (2011) estimated the total number of Australians suffering from OSA to be approximately 774,590, with women making up 24% (185,410) and men 76% (589,181) of OSA sufferers (see Table 7).

Table 7

Estimated Prevalence of Moderate to Severe OSA in Australia, 2010

Age group	Women		Men		Persons	
	%	No.	%	No.	%	No.
20-44	0.6	22,775	3.3	126,317	1.6	149,092
54-64	1.9	53,706	10.3	287,186	5.3	340,892
65+	6.7	108,929	12.6	175,677	10.2	284,606
Total	2.2	185,410	7.2	589,181	4.7	774,591

Source: Deloitte Access Economic (2011).

There are approximately 80 clinically diagnosable sleep disorders and OSA has been reported to be the most commonly diagnosed in sleep laboratories and CPAP the standard

treatment option (Pepin et al., 1999; Collard, Pieters, Aubert, Delguste, & Rodenstein, 1997; Malhotra, Ayas, & Epstein, 2000; Popescu et al., 2001; Stepnowsky & Moore, 2003). However, the insidious nature of OSA means that it often goes undiagnosed for many years and the prevalence of OSA is probably higher than estimated by the key studies mentioned above. Studies by Davies and Stradling (1996), Lindberg and Gislason (2000), and Young et al. (2002) have reported that the level of undiagnosed OSA among adults in Western countries may be as high as 5%. Wiegand and Zwillich (1994) attributed the under-diagnosis of OSA to the low awareness of OSA in the community and among health professionals and they suggested that 80% to 90% of affected individuals may not have received a clinical diagnosis (Young et al., 1993; Wiegand & Zwillich, 1994). Given the estimate of undiagnosed OSA, improving knowledge of sleep disorders at the primary care level would be a useful goal.

1.5 Quality of Life

The impact of OSA on quality of life is extensive and well documented (Young, et al., 1997; Masa et al., 2000; Baldwin, Griffith, Nieto, O'Connor, Walsleben, & Redline, 2001; Aloria et al., 2005). The consequences of OSA are commonly reflected by deficits in memory and concentration, reduced levels of alertness and increased sleepiness, fatigue, irritability, headaches, and a range of psychological symptoms such as depression and changes in mood and personality. The effects of OSA are likely to impact instrumental activities of daily functioning and living that commonly result in impaired work efficiency, relationship issues and social problems, as well as increased work-related and motor vehicle accidents. In addition, sufferers of OSA generally show increased chances of hypertension, cardiovascular disease, and other

medical implications (Flemons & Tasi, 1997; Paiva, Farinha, Martins, Batista, & Guilleminault, 1997; Wright, Johns, Watt, Melville, & Sheldon, 1997).

1.6 CPAP Treatment for OSA

CPAP use has been shown to be the most common and effective form of treatment for alleviating OSA symptoms such as loud snoring, daytime sleepiness, and excessive fatigue (Popescu et al., 2001). CPAP treatment involves connecting a patient to a CPAP device that delivers a flow of positively pressured air, via a mask worn over the nose and/or mouth, at a predetermined pressure during sleep (see Figure 5). This pressure is continuously maintained throughout phases of the respiratory cycle, acting as Sullivan et al. first described in their 1981 paper as a “pneumatic splint” holding open the upper airway to prevent the occurrence of apnoeas (Sullivan et al., 1981; McDaid et al., 2009).

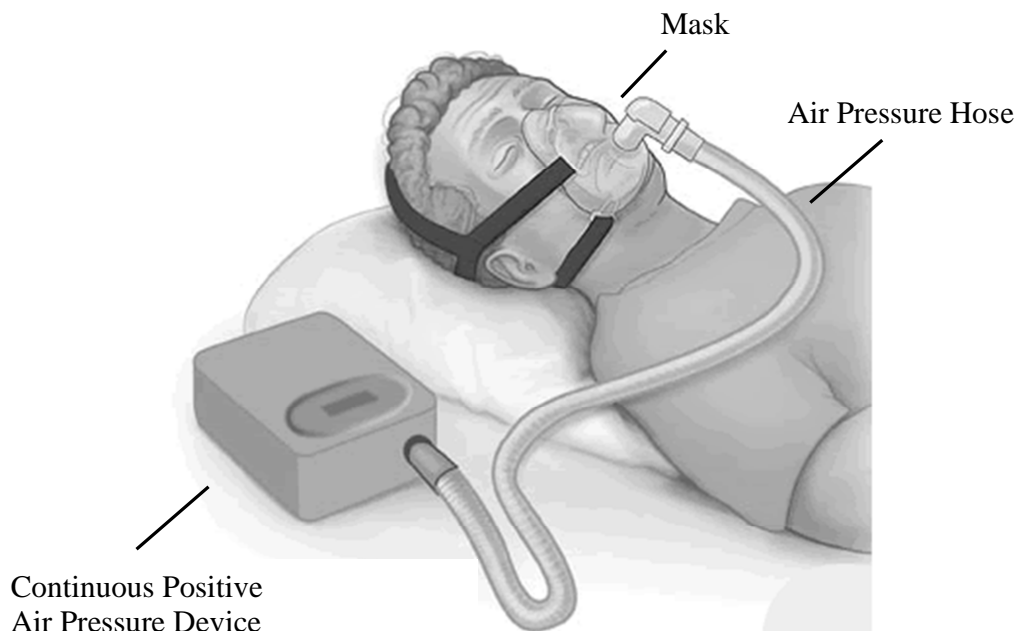


Figure 5. CPAP device. Diagram of a CPAP device that “splints” the patient’s airway open during sleep by means of a flow of pressurised air into upper airways. The patient usually wears a plastic facial mask, which is connected by a flexible tube to a small CPAP device. Adapted from Positive Airway Pressure. In Wikipedia, n.d., Retrieved May 29, 2014, from http://en.wikipedia.org/wiki/Positive_airway_pressure. Adapted with permission.

Advances have also led to the introduction of auto-titrating devices (auto-PAP). Auto-PAP is designed to deliver minimum effective pressure (often lower than the optimal fixed CPAP) that is auto-adjusted to increase pressure as required to maintain airway patency and decrease pressure if there are no events detected over time. In doing so, auto-PAP was initially argued to increase adherence to OSA treatment and offer a solution to individuals impacted by the cost and waiting times of PSGs. However research has questioned such benefits especially in light of its utility related to reported air leaks (Berry, Parish, & Hartse, 2002; Valentin, Subramanian, Quan, Berry, & Parthasarathy, 2011). Similarly, bi-level PAP therapy was developed with the idea of varying the administered pressure between the inspiratory and expiratory cycles. However, despite advancements in PAP technology a systematic Cochrane database review has concluded that bi-level PAP therapy was not superior to conventional CPAP therapy from an adherence standpoint (Antonescu-Turcu & Parthasarathy, 2010).

The beneficial effects of CPAP use have been described as rapid, with some studies reporting almost complete disappearance of OSA symptoms after one month of CPAP use (Aloia et al., 2007; Smith et al., 2008). Given the nature of OSA, CPAP treatment is considered a preventative option and not a curative measure, thus making CPAP use a long-term treatment option.

CPAP use also has a number of adverse but not serious side-effects, which can be adequately managed. These include skin sores and erosions, nasal stiffness, blocking or congestion, and discomfort due to the device itself (see Table 8). However, a large variety of commercial CPAP devices are now available with an extensive range of mask models for all shapes and forms of the human face thus minimising some of the side-effects and promoting greater comfort which leads to better ongoing usage (Berry et al., 2002; Valentin et al. 2011).

Table 8

Reports of Minor Adverse Events from CPAP

Adverse events	% reported
Dry nose/mouth/throat	41.7
Rhinitis	30.6
Noise	29.0
Nasal congestion	24.4
Sore eyes	23.4
Headaches	16.0
Mask discomfort	12.5
Chest discomfort	5.2

Source: Deloitte Access Economics (2011)

Currently CPAP treatment is considered to be the most successful medical treatment available to OSA patients. Giles, Lasserson, Smith, White, Wright, and Cates (2006) and McDaid et al. (2009) conducted two separate and comprehensive reviews of the existing literature surrounding the efficacy of CPAP use. Giles et al. reported in his Cochrane review that

CPAP treatment accounted for a significant reduction in AHI and daytime sleepiness (measured by the popular and practical Epworth Sleepiness Scale). The findings of McDaid et al.'s review mirrored those of Giles et al. (2006) in that the patients ($N = 1,334$) reported statistically significant reductions in sleepiness (similarly measured using the Epworth Sleepiness Scale) with CPAP use when compared to the placebo. Reduction in AHI was also reported to be statistically significant, especially amongst those with more severe OSA symptoms.

The common problem associated with CPAP treatment is adherence, which has been shown to be an issue in approximately 50% of patients (Bardwell, Berry, Ancoli-Israel, & Dimsdale, 1999; Popescu et al., 2001; Poulet et al., 2009). Adherence is generally defined as using CPAP for more than four hours nightly for up to 70% of nights (Wells, Freedland, Carney, Duntley, & Stepanski, 2007). Problems with adherence are generally attributed to negative experiences (i.e., side-effects of CPAP) and/or inconvenience, either socially or professionally (i.e., requiring assistance with adjusting the CPAP device or attending to its components) (Ballard, 2008). Promoting greater treatment adherence is an important goal, given the prevalence of OSA in society (Greenham-Conway, 2000; Stepnowsky et al., 2002). However, clinical studies are generally performed over short periods in sleep laboratories, making it difficult to ascertain long-term adherence.

1.6.1 Issues surrounding CPAP treatment for OSA

Over the last 25 years a large body of research has established CPAP use to be the most effective form of treatment for OSA (Popescu et al., 2001). However, as previously stated, adherence to CPAP use has been shown to be a significant problem in up to 50% of patients

(Bardwell et al., 1999; Popescu et al., 2001; Poulet et al. 2009). Therefore, ensuring greater CPAP adherence is an important goal of OSA research (Greenham-Conway, 2000; Stepnowsky, Marler, & Ancoli-Israel, 2002). Although there have been some publications documenting that symptoms of OSA are attenuated after relatively short periods of CPAP use, the general premise is that CPAP is a treatment and not a cure and must be applied nightly for an indeterminate period of time, perhaps lifelong. Being a self-administrated form of treatment, the efficacy of CPAP use is solely dependent on a patient's motivation to use the device as prescribed. This poses the problem of non-adherence to long-term treatment and it is likely that short- and medium-term adherence to CPAP use is in fact far less than believed contributing to OSA morbidity and mortality (Collard et al., 1997).

Whilst CPAP treatment has been demonstrated to produce positive results, variations in adherence from one patient to another are common and appear to be impacted by side-effects as well as the perception of clinical improvement. In a pilot study, Wiese, Boethel, Phillips, Wilson, Peters, and Viggiano (2005) investigated the benefits of video education as a mode to enhance patients' understanding of OSA to improve CPAP adherence. Wiese and colleagues recruited 100 newly diagnosed OSA patients to participate in a randomised two-group design study (treatment group = 51 & control group = 49). Patient inclusion criteria consisted of individuals who were greater than 20 years of age and had an RDI of greater than four. At the initial visit, patients in the treatment group were administered the Epworth Sleepiness Scale (EES) and the Sleep Apnoea Quality of Life Index (SAQLI) and shown a 15-minute educational video involving "two male blue-collar workers discussing their sleep problems, with one describing what sleep apnoea was in common language, what CPAP was, what CPAP felt like on a sensory basis, and how it had helped them" (Wiese et al., p.172). Those in the control group were

interviewed and asked to complete the ESS and SAQLI only. At a follow-up four weeks later, both groups were again administered the ESS and SAQLI. To illustrate the variation in CPAP adherence, 72.9% of the treatment group returned for their follow-up, compared to 48.9% in the control group ($\chi^2 = 5.65$, $p = 0.017$). Wiese et al. concluded that viewing an education video at the initial stage of OSA diagnosis and treatment implementation was in fact beneficial with regard to return at follow-up. However, they found no association between viewing the video and CPAP adherence. While it is not impossible to draw firm conclusions regarding adherence from the data presented in Wiese et al.'s pilot study, it is plausible that non-adherence to CPAP treatment may have been experienced as a result of a number of contributing factors. These could have included existing psychological problems, co-morbid illness(s), receiving poor instructions, having little ongoing support, lack of understanding, and receiving limited follow-up assistance.

In another study, Doherty, Kiely, Lawless, and McNicholas (2003) investigated the impact of CPAP treatment on the quality of life of bed partners in 55 couples where one partner had received a prior diagnosis of OSA and had commenced CPAP treatment. All couples were asked to complete the ESS, Short Form-36 health survey and the Hospital Anxiety and Depression Scale (HADS) initially and again at the six- to eight-week mark as a follow-up. Doherty et al.'s results provided evidence that CPAP treatment assisted both patient and partners, as demonstrated via significant reductions in daytime sleepiness, overall improvement in physical and mental wellbeing, and reductions in anxiety and depression measured at the six- to eight-week mark. The authors also suggested that commonly reported low adherence rate to CPAP use may generally be attributed to treatment side-effects (i.e., dry mouth, conjunctivitis, rhinorrhea, skin irritation, pressure sores, nasal congestion, and epistaxis), mask leaks, difficulty exhaling, aerophagia, chest discomfort, and bed-partner intolerance. The authors advised that

psychological problems may also impact on treatment adherence and commonly have been reported to include lack of motivation, claustrophobia, and anxiety (Doherty et al., 2003). Furthermore, it is important to note that treatment can be financially draining on the patient since CPAP devices and masks are costly, and their purchase therefore places a financial strain on both the individual and health care system since few treatment options exist for OSA patients (Collard et al., 1997).

1.6.2 Determinants of CPAP adherence

Studies have commonly reported on the correlation of daily CPAP use with objective measures of OSA severity, such as the AHI, movement arousal index, and nocturnal oxygen saturation. Patients' positive experiences at the initial titration (i.e., perceived improvement in sleep) have also been reported as factors determining subsequent CPAP usage (Drake et al., 2003). Studies have also reported finding no significant correlation between the MSLT and scores of sleepiness (using the ESS) at initial diagnosis and the subsequent use of CPAP (Pepin et al., 1999; Collard et al., 1997). This may suggest that factors relating to CPAP use are many and therefore further research is needed in this area (Pepin et al., 1999; Collard et al., 1997). It has only been within the last few decades that academics and respiratory physicians have focussed their attention on other possible factors (i.e., psychological) that are likely to contribute to poor adherence to OSA treatment, as opposed to the traditional focus on sleep-related variables. It is hoped that this shift in thinking may further our understanding of factors involved in adherence to CPAP as a long-term treatment, and contribute to the limited evidence base surrounding the efficacy of a variety of intervention strategies (see Table 9).

Table 9

Intervention Studies to Improve CPAP Adherence

Study	Sample	Intervention	CPAP adherence outcome Yes/No
Wang et al. (2012)	152	Patient education involving three nights in lab support, four-hour group education session, brochures describing the need for and benefits of CPAP, a CD containing a 20-minute video, a 24-hour treatment consultation telephone line, and progressive muscle relaxation	Yes at 12 weeks
Olsen et al. (2012)	106	Motivational interviewing	Yes at three and 12 months
Roecklein et al. (2010)	30	Personalised feedback including AHI, RDI, average and lowest level of blood oxygen, and self-reported daytime sleepiness along with brochures	No at two weeks and three months
Sparrow et al. (2010)	250	Automated telemedicine intervention (telephone-linked communications for CPAP–telephone support offering intervention based on	Yes at six and 12 months

		motivational interviewing principles)	
Smith et al. (2008)	97	20-minute audiotape with spoken directions for nightly CPAP preparation and use, educational written literature, reminder placards, and four-week diary record of CPAP use	Yes at one month; No at three and six months
Stepnowsky et al. (2007)	45	Telephone monitoring	No at six months
Richards et al. (2007)	100	Cognitive behavioral therapy	Yes at seven and 28 days
Aloia et al. (2007)	142	Motivational enhancement therapy and education interventions	Yes at one month
Meurice et al. (2007)	112	Three educational strategies	No
Golay et al. (2006)	35	Educational program/workshop involving individual treatment goal identification, treatment purpose discussion, and spouse roundtable discussion	No at three months
Smith et al. (2006)	19	Telephone-delivered intervention	Yes at 12 weeks
Wises et al. (2005)	100	Educational video	No
DeMolles et al. (2004)	30	Telephone-linked communications for CPAP use	No at two months

Aloia et al. (2001)	12	Cognitive behavioral intervention	Yes
Hui et al. (2000)	108	Video education, telephone support, and at week one and two interaction with physician/doctor	No at one month and three months
Chervin et al. (1997)	33	Positive reinforcement	No at two months
Fletcher et al. (1991)	10	Positive reinforcement	No at three months
Hoy et al. (1999)	80	Intensive support involving education at home, three nights in laboratory CPAP trial, and home visits	Yes at six months

1.7 Adherence

1.7.1 Terminology—compliance or adherence

As part of a global initiative launched in 2001, the World Health Organization (WHO) produced a report aimed at summarising existing information on compliance to promote discussion for the foundation of evidence-based policy making (WHO, 2003). In summary, the report estimated that half of the patients that engaged in prescribed treatment regimens did not follow them as directed (WHO, 2003). As well as exploring the reasons why patients did not comply, the report focused on the meaning of the term “non-compliance” suggesting that not following the directions for treatment may be attributed to “irrational behaviour or wilfully ignoring instructions”. Given the many reasons for “non-compliance”, present-day literature and health care professionals more commonly make use of the term “adherence” to a regimen rather than “compliance”.

According to Tilson (2004) and Ngoh (2003), the term “adherence” better reflected the diverse rationale for patients not following prescribed treatment regimes. Research by Bell, Airaksinen, Lyles, Chen, and Aslani (2007) and the United States National Institutes of Health (U.S. NIH) Office of Behavior and Social Sciences Research (2008), suggests that “concordance” may be a better term to describe the collaborative efforts between the patient and physician/doctor to promote greater adherence to treatment regimes. These authors suggest that the term differentiates from that of adherence pertaining to a physician/doctor-only prescribed treatment regime. Marinker and Shaw (2003) suggest that involving the patient in the treatment decision-making process fosters greater responsibility in the patient for monitoring and reporting.

The researchers at Bandolier (2004) reported that adherence to treatment regime, within this context, is improved by:

- only recommending treatments that are effective in circumstances when they are required;
- selecting treatments with lower levels of side-effects or fewer concerns for long-term use;
- prescribing the minimum number of different medications (e.g., prescribing a single antibiotic that addresses two concurrent infections);
- simplifying dosage regimen by selecting a different drug or using a sustained-release preparation that needs fewer doses during the day;
- discussing possible side-effects, and whether it is important to continue medication regardless of those effects;
- providing advice on minimising or coping with side-effects (e.g., whether to take a particular drug on an empty stomach or with food); and
- developing trust so that patients do not fear embarrassment or anger if they are unable to take a particular drug, allowing the doctor to try a better-tolerated alternative.

While the preferred terminology remains a matter of ongoing debate in some research circles (Osterberg & Blaschke, 2005; Aronson, 2007), the term “adherence” was thought to be the most appropriate for this study as it falls in line with respective clinical literature and is the preferred term used by the WHO (2003), The American Pharmacists Association (2004), and the U.S. NIH Adherence Research Network (2010).

In the most general sense, adherence pertains to the act or process of complying with a desire, demand, proposal, or regime and is often used interchangeably with the terms compliance, concordance, or capacitance (Ngoh, 2003). In the clinical arena, adherence generally describes the extent to which a patient correctly follows medical advice (Ngoh, 2003; Tilson, 2004). Traditionally, research in the area of adherence has focused its attentions on medication or drug adherence, but research in this field has extended its application to the use of appliances such as compression stockings, chronic wound care, self-directed physiotherapy exercises, attending counselling or other courses of therapy including that of CPAP use.

1.7.2 Adherence research

Historically, research surrounding adherence reported on prescription fill rates and commonly showed that while a visit to a physician/doctor may have resulted in a patient leaving with a prescription not all patients would follow through with obtaining medication from the pharmacy. In recent research, Fischer et al. (2010) reported that in the U.S., 20% to 30% of prescriptions were never filled by a pharmacist. The American Pharmacist Association (APA) (2004) and Shah et al. (2009) suggested that the probable reasons why patients fail to have their prescriptions filled included the cost of medication, questioning the need for medication, and preference for self-care measures rather than medication. These authors attributed cost as a major barrier that impacted on prescription drug adherence and reported that in a U.S. study conducted in 2001, 22% of 1,010 adults sampled choose not to fill prescriptions due to price constraints.

Course completion is noted as another commonly reported factor that impacts on adherence. Ngoh (2003) and the APA (2004) reported that once a patient committed to filling a prescription, they seldom followed treatment regimens as directed and rarely completed the prescribed course of treatment. Ngoh, the APA, and Elliott and Marriott (2009) attributed this to poor “health literacy”, which is defined as a misunderstanding of cost and treatment regime that together pose major barriers to accurately fulfilling treatment. According to the WHO report, this contributed to an estimated 50% of people who did not complete long-term therapy for chronic illnesses as prescribed, which potentially placed them at risk, prolonged recovery, and may have contributed to co-morbidities and potential mortality (WHO, 2003).

In order to assist patients to accurately complete prescribed treatment regimes, a variety of packaging approaches have been implemented. These have predominantly focused on promoting ease of remembering dosage as well as specific labelling aimed at increasing patient understanding of directions (Shrank, Avorn, Rolon, & Shekelle, 2007; Mahtani, Heneghan, Glasziou, & Perera, 2011).

WHO (2003) widely reports that failure to accurately complete treatment regimens as prescribed has significant negative health impacts. Examples given in this report of the rate and consequences of non-adherence for more commonly researched medical disorders include:

- diabetes non-adherence (98% in the U.S.) as the principal cause of complications related to diabetes including nerve damage and kidney failure;

- hypertension non-adherence (93% in the U.S., 70% in the U.K.) as the main cause of uncontrolled hypertension-associated heart attack and stroke; and
- asthma non-adherence (28-70% worldwide) increasing the risk of severe asthma attacks requiring hospitalisation

Given that only an estimated 50% of patients suffering from chronic diseases in developed countries follow treatment regimes in part or full, further research in this area is warranted. Treatment non-adherence has the potential to have many adverse effects including complications in chronic diseases, formation of resistant infections, and untreated psychological and psychiatric illnesses that may potentially impact at an individual- and at the community-level. In accordance with the research conducted by WHO (2003), the researchers at Bandolier (2004) reported that adherence rates recorded from monitored clinical studies often differed to those reported in real-life situations, with higher adherence rates observed in clinical trials. This is illustrated by the researchers at Bandolier in a review exploring the adherence rate amongst participants prescribed with Statins (cholesterol medication). The study reported a 97% adherence rate at the beginning of treatment, when monitored, and concluded that approximately 50% of patients were adherent after six months. Interestingly, these figures and those reported in the WHO report are in line with the adherence rate for OSA patients undertaking CPAP treatment as an ongoing treatment regime.

1.7.3 CPAP adherence rates

In order to determine which factors contribute to CPAP adherence it is necessary to define “how much CPAP use equates to adherence” (Weaver & Sawyer, 2010, p.247). Stradling and Davies (2000) conducted a one-month, randomised, parallel, controlled study to assess the benefits of CPAP use and explored the optimal usage rates in 101 men who reported ongoing sleepiness (ESS \geq 10). Fifty percent of the patients received CPAP at a sub-therapeutic level and the remaining 50% at therapeutic levels. The researchers concluded that five hours of CPAP use at the therapeutic level resulted in overall improvements in daytime sleepiness as measured by the ESS, Maintenance of Wakefulness Test (MWT), and the Energy/Vitality dimension of the SF-36 (health status questionnaire) after one month. Stradling and Davies concluded CPAP use was in fact effective in relieving symptoms of daytime sleepiness and determined that five hours a night of effective CPAP use was necessary to restore sleepiness to normal levels.

Other researchers have reported positive outcome measures at much lower rates of CPAP use. Engleman, Kingshott, Wraith, Mackay, Deary, and Douglas (1999) conducted a randomised, placebo-controlled study to explore levels at which patients with sleep apnoea would benefit from CPAP use. Thirty four patients (13 women and 21 men) diagnosed with mild sleep apnoea (AHI of five to 15), with a the mean age of 44 years and a mean BMI of 30, spent one month on CPAP treatment and one month on an oral placebo with randomisation of treatment orders. In contrast to receiving the placebo, patients reported significant improvement in daytime sleepiness (measured using the ESS) and overall sleep apnoea symptoms after one month of CPAP use. Engleman et al. established that effective CPAP use was estimated at an average of

2.8 hours per night (self-reported average = 4.5hr/per night), suggesting the overall rapid and short-term benefits of CPAP treatment in relieving symptoms of sleep apnoea. These findings supported an earlier pilot study conducted by Engleman et al. (1998). In the initial study, Engleman et al. recruited 23 patients (two women and 21 men) with an average BMI of 30 and an average AHI of 43 (however severity varied greatly) to participate in a randomly assigned, single-blind, placebo-controlled, cross-over study. Baseline data were collected in terms of daytime sleepiness (measured using the ESS), OSA symptoms, cognitive performance, and psychological wellbeing before 13 patients were randomised and assigned to commence with the placebo and 10 patients assigned to the CPAP treatment group. Following four weeks in the placebo or treatment group, data were again collected in terms of daytime sleepiness, OSA symptoms, cognitive performance and psychological wellbeing. The results demonstrated positive significant improvements in terms of sleep onset latency, energetic arousal, and some cognitive performance areas (not the entire suite of cognitive measures). No significant results were reported in terms of psychological wellbeing (anxiety, depression, and general health). Engleman et al. reported that effective CPAP use in cases that displayed a statistically significant positive change, on average used CPAP for 2.8 hours per night. Given there may have been some limitations to the study in terms of sample size and variability in terms of OSA severity, there are few randomised controlled trials that have investigated the impact of CPAP use on daytime functioning. Engleman and colleagues were able to objectively and reliably provide evidence to suggest significant improvements in OSA symptoms and daytime sleepiness after four weeks of CPAP use at 2.8 hours per night.

In an Australian study, Barnes et al. (2002) also conducted a randomised, controlled, cross-over study with 42 patients diagnosed with mild to severe OSA (AHI of five to 30). Patients were predominately middle-aged, overweight men (seven women and 35 men). Prior to assignment to groups, patients were administered a battery of neuro-behavioural assessments to measure memory, cognition, pre-morbid performance IQ, vigilance, and divided attention. Mood symptoms, daytime sleepiness, and quality of life were also measured. Out of the 42 patients that consented to participate in the study 28 completed both the treatment and placebo arms of the study. Outcome measures were again given after eight weeks of CPAP use and treatment with the placebo. Barnes et al. established effective CPAP use to be a mean of 3.53 hours per night, as determined by improved self-reported symptoms of OSA. However, no improvements in objective daytime sleepiness or mood were noted, suggesting no treatment effect between the CPAP use group and the placebo group. Nevertheless, the placebo group reported, statically significant results with regard to subjective daytime sleepiness, memory, cognition, and elements relating to quality of life. While Barnes et al.'s research contributed to the body of literature utilising randomised controlled cross-over designed studies, it is likely that it may have been impacted by the placebo effect or "honeymoon period". While little is known regarding the possible impacts of the "honeymoon period" and having differing measurements to define adherence, Barnes et al.'s results provided further support for CPAP use even at a low usage rates.

1.8 Psychological Determinants of CPAP Adherence

Research investigating CPAP adherence has paid little attention to the broad range of person-centred factors that are likely to impact on CPAP treatment uptake. Historically, demographic and sleep-related variables such as age, gender, degree of daytime sleepiness, greater subjective sleepiness, poorer health, high BMI, and higher AHI, have received much of the attention (Waldhorn et al., 1990; Rolfe, Olson, & Saunders, 1991; Kribbs et al., 1993; Edinger, et al., 1994; McArdle et al., 1999; Janson, Noges, Svedberg-Randt, & Lindberg, 2000; Budhiraja et al., 2007; Joo & Herdegen, 2007). However, results have been mixed, inconclusive, or confounded by other demographic variables (Ohayon, Caulet, Philip, Guilleminault, & Priest, 1997; Wiese et al., 2005; Krishnan & Collop, 2006; Amodio, Master, Yee, & Taylor, 2008; Casale, et al. 2008). It has only been within the last three decades that factors such as mood, personality, and cognitive functioning have been investigated in relation to CPAP adherence. Many studies have concluded that further research into person-centred factors is necessary to build on existing CPAP adherence research (Kribbs et al., 1993; Engleman et al., 1994; Meurice et al., 1994; Reeves-Hoche, et al., 1994; Engleman et al. 1996; Massie et al. 1999; McArdle et al., 1999; Rosenthal et al., 2000; Peppard et al., 2000).

To date, mood has received much of the attention within this domain with mixed results reported. Guilleminault et al. (1977) paved the way for research to explore the psychological factors associated with OSA. Their study consisted of 25 men aged between 25 and 65 years old ($M = 44.3$): 20% were of healthy weight and the remaining 80% were classified as being overweight. All 25 men were referred to the study due to excessive daytime sleepiness, with 14

of the patients having a prior diagnosis related to a sleep disorder (four of them were labelled with “Pickwickian syndrome” and 10 were narcoleptics). After extensive measuring procedures (including interviews with partners, parents and siblings, maintaining sleep diaries, physical examinations, administration of the Minnesota Multiphasic Personality Inventory (MMPI), blood and urine tests, and polygraphic monitoring), Guilleminault and colleagues reported that 24% of patients had seen a psychiatrist for anxiety and/or depression and 28% had high scores on the depression facet of the MMPI. While the purpose of Guilleminault and colleague’s investigation was to identify the clinical symptomatology in patients with sleep apnoea, in order to support an early clinical diagnosis so that less invasive treatments are made available, the authors also provided initial evidence to suggest a relationship between sleep disorders and negative psychological states.

1.8.1 Mood

Anxiety and depression have received much attention in treatment adherence research with regard to chronic illnesses such as cancer, end-stage renal disease, rheumatoid arthritis, and coronary disease. It is widely reported in chronic illness literature that depressed individuals report more problematic symptoms, independent of severity (Dwight, Kowdley, Russo, Ciechanowski, Larson, & Katon, 2000; Katon, Sullivan & Walker 2001; Katon & Ciechanowski, 2002).

Estimates of anxiety and depression in patients undergoing differing medical treatments vary according to measurement criteria. DiMatteo, Lepper, and Croghan (2000) conducted a

meta-analysis of the effects of anxiety and depression on treatment adherence by reviewing articles catalogued on MEDLINE and PsychLit from 1968 through to 1998. They reviewed 12 articles in total that met their inclusion criteria (involvement in a medical regime and participants were measured for adherence, anxiety and/or depression). Findings from their meta-analysis demonstrated a significant association between depression and non-adherence to medical regimes. DiMatteo et al. reported that depression was present in up to 25% of patients and that these individuals were three times more likely to not adhere to treatment than non-depressed patients. The authors concluded that this figure is likely to be increased by the presence of significant co-morbidities and/or increased disease severity. Such evidence reveals the significant role that depression plays in attenuating both acute and chronic medical conditions and the importance of screening for psychological disorders.

Depression is one of the most common disorders seen in medical practice. Thus, researchers have begun to investigate the role that mood plays in OSA patients with regards to CPAP adherence. However, while there appears to be an abundance of available literature in this area, the findings appear to be highly variable and limited only to identifying a relationship between OSA and depression and/or identification of improved mood following CPAP use rather than the impact that depression has on CPAP adherence (see Table 10).

Research surrounding depression and CPAP adherence has produced mixed findings, with early studies following on from Guilleminault et al. conducted by Reynolds, Kupfer, McEachran, Taska, Sewitch, & Coble (1984) and Millman, Fogel, McNamara, and Carlisle (1989) reporting an increased likelihood of depressive symptoms in untreated OSA patients in

comparison to the general population. In particular, Reynolds et al. provided evidence to suggest that depressive symptoms decreased following CPAP treatment, a finding that was later supported by the research conducted by Yamamoto, Akashiba, Kosaka, Ito, and Horie (2000) and Means et al. (2003).

Tables 10

Summary of the Findings from the Literature on Mood and Sleep Apnoea

Authors	Year	Subjects	Findings
Diamanti (2013)	CES-D	35 men 6 women	Overall, CPAP therapy lessened depressive symptoms.
Lee (2012)	CED-S POMS BSI	47 men 9 women	CPAP treatment improved sleep apnoea symptoms over a three-week period but did not show a specific therapeutic effect on mood.
Asghari (2012)	BDI BAI	497 men 188 women	Greater than 50% of patients had some degree of depression and anxiety with females scoring higher for depression/anxiety than males. No significant results were reported.
Soreca (2012)	HAM-D MOOD-SR	30 men 42 women	Sleep apnoea was prevalent in the sample of bipolar participants with over 50% of the sample having severe sleep apnoea.
Jackson (2011)	POMS BDI	31 men 14 women	Sleep apnoea group reported significant higher levels of depression than the control group, although depression in this cohort was minimal. Fatigue was reported as the primary factor over depression.
El-Sherbini (2011)	HDRS	24 men 13 women	Depression was prevalent in individuals with sleep apnoea, and was more severe in females. Depression was highly correlated with sleep apnoea and the severity

			of depression was reduced with the use of CPAP.
Macey (2010)	BDI	37 men	No reported links between sleep apnoea severity and depression/anxiety.
	BAI	12 women	
Peppard (2009)	SDS or antidepressants	788 men 620 women	1.8 odds ratio (OR) of developing depression within a four year interval as sleep apnoea develops or worsens
Şahbaz (2008)	65 men 15 women	HADS	12.5% of patients were previously treated for anxiety/depression. The frequency of anxiety/depression was significant in patients with excessive daytime sleepiness (EDS)
McCall (2006)	2006	92 men 26 women	Depression was more common in women with sleep apnoea than in men.
Aloia (2005)	BDI	61 men 32 women	Sleep apnoea severity and obesity contributed differentially to symptoms of depression. Depression manifested differently in men than in women.
Farney (2004)	Prescription for antidepressants	102614 men 110358 women	The likelihood of having a diagnosis of sleep apnoea increased when antidepressant medications had been prescribed—OR = 18.30 (95% CI, 10.69 to 25.66) amongst 20- to 39- years old women.
Sforza (2002)	2002	44 OSA patients, 16	Higher depression scores showed an association with reduced daytime alertness

		snorers,	
Smith (2002)	Physician diagnosis	599 men 174 women	Depression was more commonly diagnosed in the sleep apnoea patients than in the control Subjects—OR = 1.4 (95% CI, 1.0 to 1.9).
Sanchez (2001)	BDI STAI	47 men 4 women	Both depression and anxiety reduce after one and three months of CPAP use
Bardwell (1999)	1999	61 men 11 women	For subjects with sleep apnoea, depression correlated positively with deep sleep, REM, and/or hypoxemia. For subjects without sleep apnoea, vigour correlated positively with sleep quantity and negatively with hypoxemia. When age, body mass, and hypertension were controlled, results changed little for subjects without sleep apnoea; for subjects with sleep apnoea, depression no longer correlated with sleep measures.
Aikens (1999)	MMPI	155 men 23 women	Sleep apnoea patients who had core depressive symptoms without significant psychological symptoms in other areas tended to have less severe sleep apnoea, whereas those with a diverse set of other psychological symptoms tended to have greater AHI and lower O ₂ saturation.
Pillar (1998)	SCL-90	1,977 men 294 women	Neither the existence nor the severity of sleep apnoea symptoms (SAS) was associated with depression or anxiety. Women had higher anxiety and depression scores, independent of other factors, than men. Women with severe

			sleep apnoea had higher depression scores than women with mild sleep apnoea.
Enright (1996)	CES-D	2239 men 2962 women	Association between depression and observed apnoeas found in women but not in men.
Engelman (1994)	HADS	26men 6 women	Four weeks of CPAP treatment showed improvement in anxiety/depression on the HADS.
Edinger (1994)	MMPI	38 men	Those who adhered with six months of CPAP treatment eventually displayed reduced depression.
Borak (1993/1994)	BDI TMAS	20 men	Apnoeics initially had higher anxiety and depression and this was correlated with duration of illness. CPAP improved anxiety/depression.
Gall (1993)	SCL-90 POMS	20 men	No statistical significance difference was reported for depression between apnoeics vs. controls.
Ramos Platón (1992)	MMPI	23 apnoeic	Apnoeics' personality patterns were predominantly of an anxiety type. Depression, schizophrenia, and hypochondrias were the highest scales. Following one year of NCPAP treatment, there was a significant and progressive reduction for depression.
Cheshire (1992)	HADS	25 males, 4 females	Anxiety and depression contributed to cognitive impairment in sleep apnoea patients with 10 of 29 apnoeics having anxiety and seven of 29 apnoeics having

			depression.
Millman (1989)	SDS	55 apnoeics	25 of 55 patients had clinical depression, CPAP reduced depression.
Derderian (1988)	POMS	7 men	CPAP improved depression total mood disturbance, and fatigue compared with controls.
Klonoff (1987)	MMPI	10 men	When comparing sleep apnoea patients with patients undergoing coronary artery bypass surgery no difference in depression was record between the 2 groups.
Kales (1985)	MMPI	43 men 7 female	Fifty percent of the sample reported depression and 76% had suspected or mild to severe deficits in terms of thinking, perception, memory, communication, or the ability to learn new information, resulting in a greater potential for being distractible, confused, and irritable.
Reynolds (1984)	SADS	25 men	Of 25 apnoeics, three had a history of major depression, two had chronic depression, one had apnoea criteria cyclothymia, four had alcohol abuse, and 10 met the criteria for a psychiatric disorder; depression correlated with REM, REM latency, and use of HTN medications.
Sachs (1983)	MMPI	15 men 5 women	Sleep apnoea patients had higher levels of anxiety than the control group
Beutler (1981)	MMPI POMS	20 men	Men reported high depression only on the MMPI, which was significantly correlated with sleep apnoea.

Guilleminault (1977)	MMPI	25 men	Twenty-four percent had seen a psychiatrist for anxiety or depression; 28% showed elevated depression.
-------------------------	------	--------	--

BDI = Beck Depression Inventory; CES-D = Center for Epidemiologic Studies Depression Scale; HADS = Hospital Anxiety and Depression scale; SCL-90 = Symptom Checklist – 90; TMAS = Taylor Manifest Anxiety Scale; POMS = Profile of Mood States; FPI = Freiburger Personality Inventory; HDRS = The Hamilton Depression Rating Scale; STAI = The State-Trait Anxiety Inventory; SADS = Schedule for Affective Disorders and Schizophrenia; SDS = Zung Self-Rating Depression Scale; HAM-D = Hamilton Rating Scale for Depression; MOOD-SR = MOOD questionnaire; and BSI = Brief Symptoms Inventory.

Yamamoto et al.'s (2000) study investigated the long-term effects of CPAP use on the rate of traffic car accidents, excessive daytime sleepiness (as measured by the ESS) and mood in 75 male patients with severe OSA as determined via PSG. Using a self-report questionnaire to measure depression (the Self-related Depression Scale–SDS), Yamamoto and colleagues concluded that after two years of CPAP use, depressive symptoms decreased in 46 patients. Of the 46 patients, Yamamoto et al. identified that 26 patients had high scores on the SDS for depression prior to CPAP use. Specifically, Yamamoto and colleagues reported that 13 of the 26 patients reported a significant reduction in depressive symptom at post-testing, concluding that “this effect may contribute to an improvement of patients' quality of life because most patients with severe OSA compromise their activities in social life” (p. 89). Statistically significant reductions in ESS scores were also reported, which suggested overall improvement in daytime sleepiness with CPAP use. Similar results in other studies, especially those of Engleman and colleagues, have consistently reported significant reductions in negative mood, particularly anxiety and depression, as early as one month on CPAP treatment (Engleman et al, 1994; Engleman et al., 1997).

However, the findings of Yamamoto et al. (2000) were not universal across OSA research, raising the possibility that the association between depression and OSA was coincidental or even the results of uncontrolled factors such as age, BMI, fatigue, and other co-morbidities. Borak, Cieslicki, Koziej, Matuszewski, and Zielinski (1996) investigated the impact of CPAP treatment on emotional state in their study of 20 obese men with moderate to severe sleep apnoea. Prior to CPAP use, psychometric testing using the Taylor's Manifest Anxiety Scale, Beck Depression Scale, and Tylka's Psychological Evaluation Scale of the Effectiveness of

Rehabilitation (a measure of mental stress) reported that a majority of the patients reported signs of anxiety (14 patients), depression (11 patients) and stress (15 patients). After three and 12 month of CPAP use, the same psychometric measures were re-administrated and Borak and colleagues concluded no significant improvement in anxiety, depression, and/or mental stress.

Similarly, in a randomised controlled study, Engleman et al. (1998) measured psychological wellbeing using the HADS, General Health Questionnaire, and the Nottingham Health Profile in 23 (two women and 21 men) obese (mean BMI = 30) patients. All patients were exposed to four weeks in the treatment group and four weeks in a placebo control group, assigned in a random order. They concluded that there was no statistically significant improvement in the psychological wellbeing of patients when treated with CPAP or the placebo. Nevertheless, Engleman et al.'s study contributed to the limited number of randomised controlled group studies that currently exist within the OSA domain. They did provided valuable clinical and statistically significant findings to show that daytime sleepiness improved (as measured by the ESS) improved with CPAP treatment.

Interestingly, the results of a large long-term, prospective controlled study conducted by Munoz, Mayoralas, Barbe, Pericas, and Agusti (2000) also reported no statistically significant short-term or long-term improvements in anxiety and depression following 12 months of CPAP treatment. Munoz et al. recruited 80 patients with mild to severe sleep apnoea ($M_{AHI} = 60$) and 80 healthy control subjects matched for sex to participate in their control group study. It was noted that patients in the treatment group were marginally, yet significantly, older and more obese than

those in the control group. Both the treatment and control groups were administered the Beck Anxiety and Depression scales post-CPAP use, and these scales showed that patients in the treatment group were more anxious and depressed than those in the control group. At the three-month mark, only patients in the treatment group were again assessed using the Beck Anxiety and Depression scales. While Munoz and colleagues reported significant improvement in daytime sleepiness (as measured by the ESS), suggesting that short-term CPAP use improved sleepiness, no significant changes in anxiety and/or depression were noted. Identical results were reported after 12 months of CPAP use. While these findings are generally in line with the previously reported studies of Boreak et al. (1996) and Engleman et al. (1998) they were somewhat surprising given the large sample size, rigorous study design, and acceptable CPAP usage (i.e., 5.8 hours per night). Munoz et al. concluded that it was plausible that CPAP use itself was likely to be anxiety provoking and perhaps contributed to depressive symptoms given that CPAP treatment is a symptomatic and not a curative treatment option.

While a large amount of the research on psychopathology in OSA patients has predominantly focused on anxiety and depression, in line with the general adherence research on other chronic medical illnesses and conditions, the high prevalence of other psychological symptoms is also starting to be explored. One of the first to begin to investigate a broader range of mood states using the Profile of Mood States (POMS) was Derderian, Bridenbaugh, and Rajagopal (1988). In a controlled study of seven middle-aged men ($M = 59.3$ years) with severe OSA (mean AHI = 40.7), Derderian et al. administered the POMS before and after two months of CPAP use. A control group was also used for comparison. Derderian and colleagues reported that

two of the six mood states, depression/dejection and fatigue/inertia, had improved significantly following CPAP use. Overall Total Mood Disturbance (TMD) had also decreased following CPAP treatment, which suggested improvement in overall general mood.

Similar results were also reported in an early study by Kribbs et al. (1993), who investigated the impact of one night without CPAP use in 15 obese patients (one = female & 14 = males) aged between 36 and 63 who were diagnosed with moderate to severe OSA (mean RDI = 56.6). Following diagnosis via an overnight sleep study and education surrounding CPAP use, patients in Kribbs et al.'s study were prescribed one month's CPAP treatment before returning to the sleep centre for a follow-up. At that stage patients were required to spend the next night at the sleep centre. During the first night on return, patients were asked to complete a battery of assessments that involved the completion of the POMS and they were requested to use their CPAP device as prescribed. On the second night the POMS was again administered, but CPAP was withdrawn. Kribbs and colleagues reported that mood as measured by the POMS had shown an overall improvement when CPAP was used, but quickly declined when patients were off CPAP. The authors also reported some amazement at these results in that after one month of prescribed CPAP use, missing one night would have such an impact on mood, placing patients at risk. Given the decline of mood when CPAP use was removed, Kribbs et al. believed that the benefits of CPAP is likely to be much more than just abolishment of respiratory events.

While the results from the Derderian et al. (1988) and Kribbs et al. (1993) showed that CPAP treatment led to improved mood, an interesting study conducted

by Bardwell, Moore, Ancoli-Israel, and Dimsdale (2003) concluded that depression rather than OSA accounted for the fatigue that was commonly reported by OSA patients. Bardwell et al. administered the POMS and the Center for Epidemiological Studies Depression Scale (CES-D Scale) to 60 OSA patients (9 women and 51 men) with moderate to severe OSA (mean RDI = 48.8). The results from correlational analysis suggested that depression was statistically significantly related to fatigue and not significantly related to OSA severity. Bardwell and colleagues concluded that it is probable that depressive symptoms directly contributed to fatigue rather than the respiratory disturbances experienced by patients with OSA. The findings of Bardwell et al. demonstrated the impact mood plays in those with OSA, especially in terms of identifying factors that contribute to fatigue.

Other studies that have used the POMS to measure mood have adopted more sophisticated methodologies to explore the impact of mood in OSA patients before and after a period of time on CPAP treatment. In particular, Yu, Ancoli-Israel, and Dimsdale (1999) hypothesised that the positive findings of previous studies on mood and CPAP treatment may have failed to consider the placebo effect associated with CPAP use. During the initial weeks of CPAP use, it is plausible that patients are more likely to initially adhere and/or perceive improvement in OSA symptoms. Yu et al. administered the POMS to nine women and 25 men, aged 32 to 60 years old, who after an overnight PSG were randomised to either a treatment or placebo group. A mean AHI of 20 placed patients in the moderate to severe range for sleep apnoea (baseline treatment group AHI = 20.7; baseline placebo group AHI = 17.4). After seven days the groups were re-administered the POMS and asked to undergo a second PSG. The results showed positive improvements in AHI and oxygen saturations after

one week of CPAP use in the treatment group only, but mood was reported to improve significantly in both the CPAP treatment group and the placebo group. This suggested that improvements may have been due to a placebo effect or short-term CPAP use.

The results from Yu et al. (1999) were later supported by Stepnowsky, Mao, Bardwell, Lored, and Dimsdale (2012) in a similar study. Stepnowsky et al. were interested in assessing the baseline psychological factors in relation to CPAP use in a double-blind randomised controlled study. Twenty-five mildly obese CPAP naïve patients (three women and 22 men) aged 33 to 60 years old were randomly assigned to a treatment group or a placebo group that received sub-therapeutic CPAP pressure. A mean baseline AHI of 45.2 placed the majority of patients within the severe range with regards to OSA. Patients' mood was assessed using the POMS and they were considered to be mildly to moderately emotionally distressed (mean TMD = 37.7). Patients were split into two groups: those in Study One returned for a follow-up PSG and re-administered the POMS after one week of CPAP use and those in Study Two returned for a follow-up PSG and re-administered the POMS after 2 weeks. Results from Stepnowsky and colleagues study showed that AHI (mean AHI difference from 45.2 to 38.9) and emotional distress improved in the placebo group in both Study One and Two following PSG at weeks one and two respectively. Stepnowsky et al. reported that those that were classified as highly distressed showed the most significant improvement with CPAP use over time. The findings suggested that psychological factors may play an important role regarding CPAP use and adherence and also that the placebo effect is something that also needs to be considered, especially within the short-term "honeymoon period".

Kjelsberg, Ruud, and Stavem (2005) also assessed the predictive association of mood in a sample of 178 diagnosed OSA patients in terms of its impact on adherence to CPAP use. The samples consisted of 43 women and 134 men with a mean age of 55.5, a mean AHI of 29, and a mean BMI of 31.1 that placed most patients in the obese range. Patients were administered the HADS to assess anxiety and depression and the ESS to assess for daytime sleepiness. The authors reported that patients who scored high for depressive symptoms also self-reported high levels of daytime sleepiness. Depression was also determined to be a statistically significant independent predictor of self-reported sleep quality. In addition, patients who were identified with anxiety and/or depressive symptoms were found to be non-adherent to CPAP use measured by fitted CPAP meters. Kjelsberg et al.'s results showed that OSA patients who presented with symptoms associated with mood disorders were more likely to report complaints regarding sleepiness than non-anxious and non-depressed individuals. This study was one of the first to report a significant negative relationship between mood and CPAP adherence, as previous studies found no association between CPAP use and mood (Pillar & Lavie, 1998; Bliwise et al., 1986). Kjelsberg and colleagues study suggested a link between patients' psychological symptoms and a lower level of adherence to CPAP use. The authors concluded that mood is likely to play an important role in treatment adherence as it was likely to impact on an individuals' tolerance towards treatment. Kjelsberg et al. recommended that "treatment of their mental symptoms may be a possible therapeutic option in patients with OSA and symptoms of anxiety or depression that may contribute to improved compliance with CPAP therapy and break the vicious circle" (p. 343).

Similarly, Greenham-Conway (2000) examined the relationship between CPAP adherence and improved mood and psychological functioning in patients diagnosed with sleep apnoea-hypopnea syndrome. Greenham-Conway employed a comparative study design using pre- and post-treatment data to evaluate effectiveness in 37 patients engaging in CPAP use for at least 30 days. The POMS and the ESS were given prior to and following 30 days of CPAP use. A follow-up, self-evaluation questionnaire was also given post-treatment. Greenham-Conway reported a significant improvement post-treatment in regards to mood. Specifically, a significant decrease in anger and hostility, a decrease in fatigue and inertia, and an increase in vigour and activity was reported following CPAP use. Adherence was defined as four hours or more of CPAP use per night. Overall, the patients used CPAP for an average of 4.2 hours each night. The authors reported no difference between groups in pre- and post-scores for sleepiness at four or five hours of adherence. Only when CPAP adherence was defined as six or more hours of nightly use, were there any significant differences between groups in terms of sleepiness, mood, and overall psychological functioning. Adherent and non-adherent patients in Greenham-Conway's study improved equally, regardless of how little or how much they engaged in CPAP use. The findings by Kjelsberg et al. (2005) and Greenham-Conway suggest that any improvements in daytime sleepiness attributed to CPAP use may not be as salient in the presence of mental health or psychological issues such as a mood disorder.

On the whole, the aforementioned studies generally suggest that individuals who experience and report negative symptomatology associated with mood are less likely to adhere to treatment regimes. A common finding reported in these studies was that depressed individuals demonstrated poorer adherence to CPAP treatment

(Stammnitz et al., 2004; Hassaballa, Tulaimat, Herdegen, & Mokhlesi, 2005; Casale et al., 2008).

1.8.2 Personality

Research surrounding the impact of personality traits on CPAP adherence is still in its infancy, but it is well established within the realms of adherence research for other chronic medical illnesses and conditions such as HIV/AIDS, diabetes, depression, hypertension, cancer, and coronary heart disease (Courneya, Friedenreich, Sela, Quinney, & Rhodes, 2002; Brickman, Yount, Blaney, Rothberg, & De-Nour, 1996; Penedo et al., 2003; Cohen, Ross, Bagby, Farvolden, & Kennedy 2004). One study investigating the antecedents of adherence to medical recommendations found that patients with personality types that relied upon avoidant coping strategies had poorer adherence to set treatment regimes (Sherbourne, Meredith, Rogers, & Ware, 1992). Sherbourne et al. specifically investigated the impact that social support and stressful life events had on the long-term physical functioning and emotional wellbeing of ill people. An initial sample of 20,223 patients that participated in a Medical Outcomes Study was utilised for this study. A subset of the initial sample was selected based on severity of illness (e.g., diagnosed depression as opposed to depressive symptoms only, diagnosed long-term diabetes, medicated hypertensives). The design involved a two-year longitudinal study where patients were required to complete the self-administered Form B Patient Assessment Questionnaire (PAQ) and participate in a two year follow-up assessment. The resultant participant count was 1,402 chronically ill patients aged between 19 and 98 years of age. Sherbourne and colleagues' study found that it was probable that "avoiders" were less attentive and

responsive to information about threatening events, such as health problems. They also reported that those who were more extroverted and reported having solid social support networks maintained healthier lifestyles and showed decreased impact of illness over the longer term as opposed to those who had depressive personality styles and were more introverted. Similar findings were supported in later research regarding CPAP treatment and use (Stradling, Hardinge, & Smith, 2004). Generally, such studies have concluded that personality is indeed an independent predictor of long-term mortality, adding further support for the need for personality research in regard to OSA and CPAP adherence (Moran et al., 2011). Personality and its impact on CPAP adherence have received little attention in the growing body of literature surrounding psychological predictors to non-adherence. Available literature in this area show that personality traits such as depression and hypochondriasis, as measured by the MMPI, may be valid predictors of CPAP adherence (Edinger, et al., 1994).

Perhaps the most well-known study that has investigated personality as a correlate to CPAP adherence is that of Edinger and colleagues (1994). In their study of 38 male veterans (mean age = 53) diagnosed with OSA, Edinger et al. initially gathered two weeks' worth of patient data. Predictors included results from diagnostic PSG and physical examinations (RDI, O₂ saturation, age, and BMI); results from a diary rating each night's sleep quality and how well rested they felt; data collected from the Stanford Sleepiness Scale and an analogous fatigue-rating scale gathered at breakfast, lunch, dinner, and bedtime each day; and psychological data assessed by the MMPI (specifically, scores were obtained on the Lie, Infrequency, Defensiveness, Hypochondriasis, Depression, Hysteria, Psychopathic Deviate, Masculinity/Femininity, Paranoia, Psychasthenia, Schizophrenia, Hypomania, and

Social Introversion scales). After six months of CPAP use, 26 (72.2%) of the 36 patients were available for follow-up. Using regression analysis of their data, Edinger and colleagues found that these 26 patients, who displayed common OSA characteristics pre-CPAP use (i.e., high BMI, high daytime sleepiness, high nocturnal apnoea, and high score depression and hypochondriasis sub-scales of the MMPI), continued to use CPAP and displayed greater adherence than the 12 patients that did not return after six months. Edinger et al. reported that the aforementioned pre-treatment measures independently predicted CPAP adherence, accounted for 63% of the variance. While post- CPAP use analysis reported BMI to be high in this sample, daytime sleepiness, nocturnal apnoea, and scores on the Hypochondriasis and Depression sub-scales of the MMPI decreased, which demonstrated the efficacy of CPAP treatment. This study provided initial evidence to suggest that personality can be a determinant to CPAP adherence—although at the time Edinger and colleagues noted that influence of pre-treatment psychological status is generally ignored in many studies.

Despite limited research surrounding the impact of personality on CPAP adherence, the research that has been carried out in this area has predominantly focused on the use of the MMPI to measure personality, a tool designed to assess psychopathology rather than temperament or traits. In a move away from the MMPI, Brostrom et al. (2007) were the first to investigate the prevalence and impact of Type D personality in OSA patients in relation to CPAP adherence. Denollet (2000) defines Type D or distressed personality as consisting of negative affectivity (NA) and social inhibition (SI). Negative Affectivity is described as subjective distress where an individual is likely to display a tendency to demonstrate negative emotions such as

anger, contempt, disgust, guilt, fear, and nervousness as opposed to a state of calmness (Denollet, 2000). Conversely, SI is described as an individual's conscious or unconscious tendency to constrain their emotions, behaviours, or appearance (Denollet, 2000). Initially, research conducted by Costa and McCrae (1987, cited in Brostrom et al., 2007) suggested a correlational relationship between NA and subjective reports of health complaints and quality of life. This was especially evident when both NA and SI were explored in a correlational study of cardiovascular patients regarding morbidity and mortality (Pedersen & Denollet, 2003, cited in Brostrom et al., 2007). However, the association of Type D personality with CPAP adherence in OSA patients is novel. In a cross-sectional design study, Brostrom et al. administered the Type D Scale 14 (DS-14), the Side Effects of CPAP Inventory (SECI), and the ESS to 247 patients (mean age = 60; 44 women and 203 men) diagnosed with moderate to severe OSA who had been using CPAP for a period of more than six months. Data was also collected from medical records that identified the following co-morbid conditions: hypertension, obesity, angina pectoris, acute myocardial infarction, stroke, diabetes, and daytime sleepiness. Dependent on the results from the DS-14 patients were into Type D (74 patients) and non-Type D (173 patients) cohorts. The authors found that objective mean CPAP use in the Type D cohort was 4.8 hours per night compared to 6.3 hours per night for the non-Type D cohort ($p < 0.001$). Their results show that CPAP use in patients who experienced negative emotions, but at the same time inhibited expressing those emotions displayed significantly less CPAP use. However, it must be noted that whilst Brostrom et al. reported a statistically significant difference in average CPAP use for both groups, the reported times were both within adherence rate ranges which have displayed improvements in overall OSA symptoms as described in other studies (Engleman et

al., 1998; Engleman et al., 1999; Stradling & Davies, 2000; Barnes et al., 2002).

Brostrom and colleagues results also showed that Type D patients were frequently inclined to report significantly more side-effects associated with their CPAP use as well as daytime sleepiness. This finding mirror those reported in chronic illness literature, suggesting that individuals with mood disorders are likely to report more problematic symptoms, independent of the physiological severity (Dwight et al., 2000; Katon et al., 2001; Katon & Ciechanowski, 2002). In Brostrom et al.'s study, these patients affirmed that reported side effects and continued daytime sleepiness contributed to their decision to cease CPAP use. Brostrom et al.'s study showed similar results to other studies that have attempted to investigate the impact of personality on CPAP adherence. They concluded that having a Type D personality was associated with non-adherence to CPAP use in OSA patients, but acknowledged that further research in this area is needed as “the question remains as to whether a Type D personality is a result of being sleep deprived (i.e., caused by OSA or a non-adherent CPAP use) and having a chronic condition, or existing prior to developing OSA” (p. 444).

In another study, Moran et al., (2011) utilised the mini-International Personality Item Pool (mini-IPIP), a 20-item questionnaire developed by Goldberg in 1999 (Donnellan, Oswald, Baird, & Lucas, 2006), to investigate the extent to which personality may impact on the behavioural inhibition system (BIS) and/or behavioural activation systems (BAS) to predict CPAP adherence. To date, this is the first study to employ a personality measure modelled after Costa and McCrae's Big five framework of personality—neuroticism, extraversion, intellect/imagination, agreeableness, and conscientiousness. Following an initial sleep study Moran et al.

collected data from 63 obese ($M_{BMI} = 37.6$) patients (32 women and 31 men). Patients completed the mini-IPIP questionnaire, the Ways of Coping Questionnaire (66 items that measure the thoughts and actions people use to handle stressful encounters), and Carver and White's questionnaire to measure the BIS and BAS models of personality. In line with other studies (Engleman et al., 1999; Greenham-Conway, 2000; Barnes et al., 2002; Wells et al., 2007), adherence was defined as four hours or greater of CPAP use per night. Moran and colleagues' results yielded a statistically significant moderate relationship between neuroticism and the BIS. This suggested that those who scored high on neuroticism were more likely to rely upon their BIS as a means of coping with their OSA and hence not adhere to CPAP use as a treatment regime. Moran et al. reported that 62% of the patients relied upon their BAS and as such did in fact adhere with CPAP use. Such research had contributed greatly to the limited body of literature investigating the impact that personality factors may have on CPAP adherence. Whilst it has been suggested that certain personality traits, as measured by the MMPI, may in fact predict CPAP adherence, broadening the scope of psychometric tools used and exploring a range of theoretical orientations are likely to further contribute to the body of evidence in this area.

1.8.3 Health Belief Model

To better understand the role that psychological factors play in CPAP adherence, researchers have begun to explore the utility of specific models in the prediction of CPAP use (Edinger et al., 1994; McFadyen, Espie, McArdle, Douglas, & Englemann, 2001; Stepnowsky, Bardwell, Moore, Ancoli-Israel, & Dimsdale, 2002; Stepnowsky et al., 2002; Wild, Englemann, Douglas, & Espie, 2004; Aloia,

Arnedt, Stepnowsky, Hecht, & Borrelli, 2005). The Health Belief Model (HBM) is the most widely used theory in health related research that aims to understand health behaviour change (Glanz, Rimer, & Lewis, 2002).

First introduced by the U.S. Public Health Services in the 1950s as a means to understand why certain medical screening programs were unsuccessful, the underlying premise of the HBM is that “health behaviour is determined by personal beliefs or perceptions about a disease and strategies available to decrease its occurrence” (Hochbaum, 1958 cited in Glanz et al., 2002, p.31). There are four perceptions that contribute to the main construct of the HBM: perceived seriousness, perceived susceptibility, perceived benefits, and perceived barriers.

Perceived seriousness: an individual’s belief about the seriousness or severity of the disease, syndrome, or condition. This belief is generally derived from medical information as well as the belief an individual develops pertaining to the difficulties a disease, syndrome, or condition is likely to create or the effects it would have on his or her life in general (McCormick-Brown, 1999). For example, most individuals would not think twice about contracting the common flu annually, as it may mean a day or two of bed rest. However, if you suffer from severe asthma or chronic fatigue, contracting the flu could lead to hospitalisation, hence the perceived seriousness of contracting the flu may be considerably higher.

Perceived susceptibility: personal risk is considered to be a great influence in motivating people to adopt healthier behaviours. Generally speaking, the higher the perceived risk, the greater the likelihood is of accepting behaviours to reduce the risk.

Literature shows that perceived susceptibility is an influential factor to motivating individuals to be vaccinated for influenza; to use sunscreen to prevent skin cancer; to brush their teeth to prevent gum disease and tooth loss; and use condoms in an effort to decrease susceptibility to HIV infection, contraction of sexually transmitted infections and unplanned pregnancies (Belcher, Sternberg, Wolitski, Halkitis, & Hoff, 2005; de Wit, Vet, Schutten, & van Steenberg, 2005; Chen, Fox, Cantrell, Stockdale, & Kagawa-Singer, 2007).

Perceived benefits: the value or usefulness an individual places on a new behaviour to reduce the risk of developing a disease, syndrome, or condition. People are motivated to adopt healthier behaviours when there is a belief that the new behaviour will reduce the risk of developing a disease. It is suggested that this is why individuals strive to eat five servings of fruits and vegetables a day, quit smoking, and use sunscreen (Glanz et al., 2002).

Perceived barriers: an individual's self-assessment of the perceived obstacles or barriers that may stand in the way of them adopting a new behaviour. This construct is likely to be the most significant in determining behaviour change (Janz & Becker, 1984). In order for a barrier to be overcome and the new behaviour to be adopted, an individual needs to believe that the benefits of the new behaviour outweigh the consequences of continuing the old behaviour (Centre for Disease Control and Prevention, 2004).

There is limited consistent literature regarding HBM and CPAP adherence, but it is likely that patients begin to develop perceptions, expectations, and beliefs

surrounding treatment before commencement of CPAP use at home (Smith, Lang, Sullivan, & Warren, 2004). Thus, CPAP adherence is likely to be greatly influenced by initial beliefs derived from patients' subjective experiences, self-reported benefits and side-effects (Aloia et al., 2005). Those that develop negative perceptions, expectations, and beliefs towards treatment may be less likely to try to accept CPAP as a form of OSA treatment in the first place.

Sage, Southcott, and Brown (2001) were one of the first to apply the HBM construct to OSA research regarding CPAP use via the development of a specific CPAP questionnaire based on the HBM. Forty patients with a mean age of 54 (10 women and 30 men) diagnosed with OSA ($RDI = > 10$) completed the specifically designed HBM-based questionnaire post-CPAP titration. When CPAP adherence was measured after one month, Sage et al. found a statistically significant moderate correlation with the perceived benefits and perceived barriers of CPAP use. Predictive analysis showed that these two HBM constructs explained 23% of the variance in adherence over the traditional sleep-related variables. Sage and colleagues explained that such results are common findings in HBM and adherence literature regarding other medical conditions such as diabetes and in psychology in general. Their results imply that a patient's belief in relation to perceived benefits and perceived barriers are probably linked to the short-term and long-term costs and benefits of CPAP use. Interestingly, the authors found no statistically significant association between perceived susceptibility and CPAP adherence, despite patients being informed about the long-term consequences of untreated OSA (i.e., cardiovascular disease). However, given the sample size, such results are best deemed exploratory in nature, but they

nonetheless successfully provided initial links between HBM and CPAP adherence and indicated the potential value of using the HBM for predicting CPAP adherence.

Closer to a decade later, Olsen, Smith, Oei, and Douglas (2008) employed the HBM to compare the role of psychological constructs with the commonly used sleep-related variables to determine CPAP adherence and acceptance. Their sample comprised 77 CPAP naïve patients (30 women and 47 men) aged between 26 and 80 years old ($M = 55.25$) with a mean BMI of 35.11 and a mean RDI of 38.36 (placing on average most patients in the obese range with severe OSA). In addition to the demographic information collected, each patient completed the ESS, the Functional Outcomes of Sleep Questionnaire (FOSQ), the Self-Efficacy Measure for Sleep Apnoea (SEMSA), the Depression Anxiety Stress Scales (DASS) and a standard PSG. Following four months of CPAP use, patients were contacted to gather adherence data from their CPAP device meter and to discuss any ongoing difficulties with treatment. Olsen et al. found that patients on average used CPAP 4.57 hours per night. Results from their multiple regression analysis showed that the HBM constructs accounted for 22% of the variance in CPAP adherence compared to 32% when the sleep-related variables were included. Olsen et al. reported that their results suggested that patients had developed perceptions, expectations and beliefs about OSA and CPAP use before implementation. Olsen et al.'s findings provide valuable support for the HBM as a solid model for understanding individuals' motivations to accept and adhere to CPAP use as a treatment.

While research in this domain is still somewhat limited, Tzischinsky, Shahrabani, and Peled (2011) investigated the possible factors that influenced the

decision to purchase a CPAP device at the pre-PSG stage. Their study initially consisted of 83 patients with a mean age of 55 years and a mean BMI of 30 who completed a demographic questionnaire, the Mini Sleep Questionnaire (MSQ), the Pittsburgh Sleep Quality Index (PSQI), the ESS, and their own developed HMB measure. Three month post-PSG, patients diagnosed with OSA were contacted via telephone to complete a short survey. Post-PSG out of the 66 patients diagnosed with OSA ($M_{RDI} = 21.58$), 50 (23 women and 27 men) participated in the short survey. Applying the specifically developed questionnaire based on the HBM constructs, Tzischinsky et al. found that only perceived susceptibility significantly explained the decision to purchase a CPAP device post-PSG, which was contrary to their expectations. While limited, these results do suggest that potential OSA sufferers who initially believed they were susceptible to having OSA were more likely to purchase a CPAP device, compared to individuals who did not feel at risk. The results from Tzischinsky et al.'s research did, however, provide statistically significant evidence to suggesting that sleep-related variables and patients' demographic information as well as having a higher level of OSA knowledge and higher levels of health motivation positively affected the decision to purchase a CPAP device. These results were similar to the findings of Smith et al., which suggested that OSA patients who were more informed about OSA had greater positive attitudes towards CPAP treatment and use. Tzischinsky and colleagues concluded that their results contributed to the body of evidence suggesting that individuals who were aware of their own health conditions had a greater tendency to invest in their own treatment. While the research of Tzischinsky et al. is somewhat removed from that of Olsen et al. and Sage et al. with regards to their focus on predicting CPAP adherence, it does contribute to the

limited literature in this domain and suggests that HBM constructs are likely to be an important overall factor.

1.8.4 Locus of Control

Locus of control is another widely studied construct of social cognitive factors that have received much attention in the field of adherence research with regards to, and not limited to, smoking cessation, diabetes, tablet-treated diabetes, hypertension, arthritis, cancer, and heart and lung disease (Nicasio et al., 1985; Allison, 1987; Desmond & Roberts, 1987; Stantion, 1987; Pruyn et al., 1988; Georgio & Bradley, 1992; Ferraro et al., 1990 all cited in Norman & Bennett, 1995). Locus of control is described as a stable personality trait that is likely to affect an individual's motivation to adhere to treatment (Rotter, 1966). Derived from Rotter's Social Learning Theory in 1966, the construct of health locus of control (HLC) offers some understanding to predicting and explaining specific health-related behaviours. This construct proposes that through learning, individuals will develop the belief that certain outcomes are a result of their actions (internals) or a result of other forces independent of themselves (externals). The use of HLC in research has assisted with understanding smoking reduction, birth control utilisation, weight loss, information-seeking, adherence to medication regimens, and other health or "sick-role" behaviours. As with other psychological factors, currently there is a paucity of research into the impact of HLC on CPAP adherence.

According to Wallston, a "patient's perception about the level of control they have over their illness will determine whether they change their behaviour to improve

their health” (1992, cited in Olsen et al., 2008, p. 16). In their discussion of locus of control, Norman et al. (1998) have suggested that behaviour specific locus of control measures has generated positive results in adherence literature—this is also reflected in the limited body of research available pertaining to CPAP adherence (Engleman & Wild, 2003; Wild et al., 2004). Wallston, Stein, and Smith (1994) described those that scored high on internal locus of control as having control over their health and therefore were more likely to adhere to treatment. Conversely, those who believed that their health was influenced by others, such as a doctor, or determined as a matter of chance, scored high on an external locus of control. This subgroup of individuals is generally less likely to adhere to treatment.

Wild et al. (2004) were the first to explore the construct of locus of control to improve CPAP use in OSA patients. They applied a social cognition model to a sample of OSA patients that were CPAP naïve to determine whether pre-treatment social cognitive factors influenced CPAP adherence. Their sample consisted of 119 (24 women and 94 men) obese (mean BMI = 33) patients with a mean AHI of 45, which placed patients within the severe range for OSA. Wild et al. provided each patient with a battery of psychological measures to assess locus of control, health value, and self-efficacy. Sleep-related variables were also collected along with information relating to daytime sleepiness (measured using the ESS) and adherence. Patients were provided with ongoing support and visitation during CPAP treatment, and a formal follow-up was conducted after three months of CPAP use. Statistically significant results from logistic regression showed that the predictors ESS, AHI, BMI, and the psychological factors of internality, powerful others, and health value accounted for 24% of the variance in CPAP adherence. Wild et al. also found that

improved adherence was significantly associated with a stronger internal locus of control and less belief in powerful others. These results suggest that individuals who possessed beliefs associated with internality were better equipped to take significant control over their own circumstances and environment. These individuals were therefore likely to internalise medical and health-related advice provided by their physicians and persevere with CPAP use, regardless of inconveniences or associated side-effects. Wild et al.'s findings were in line with those of other researchers (e.g., Hoy, Vennelle, Kingshott, Engleman, & Douglas, 1999; Stepnowsky et al., 2002), who generally suggested that adherers were more likely to take control over their treatment options and proactively engage in coping strategies if they believed they had some control over their OSA treatment.

One of the most recent and comprehensive studies of OSA and locus of control was conducted by de Zeeuw et al. (2007). Using a sample of 85 patients diagnosed with OSA, de Zeeuw et al. aimed to identify the likelihood of a person being a low adherer prior to initiation of CPAP treatment. Using interviews and self-report questionnaires (the Nottingham Health Profile, the von Zerssen's Depression Scale, the State Trait Anxiety Inventory, and the IPC-Scale), de Zeeuw and colleagues reported that eight months later 66 patients continued regular CPAP use and 19 individuals (approximately 25%) had discontinued CPAP treatment. De Zeeuw et al. found that while all patients reported no anxiety and/or depression, those who discontinued CPAP treatment had significantly less external control belief, which suggested that these patients may not have been convinced of the benefits of CPAP use. They concluded that identifying individuals with a diminished external control

belief prior to the commencement of OSA treatment using CPAP is likely to be useful.

1.8.5 Self-Efficacy

Self-efficacy is another commonly investigated social cognitive factor in adherence research that has been widely acknowledged in many studies as an important contributing factor to the treatment and management of chronic illnesses and conditions. Similar to locus of control, self-efficacy has generally been reported to be a good predictor of adherence in a range of other chronic medical illnesses and conditions, such as diabetes (Connor & Norman, 1996; Jones, 2002, cited in Olsen et al., 2008). In fact, self-efficacy has been reported to contribute up to 40% of the variance explained in those engaged in treatment for diabetes (Kavanagh, Gooley, & Wilson, 1993). However, its application to aid the understanding surrounding beliefs about OSA and its management via CPAP use has been limited but positive in identifying self-efficacy's predictive value with regard to CPAP adherence (Sage et al., 2001; Stepnowsky, Marler, & Ancoli-Israel, 2002; Aloia et al., 2005).

Albert Bandura defines self-efficacy as an individual's "belief in one's capabilities to organise and execute the course of action required to produce given attainment" (Bandura, 1997, p. 3). According to Bandura, self-efficacy is the belief that one can successfully execute the behaviour required to cope with potentially threatening situations. Individuals with a high level of self-efficacy are likely to exert considerable efforts in order to cope with situations that may demand new behaviour patterns. Commonly, research surrounding chronic illnesses has applied Bandura's

social learning theory to help understand the way individuals adhere with treatment. Building upon Bandura's definition, Wild et al. (2004) defined self-efficacy as "an individual's belief in their capability to organise and carry out the courses of action required to deal with prospective situations" (p. 462).

To date, the study conducted by Stepnowsky, Marler, and Ancoli-Israel (2002) has been the only study to comprehensively examine beliefs as they relate to CPAP adherence using Bandura's Social Cognitive Theory. In their dissertation, Stepnowsky et al. identified the need for OSA research to shift from a narrow focus based on sleep-related factors and medical issues to a perspective that considers other factors that may influence health behaviours. Stepnowsky and colleagues investigated the relationship between Social Cognitive Theory (SCT) and Transtheoretical Model (TM) variables and objectively measured CPAP adherence over a period of one month in 51 patients (two women and 49 men). Patients in this study had a mean AHI of 40, a mean BMI of 36 and a mean age was of 54 years, making the sample predominately middle-aged obese men with severe OSA. The SCT variables in Stepnowsky et al.'s study included measures of self-efficacy, outcome expectations, social support, and knowledge. The TM variables included measures of stage of change, a decisional balance index (consisting of a summary of the pros and cons of engaging in the behaviour), and processes of change. Information on daytime sleepiness was also gathered via administration of the ESS. All patients were administered the test battery at one week post-CPAP fitting and then again one month pre-CPAP fitting. Stepnowsky and colleagues' findings showed that SCT measured at time one (initial testing) were not predictive, while SCT variables measured at time two were predictive of CPAP adherence at one month. Similarly, TM variables

measured at time one were not predictive, but were predictive of CPAP adherence at one month. When measuring both SCT and TM Stepnowsky et al. reported that while initial statistical analysis displayed no significant findings, after a short period of time (one month) hierarchical regression applied to SCT and TM accounted for 40% and 33% of the variance in CPAP adherence when CPAP was effectively and continuously used for 3.4 hours per night. This study has therefore provided important information with regards to the future direction of OSA and CPAP adherence research. Stepnowsky et al. suggested that future behavioural interventions designed to increase CPAP adherence may prove to be effective if based on such models. These results are akin to those exploring the impact of self-efficacy on adherence to various chronic diseases, suggesting that self-management, belief, and control are likely to contribute to the prediction of adherence to treatment.

Building on Stepnowsky et al.'s initial research, Aloia et al. (2005) found that self-efficacy measured at one week and then again at three months after CPAP use significantly predicted CPAP adherence at six months. Stepnowsky was also part of the Aloia research team's longitudinal study to determine the predictive value of behaviour change principles on treatment adherence in individuals with OSA. Utilising a sample of 98 CPAP naïve patients diagnosed with moderate to severe OSA, Aloia and colleagues collected data measuring of behaviour change at baseline and continually throughout a six month period. Adherence data was also collected via the fitted CPAP device meters. The same two behaviour change measures (SCT and TM) used in Stepnowsky et al.'s initial research were investigated, and results showed that behaviour change predicted CPAP adherence at the six months post-CPAP treatment when assessed at one week and at three months. However, when the two

behavioural measures were assessed at baseline, they were not predictive of CPAP use at six months, suggesting that SCT and TM do not improve upon the prediction of adherence at six months. Aloia et al.'s study differed from that of Stepnowsky et al.'s original research with regard to collecting adherence results at one and six months respectively. However, both studies showed while that SCT and TM are not predictors of CPAP adherence pre-CPAP use, they become predictors as patients become more familiar with CPAP treatment and received appropriate ongoing intervention and support. This finding provided important evidence for the ongoing monitoring of patients' self-efficacy, as the ability to identify issues and intervene is likely to enhance adherence and maintain self-efficacy during treatment (Trupp, Corwin, Ahijevych, & Nygren, 2011).

Follow Stepnowsky et al.'s study, Weaver and colleagues (2003) aimed to build the knowledge base surrounding self-efficacy pertaining to OSA treatment and CPAP adherence. Weaver et al. developed the Self-Efficacy Measure for Sleep Apnoea (SEMSA) that assessed and identified individuals who were not likely to adhere to CPAP use. Two hundred and thirteen newly diagnosed OSA patients participated in the study to validate the SEMSA. The sample comprised 85 women and 128 men with a mean age of 47 and a mean BMI of 38. Mean ESS score was 12 and mean RDI was 43. The content validity and internal consistency of the SEMSA (0.92) was found to be more than adequate. Confirmatory factor analysis validated the authors' *apriori* sub-scales: Risk Perception, Outcome Expectancies, and Treatment Self-Efficacy. The test-retest reliability coefficients ($n = 20$) were estimated to be 0.68, $p = 0.001$, for Perceived Risk; 0.77, $p < 0.0001$, for Outcome Expectancies; and 0.71, $p = 0.0005$, for Treatment Self-Efficacy. Weaver et al. were the first to develop

an OSA-specific measure with strong psychometric properties for identifying individuals who were not be likely to adhere to CPAP use. While the study by Weaver and colleague aimed to develop a specific self-efficacy measure rather than investigate the impact or predictive value of self-efficacy in association with CPAP adherence, it did report some interesting findings. Weaver et al. found that 50% of the newly diagnosed OSA patients in their sample had limited knowledge pertaining to the perceived associated risks of OSA. However, more than 60% of patients associated CPAP use with feeling better, snoring less, being more active, having improved relationships, having a decreased chance of a driving accident, and having enhanced alertness and job performance. Such a tool is therefore likely to provide a direction for examining the impacts of self-efficacy on CPAP adherence in future research.

1.8.6 Daytime sleepiness

While daytime sleepiness is not a psychological construct, it has and continues to play an important and significant role in OSA and CPAP adherence research. As one of the main symptoms associated with OSA, researchers have collected an abundance of data on daytime sleepiness and have adequately reported on it, as evident in the above literature review. As such, daytime sleepiness has established its place clinically in the diagnosis of OSA, as sleep restriction generally and significantly manifests as sleepiness. Central to the diagnosis of OSA, sleepiness is generally assessed through the self-report ESS and/or the MSLT (Reynolds, Coble, Kupfer, & Holzer, 1982; Carskadon, Dement, Mitler, Roth, Westbrook, & Keenan, 1986; Johns, 1993). Specifically, the utility of the ESS in the screening and diagnosis

of OSA has contributed to its prevalent use in measuring subjective sleeping in OSA research (Roehrs, Zorick, Wittig, Conway, & Roth, 1989; Johns 1993). This was again evident in the above literature review pertaining to the psychological determinants of CPAP adherence.

In general, daytime sleepiness is associated with many serious and incapacitating effects in individuals diagnosed with OSA. These effects are commonly experienced not just in isolation, but often impact on family, friends, employment, and co-workers (Zammit, 2008). Research surrounding daytime sleepiness has shown the predictive value the ESS in the arena of CPAP adherence (Englemann, et al., 1996; Hui et al., 2000; McFadyen et al., 2001; Lewis, Seale, Bartle, Watkins, & Ebden, 2004; Tanaka, Nakano, Sudo, & Kubo, 2009). This perspective is widely reflected in research, with the continued use of the ESS as a subjective measure of daytime sleepiness.

1.9 Statement of Significance and Contribution to Knowledge

Historically, it was Gastaut, Tassinari, and Duron who first provided a comprehensive account of OSA in the 1960s (cited in Lavie, 2003) that led to an increase in sleep apnoea-related publications in the 1970s. However, the surge in research during this period had been described as ineffectual due to the perception that such research had little chance to impact the everyday practice of medicine, hindering any further progression (Lavie, 2008). It was not until two decades later that Young et al.'s (1993) population-based study on the prevalence of sleep apnoea led to its recognition and acknowledgement as a serious medical disorder with severe health

consequences. While it is important to acknowledge past prevalence studies within this domain, along with the increased number of related publications, OSA research remains in its infancy—especially in regards to the role that psychological factors play in relation to CPAP adherence.

To illustrate the limited amount of research conducted in this area, Lavie (2008) performed a review of publications with the key words “sleep apnoea”, “sleep apnoea”, “sleep disordered breathing”, “Pickwickian syndrome”, “Upper airway occlusion during sleep”, and “snoring” in the title, abstract, or body of publications. Lavie found a total of 15,064 related publications (inclusive of articles, letters, reviews, and editorials) using these key words between 1965 and 2006. The rate of sleep apnoea-related publications doubled from 1990 to 1991 and had increased sixfold since 1965, with the U.S. (40%) contributing the most towards sleep apnoea related research. At the time of Lavie’s review, Australia produced 5% of the available literature surrounding sleep apnoea that highlighting the need for further research in this field at a global and domestic level.

Specifically, there are few treatment options available to OSA patients apart from CPAP (see Table 11). Those that are currently under consideration include a range of pharmacologic treatments as well as surgery. However, no one pharmacologic agent is universally useful in the treatment of OSA, and more research on the effectiveness of drugs and possible side-effects is required. In addition, there is concern about surgical risk in the treatment of OSA, especially since most surgical techniques for the treatment of OSA are far from established therapies and/or have a very low success rate—in most cases less than 50% (Collard et al., 1997).

Table 11

Treatment Options for Obstructive Sleep Apnoea

Treatment
1. Lifestyle modification for obesity
– Weight reduction
– Bariatric surgery in some patients with morbid obesity
2. Treatment of nasal congestion and deviated nasal septum
– Use of nasal anti-histaminic drops or sprays and topical steroids
– Surgery in subjects with deviated nasal septum
3. Body positioning during sleep
– OSA patients should sleep on their sides or in prone posture
– Avoidance of supine sleeping accomplished with a tennis ball
4. Thyroid hormone replacement therapy for hypothyroidism
5. Pharmacologic treatment of excessive daytime sleepiness in selected patients
– Modafinil
– Armodafinil
– Tricyclic Antidepressants
– Serotonin Agents
– Nicotine Products
– Methylxanthine Derivatives
– Inhaled Corticosteroids
– Leukotriene Antagonists
6. Surgery
– Uvullectomy
– Pillar system procedure

-
- Nasal reconstruction
 - Adenotonsillectomy
 - Palatal surgery
 - Genioglossal advancement
 - Thyrohyoid suspension
 - Maxillary-mandibular advancement
 - Tongue-base surgery
 - Radiofrequency ablation
 - Tracheotomy
-

1.10 Aims

This study aims to explore a range of psychological factors (mood, personality, self-efficacy, health locus of control, and health belief) that may impact adherence to CPAP use as a treatment option for OSA. The identification of adherent and non-adherent patients is particularly pertinent since there are few successful and viable treatment options available to OSA patients, and CPAP use has been identified, by both users and research, as the most effective treatment that currently exists.

Therefore, given that CPAP use is viewed as a treatment option rather than a cure and taking into account the high prevalence of OSA in the community, ongoing research is imperative. The possibility that psychological factors associated with OSA and CPAP use can be identified to improve adherence is of particular interest and deserves attention.

1.11 Exploratory Question

The present study set out to explore what psychological factors, measured at the diagnostic phase, would likely predict CPAP adherence and non-adherence for patients with OSA. The Literature review exploring the role that psychological factors play as possible predictors of CPAP adherence and non-adherence revealed that research in this domain is still in its infancy. Within the research available for examination, much of it has focussed only on depression rather than other psychological determinants. It is likely that this pre-occupation with depression alone has set research on adherence to CPAP treatment in the area of OSA behind research in other areas of chronic disease (e.g., lower back pain, arthritis, asthma, diabetes, and HIV/AIDS). Thus to further explore the role that psychological factors play as possible predictors of CPAP adherence and non-adherence the following were explored:

1. The combination of psychological factors—mood, personality self-efficacy, health locus of control, and health belief—that best predict adherence and non-adherence to CPAP use.
2. The impact of adherent CPAP use on mood following the implementation phase.
3. The impact of adherent CPAP use on sleep-related variables collected from PSG at the diagnostic phase.

CHAPTER 2

2.1 METHOD

2.1.1 Patients

Between 2005 and 2009, a total of 156 sleep study patients were initially invited to participate in the present study. Participants consisted of newly diagnosed patients with moderate to severe OSA as determined by an AHI greater than 15. The sample included 52 women and 104 men aged between 18 and 74 years ($M = 50.3$ years, $SD = 10.9$). No exclusion criteria were set, and those who were non-English-speaking, greater than 65 years of age, and diagnosed with other medical illnesses and conditions were included in the present study. This decision to exclude certain common selection criteria was based on the fact that the prevalence of being diagnosed with OSA increased with age, and patients with OSA commonly presented with other co-morbid illnesses. This decision also aided in increasing the sample size, as it has been noted that one of the main limiting factors associated with research in this field is small sample sizes. Patients were recruited using a convenience sampling technique from the respiratory and sleep medicine departments at Southern Health ($n = 41$) and Austin Health ($n = 115$) following a referral by their physician for an overnight PSG sleep study. Sixty-nine patients participated in both the diagnostic and implementation phase, resulting in a 56% “drop off” rate.

2.1.2 Materials

A battery of paper-based measures were utilised in the present study: an explanatory statement, demographic questions, and five valid and reliable measures for daytime sleepiness, mood, personality, self-efficacy, and health locus of control. A health belief scale modelled on the HBM was also designed for the present study to measure patient's perceptions regarding OSA and CPAP use.

2.1.2.1 Explanatory statement.

A one-page explanatory statement in plain language was provided to patients, inviting them to participate in the present study. The statement described the nature, objectives, and aims of the study and also provided contact details of the principal researcher and student researcher (see Appendix 1).

2.1.2.2 Demographics questionnaire.

The demographic questions were designed to elicit information about patient age, gender, and educational level (see Appendix 2). The following five additional yes/no questions were also included:

1. Do you suffer from any other illness apart from Obstructive Sleep Apnoea (OSA)?
2. Do you believe you actually have OSA?
3. Do you THINK it will be difficult to use CPAP?
4. Do you THINK it will be uncomfortable to use CPAP?

5. Do you THINK CPAP treatment alleviates OSA symptoms for you?

2.1.2.3 Profile of Mood States–Short Form.

The Profile of Mood States Short Form (POMS-SF) is a 37-item adjective rating form used to assess present mood state. It is not a diagnostic measure, and it assesses sub-clinical symptoms of mood. The form has a wide application in many areas including psychotherapy, drug treatments, and psychological interventions such as relaxation, stress management, and exercise. McNair, Lorr, and Droppleman conducted a factor analysis to determine the six mood dimensions from the original POMS that would be measured by the POMS-SF: tension/anxiety, depression/dejection, anger/hostility, vigour/activity, fatigue/inertia, and confusion/bewilderment. Respondents are asked to indicate their mood reactions “during the past week including today” or for shorter periods such as “right now” on a five-point Likert scale ranging from 0 (“not at all”) to 4 (“extremely”).

In order to obtain an individual’s TMD score as well as scale scores for the six dimensions, the sum of the responses are obtained for tension/anxiety (1, 10, 15, 16, 22, 27), depression/dejection (4, 8, 12, 14, 20, 23, 28, 33), anger/hostility (2, 7, 11, 19, 21, 25, 31), vigour/activity (5, 9, 13, 24, 32, 35), fatigue/inertia (3, 18, 26, 29, 37), and confusion/bewilderment (6, 17, 30, 34, 36). To obtain the TMD score, the sum of tension/anxiety, depression/dejection, anger/hostility, fatigue/inertia, and confusion/bewilderment are added minus the sum of vigour/activity. The higher the TMD score the greater the overall mood disturbance.

Contributing to the profiles psychometric properties, test-retest reliability of the POMS-SF has been reported to be between 0.65 and 0.74 (Curran, Andrykowski, & Studts, 1995). Whilst this statistic is not overly high, it is important to highlight that the POMS-SF was developed to assess an individual's immediate and transitory mood state, and as such these reliability scores are considered reliable and appropriate. Internal consistency reliability for each of the six dimensions is reported as follows: 0.70 for tension/anxiety, 0.74 for depression/dejection, 0.71 for anger/hostility, 0.65 for vigour/activity, 0.66 for fatigue/inertia, and 0.68 for confusion/bewilderment. These scores suggest that the POMS-SF is likely to yield reliable and appropriate internal consistency.

Five of the six POMS-SF dimensions also display high face validity. The sixth dimension, tension/anxiety scale, measures cognitive tension or emotional anxiety and was initially included to measure an individual's physical and musculo-skeletal tension. Literature suggests that this is likely to have a negative impact on the measure of overall face validity and could be a source of error variance (Curran et al., 1995).

2.1.2.4 Eysenck Personality Questionnaire-Revised Short Form.

The Eysenck Personality Questionnaire-Revised Short Form (EPQ-RS) is a 48-item questionnaire developed to measure three personality dimensions containing 12-items in each: neuroticism (a predisposition to anxiety—items 1, 5, 9, 13, 17, 21, 25, 30, 34, 38, 42, 46), extraversion (a predisposition to sociability—items 3, 7, 11, 15, 19, 23, 27, 32, 36, 41, 44, 48), and psychoticism (a predisposition to antisocial

behaviour—items 2, 6, 10, 14, 18, 22, 26, 28, 31, 35, 39, 43) (Eysenck, Eysenck, & Barrett, 1985). A lie scale to measure social desirability also makes up part of the EPQ-RS (items 4, 8, 12, 16, 20, 24, 29, 33, 37, 40, 45, 47). Patients are asked to respond to items with either a “yes” = 1 or a “no” = 0. These include questions such as “Do you ever feel just miserable for no reason?”, “Are you a worrier?” (neuroticism); “Are you a talkative person?”, “Can you usually let yourself go and enjoy yourself at a lively party?” (extraversion); “Would being in debt worry you?” and “Do you enjoy co-operating with others?” (psychoticism); and “Are all your habits good and desirable ones?”, “Have you ever said anything bad or nasty about anyone?” (lie scale). Scores are then summed to derive a total for each of the EPQ-RS sub-scales.

The EPQ-RS is a widely used and statistically sound measure of personality (Robbins, Francis, & Rutledge, 1997; Francis & Wilcox, 1998; Martin & Kirkaldy, 1998; Chivers & Blagrove, 1999; Francis, 1999; Halamandaris & Power, 1999; Aleixo & Norris, 2000; Chan & Joseph, 2000; Glicksohn & Bozna, 2000; Blagrove & Akehurst, 2001; Creed, Muller, & Machin, 2001; Glicksohn & Golan, 2001; Linton & Wiener, 2001). Test-retest reliability and internal consistency has been reported as strong, yielding the following coefficients for women and men respectively: neuroticism 0.80 and 0.84, extraversion 0.84 and 0.88, psychoticism 0.61 and 0.62, and the lie scale 0.73 and 0.77 (Eysenck et al., 1985).

2.1.2.5 General Self-Efficacy Scale.

Developed by Jerusalem and Schwarzer in 1981 in Germany, the General Self-Efficacy Scale (GSE) is a brief self-administered 10-item scale. It is designed to

measure an individual's optimistic self-belief about coping with a variety of daily hassles as well as adaptation after experiencing all kinds of stressful life events. The GSE was designed to assess perceived self-efficacy, with responses made on a four-point Likert scale (1 = "not all true" to 4 = "exactly true") that is then summed to form part of an overall total score ranging between 10 and 40. Higher scores indicate stronger individual belief, suggesting that one can perform an unfamiliar or difficult task or have little difficulty coping with adversity. Those with a perceived high level of self-efficacy are said to facilitate goal-setting and show persistence in the face of barriers and recovery from setbacks. Perceived self-efficacy is described as an operative construct because it is related to subsequent behaviour, and it is therefore, relevant for clinical practice and behaviour change.

The GSE has demonstrated high reliability, stability, and construct validity, yielding statistically significant Cronbach's alpha coefficients ranging from 0.75 to 0.94. (Rimm & Jerusalem, 1999; Schwarzer, Mueller, & Greenglass 1999; Leganger, Kraft, & Roysamb, 2000; Luszczynska, Scholz, & Schwarzer, 2005). Comparisons have been made to other social cognitive variables (intention, implementation of intentions, outcome expectations, and self-regulation) that confirm and contribute to the validity of the scale (Luszczynska et al., 2005).

2.1.2.6 Multidimensional Health Locus of Control-Form C.

The Multidimensional Health Locus of Control-Form C (MHLC-C) scales have been cited as the most used and efficient measures of health-related beliefs. Locus of control is recognised as an important construct in understanding and

predicting health behaviours, and it has helped to develop thinking about the role that beliefs play in the context of health behaviours, health outcomes, and health care (Malcarne, Drahota, & Hamilton, 2005). According to Rotter's (1966) social learning theory, individuals may have an internal or external locus of control. An individual with an internal locus of control believes that they control their life, as opposed to an individual with an external locus of control, who believes that their environment, some higher power, or other people control their decisions and their life. Individuals with a high internal locus of control believe that events result primarily from their own behaviour and actions. Those with a high external locus of control believe that powerful others, fate, or chance primarily determine events.

Developed by Wallston, Wallston, and DeVellis in the mid 1970s at Vanderbilt University, the MHLC consists of three forms (A, B, and C). Forms A and B have been applied in more than a thousand clinical studies over the last 30 years and measures "general" health locus of control (Wallston, n.d.) . Form C (MHLC-C) is an 18-item measure developed by Wallston, Stein, & Smith (1994) to be "condition-specific" and was used in place of Form A and B in the present study. Patients were asked to rate items on a six-point Likert scale ranging from 1 "strongly disagree" to 6 "strongly agree". The values on each sub-scale are summed (internal-items 1, 6, 8, 12, 13, 17; chance-items 2, 4, 9, 11, 15, 16; doctors-items 3, 5, 14; others people-items 7, 10, 18) to produce an independent score for a patient's condition-specific health locus of control. Form C has been described as a better measure than Forms A and B at discriminating the role that the physician plays in determining health status, particularly in individuals that already have a diagnosis (Wallston et al., 1994).

Given that health belief's can change through learning and experience, it is not surprising that lower reliability coefficients can in some situations be considered acceptable for the MHLC-C. The MHLC-C has displayed its potential, yielding stability over time and producing reliability coefficients ranging between moderate and high values ($\alpha = 0.58$ to 0.80) (Wallston et al., 1994). Internal consistency also yielded favourable and acceptable Alpha coefficients of ≥ 0.70 . The MHLC-C has also demonstrated concurrent validity when compared to Form A and B.

2.1.2.7 Epworth Sleepiness Scale.

Developed in 1990 at Epworth Hospital, Melbourne, Australia, the Epworth Sleepiness Scale (ESS) is a brief questionnaire designed to measure an individual's subjective level of daytime sleepiness as opposed to how much sleep or "sleep debt" they experience. Items on the ESS are self-rated on a four-point scale relating to an individual's chances of dozing off (0 = "would never doze" to 3 = "high chance of dozing") in eight different situations that differ in "sleep-inducing characteristics". Sleep-inducing characteristics are dependent on the time of day and variables such as what the individual is doing at a particular time (e.g., lying down as opposed to standing up) that increases the "propensity" of falling asleep. "Sleep propensity" is measured within the context of the situation and activity at the time. Thus, an individual's "sleep propensity" in the same situation is known as "situational sleep propensity", for example sitting and watching television.

Response to the ESS relies on retrospective reports of dozing behaviour in the course of a day during activities such as sitting or watching television. The ESS item

scores representing different “situational sleep propensities” are added together to provide a total ESS score. This is a measure of an individual’s “average sleep propensity” (ASP) and can range between zero and 24, with the normal range defined as zero to 10 (Hardinge, Pitson, & Stradling, 1995). The ESS ASP is not a measure of fatigue or tiredness. Therefore, a moderately high ASP is not the same as falling asleep, especially if the individual avoids sleep-inducing situations, for example, keeping busy and not sitting down. Similarly, those with a high ASP may not necessarily be impacted by sleep-inducing situations such as lying down.

Test-retest reliability for the ESS has been demonstrated, yielding positive and strong coefficients over months using Spearman’s correlation ($\rho = 0.82$, $n = 87$, $p < 0.001$). The ESS has also demonstrated a high level of internal consistency, as assessed by Cronbach’s alpha ($\alpha = 0.88$ – 0.74 in four different groups of subjects). Given the ESS is generally used as a screening tool to assess “sleep propensity”, rather than as a diagnostic measure. It is also widely acknowledged to be a valid questionnaire comparable to the MSLT ($\rho = -0.42$, $n = 44$, $p < 0.01$) (Thorpy, 1992; Johns, 2002).

In addition, validity studies have demonstrated change in ESS scores for experimental findings between normal individuals and those with OSA (Johns, 1991; Johns, 1993). Higher than normal ESS scores are common in those with OSA and have been found to return to the normal range once individuals engage in successful CPAP treatment (Johns, 1993; Smolley, Ivey, Farkas, Faucette, & Murphy, 1993; Manni, Politini, Ratti, & Tartara, 1999).

2.1.3 Procedure

The present study received ethical approval from the Human Research Ethics Committees at Victoria University, Southern Health and Austin Health.

A quasi-experimental design was employed using pre- (diagnostic) and post- (implementation) testing without a control group. It is important to note that an experimental design with a control group, such as a randomised control trial, was considered and deemed unsuitable for the present study. Such a design is likely to be better applied to medical research where effect is unlikely to vary across individuals, as opposed to psychological research that tends to interact with factors such as gender. Control group designs are also likely to further reduce the sample size when taking into consideration confounding factors such as co-morbidity (that is common in OSA patients') in order to achieve greater statistical significance and avoid central tendency. In addition, it has been argued that volunteers to control group designs are not necessarily reflective of the general population thus results may not necessarily be externally valid (Shadish, Clark, & Steiner, 2008). Testing was conducted in two phases in a staggered format (meaning that patients were able to participate anytime within the designated time period). Data was gathered in two forms: subjective patient reports and objective CPAP-use figures (from the CPAP devices fitted with usage meters). Patients recruited from the respiratory and sleep medicine departments at Southern Health and Austin Health were assessed twice during the six month study period. This allowed for the measurement of medium-term adherence to CPAP treatment.

Adherence is generally defined in terms of CPAP use as a percentage of hours prescribed by the patient's physician/doctor. In line with previous research (Engleman, 1999; Barnes, 2002; Wells et al., 2007), adherence was initially considered adequate if CPAP was used for at least 2.5 to 4 hours per night. Such studies had achieved improvements in self-reported OSA symptoms at relatively low nightly CPAP use.

In this study, data were initially obtained from the CPAP devices fitted with meters to verify reports of adherence from patients. As a considerable amount of variability and associated errors were encountered with faulty CPAP meters, adherence was re-defined as completion of the diagnostic phase and return for completion of the implementation phase. This decision is in line with previous research that employed a pre- and post-experimental design to determine CPAP adherence (Kribbs et al., 1993; Edinger, et al., 1994; Yu et al., 1999; Doherty et al., 2003; Wiese et al., 2005; Stepnowsky et al. 2012)

2.1.3.1 Diagnostic phase.

Patients were requested to complete the range of psychometric measures during the diagnostic phase prior to PSG. This phase was designed to gather baseline patient data on mood, personality, self-efficacy, health locus of control, and health belief. Data pertaining to sleep-related variables was also collected from PSG.

2.1.3.2 Implementation phase.

Following the diagnostic phase, patients had obtained or were provided with CPAP devices fitted with a usage meter. Details regarding the use of the CPAP device were based on the physician's/doctor's prescription, and the usage meters provided objective data with regards to adherence. Patients were requested to return for a follow-up PSG, where data pertaining to sleep-related variables were again collected. At the end of a six month period, patients were requested to complete the same range of psychometric measures as at the diagnostic phase.

2.1.3.3 Data analysis phase.

Data were collated and analysed using the Statistical Package for Social Science (SPSS) for Windows version 22.0. Four distinct analyses were conducted:

- 1) identification and treatment of missing values and outliers;
- 2) calculations of reliabilities for all corresponding measures;
- 3) descriptive analyses; and
- 4) inferential analyses utilising Discriminant Function Analysis (DA), ROC curve analysis, and Multiple Analysis of Variances (MANOVA) to explore the combination of psychological factors contributing to CPAP adherence and non-adherence; and Paired Samples t-tests along with Analysis of Variances (ANOVA) to explore potential improvements in mood and sleep related variables determined by PSG following the implementation phase with adherent CPAP use.

2.1.4 Preparation

Prior to attending their sleep study, each patient was contacted via telephone to inform them about the study and to explain confidentiality. A plain language statement explaining the study was also mailed to each patient that expressed an interest in participating in this study. Patients were informed that on the day of their sleep study they would be introduced to the researcher who would explain to them the requirements of the study and assisted with the administration of the various psychometric measures. Patients who wished to enter the study were requested to read and sign the consent form.

2.1.4.1 Polysomnograph procedure.

Patients were requested to attend their pre-scheduled PSG studies on the arranged dates. They usually reported for the studies at 1900h. The studies were conducted in the department(s) of respiratory and sleep medicine at both Southern Health and Austin Health.

Sleep study patients were requested to:

- not eat or drink caffeine after 1200h on the day of their study–this included coffee, chocolate, and most teas;
- not to consume any alcoholic beverages or intoxicating substances on the day of their study;
- avoid naps all day;

- to consume a normal meal, wash and dry their hair (and not to apply hair sprays, oils or gels) prior to attending their sleep study; and
- bring their bedtime attire, toiletries, any prescribed medication and a change of clothing for the next day.

Upon arrival at the respiratory and sleep medicine departments at Southern Health and Austin Health, a sleep technician described the PSG procedure to each patient. The patient then watched a short informative video describing the purpose of a sleep study. Patients were asked to change into their nightclothes and were then marked and measured to apply the proper placements of electrodes. It was explained that none of the monitoring devices would be painful or harmful. The entire preparation for the PSG study took approximately 40 to 60 minutes.

Patients who had previously agreed to participate in the study were requested to complete the questionnaires (POMS-SF, EPQ-RS, GSE, MHL-C, and Health Belief Scale) after they had been wired for the PSG and were waiting to go to bed. The ESS was administered by the sleep technician as part of the standard administration procedure prior to PSG. Patients that were scheduled to attend their sleep study later in the evening or those that elected to complete the questionnaires at a later date were provided with a reply-paid envelope.

Commencement of sleep monitoring typically began between 2200h and 2300h, and patients were encouraged to sleep as they would normally at home. This included sleeping in any position during the night; however, spending some time in the supine position was requested. During the night each patient was free to use the

adjacent bathrooms. The monitoring devices were all connected to a small “jack-box” that was easily disconnected and carried. If a monitoring device became disconnected during the night, the sleep technician would attend to it. Some patients underwent split diagnostic-implementation studies while most had a standard diagnostic PSG. The sleep study was concluded between 0600h and 0700h and the sleep technician or morning nurse removed all of the monitoring devices, which took approximately 15 to 20 minutes.

2.1.4.2 Monitoring parameters.

A PSG typically records a minimum of 12 channels requiring a minimum of 22 wires to be attached to each patient. This varies between different sleep laboratories according to the requirements of physicians, patient needs, and budgetary constraints. In this study, a minimum of three channels were set aside for the EEG, one to two measured airflow, one to two for chin muscle tone, one or more for leg movements, two for eye movements (EOG), one to two for heart rate and rhythm, one for oxygen saturation, and one each for the belts that measured chest wall movement and upper abdominal wall movement. The movement of the belts was measured with piezoelectric sensors or respiratory inductance plethysmography. This movement was equated to effort and produced a low-frequency sinusoidal waveform as the patient inhaled and exhaled. Wires for each channel of recorded data led from the patient and converged into the “jack-box”, which in turn was connected to a computer system for recording, storing, and displaying the data (see Figure 6). During sleep the computer monitor displayed multiple channels continuously. In addition, each sleep laboratory

had a small video camera in the room so the sleep technician could observe each patient visually (Iber, Ancoli-Israel, Chesson, & Quan, 2007).

The electroencephalogram (EEG). Eight electrodes (six exploratory and two reference) were applied to each patient. The exploratory electrodes were attached to the frontal, central, and occipital areas of the patient's head using a conducting gel. These electrodes provided information on brain activity that was "scored" into different stages of sleep (Iber et al., 2007).

The electrooculogram (EOG). Two electrodes, one placed above the corner of the right eye and one below the corner of the left eye, monitored and recorded activity of the eyes. This information provided data relating to the onset of REM sleep (Iber et al., 2007).

The electromyogram (EMG). Six electrodes were applied, two to each anterior tibia of each leg to measure muscle tension (to determine periodic limb movement disorder) and two on the chin (one above the jaw line and one below to establish sleep onset and REM sleep) (Iber et al., 2007).

The electrocardiogram (ECG or EKG). For a standard PSG, two to three electrodes were applied either under the collar bone on each side of the chest or one under the collar bone and the other above either waist to record the electrical activity of the heart (i.e., "P" wave, "QRS" complex, and "T" wave) (Iber et al., 2007).

Respiration. Pressure transducers and/or a thermocouple were generally fitted near the nostrils to measure nasal and oral airflow. These were applied to measure the rate of respiration and identify interruptions in breathing. Belts that expanded and contracted upon breathing effort were also applied to measure respiratory effort (Iber et al., 2007).

Oximetry. A sensor was fitted over the finger or ear lobe to determine changes in blood oxygen levels that often occur with sleep apnoea and other respiratory problems (Iber et al., 2007).

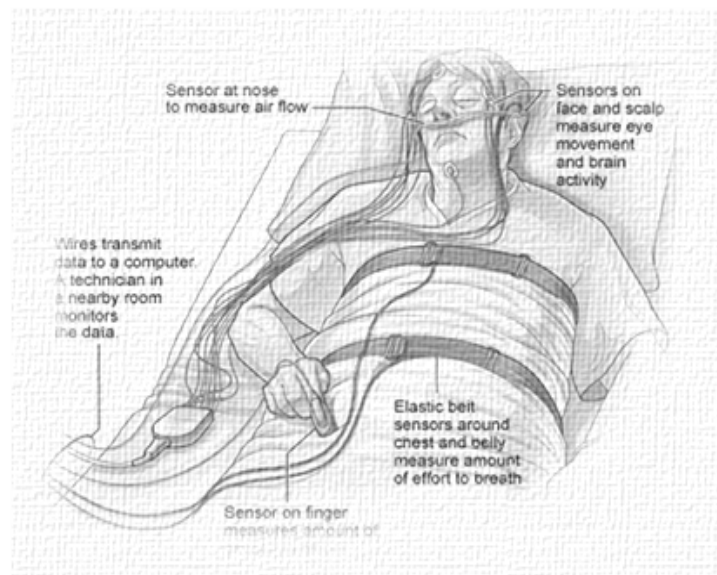


Figure 6. Patient connected to a polysomnography. Adapted from Benefits of a Polysomnography, Patrick, 2013, *Sleep Solutions*, Retrieved from <http://sleepsasolutions.com/benefits-of-a-polysomnography>.

On completion of the PSG, the results were collected and analysed to provide information on the following sleep-related data:

- Sleep efficiency—the number of minutes of sleep divided by the number of minutes in bed. This is normally calculated to be approximately 85 to 90% or higher;
- Sleep onset latency—onset of sleep from time the lights were turned off. This is normally less than 20 minutes and is measured by EEG results;
- Stage of sleep—determined by the data collected from the EEG, EOG, and EMG and recorded in 30-second epochs. It is normally recorded as “awake”, non-REM sleep (sleep Stages 1, 2, 3) or REM sleep. Non-REM sleep Stage 1 and 2 are referred to as “light sleep”, and sleep Stage 3 is referred to as “deep sleep” or “slow wave sleep” and is demonstrated by wide brain waves compared to other stages (see Figures 7 and 8; Table 12);
- Apnoeas and hypopnoea—identified by the complete or partial cessation of airflow for at least 10 seconds followed by an arousal and/or a decrease in oxygen desaturation;
- Arousals or sudden shifts in brain wave activity—generally as a result of breathing abnormalities, leg movements and environmental noises;
- Cardiac rhythm abnormalities;
- Leg movements;
- Body position during sleep; and
- Oxygen saturation during sleep;

Interpretations were made by a sleep medicine physician/doctor taking into account the patient's medical history, a complete list of medications the patient was taking, and any other relevant information that might impact the sleep study such as napping done before the PSG. A report was completed and sent to the referring physician with specific recommendations based on the test results.

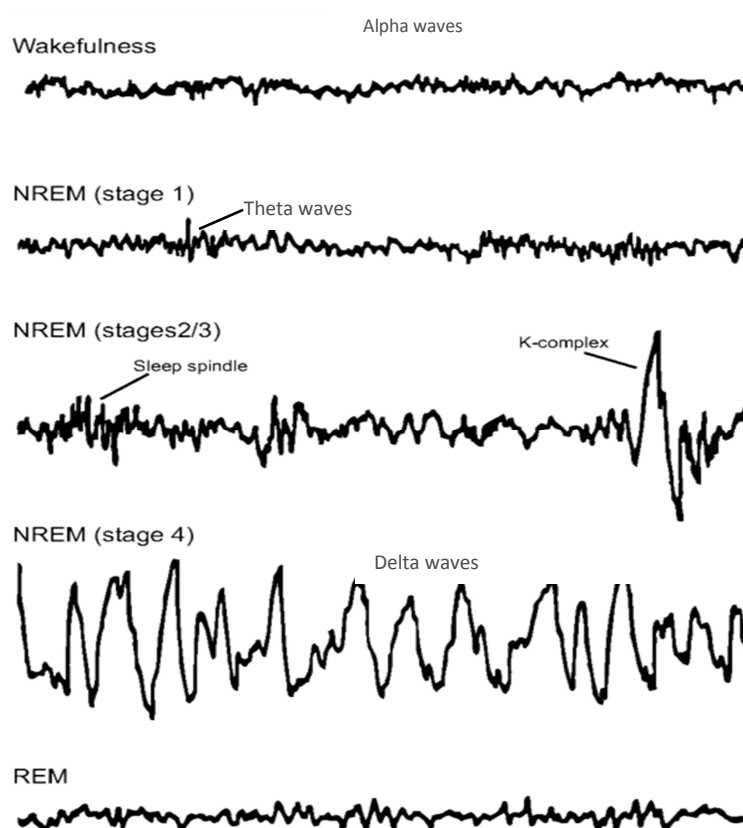


Figure 7. Polysomnography results. Results highlighting Alpha wave, Theta waves, Sleep spindles, K-complex and Delta wave from Stage 1 sleep through to Stage 4 sleep including REM. Adapted from Control of Sleep and Wakefulness, by R. Brown, R. Basheer, J. McKenna, R. Strecker, R. McCarley, 2012, *Physiological Reviews* 1(92), 1087-1187. Copyright 2014 by the American Physiological Society. Adapted with permission.

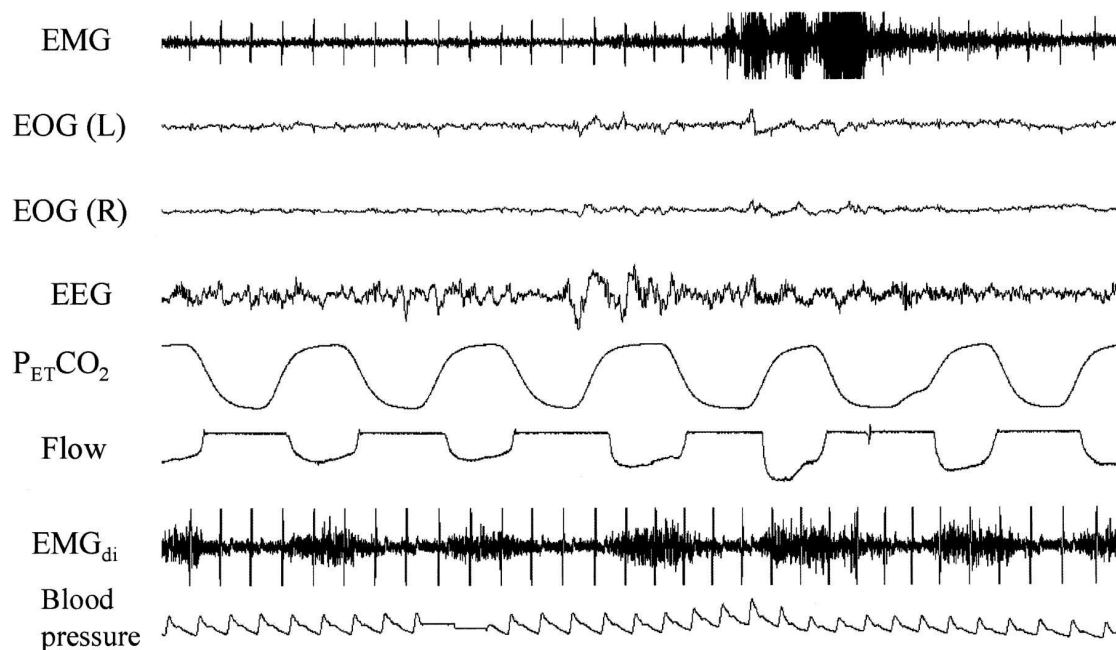


Figure 8: Parameters monitored during a PSG. The recorded variables are shown over 30-second epochs. Data exhibits passive breathing during stage 2 sleep, followed by an arousal (apnoea) resulting in increased EMG, EOG, and EEG activity. From Cardiac and respiratory activity at arousal from sleep under controlled ventilation conditions, by J. Trinder et al., 2001, *Journal of Applied Physiology*, 90(4), 1455-1463. Copyright 2001 the American Physiological Society. Reprinted with permission.

Table 12

Stages of Sleep

Stage	Total Sleep, %	EEG Wave	Defining Traits
1	2–5%	Theta wave	Light sleep, hypnic jerks, conscious awareness.
2	45–55%	Sleep spindles and K complexes	Consolidated sleep, loss of conscious awareness, slowed heart rate, decreased body temperature.
3 & 4	3–8%	Delta/slow wave	Deep/restorative sleep, difficult to arouse.
REM	20–25%	Alpha (wakefulness)	Dream sleep, paradoxical sleep, paralysis, high cortical activity.

CHAPTER 3**3.1 Results**

The data were collated and then analysed using the SPSS for Windows version 22.0. Four distinct analyses were conducted and included:

- 1) identification and treatment of missing values and outliers;
- 2) calculations of reliabilities for all corresponding measures along, with tests of assumptions to establish the validity for conducting various statistical analyses;

- 3) descriptive analyses of demographical information (using McNemar Chi-Square analysis), sleep-related variables and psychological factors incorporating a list of means, standard deviations, and percentages for comparative purposes; and
- 4) inferential analyses utilising Paired Samples t-tests, ANOVA, DA, ROC curve analysis, and MANOVA.

3.1.1 Treatment of Missing Values and Outliers

3.1.1.1 Missing values.

The data was inspected for missing values and outliers prior to the calculation of reliabilities and assumption testing. Frequency tables were generated to search for missing data and to determine the nature of missing data so that appropriate correctional procedures could be executed. Missing data was mostly found amongst the sleep-related variables collected at the diagnostic and implementation phases. This predominantly included adherence data gathered from the CPAP device meters (due to faulty equipment) as well as some incomplete O₂ saturation readings and AHI records. As such, the missing values were deemed to be missing completely at random (MCAR), suggesting that the probability that an observation (X_i) that was missing was unrelated to the value of X_i or to the value of other variables (Howell, 2007).

Given the small number of patients that completed both the diagnostic and implementation phases ($n = 69$), an imputation procedure, instead of deletion, using

Missing Value Analysis (MVA) to address values that were MCAR was utilised (Schafer, 1999). By examining the pattern, location, prevalence, significance, and randomness of the missing values within a data set, the MVA procedure used estimated means, standard deviations, co-variances, and correlations for imputation of missing values via a method of expectation-maximization (EM). The EM algorithm is a well-accepted approach in statistical estimation problems to gain the maximum likelihood estimators to treat values that are MCAR (Schafer, 1997; Schafer 1999; Schafer & Olsen 1998). It was not deemed appropriate to apply similar imputations procedures on the remaining data ($n = 87$) as missing values were identified as high.

To ensure the appropriateness of the MVA procedure in the present sample and to statistically confirm the nature of the missing values, Little's Chi-square statistic was used. Little's Chi-square statistic tests and confirms whether missing values are in fact MCAR on the premise that the null hypothesis is rejected—i.e., the values are MCAR, when the p value is significant at the 0.05 level. Thus, if the p value is less than 0.05, the data is noted as not MCAR and consideration needs to be given to it being missing at random or not missing at random and therefore an alternative approach to imputation might need to be considered. In this study, the results of Little's Chi-square statistic for MCAR were non-significant, which provided support and confidence that values were in fact MCAR (Little's MCAR test Chi-Square = 2.96.71 (292), $p = 0.412$).

Missing values were also noted amongst the collection of psychological factors explored in the present study. In this study, 44% of patients completed the battery of psychometric measures at the diagnostic phase and six months after the

implementation phase. The remaining 56% of patients only completed the psychometric measures at the diagnostic phase before discontinuing their participation and/or “dropping out”. Although the examination of frequency tables reveals that some data was missing in relation to psychological information, those patients that “dropped out” presented with the largest amount of missing data at the diagnostic phase compared to patients who completed the implementation phase and six month psychometric testing.

As the missing data amongst psychological factors in patients that “dropped out” posed a problem due to sample size and inability to conduct accurate and valid statistical analysis, a further imputation procedure was conducted. Further exploration of missing data, using a missing data pattern chart, suggested that the missing values amongst psychological factors were again missing at random. However, given the high likelihood that the missing values were in fact not missing at random, as data was predominantly missing amongst those who “dropped out” of the study, Multiple Imputation (MI) was used instead of MVA or other tradition imputation methods, such hot-deck imputation. MI is a procedure generally used to deal with missing data so that statistical analysis can continue (Schafer, 1997).

Multiple Imputation is a Monte Carlo technique common in biomedical, behavioral, and social sciences research to overcome the limitations of missing data (Schafer, 1997). This procedure averages outcomes across the existing data to replace missing values by $m > 1$ simulated versions, where m is typically small (Rubin, 1987). Similar to a stochastic regression procedure, several (five with the present data set) imputations are run for the same data multiple times. Using the SPSS, a separate

analysis was performed on each of the five completed imputations. This analysis took into account standard error (SE), which was calculated by adding the within and between variance of each of the five data sets, and the squared root of this figure was introduced into a final regression model.

3.1.1.2 Outliers.

Outliers in the present data set were detected via histograms, boxplots, and descriptive tables that were produced for this purpose. Inspection of the histograms provided information on normality of distribution while inspection of the respective boxplots provided information and location of identified outliers. In addition, inspection of the corresponding descriptive tables provided an indication of the extent of the problem associated with the identified outlier. This was done by comparing the original mean and the new trimmed mean values to determine whether extreme scores were likely to have a significant influence on the study's data set. Further investigations to determine whether the outliers were genuine or an error were conducted when:

1. histograms presented with tailed distributions;
2. outliers were identified on the boxplots; and/or
3. the two mean values were noted to be significantly different in the descriptive tables.

To reduce the chance of losing more data as a result of outliers, further reducing the present sample size, a winsorising procedure—as opposed to a trimming

procedure—was deemed an appropriate method for treating outliers. Winsorisation required identification of outliers and recoding these to their nearest acceptable value (upper or lower bound values). The winsorising procedure was only applied where outliers contributed to less than 20% of the variable (Lewis, 2007).

3.2 Alpha

Where possible and unless otherwise indicated, Alpha was set at 0.05. However, given the sample size, it was also deemed appropriate to report statistical significant at the 0.10 level to increase power (the probability of rejecting the null hypothesis when the alternative hypothesis is true, i.e., the probability of not committing a Type II error). The typical level of Alpha is commonly set at 0.05, but it is noted that this is simply a convention and is not based on any statistical science, theory, or criteria other than conventional practice (Noymer, 2008; Price, 2000). Amongst social science literature, an Alpha of 0.01, 0.05, and 0.10 are the most commonly used values, representing a 1%, 5%, and 10% chance of a Type I error occurring (i.e., rejecting the null hypothesis when it is in fact true). Within the context of exploratory studies where limited research is available, it is common to be more liberal with setting criteria around Alpha and to relax the level of significance to 0.10 or even 0.20 (Price, 2000).

3.3 Test of Assumptions

Prior to analysis, tests for the assumption of normality (Gaussian distribution), linearity, and homoscedasticity were also conducted, providing the justification for

utilisation of the various statistical procedures. There are two common methods for testing the assumptions of normality, linearity, and homoscedasticity in a sample: statistical tests and visual inspection of residual plots.

Statistical tests such as the Kolmogorov-Smirnov Test and the Shapiro-Wilks Test are frequently employed to test the assumption of normality, while correlations and Levene's statistics are common tests for linearity and homoscedasticity. Generally, statistical tests are preferred as they provide for objective judgement of the violation of assumptions. However, such statistical tests are sensitive to sample size, a limiting factor in the present study. Thus, this study initially used subjective visual inspection of residual plots including—normal Q-Q plots, scatter plots, and boxplots—to determine violations of normality, linearity, and homoscedasticity (Laerd Statistics, n.d.).

Interpretations of the normal Q-Q plot data points showed all points to be close to a diagonal line and not deviate greatly in a highly obvious non-linear fashion. Thus, the assumption of normality of all variables in the present sample was deemed to have been met. Inferential analyses for hypothesis testing provided information pertaining to linearity and homoscedasticity, and are provided in the respective sections within this chapter. It is noted that where assumptions for linearity and homoscedasticity were not deemed to have been met, exploratory analyses were conducted and reported where significant. This decision was based on the actuality that violations of the assumptions for linearity and homoscedasticity do not necessarily invalidate a statistical procedure, but tend more to weaken it (Tabachnick & Fidell, 2006).

3.4 Reliability Analyses

Confirmation of the reliability of all measures used was deemed necessary to ensure the integrity of the findings. While all measures used in the study have previously displayed positive moderate to strong reliabilities (see section 2.1.2) it was considered necessary to provide study specific psychometric properties exclusive to the present sample especially given the multivariate nature of the study. Such rigorous practises have been supported by academics and editors of respected journals who have recommended computing reliability coefficients for each administration of a measure for the specific population under study (DeVon et al., 2007).

Reliability analyses were calculated for each of the measures used in the present study. Given the nature of the variables and study design, Cronbach's Alpha was utilised to measure internal consistency for the POMS, MHLC-C, GSE, EPQ-RS, and Health Belief measure at the diagnostic and implementation phase. Cronbach's Alpha is expressed as a number between one and zero. While there are different reports about the acceptable values of Alpha, the general consensus is that an acceptable range is 0.70 to 0.90 (Nunnally & Bernstein, 1994; Bland & Altman, 1997). Internal consistency describes the extent to which all items in a test measure the same concept and/or construct, and it is therefore connected to the inter-relatedness of the test items. Given the study design (pre- and post-testing), internal consistency was calculated for data gathered at the diagnostic phase and again for data gathered six months after the implementation phase. Cronbach's Alpha reliabilities are reported in Table 13.

Table 13

Reliability Coefficients for the POMS, EPQ-RS, MHLC-C, GSE, and Health Belief

Measure at the Diagnostic and Implementation Phases (n = 69)

	Diagnostic		Implementation	
	Cronbach's	M(SD)	Cronbach's	M(SD)
	Alpha		Alpha	
POMS				
<i>Tension-Anxiety</i>	0.88	12.91(5.34)	0.92	13.36(6.05)
<i>Depression-Dejection</i>	0.92	14.24(6.39)	0.95	14.55(7.63)
<i>Anger-Hostility</i>	0.89	13.27(5.49)	0.93	13.62(6.34)
<i>Vigour-Activity</i>	0.90	14.04(4.71)	0.90	15.10(5.02)
<i>Fatigue-Inertia</i>	0.89	15.01(5.20)	0.92	12.65(5.40)
<i>Confusion-Bewilderment</i>	0.82	10.56(4.19)	0.90	10.07(4.86)
<i>TMD</i>	0.94	51.97(24.4)	0.95	49.15(31.3)
EPQ-RS				
<i>Neuroticism</i>	0.81	6.30(3.37)	0.80	6.11(3.34)
<i>Extraversion</i>	0.74	5.47(2.59)	0.53	6.08(2.18)
<i>Psychoticism</i>	0.41	5.00(1.48)	0.02	5.37(1.30)
<i>Lie</i>	0.60	5.56(2.04)	0.42	5.86(1.94)
MHLC- C				
<i>Internal</i>	0.76	25.49(6.41)	0.65	24.3(5.51)
<i>Chance</i>	0.83	15.73(6.65)	0.77	16.4(6.24)
<i>Doctors</i>	0.40	14.49(2.64)	0.61	13.4(2.81)
<i>Powerful Others</i>	0.55	9.88(3.62)	0.81	9.7 (4.13)
GSE	0.91	29.79(5.24)	0.91	29.4(5.88)

Health Belief

<i>Perceived Susceptibility</i>	0.58	15.43(2.03)	0.63	15.07(2.37)
<i>Perceived Severity</i>	0.59	13.27(1.79)	0.61	12.95(2.71)
<i>Perceived Benefits</i>	0.89	11.36(1.94)	0.76	11.24(4.36)
<i>Perceived Barriers</i>	0.74	9.34(2.85)	0.73	14.52(9.34)

Table 13 shows strong reliability coefficients for all the sub-scales within the POMS-SF, with Cronbach's Alpha ranging from 0.82 to 0.94. Similar results were noted for the GSE, which yielded a Cronbach's Alpha reliability coefficient of 0.91. Reliability coefficients for the MHLC-C, EPQ-RS, and Health Belief measures show mixed results ranging from 0.41 to 0.89. Excluding the Health Belief measure, the aforementioned reliability coefficients were comparable with those reported in previous reliability studies identified in Section 2.1.2 of the Methods.

The neuroticism sub-scale from the EPQ-RS yielded a strong reliability coefficient of 0.81. It is noted that the extraversion, psychoticism, and lie sub-scales were questionable before item deletion, with reliability coefficients ranging from 0.33 to 0.63. Following deletion of the seventh item in the extraversion and psychoticism sub-scales and the tenth item in the lie sub-scale, reliability increased and ranged from 0.41 to 0.74. The sub-scale psychoticism, however, remained questionable.

Internal consistency for the sub-scales of internal and chance on the MHLC-C yielded moderate to strong reliability coefficients ranging from 0.76 to 0.83. Prior to item deletion, the sub-scales doctors and other people showed questionable reliability coefficients of 0.40 and 0.55 respectively. Following deletion of the first item in the

doctor sub-scale and the second item in the other people sub-scale, reliability slightly increased to 0.40 and 0.56 respectively but such increase was noted to be minimal and non-effective. While these items remained questionable, they were in line with the reliabilities coefficient reported in previous studies identified in Section 2.1.2 of the Methods.

Cronbach's Alpha coefficients for the Health Belief measure were also questionable, with reliability prior to item deletion initially ranging from -0.20 to 0.74. Moderate to high reliabilities ranging from 0.58 to 0.89 were obtained following deletion of items four, five, nine, and 15 within the perceived susceptibility, perceived severity, perceived benefits, and perceived barriers sub-scales respectively.

Given the repeated design of the present study, the Cronbach's Alpha reliability coefficients were also calculated following administration of the psychometric measures at the six months mark following the implementation phase for comparative purposes (see Table 13). Associated means and standard deviations displayed minimal changes between the diagnostic phase and six months following the implementation phase suggesting strong consistency and little variation in reliability.

3.5 Statistical Analysis

3.5.1 Patient characteristics

Of the 156 patients who initially participated in this study, 69 patients completed the same psychometric test battery at the diagnostic phase and then again six months after the implementation phase. The remaining 87 patients only completed psychometric testing at the diagnostic phase, “dropping out” prior to the implementation phase. No exclusion criteria were set for this study. The total mean age was 50.3 years ($SD = 10.97$) with an age range of 25 to 74 years. Mean age for women was 52.37 ($SD = 11.86$) and 49.28 ($SD = 10.44$) for men. Patients were deemed adherent if they completed both the diagnostic and implementation phases. Patients initially grouped as adherent displayed between four and six hours of CPAP use per night. Further breakdown of the patient demographic characteristics are provided in Figure 9 and Table 14.

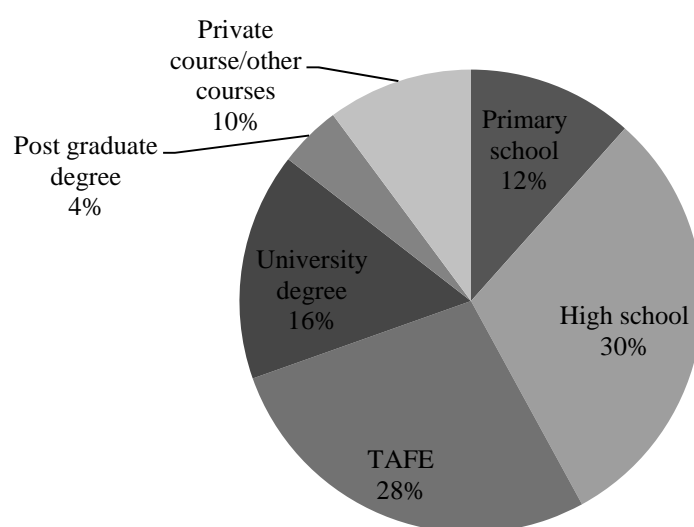


Figure 9. Educational level of the study participants.

Figure 9 highlighted that a majority of the patients reported achieving a high-school education (30%) and/or completion of TAFE (28%) as their highest completed level of schooling. Information elicited at the diagnostic phase describing patient perceptions in relation to OSA and CPAP was analysed using McNemar Chi-Square. McNemar Chi-Square analysis, using the Binomial test with Yates correction for continuity, requires variables to be binominal in nature (i.e., “yes”, “no” nominal responses) and paired. Table 14 displays the marginal frequencies associated with the demographic questions.

Table 14

Perceptions of OSA and CPAP Use at Diagnostic and Implementation Phases (n = 69)

	Diagnostic		Implementation		<i>p</i>
	Yes	No	Yes	No	
	(%)	(%)	(%)	(%)	
Do you suffer from any other illness apart from OSA?	62.3	37.7	43.5	6.5	0.01
Do you believe you actually have OSA?	94.2	5.8	72.5	27.5	0.00
Do you THINK it will be difficult to use CPAP?	46.4	53.6			
Are you currently using CPAP for your OSA treatment?			55.1	44.9	
Do you THINK it will be uncomfortable to use CPAP?	75.4	24.6	55.1	44.9	0.02

Do you THINK CPAP treatment					
alleviates OSA symptoms for you?	92.8	7.2	68.1	31.9	0.005
Did you receive specific					
instructions on how to use your			87.0	13.0	
CPAP machine?					
If yes, did you understand the					
instructions on how to use your			92.8	7.2	
CPAP machine?					

Table 14 shows that a statistically significant result was reported on responses to the demographic question “Do you suffer from any other illness apart from OSA?” It was noted that at the diagnostic phase 62.3% of patients responded “yes” to this question and the response percentage showed a significant decrease to 43.5% when asked again six months after the implementation phase. Similar results were recorded for the demographic question “Do you believe you actually have OSA?” with 94.2% of patients responding “yes” at the diagnostic phase compared to a significant decrease (72.5%) six months after the implementation phase. A significant decrease in percentage between the diagnostic phase and six months after the implementation phase was also recorded for the demographic question “Do you THINK it will be uncomfortable to use CPAP?” with 75.4% of patients responding “yes” at the diagnostic phase compared to 55.1% responding “yes” six month following the implementation phase. Furthermore, a McNemar Chi-Square analysis conducted on the demographic question “Do you THINK CPAP treatment will alleviate OSA symptoms for you?” yielded statistical significant results with 92.8% of patients

responding “yes” to this question at the diagnostic phase compared with 68.1% six months following the implementation phase.

McNemar Chi-Square analysis was not conducted for the following demographic questions: “Do you THINK it will be difficult to use CPAP?”, “Are you currently using CPAP for your OSA treatment?”, “Did you receive specific instructions on how to use your CPAP machine?”, and “If yes, did you understand the instructions on how to use your CPAP machine?” as these questions were asked in isolation and therefore did not meet the requirement of this analysis of being paired.

3.5.2 Examination of Sleep-Related Variables

Data on sleep-related variables were collected at the diagnostic and implementation phases. The variables selected for investigation included PSG data and were based on previous key research papers (e.g., Ohayon et al., 1997; Sage et al., 2001; Wiese et al., 2005; Krishnan & Collop, 2006; Amodio et al., 2008; Casale et al. 2008; Olsen et al., 2008; Tzischinsky et al., 2011). Sleep-related variables included:

- Sleep Efficiency (normal ≥ 85);
- Sleep Latency (normal ≥ 15 minutes);
- NREM O₂ saturation–blood oxygen saturation measured during non-rapid eye movement sleep (normal $\geq 94\%$);
- REM O₂ saturation–blood oxygen saturation measured during rapid eye movement sleep (normal $\geq 94\%$);

- NREM AHI–apnoea-hypopnea index gathered during non-rapid eye movement sleep (normal ≤ 15);
- REM AHI–apnoea-hypopnea index gathered during rapid eye movement sleep (normal ≤ 15); and
- BMI–Body Mass Index (normal = 18.5 to 24.9).

A summary of the data pertaining to the sleep-related variables collected at the diagnostic and implementation phases is shown in Table 15.

Table 15

Means, Standard Deviations and Percentage for the Sleep-Related Variables (Sleep Efficiency, Sleep Latency, O₂ saturation, AHI, BMI, and Sleepiness) at the Diagnostic and Implementation Phases

	Diagnostic		Implementation		<i>p</i>
	<i>M (SD)</i>	%	<i>M (SD)</i>	%	
Sleep Efficiency	60.7 (29.7)	81.2	62.2 (29.1)	82.6	.434
Sleep Latency	26.2 (29.6)	31.9	25.7 (29.6)	31.9	.216
NREM O ₂ saturation	73.3 (34.7)	71.0	78.6 (34.8)	34.8	.0005
REM O ₂ saturation	79.6 (30.4)	56.5	86.8 (27.1)	15.9	.0005
NREM AHI	38.1 (33.6)	94.2	8.8 (14.8)	53.6	.0005
REM AHI	43.0 (28.9)	97.1	11.4 (19.9)	69.6	.0005
BMI	31.2 (11.0)	92.8	30.8 (10.7)	95.7	.972
Sleepiness	9.79 (4.74)	59.4	9.55 (4.46)	58.0	.643

Table 15 shows the associated means and standard deviations as well as the percentage of patients who recorded PSG results outside of the normal ranges at the diagnostic and implementation phases. In order to identify and compare any significant mean differences between the sleep-related variables at the diagnostic and implementation phases, a series of Paired Samples t-tests were conducted. Assumptions for this statistical procedure ensured that the dependent variable was continuous in nature; the independent variable consisted of at least two categorical, related groups or matched pairs; there be no significant outliers in the differences between the two related groups; the sample was approximately normally distributed; and the variances of the differences between all combinations of related groups must be equal, known as sphericity.

Paired Samples t-tests determined that the mean for NREM O₂ ($t(68) = -7.22$, $p = .0005$) and REM O₂ saturation ($t(68) = -6.36$, $p = .0005$) showed a statistically significant difference between the diagnostic and implementation phases. Observation of mean changes noted in Table 15 suggests a positive moderate significant increase in oxygen saturation in patients following CPAP implementation. Specifically, 71% of patients initially recorded a NREM O₂ saturation within the mild to severe range at the diagnostic phase, a figure that largely and positively decreased to 34.8% and placed patients within the normal range after CPAP implementation. Similar results were recorded for REM O₂ saturation, with 56.5% of patients having a REM O₂ saturation within the mild to severe range at the diagnostic phase compared to 15.9% at the implementation phase. With CPAP use, this change showed a large positive decrease in the number of patients who still presented with concerning oxygen saturation levels.

In addition, a further set of Paired Samples t-test also determined that the mean for NREM AHI ($t(68) = 10.88, p = .0005$) and REM AHI ($t(68) = 10.45, p = .0005$) showed a statistically significant difference between the diagnostic and implementation phases. The direction of mean change noted in Table 15 shows a significant change in AHI following CPAP implementation. At the diagnostic phase, 94.2% of patients had an NREM AHI within the mild to severe range compared to 53.6% following CPAP implementation. This shows a significant moderate to large positive decrease in NREM AHI. The REM AHI also yielded statistically significant results, with 97.1% of patients having a REM AHI within the mild to severe range at the diagnostic phase compared to 69.6% at the implementation phase following CPAP use. There was therefore a significant moderate to large positive decrease in REM AHI. No differences were noted in the remaining sleep-related variables following CPAP implementation.

3.5.3 Exploration of Psychological Factors

Psychological factors were collected from the range of self-reported psychometric measures administered at the diagnostic phase and again six months following the implementation phase. The variables selected for investigation included:

- Mood (tension/anxiety, depression/dejection, anger/hostility, fatigue/inertia, vigour/activity, and confusion/bewilderment);
- Personality (neuroticism, extraversion, psychoticism, and lie);
- Self-Efficacy;

- Health Locus of Control (internal, chance, doctor and powerful others);
- Health Belief (perceived susceptibility, perceived severity, perceived benefits, and perceived barriers).

A detailed breakdown and description of the psychological factors collected at the diagnostic phase and six months after the implementation phase are displayed in Table 16.

Table 16

Means, Standard Deviations and Percentages Associated with Psychological Factors (Mood, Personality, General Self-Efficacy, Health Locus of Control, Health Belief) at the Diagnostic Phase and Six Months following the Implementation Phase (n = 69)

	Diagnostic		Implementation		<i>p</i>
	<i>M (SD)</i>	%	<i>M (SD)</i>	%	
Mood					
<i>Tension/Anxiety</i>	6.85 (5.17)	17.4	6.46 (4.00)	17.4	.570
<i>Depression/Dejection</i>	6.21 (6.32)	14.5	6.21 (6.82)	15.9	1.00
<i>Anger/Hostility</i>	6.17 (5.20)	10.1	6.60 (6.30)	14.5	.617
<i>Vigour/Activity</i>	7.97 (4.51)	78.3	9.10 (5.02)	66.7	.097
<i>Fatigue/Inertia</i>	10.01 (5.20)	50.7	7.62 (5.34)	26.1	.003
<i>Confusion/Bewilderment</i>	5.50 (4.04)	20.3	4.76 (4.16)	20.3	.219
<i>TMD</i>	26.89 (24.29)	20.3	22.85 (28.13)	18.8	.304
Personality					
<i>Neuroticism</i>	6.69 (2.97)	52.2	6.59 (2.86)	49.3	.823
<i>Extraversion</i>	5.47 (2.59)	39.1	6.08 (2.18)	42.0	.083

<i>Psychoticism</i>	5.00 (1.48)	14.5	5.38 (0.95)	15.9	.035
<i>Lie</i>	5.56 (2.04)	34.8	5.86 (1.94)	40.6	.339
Health Locus of control					
<i>Internal</i>	25.75 (5.95)	1.4	24.51 (4.66)	0.0	.069
<i>Chance</i>	15.73 (6.65)	46.4	16.46 (6.24)	56.5	.437
<i>Doctors</i>	14.49 (2.64)	0.0	13.39 (2.81)	0.0	.002
<i>Powerful Others</i>	9.88 (3.62)	79.7	9.76 (4.13)	72.5	.830
Self-Efficacy	30.18 (4.61)	0.0	29.95 (5.02)	0.0	.714
Health Belief					
<i>Perceived Susceptibility</i>	15.43 (2.03)	0.0	15.07 (2.37)	0.0	.283
<i>Perceived Severity</i>	13.27 (1.79)	4.3	12.95 (2.71)	8.7	.001
<i>Perceived Benefits</i>	11.36 (1.94)	0.0	11.24 (4.36)	7.2	.699
<i>Perceived Barriers</i>	9.34 (2.85)	5.8	14.52 (9.34)	7.2	.720

Table 16 shows the means and standard deviations associated with the data collected pertaining to the psychological factors at the diagnostic phase and six months after the implementation phase along with corresponding percentages depicting response patterns. To identify and compare any significant mean differences amongst the psychological factors collected at the diagnostic phase and six months following the implementation phase, a series of Paired Samples t-tests were conducted. Assumptions for this statistical procedure ensured that the dependent variable was continuous in nature; the independent variable consisted of at least two categorical, related groups or matched pairs; there be no significant outliers in the differences between the two related groups; the sample was approximately normally

distributed; and the variances of the differences between all combinations of related groups must be equal, known as sphericity.

A Paired Samples t-test determined that the mean score for the self-reported mood sub-scales vigour/activity ($t(68) = -1.68, p = .097$) and fatigue/inertia ($t(68) = 3.12, p = .003$) showed statistically significant differences between the diagnostic phase and six months after the implementation phase. Observation of the mean changes noted in Table 16, suggests a positive significant increase in patients' energy levels (as measured by the POMS sub-scale vigour/activity) following CPAP implementation. Specifically, 78.3% of patients initially reported that their energy and activity levels were within the mild to severe range at the diagnostic phase, a figure that moderately decreased to 66.7% suggesting that reported energy levels were higher following implementation of CPAP. Similar positive results were seen for the POMS sub-scale of fatigue/inertia, which measured patients' degree of overall fatigue or exhaustion. At the diagnostic phase, 50.7% of patients reported their fatigue levels to be within the mild to severe range compared to 26.1% six months following the implementation phase. This shows a large decrease in patients' self-reported fatigue following CPAP implementation.

A second Paired Samples t-test determined that the mean score for the health locus of control sub-scales of internal ($t(68) = 1.84, p = .069$) and doctors ($t(68) = 3.28, p = .002$) yielded statistically significant differences between the diagnostic phase and six months following the implementation phase. Examination of the data presented in Table 16, shows that while a majority of patients (99.6%) reported having control over their OSA treatment (having an internal locus of control),

following CPAP implementation 100% self-reported having an internal health locus of control, suggesting a slight yet significant increase. Similarity, with relation to the health locus of control sub-scale doctors 100% of patients self-reported relying on their physicians'/doctors' advice regarding their OSA and CPAP treatment. Mean between comparisons from the diagnostic phase and six months after the implementation phase showed a slight yet significant mean decrease with 100% of patients still reporting a reliance on their doctors and or physicians.

A third Paired Samples t-test determined that the mean score for the self-reported measure of personality, in particular the sub-scales extraversion ($t(68) = -1.75, p = .083$) and psychoticism ($t(68) = -2.15, p = .035$), also showed a statistically significant difference between the diagnostic phase and six months following the implementation phase. Specifically, 39.1% of patients reported being extraverted at the diagnostic phase compared to 42% following the implementation phase. This result suggests a slight yet significant improvement in relation to patients "outgoingness" and social interactions following CPAP implementation. Similarity, a slight significant increase in patients' psychoticism scores was also shown in Table 16, where 14.5% of patients reported traits associated with sensation seeking and/or risk taking at the diagnostic phase compared to 15.9% following the implementation phase.

The forth Paired Samples t-test showed that the mean score for the perceived severity sub-scale of the health belief measure ($t(68) = 3.36, p = .001$) was statistically significantly different between the diagnostic phase and six months following the implementation phase. Table 16 shows that 4.3% of patients may not have been fully

aware of the perceived risks or seriousness associated with OSA at the diagnostic phase—a figure that slightly and significantly increased following the implementation phase—even though slightly more patients reported (8.75%) less understanding of that potential seriousness of their symptoms following CPAP implementation.

No further statistical significant findings were noted amongst the remaining psychological factors that were measured at the diagnostic phase and six months following the implementation phase. A detailed account of the significant results achieved at the diagnostic phase and six months following the implementation phase will be further explored in relationship to existing research and literature in the Discussion.

3.5.3.1 Psychological predictors of CPAP adherence.

To explore the psychological factors that best predicted adherence and/or non-adherence to CPAP use, a stepwise DA was conducted. Predictive DA addresses the question of how to assign cases to groups. The DA function uses an individual's scores on the predictor variables to predict the category in which the individual belongs. In the present study, patients were classified *a priori* into two groups, adherent and non-adherent, determined by completion of the diagnostic and implementation phases of the study (i.e., patients who only completed the diagnostic phase were categorised as non-adherent, and patients who completed both diagnostic and implementation phases were categorised as adherent).

A stepwise estimation procedure was used to predict group membership in either the adherent or non-adherent categories and explore the impact of the following psychological factors: mood, personality, self-efficacy, locus of control, and health belief. The stepwise estimation procedure was employed. The stepwise procedure involves entering the psychological factors into the discriminant function one at a time on the basis of their discriminating power. Eventually, either all independent variables will have been included in the function or the excluded variables will have been judged as not contributing significantly to further discrimination.

Assumption testing determined the appropriateness of DA and included tests for normality, non-multicollinearity, and the absence of outliers. It must be noted that the assumption of homogeneity of variances/co-variances was not supported; however, given that the multivariate Box's M test for homogeneity is particularly sensitive, the stepwise DA procedure was still carried out (Tabachnick & Fidell, 2006). Initial results of the stepwise DA procedure are shown in Table 17.

Table 17

Stepwise Statistics Loadings Ranked According to Relative Importance of the Predictors for CPAP Adherence and Non-Adherence

Entered	Function	<i>p</i>
Anger/Hostility	.624	.0005
Vigour/Activity	.530	.0005
Self-Efficacy	.491	.0005
Internal Health Locus of Control	.465	.0005
Perceived Susceptibility	.442	.0005

Depression/Dejection	.424	.0005
Perceived Benefits	.412	.0005

Statistically significant predictor variables entered into the model ranked according to their discriminating power included anger/hostility, self-efficacy, internal health locus of control, perceived susceptibility, depression/dejection, and perceived benefits. Significant mean differences were observed for all identified predictors. The discriminant function revealed a significant association between groups and all predictors, accounting for 58.8% of between-group variability. The absence of the remaining psychological factors meant that they provided no statistically significant loading on the discriminant function, and they were therefore deemed to have poor predictive value and were excluded from the model. The cross-validated classification showed that overall, 87.8% of patients were correctly classified, with predictions for the non-adherent group (92%) being more accurate than those for the adherent group (82.6%). In order to determine if discrimination was clinically significant as well as statistically significant, that is, the ability of the discriminate function to correctly classify those that are CPAP adherent a ROC curve was also constructed and displayed in Figure 10.

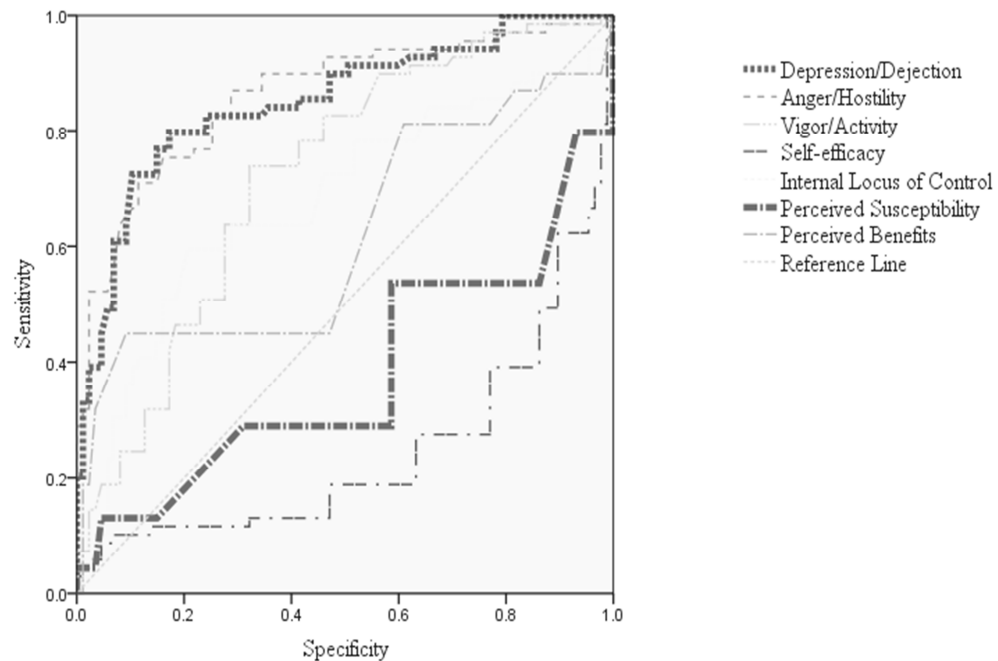


Figure 10. ROC curve differentiating clinically significant psychological predictors of CPAP adherence.

Figure 11 provides a graphical representation of the sensitivity and specificity for clinical significance. The accuracy is expressed as the area under the ROC curve (AUC) and provides a useful parameter for comparing the statistically significant psychological factors that predict CPAP adherence from the DA. The results from the ROC curve showed strong clinical significance associated with the psychological factors depression/dejection (AUC = .852), anger/hostility (AUC = .862), and vigor/activity (AUC = .724); moderate clinical significance was associated with the psychological factors for internal locus of control and perceived benefits, while weak clinical significant results are exhibited for the psychological factors self-efficacy and perceived susceptibility.

To further explore mean difference at the diagnostic phase amongst the identified psychological predictors from the stepwise DA procedure, a MANOVA was conducted. The MANOVA procedure highlights any specific mean differences between the CPAP-adherent and non-adherent groups in relation to the identified predictors. Assumptions testing mirrored those from the DA and the outcome of the MANOVA is shown in Table 18.

Table 18

Means and Standard Deviations for Identified Predictor Variables for the Adherent (n = 69) and Non-Adherent Groups (n = 87)

	Adherent	Non-adherent	<i>p</i>
	M (SD)	M (SD)	
Anger/Hostility	13.27 (5.49)	21.87 (5.57)	.0005
Vigour/Activity	14.04 (4.71)	19.35 (6.08)	.0005
Self-Efficacy	29.79 (5.24)	25.59 (3.59)	.0005
Internal Health Locus of Control	25.49 (6.41)	29.45 (4.75)	.0005
Perceived Susceptibility	15.43 (2.03)	14.66 (1.23)	.004
Depression/Dejection	14.24 (6.39)	23.49 (6.97)	.0005
Perceived Benefits	11.36 (1.94)	12.11 (1.32)	.005

Results from the MANOVA yielded a statistically significant difference amongst the identified predictive psychological factors based on a patient's group membership to the CPAP-adherent or non-adherent group ($F(7, 148) = 30.166, p = .0005$; *Wilk's Λ* = 0.412, partial $\eta^2 = .58$). Specifically, means and ANOVA

(conducted for comparative purposes) results showed that the greatest mean difference is noted on the mood sub-scales anger/hostility ($F(1,68) = 92.746, p = .0005$) and depression/dejection ($F(1,68) = 72.742, p = .0005$). This result suggests that patients who were identified as non-adherent reported more symptoms associated with these mood states than the adherent group. The remaining psychological factors—vigour/activity ($F(1,68) = 35.628, p = .0005$), self-efficacy ($F(1,68) = 35.100, p = .0005$), internal health locus of control ($F(1,68) = 19.604, p = .0005$), perceived susceptibility ($F(1,68) = 8.409, p = .004$), and perceived benefits ($F(1,68) = 8.086, p = .005$)—showed significant yet small mean differences between groups. Interestingly, non-adherent patients at the diagnostic phase reported more vigour/activity, a greater sense of internality, and slightly better understanding of the perceived benefits associated with CPAP use to treat OSA. The above results are further discussed in greater detail in the Discussion.

CHAPTER 4

4.1 Discussion

Over the last three decades much of the research surrounding OSA and CPAP use has focused on improving poor adherence to treatment. Much of the earlier research attributed poor CPAP adherence to a range of sleep-related variables that have been shown to have low predictive validity when considering CPAP adherence (Ohayon et al., 1997; Wiese et al., 2005; Krishnan & Collop, 2006; Amodio et al., 2008; Casale et al., 2008). However, recent research has shifted its attention to identifying psychological factors that may possibly contribute to poor CPAP adherence (Kribbs et al., 1993; Engleman et al., 1994; Meurice et al., 1994; Reeves-

Hoche et al., 1994; Engleman et al., 1996; Massie et al., 1999; McAdle et al., 1999; Rosenthal et al., 2000). It is widely accepted that untreated moderate to severe OSA poses a serious long-term threat to health. Thus, the aim of the present study was to explore a range of psychological factors that are likely to predict non-adherence and adherence to CPAP use.

Specifically, this study focused on examining the psychological factors mood, personality, self-efficacy, health locus of control, and health belief as predictors of adherence to CPAP use. In order to achieve the study's aim, 156 patients with moderate to severe OSA from the respiratory departments at Southern Health and Austin Health were invited to participate in the present study. Patients consented to data being collected from a PSG at a diagnostic phase and implementation phase. Sleep-related variables (sleep efficiency, sleep latency, AHI, O₂ saturation, BMI, and sleepiness score from the ESS) were collected at the diagnostic and implementation phases. Data pertaining to the psychological factors (collected using the POMS-SF, EQP-RS, GSE, MHLC-C, and Health Belief measure), were collected at the diagnostic phase and six months following the implementation phase. Exploration of the psychological factors mood, personality, self-efficacy, health locus of control, and health belief were carried out to explore their significance in predicting adherence and non-adherence to CPAP use. Literature surrounding OSA and CPAP adherence and adherence literature pertaining to other common chronic illnesses and diseases was also explored (see Section 1.8: Psychological Determinants of CPAP Adherence).

4.2 Review of Exploratory Questions and Explanation of Findings

4.2.1 Characteristics of the sample

For a long time, OSA sufferers have been characterised as obese, middle-aged men with excessive daytime sleepiness (Guilleminault et al., 1978). The current sample reflected these typical characteristics. Making up the majority of the sample were men (65%) with a mean age of 49 years and a mean BMI of 32, which placed patients within the obese range. Similarly, women presented with a mean age of 52 years and a mean BMI of 34, also placing them within the obese range. OSA severity was determined by AHI, which placed approximately 90% of patients in the moderate to severe range for OSA at the diagnostic phase. Polysomnography data collected at the diagnostic phase also showed that patients in this study presented with an average NREM and REM O₂ saturation of 74% and 77% respectively—below what is considered acceptable (normal = > 94%).

While on the whole patients were deemed to adhere to CPAP use—determined by adequate CPAP usage of greater than four hours per night and improved changes in sleep-related variables—no statistically significant differences in BMI or sleepiness were noted between the diagnostic and the implementation phases. However, as patients were requested to return for the follow-up PSA several weeks following the initial diagnostic phase, there may not have been enough time to observe any reduction in BMI. Such a reduction is probably only likely to occur with an overall OSA management plan incorporating diet, education, exercise, efficient CPAP usage, and ongoing support/intervention. It is also important to note that in agreement with

existing OSA literature (e.g., Popescu et al., 2001), only 55% of patients in this study continued CPAP use following the diagnostic phase. This figure is also in line with current CPAP adherence rates reported in patients with moderate to severe OSA as well as literature surrounding treatment adherence in other chronic diseases and illnesses such as HIV/AIDS and diabetes (Bandolier, 2004; WHO, 2003; Collard et al., 1997).

Patients in the present study presented with minimal negative symptoms associated with mood. Fewer than 20% presented with anxiety, depression, anger, and/or confusion as measured by the POMS-SF. Given the low percentage of negative symptoms reported by these patients, it is probable that they were experiencing acute, short-term, or situational symptoms associated with a mood disorder rather than chronic symptoms. It is therefore highly likely that having a sample with minimal associated symptoms of mood disorders contributed to the higher than expected adherence rates in the present study. The main reported symptoms associated with mood were low levels of vigour and high levels of fatigue, which are symptoms commonly described by moderate to severe OSA sufferers.

Interestingly, patients in the present study reported mixed beliefs relating to the type of control they had over their condition and treatment as measured by the MHLC-C. On the whole, they reported having both a healthy level of internal and external health locus of control, and all reported a reliance on doctors (external health locus of control). While this result will be explored further within this chapter, it is highly probable that given the nature of OSA, relying on health professionals/specialists such as respiratory physicians and doctors would contribute

to high adherence rates. Patients also presented with a high level of self-efficacy, and therefore their perception regarding their ability to succeed with CPAP treatment was deemed high. It was also noted that while only 17% of patients in this study reported symptoms of anxiety, 52% described having personality traits associated with neuroticism. It is likely that while patients' reliance on the support of their respiratory physicians was beneficial, having some degree of anxiety may have also allowed them to respond appropriately to their OSA symptoms or helped motivate them to adhere to CPAP use. In contrast, a high degree of neuroticism and/or anxiety may cause the patient to be too fearful to act and follow through with the implementation phase and CPAP use.

Those patients who "dropped out" of the study reported higher levels of negative symptoms associated with mood as well as their overall level of distress when compared with adherent patients. Non-adherent patients also reported lower self-efficacy when compared with patients who followed through with the implementation phase and completion of psychometric measure six months later. Although the "drop outs" represented 56% of the sample, this figure may indicate that "drop outs" differ from patients who continued to attend (whether adherent or not). As in other clinical studies, those who agree to take part in and then complete the study are, to some extent, a self-selected group. Therefore, adherence criteria were re-established and based on participation and completion of the diagnostic and implementation phases. Nonetheless, patient characteristics in this sample mirror those commonly reported for the OSA population in previous reported research literature.

4.2.2 Exploratory Question One

Exploration of a combination of psychological factors—mood, personality, self-efficacy, locus of control, and health belief—will predict adherence and non-adherence to CPAP use.

The results from the present study utilising DA highlighted the predictive value of the following combined psychological factors in predicting adherence and non-adherence to CPAP use: mood; self-efficacy, locus of control, and health belief. Specifically, the results show that the mood sub-scales anger/hostility, vigour/activity, and depression/dejection; self-efficacy; internal health locus of control; and perceived susceptibility and benefits sub-scales of the health belief measure were significant predictors of CPAP adherence and non-adherence. The combination of statistically significant psychological predictors accounted for 59% of between-group variability. The remaining psychological factors and sub-scales were deemed to have poor predictive powers. These results support the findings of research conducted on other chronic diseases and illnesses in relation to treatment adherence (Sherbourne et al., 1992; Edinger et al., 1994; Norman & Bennett, 1995; Connor & Norman, 1996; DiMatteo et al., 2000; McFadyen et al., 2001; Stepnowsky et al., 2002; Stepnowsky et al., 2002; Wild et al., 2004; Aloia et al., 2005; Olsen et al., 2008). In particular, the results provide considerable evidence to suggest that psychological factors play an important predictive role in treatment adherence to CPAP use. Figure 11 displays the comparison profile between the CPAP adherent group and non-adherent group.

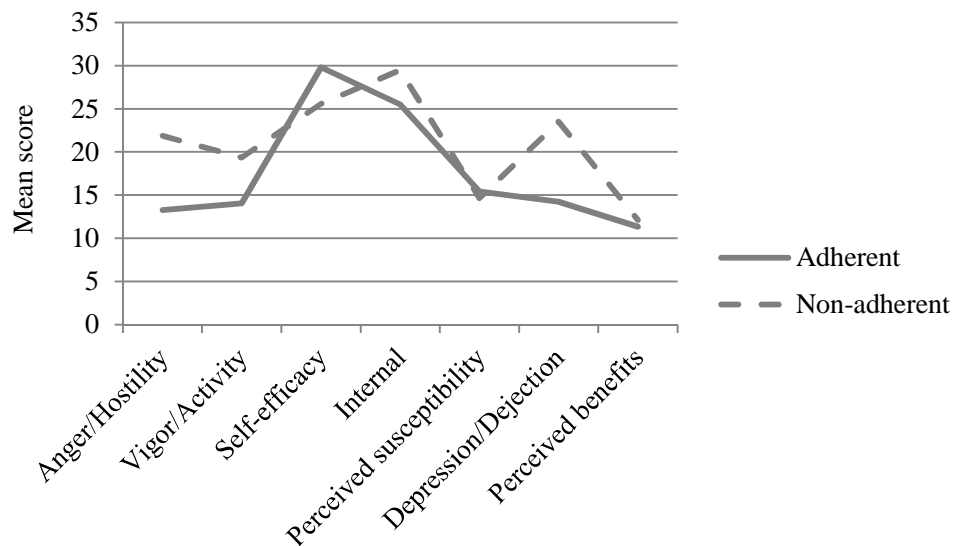


Figure 11. Profile of CPAP-adherent patients compared to CPAP non-adherent patients based on psychological predictors (mood, self-efficacy, health locus of control, and health belief).

Figure 12 depicts the significant group mean differences between the CPAP adherent group and CPAP non-adherent group. The group profile shows that patients classified as CPAP adherent reported less negative symptoms associated with mood (anger/hostility, vigor/activity, depression/dejection) than patients in the non-adherent group. CPAP adherent patients also reported more self-efficacy, having an internal locus of control, and understanding the perceived susceptibilities and perceived benefits of CPAP use. Whilst the latter two psychological factors displayed slight mean differences between groups these differences remain statistically significant.

4.2.2.1 Mood.

In the present study, mood was found to be the strongest predictor of adherence and non-adherence to CPAP use. In particular, anger/hostility and

vigour/activities were identified through DA to be the strongest psychological predictors of group membership depicting adherence and non-adherence. Despite limited predictive research surrounding the mood construct anger, results from Brostrom et al.'s research supports the predictive findings of the present study. Brostrom et al. (2007) were the first to investigate the prevalence and impact of negative affectivity (such as anger) in OSA patients in relation to CPAP adherence. In their study, patients who reported higher levels of anger displayed significantly less CPAP use. It is likely that anger observed in non-adherent patients in the present study may have contributed to low motivation or readiness to accept a likely diagnosis of OSA and subsequently commence CPAP use.

General literature surrounding adherence treatment and medication regime has also identified anger as a precipitating factor leading to poor adherence. For example, the researchers at Bandolier (2004) reported that adherence to treatment regime within the aforementioned context was generally improved by developing trust with patients so that anger did not impact on adherence. While it is noted that the POMS has been utilised as a measure of mood in relation to CPAP adherence studies conducted by Derderian et al. (1988), Kribbs et al. (1993), Yu et al. (1999), Bardwell et al. (2003), and Stepnowsky et al. (2012), the central focus of these studies was on depression and/or overall emotional distress as measured by the POMS sub-scales depression/dejection and TMD. A breakdown of the entire POMS sub-scales from these studies for comparative reasons is therefore not possible. Nevertheless, Derderian et al. and Kribbs et al. did provide evidence to suggest that with effective CPAP use adherent patients displayed a decrease in anger. This evidence supports the findings of the present study.

Similarly, few studies have focused on the predictive powers of vigour/activity in relation to OSA and CPAP adherence. The present study has shown that high vigour/activity is predictive of non-adherence to CPAP use. Given this cohort of patients reported statistically significant higher levels of activity compared to the CPAP-adherent group, it is likely that regardless of mean AHI scores, they may not have been experiencing severe enough symptoms associated with OSA. It is possible that patients may have questioned the validity of their OSA diagnosis, given they might have been carrying out their daily activities “as usual”, without the identifying of symptoms of low energy and/or fatigue. However, those researchers identified above that have previously utilised the POMS have also reported limited results pertaining to the sub-scale vigour/activity making comparisons difficult. Derderian et al. (1988) and Kribbs et al. (1993) did however identify that patients who were adherent to CPAP use displayed an overall increase in vigour.

Depression was also identified as a predictor of non-adherence in the present study, a result that supports the general findings of the meta-analysis conducted by DiMatteo et al. (2000). DiMatteo et al. reported that depressed patients were three times less likely than non-depressed patients to adhere to medical treatment. However, research related to the association of depression and CPAP use has been limited to reporting positive changes in depressed patients as a result of adherence to CPAP use and has not been based on whether the presence of depression predicted non-adherence to CPAP use.

Kjelsberg et al., (2005) were one of the first to explore the predictive association between mood (anxiety and depression) and adherence to CPAP use. The

authors found that depression was a statistically significant independent predictor of CPAP non-adherence. Thus, patients who exhibited symptoms anxiety and/or depression were non-adherent to CPAP use. Kjelsberg et al.'s study was one of the first to report a significant negative relationship between mood and CPAP adherence, as previous landmark studies had found mixed associations between CPAP use and mood (Bliwise et al., 1986; Pillar & Lavie, 1998). Kjelsberg and colleagues' study suggested a link between patients' psychological symptoms and lower levels of adherence to CPAP use. The authors concluded that mood is likely to play an important role in treatment adherence as it was likely to impact on an individual's tolerance towards treatment. Kjelsberg et al. recommended that "treatment of their mental symptoms may be a possible therapeutic option in patients with OSA and symptoms of anxiety or depression that may contribute to improved compliance with CPAP therapy and break the vicious circle" (p. 343). However, it is important to distinguish whether identified mood disorders in OSA patients are a result of being sleep deprived (i.e., caused by OSA or a non-adherent CPAP use) or existed prior to developing OSA.

4.2.3 Social Cognitive Factors

Given their inter-relatedness, it is not surprising that the social constructs of self-efficacy, health locus of control, and health belief were also predictors of CPAP adherence. Overall, both adherent and non-adherent patients reported high levels of self-efficacy and internal health locus of control as well as similar health beliefs associated with perceived susceptibility and perceived barriers to OSA and CPAP use. There can be an implicit assumption that self-efficacy and internality contribute to

patients having a greater tendency to engage in health-promoting activities. As such, patients with the confidence to take control of their treatment and who believe that, in general, their actions play a large role in determining their circumstances may tend to adhere to a prescribed treatment regimen because they believe that they can affect their own health. However, the results from the present study showed that patients with high self-efficacy and an internal health locus of control were also non-adherent to CPAP use. It is probable that there are situations when a strong belief in others would be advantageous regardless of self-efficacy—for example, during hospitalisation where there is little that one can do to change health status, an external health locus of control (chance or doctor), and strong self-efficacy may be most adaptive. This is likely to be the case given the nature of OSA, as CPAP treatment is considered a preventative option and not a curative measure, making it a long-term treatment option where there is no option for cure.

The findings of the present study were consistent with those of de Vellis et al., (1980), who showed that patients with epilepsy who possessed an internal locus of control were more likely to be engaged in preventive behaviour or lifestyle changes and seek information about their condition but were less willing to adhere with the drug therapy. Conversely, in a frequently cited study by Wild et al. (2004) that also investigated the psychological constructs of locus of control, self-efficacy, and health beliefs in 119 CPAP naïve OSA patients, patients who received a higher adherence rating reported a higher internal locus of control and lower scores on chance, doctors, and powerful others. Wild et al.'s findings support this present study's results for the CPAP adherent group.

4.2.3.1 Health Locus of Control.

Similar to mood, there is extensive literature on the social construct locus of control and its association with adherence to medical treatments. Locus of control has been linked to a wide range of behaviours, with internality associated with a number of adaptive behaviours—for example, better management of chronic illnesses and diseases such as diabetes, hypertension, cancer, and heart and lung disease. Adaptive behaviours to these illnesses and diseases may include taking steps to improve health, stopping smoking, losing excess weight, and adhering to medication regimes and check-ups (Georgio & Bradley, 1992; Ferraro, Price, Desmond, & Roberts, 1987; Bradley, Lewis, Jennings, & Ward, 1990; Stantion, 1987; Nicasio et al., 1985; Pruyn et al., 1988; Allison, 1987, all cited in Norman & Bennett, 1995). Externally-oriented patients believe that illness is caused by something or someone elsewhere, while internally-oriented individuals believe that illness is caused by their own unhealthy attitudes and actions (Rotter, 1966). In line with previous studies that have shown that locus of control correlates positively with behaviours that affect adherence (Hoy et al., 1999; Stepnowsky et al., 2002; Engleman & Wild, 2003; Wild et al., 2004; de Zeeuw et al., 2007; Olsen et al., 2008), this study investigated the effect of locus of control on adherence to CPAP by patients diagnosed with OSA.

In this study, results indicated that a high level of internality at the diagnostic phase was likely to contribute to and be predictive of CPAP adherence. Comparisons of scores for overall external health locus of control, especially reliance on doctors, showed that patients' health locus of control decreased significantly between the diagnostic phase and six months following the implementation CPAP use. These

findings suggest that patients who held the belief that they had control over treatment and recovery were more likely to adhere to CPAP use. Literature on existing CPAP adherence also commonly reports the predictive power of internal locus of control, suggesting that individuals who have control over their treatment and recovery are likely to adhere to CPAP treatment in the long-term (Wild et al., 1999 Hoy et al., 2002; Stepnowsky et al., 2004).

Of particular interest in this study, was the fact that the entire sample reported high scores for reliance on doctors, which is an external construct for the MHLC-C measure. Wild et al. (1999), Hoy et al. (2002) and Stepnowsky et al. (2004) reported similar results in their research, although they viewed seeking advice from doctors as an internal aspect as it was likely to contribute to independent self-management of OSA. Results from the present study suggest that patients who are externally-oriented also display a healthy level of internality and are likely to internalise treatment advice provided to them by their respiratory physician/doctors and thus persist with CPAP use over time. It is probable that these patients had a better understanding of the nature of OSA and its long-term treatment option of CPAP use. From a theoretical standpoint, it makes sense that individuals who strongly believe that health care professionals control their health would be more likely to follow the advice of their doctors. This could include following a physician's/doctor's directions for proper CPAP use as well as an overall treatment plan for lifestyle changes and modifications. Such findings are starting to emerge from adherence research on other long-term diseases and illnesses—for example, taking anti-retroviral medications for HIV.

4.2.3.2 Self-Efficacy.

Self-efficacy is a universally investigated social cognitive factor in adherence research. It is likely to impact upon individuals' choices, the amount of energy and time they put in to achieving certain goals, and how long they decide to persevere with setbacks, failures, and side-effects. Moreover, self-efficacy has been commonly reported to be a predictor of treatment adherence and this finding warrants further explorations on its predictive value regarding CPAP adherence (Connor & Norman, 1996; Jones, 2002 cited in Olsen et al., 2008).

Research on OSA has frequently reported on predictive value of self-efficacy with regards to CPAP adherence (Sage et al., 2001; Stepnowsky et al., 2002; Aloia et al., 2005; Olsen et al., 2008). Specifically, the importance of self-efficacy was clearly conveyed in research conducted by Stepnowsky et al. and Aloia et al., who investigated the predictive value of SCT and the TM in CPAP adherence. Both studies were pivotal in drawing a link between self-efficacy and CPAP adherence, and they provided evidence to suggest that regular ongoing monitoring and support post-CPAP implementation was essential in order to maintain long-term adherence as well as continued self-efficacy.

The findings of the present study showed that while self-efficacy was a predictor of CPAP adherence, no statistically significant differences were noted in patients' levels of self-efficacy following the implementation phase of CPAP use. It must be noted, however, that 100% of patients in the study's sample reported high levels of self-efficacy at the diagnostic phase and six months following the

implementation phase. Similar to the internal measure for health locus of control the lack of variance within patients may have contributed to higher than expected levels of overall adherence within this sample. This may have also impacted the possibility of achieving statistically significant findings following CPAP adherence after the implementation phase. Whilst the results of the present study did not show significant changes between the diagnostic phase and six months following the implementation phase they did provide insight into the predictive role of self-efficacy. However, the decision to use a general self-efficacy measure—unlikely Stepnowsky et al.’s study—may have also contributed to the lack of sensitivity in identifying specific treatment-related self-efficacy.

In their study, Wild et al. (1999) and Olsen et al. (2008) both reported that self-efficacy was not a predictor of CPAP adherence. Each concluded that the use of a treatment-related self-efficacy measure, such as the SEMSA (Weaver et al., 2003), might have better discriminatory value and provide better predictive validity. Using the SEMSA, Baron et al. (2011) found that self-efficacy was significantly correlated to CPAP treatment adherence. They proposed that patients with high levels of self-efficacy reported higher levels of CPAP use that also coincided with decreased levels of sleepiness. Similar to the conclusions of Trupp et al. (2011), Baron and colleagues identified the need for ongoing support and intervention in order to enhance CPAP adherence and maintain self-efficacy to improved CPAP use beyond the initial “honeymoon” period of treatment.

4.2.3.3 Health Belief.

The HBM is one of the most researched and utilised models in health education and health promotion (Glanz et al., 2002). While closely related to self-efficacy, the fundamental concept of the HBM is that health behaviour is influenced by personal belief and perceptions. The HBM is widely utilised to explain adherence to preventive health care regimens and to predict treatment adherence (e.g., i.e., flu vaccination) (Shahrabani, Benzion, & Yom Din; 2009). Only a few researchers have employed the HBM to predict adherence to CPAP treatment in OSA patients (Sage et al., 2001; Olsen et al., 2008).

The HBM identifies four categories of beliefs to that are likely to dictate behaviour change:

1. perceived susceptibility;
2. perceived severity;
3. benefits; and
4. barriers.

Sage and colleagues (2001) and Olsen et al. (2008) are among the few researchers to have incorporated the HBM construct into OSA research regarding CPAP adherence. Sage et al. administered it post- CPAP titration to 40 patients diagnosed with OSA. After one-month, they found a moderate relationship between the perceived benefits and perceived barriers constructs of their HBM in relation to CPAP treatment. The authors reported that this explained 23% of the variance in

CPAP adherence over the more commonly explored sleep-related variables. Similarly, Olsen et al. also reported that HBM constructs in their study accounted for 22% of the variance in CPAP adherence. Such findings provide support and evidence for the use of HBM constructs to predict CPAP adherence.

Consistent with the findings associated with self-efficacy and health locus of control, the majority of the patients in this study reported understanding the perceived susceptibility, perceived severity, and perceived benefits associated with CPAP use at the diagnostic phase and at six months following the implementation phase. Interestingly, there was an increase in the perceived barriers sub-scale scores at the six months mark following CPAP use. This result indicates that patients identified more obstacles to CPAP use at this time, despite high perceptions of perceived susceptibility, perceived severity, and perceived benefits of treatment. Given that patients were generally initially naïve to CPAP exposure at the diagnostic phase, the measurement of perceived benefits at the six months mark after exposure to CPAP is likely to account for these results. Alternatively, various issues may have arisen during the six months that have pushed home the fact that there are many issues to face when using CPAP. As such, ongoing support is very important over the life span of CPAP use, but it is likely that patients are often left to battle on alone and are misinformed or have misunderstood instructional information.

The results of this study show that HBM construct sub-scales perceived susceptibility and perceived benefits were predictive of CPAP adherence but yielded similar predictive value to non-adherence. This suggests that patients' perception of the risk associated with OSA may have negatively impacted on their adherence to

CPAP use in the current study. These results were unexpected as literature on the HBM and adherence has generally reported a positive association between increased perceived susceptibility and adherence. However, it is possible that with the identification of factors associated with the susceptibility of having OSA, individuals were left initially feeling overwhelmed, especially following six months' worth of CPAP use and exposure.

4.2.3.4 Personality.

While personality was proven to be a poor predictor of CPAP adherence in the present study, it is nevertheless important to explore non-significant results, especially since changes in personality from the diagnostic phase following CPAP use were noted. The role that personality plays in adherence to CPAP treatment has received little attention in the literature on OSA treatment. Nevertheless, it has been suggested that personality traits such as hypochondriasis and depression, as measured by the MMPI, may be predictors of adherence to CPAP treatment, possibly explaining up to 63% of the variance in adherence (Edinger, et al., 1994).

In the present study the EPQ-RS was used to measure personality. The EPQ-RS provides a measure of personality based on temperament (neuroticism, extraversion, psychoticism, and lying) as opposed to the normative psychopathology of other commonly used measures. Statistically significant increases in scores on the sub-scales for extraversion and psychoticism were noted following the implementation phase and six month of CPAP use.

While it is not clear what these results mean, in the limited literature available pertaining to personality and treatment adherence, Cohen et al. (2004) found a negative relationship between extraversion and adherence to antidepressant treatment suggesting that extraverted individuals may be “too busy” or “too engaged” to remember or prioritise taking medications. Conversely, in another study, Courneya et al. (2002) found a positive relationship between extraversion and exercise adherence. Keeping in mind Courneya et al.’s findings, this present study’s results may indicate that effective CPAP use is associated with increases in energy (as noted by the vigour/activity sub-scale of the POMS); therefore, patients who score high on extraversion (following CPAP implementation) might also start to live a more physically active lifestyle again. Similarly, Olsen et al. (2008) reported that having solid social support, as depicted by individuals with extraversion traits, was also likely to contribute to improved treatment adherence. This is an important finding given that such improvements are likely to lead to better coping skills, healthier behaviours, and adherence to medical regimens in general.

Similarly, elevated scores on the psychoticism sub-scale, which indicates low constraint and high impulsivity, were reported following the implementation phase with CPAP use. While the reasons for this finding are also not clear, it is probable that patients in the present study may have been prone to such negative affects, especially non-adherers, which were therefore likely to impact upon decisions regarding OSA treatment options. Such personal responsibility for making important health decisions is likely to be anxiety-provoking or otherwise distressing, and therefore possessing impulsive personality traits is likely to have a potential, impact on adherence to OSA treatment in general. In support of this idea, it is noted that patients who reported

traits of psychoticism were predominantly in this study's non-adherent group. Moran et al. (2011) drew similar conclusions relating to negative affects in their study, which utilised the Costa and McCrae's Big Five framework of personality to measure neuroticism, extraversion, intellect/imagination, agreeableness, and conscientiousness. Moran et al. concluded that patients affected by negative traits such as neuroticism were negatively correlated to CPAP use and adherence, suggesting that these patients were more likely to rely upon their behavioural inhibition systems as a means of coping with OSA and hence were unlikely to adhere to CPAP use and other treatment regimes.

While personality was not a significant predictor of CPAP adherence or non-adherence, all of these findings lend support to a possible relationship between personality and adherence to CPAP use. Additionally, the data illustrate how these relationships may change as a function of the particular treatment in question—for example, medication adherence versus exercise adherence or the treatment of one disease process over another. Thus, further research is needed that focuses on adherence to prescribed CPAP use, as it would allow for a better understanding of the relationship between personality and adherence to CPAP treatment.

4.2.4 Exploratory Question Two

Exploration of the impact of adherent CPAP use on mood following the implementation phase.

The prevalence of affective disorders such as anxiety and depression in relation to the treatment adherence of chronic illnesses and diseases has received much attention over the past 50 years. While initially the focus of research was on chronic illnesses and diseases such as cancer, renal disease, rheumatoid arthritis, diabetes, coronary disease, and HIV (Dwight et al., 2000; Katon et al. 2001; Katon & Ciechanowski, 2002), researchers of sleep disorders have also paid attention to investigating the role that mood plays with regard to CPAP adherence (see Section 1.8.8: Table 10).

Generally, research has presented mixed findings on this topic, and the association between mood (particularly depression) and OSA has been deemed coincidental or perhaps the results of uncontrolled factors such as age, BMI, fatigue, and other co-morbidities (Reynolds et al., 1984; Millman et al., 1989; Engleman et al., 1994; Borak et al., 1996; Engleman et al., 1997; Engleman et al., 1998; Charbonneau et al., 1999; Munoz et al., 2000; Yamamoto et al., 2000; Means et al., 2003). The current study, however, extended its focus beyond examining only anxiety and depression to examine a range of moods measured by the POMS-SF. Overall, the findings demonstrated observable and statistically significant improvements in vigour/activity and fatigue/inertia after six months of CPAP use. Observable, but non-significant improvement in symptoms of tension/anxiety, depression/dejection, anger/hostility, confusion/bewilderment, and TMD following CPAP implementation in adherent patients were also noted.

Less than 21% of patients reported negative symptoms of tension/anxiety, depression/dejection, anger/hostility, confusion/bewilderment, and TMD at the

diagnostic phase. This suggests patients presented with minimal negative mood-related symptoms, which may account for the non-significant findings following the implementation phase. Yu et al. (1999) and Means et al. (2003) also reported similar findings for mood to those of the present study. In their study, Means and colleagues reported that approximately 35% of their sample reported depressive symptoms pre-CPAP use at diagnosis. This level is considered to be low and is akin to the figure of approximately 15% reported in the present study. Yu et al. also reported similar findings in their study, which investigated the effect of CPAP use on mood states by comparing results between a CPAP treatment and placebo group. In their study, Yu and colleagues excluded patients with major psychiatric illness, and therefore low scores on the POMS at pre-CPAP use were reported for both the treatment and placebo groups. Similar to the present study, Yu et al. reported improvement in mood post-CPAP use in both the treatment and placebo groups, but this was not statistically significant. Yu et al. attributed their findings to the placebo effect, the short CPAP use period (one week) and the relatively low initial scores on all POMS sub-scales. It is therefore likely that in the present study CPAP treatment would have had a more positive impact on mood in patients where their symptomatology was assessed as moderate to severe. In this study's sample, mood scores following the implementation phase were predominantly in the normal to mild range. It is likely that patients' mood scores were not high enough initially to detect any statistically significant improvements after six months of CPAP use on all of the POMS-SF sub-scales.

In this study, 78% of patients did however report negative symptoms associated with vigour/activity and 51% with fatigue/inertia at the diagnosis phase.

This finding that possibility suggests that energy levels and exhaustion were the key presenting symptoms in patients with moderate to severe OSA. These figures also suggest that change is more observable in patients that present with moderate to severe symptomatology. Such a finding corresponds with those of Derderian et al. (1988) and Kribbs et al. (1993), who were one of the first to utilise the POMS to measure positive changes in mood after a period of CPAP therapy. Derderian et al. reported improvement in depression/dejection and overall TMD, and Kribbs et al. reported improvement in vigour/activity post-CPAP use. Both studies also, reported significant improvement in fatigue/inertia as measured by the POMS. The current study mirrored the findings of Derderian et al. and Kribbs et al. and showed observable improvements in vigour-activity and fatigue/inertia following CPAP implementation at the six month mark. Further exploration using Paired Sample t-tests showed that symptoms of fatigue/inertia statistically significantly improved following the implementation phase with CPAP use. As previously noted, 51% of patients presented with fatigue at the diagnostic phase, which decreased to 26% following a six month period of CPAP use. Vigour/activity also yielded statistically significant results that showed an improvement in overall level of activity after six months of CPAP use. While there was an observed improvement in the percentage of patients who reported an improved overall TMD, the results of a Paired Sample t-test showed that this was not statistically significant. Nonetheless, such noticeable improvements may still play an important role in an individual's improvement of health and quality of life.

Interestingly, while not statistically significant, there was also a notable increase in anger following the implementation phase with six months of CPAP use.

Although patients who reported increased levels of anger at the implementation phase were predominantly patients in the non-adherent group, a small proportion of adherent patients also reported a slight increase in anger. It is plausible that reasoning relating to OSA being symptomatic and not curative may account for the slight observable increase in anger scores within the adherent group as patients begin to realise the permanence of CPAP use (Munoz et al., 2000).

4.2.5 Exploratory Question Three

Exploration of the impact of adherent CPAP use on sleep-related variables collected from PSG at the diagnostic phase.

The results of this study showed that following the diagnostic phase, CPAP-adherent patients displayed positive and statistically significant improvements in the sleep-related variables NREM AHI, REM AHI, NREM O₂ saturation, and REM O₂ saturation. Patients did not show any significant improvements in sleep efficiency (the percentage of time in bed asleep) and sleep latency (time taken to fall asleep) following the diagnostic phase. The reason for the lack of improvement in these two sleep parameters is likely due to a number of factors, including but not limited to the “sleep laboratory effect”, age (poorer sleep in older adults, such as decreased total sleep time, more frequent awakenings, and decreased sleep depth), co-morbid chronic medical conditions, negative polypharmacological interactions (medications), and other diagnosed or not diagnosed sleep disorders (e.g., insomnia, restless leg syndrome, periodic limb disorder, circadian rhythm disorders).

Psychological/psychiatric factors, such as anxiety and/or depression, are also known to cause disruptions to sleep latency, sleep consolidation, and efficiency.

A study conducted by Drake et al. (2002) found similar results when exploring sleep-related variables as predictors of CPAP adherence during titration. Drake and colleagues hypothesised that improved PSG result/sleep-related variable (e.g., reductions in AHI and increased in O₂ saturations), representing improved sleep at this time, would predict higher subsequent CPAP adherence. The researchers reported that overall improvement in sleep-related variables, measured approximately 48 days following an initial PSG, was associated with improvements in scores for sleep-related variables collected from a follow-up PSG. Drake and colleagues concluded that while sleep-related variable were generally a significant predictor of adherence in their study, they also reported no significant correlation between CPAP adherence and the sleep-related variables of sleep efficiency and sleep latency. The authors acknowledged that such a finding was not likely to reflect disease severity alone, as it was likely that more complex factors (i.e., psychological and medical/physiological conditions) may have also had an impact on adherence to CPAP treatment.

Fleck et al. (2005) support this viewpoint, having reported that poor adherence to health-related interventions is a common problem with chronic illnesses and diseases and may have a “multifactorial etiology”. These authors believe that factors such as ethnicity, level of education, family support, and patients’ perception of their disease and the benefits of intervention could influence adherence to treatment. In addition, behavioural, psychological, social, anatomic, and financial difficulties may

underlie differences in adherence to therapies. However, it remains unclear what role or to what extent these complex factors play in CPAP adherence.

While the results surrounding sleep-related variables have been generally mixed (Ohayon et al., 1997; Wiese et al., 2005; Krishnan & Collop, 2006; Amodio et al., 2008; Casale, et al. 2008), given the result of the present study pertaining to improvements in AHI and O₂ saturations, an element of presumption can be attributed to these present finding. As such it is possible that if effective and adherent CPAP use is continued, over time it would be expected that AHI and O₂ saturations would improve markedly. Such conclusions have also been drawn by Englemann et al. (1996), Lewis et al. (2004), Hui et al. (2000), and McFadyen et al. (2001).

Nevertheless, given the significant improvements in AHI and O₂ saturations following CPAP use one would presume that similar improvements would follow in regards to sleep efficiency and sleep latency. One possible explanation for the absence of such results is that people with OSA require further diagnosis and treatment if additional sleep issues are identified—for example, insomnia, which is probably more common than previously thought. In addition, differential diagnosis of conditions such as periodic limb movement disorder (which is likely to impact sleep efficiency and sleep latency) can best be achieved once OSA is treated and persistent nocturnal movements of the limbs can be observed as a separate and treatable condition. This alludes to the possible dangers of specialised treatment by specialised respiratory physicians. What is required is a holistic approach to treating sleeping difficulties so that each separate and independent disorder can be appropriately treated—similar to the necessary treatment of co-morbid psychological/psychiatric disorders. Such an

approach may involve specialised treatment by one or more specialists who can identify and apply the appropriate treatment whether it is behavioural or psychological.

4.2.5.1 Sleepiness.

While it was not the intention of the present study to focus on daytime sleepiness as measured by the ESS, its prevalence in OSA literature (e.g., Englemann et al., 1996; Hui et al., 2000; McFadyen et al., 2001; Lewis et al., 2004) makes it noteworthy. In this study, daytime sleepiness of patients scored on average within the 0 to 10 range ($M = 9.79$) at the diagnostic and implementation phases ($M = 9.55$), suggesting that patients reported sleepiness to be within the normal range overall. However, despite its widespread use as a measure of daytime sleepiness, studies of patients with OSA by Hoy and colleagues (1999) and Stepnowsky et al. (2002) have reported that the ESS has little predictive value as an indicator of CPAP adherence. Thus, the continued use of the ESS in studies of CPAP adherence may be questionable.

In a previous study, Tanaka et al. (2009) reported a weak but significant correlation between the ESS and AHI in patients with mild to moderate OSA. In the present study, patients' daytime sleepiness scores were on the high side of average, while AHI scores were reported to range between moderate to severe at the diagnosis phase. Using AHI as a potential objective indicator of OSA severity, 90% of the current sample at the diagnosis phase was in the moderate to severe range for OSA. While measures of AHI and daytime sleepiness provided separate indicators of

severity, one would generally assume a link between the two. That is, a decrease in AHI would coincide with a decrease in daytime sleepiness as measured by the ESS. Such a relationship has been demonstrated in other studies (Hui, et al. 2000; McFadyen et al., 2001). However, other studies, such as Barbe et al. (2001) have found that reported daytime sleepiness as measured by the ESS can be within the normal range, even in patients with an AHI greater than 30. This was also found in the current sample with patients self-report of daytime sleepiness being within the normal range ($M = 9.79$) and AHI scores ($M = 40.55$) in the mild to severe range for OSA. Barbe et al. found that these patients, showed less of a tendency to have cognitive issues and/or any concerns with their quality of life and therefore, it appears that the relationship between sleepiness as measured by the ESS and OSA severity is not a simple one. It is probable that patients may develop compensatory responses to deal with the slow and insidious onset of OSA. Furthermore, as has been demonstrated in other areas where people are asked to estimate sleepiness or the converse—alertness, they may be notoriously poor in actually judging their own sleepiness/alertness states. Thus, there is a need to develop methods that more accurately assess sleepiness, fatigue, and alertness for both medical assessments and also for road- and work-related safety concerns.

The findings of the present study with respect to the ESS were similar to those reported by Hoy et al. (1999) and Stepnowsky et al. (2002). Both Hoy et al. and Stepnowsky et al. found that ongoing CPAP usage reduced symptoms of OSA but did not always result in significant changes in excessive daytime sleepiness, as measured by the ESS. Similarly, no mean differences in daytime sleepiness scores were observed from the diagnosis phase to the implementation phase, with approximately

60% of patients reporting symptoms of excessive daytime sleepiness at both measurement points. Excessive daytime sleepiness scores at the diagnosis phase showed that approximately 50% of patients scored within the average range for daytime sleepiness, depicting little change over time. Similar to the studies of Hoy et al. and Stepnowsky et al., in the present study the length of time between the diagnostic and implementation phases when patients completed the ESS may have been a relatively short period in terms of long-term treatment. However, some studies have reported improvements in excessive daytime sleepiness over much shorter periods of CPAP use (Edinger et al., 1994; Engleman et al., 1998; Greenham-Conway, 2000; Munoz et al., 2000; Wild et al., 2004; Aloia et al., 2005; Budhiraja et al., 2007). Given that OSA treatment with CPAP use is usually considered a lifelong therapy option perhaps a longer interval for re-assessment of sleepiness (i.e., 12 to 24 months) to determine and increase the chances of detecting positive changes in excessive daytime sleepiness may be warranted.

It is important to acknowledge that the ESS used to measure daytime sleepiness was completed as part of the intake process by sleep technicians and/or nurses in the respiratory sleep departments at the diagnostic and implementation phases. Further in-depth exploration into the impact of mode of administration of the ESS was beyond the scope of the present study, but it has been the focus of research conducted by Kaminska et al., (2010). Kaminska and colleagues reported that on average, scores from a self-administered ESS were statistically significantly higher than when administered by a specialist. In the present study, lower ESS scores may have been due to their administration by a sleep technician and/or nurse. Kaminska et al. reported that self-administered questionnaires answered in private provide

anonymity to patients, and as a result patients may be more open in disclosing perceived problems. Having a specialist ask the questions may introduce a social desirability bias, where patients may be inclined to under-report perceived problems (Kaminska et al., 2010). It is also likely that reported differences in results between the various aforementioned studies could be due to OSA severity, as patients with greater levels of severity often report greater levels of improvement. Thus, ESS scores within the normal range may have a similar impact on the results to some of the psychological factors explored in the present study (i.e., depression/dejection, self-efficacy).

Interestingly, while patients' ESS scores were deemed to be predominantly within the normal range, patients' rating of fatigue/inertia as measured on the POMS-SF bordered on the moderate side. Possible reasons for higher levels of fatigue may include patients having other concomittant sleep- or health-related conditions (e.g., insomnia, restless legs, depression, lack of energy, and sleepiness) at the diagnosis phase of OSA that were not recognised and differentially diagnosed. Furthermore, one could argue that sleepiness and fatigue are in fact measuring separate constructs.

There is a growing amount of evidence, suggesting that fatigue is a better indicator of OSA severity than sleepiness (Bailes et al., 2011). Again, while it was beyond the scope of the present study to distinguish between impact of daytime sleepiness over fatigue, this present study's results suggest that fatigue, as measured by the POMS-SF, significantly improved following the implementation of CPAP use. Although initial investigations surrounding fatigue and its impact on insomnia are now taking place, the role of fatigue in association with OSA has received little

attention. Nevertheless, to date, the limited amount of research focusing on the role fatigue plays in OSA suggests that sleepiness and fatigue are in fact independent constructs (Hossain et al., 2005; Chervin, 2000). This finding provides valuable future direction for OSA research, especially in relation to the psychological impacts that generally accompany severe fatigue.

4.3 Clinical Implications

The diagnosis of OSA and adherence to CPAP use has significant clinical implications. The adverse consequences are numerous and include impaired quality of life and overall mortality (see Section 1.2: Table 2). To date, research has focused on the predictive value of sleep-related variables and has only recently begun exploring potential psychological determinants of CPAP adherence (namely anxiety and depression in line with adherence literature for other chronic illnesses and diseases). Thus, this present study aimed to expand our understanding of a range of potential psychological predictors for CPAP adherence. The study identified mood, self-efficacy, health locus of control, and health beliefs as predictors of CPAP adherence. The inclusion of these constructs in a thorough psychosocial assessment of newly diagnosed OSA individuals is therefore likely to be beneficial. The findings of this study point to the need for a thorough understanding of an individual's psychological wellbeing and social constructs when undertaking treatment for OSA through CPAP use. Such an understanding becomes especially important when considering intervention strategies to promote adherence to CPAP use and it is likely to require a holistic approach incorporating a range of professionals and supports—doctors, respiratory specialists, psychologists, dieticians, educators, family, and friends.

4.3.1 Intervention

Key to intervention is the identification of a non-adherent patient, which often is as simple as monitoring missed appointments, identifying non-responders to past treatment, and asking patients about past medicinal adherence (Burke & Ockene, 2001). By identifying non-adherence, it becomes possible to intervene before secondary health issues become problematic. Once identified, exploring the underlying causes of poor adherence is important. Administration of the POMS-SF or alternative forms of psychometric measures to screen for potential mood disorders will provide additional information on a patient's psychological status in relation to mood. The administration of a self-efficacy measure as well as health locus of control, and health belief measures will also provide further insights regarding the reinforcing motivators of health-specific behaviours. Non-adherent patients scoring high on these social constructs, as in the present study, may require further explanation of OSA and CPAP treatment to motivate health-behaviour modification. Non-adherent patients scoring high on external health locus of control (including reliance on doctors as measured by the MHLC-C) may require step-by-step specific instructions regarding the importance, value, and benefits of CPAP use as well as ongoing support, monitoring, and education. When using the MHLC-C, non-adherent patients scoring high on the chance sub-scale are likely to require stringent counselling to avoid poor health outcomes. Similarly, non-adherent patients presenting with symptoms associated with mood disorders, poor self-efficacy, and health belief are likely to benefit from counselling, ongoing education, and monitoring until their related symptomatology is addressed.

4.3.2 Intervention Options to Increase CPAP Adherence

In a review of available literature surrounding interventions aimed at improving CPAP adherence, Weaver and Sawyer (2010) categorised intervention options as supportive (aimed at supporting and enhancing access to sleep-specific healthcare resources), educational (focused on enhancing knowledge on the diagnosis and treatment of OSA), cognitive behavioural (theoretically derived intervention strategies), or mixed strategy (a combination of support and education).

4.3.2.1 Supportive interventions.

Interventions to promote CPAP adherence have been predominantly supportive in nature and included activities such as patient phone calls, handing out printed information/documents, and clinical follow-ups. Advancements in supportive interventions have seen the introduction of more sophisticated methodologies such as the utilisation of a computerised telephone system or wireless tele-monitoring to monitor CPAP use and reinforcing feedback to patients. Studies surrounding the efficacy of utilising supportive interventions to improve CPAP adherence have commonly reported non-significant findings (Weaver & Sayer, 2010). However, it is probable that when combined with real-time assessment of CPAP use and the availability of on-call support, this form of intervention is likely to promote CPAP adherence—especially in patients with poor self-efficacy and/or an external locus of control.

4.3.2.2 Educational interventions.

Educational interventions have only recently been examined for efficacy in promoting CPAP adherence. Such practises include education carried out via videos, through demonstrations, and by discussions—some of which are based on the construct surrounding the HBM. Such educational interventions are generally provided by the respiratory physician and best reinforced by the primary healthcare provider in conjunction with a homecare provider (i.e. partner, friend, or other supports) (Weaver & Sawyers, 2010). To date, limited studies exploring the efficacy of educational interventions alone in improving CPAP adherence have also reported non-significant findings. Nevertheless, this form of intervention is currently widely utilised.

4.3.2.3 Cognitive behavioural interventions.

Growing in popularity, cognitive behavioural interventions have shown some success in improving adherence to CPAP use over time, as well as improvements in patient self-efficacy (Weaver & Sawyer, 2010). In particular, motivational enhancement therapy (MET) has been reported to be beneficial in providing clarity to negative self-perceptions and beliefs that cause and re-enforce ambivalence to treatment. When combined with educational interventions, cognitive behavioural intervention such as MET has shown lower CPAP “drop out” rates and higher CPAP adherence (Weaver & Sawyer, 2010).

4.3.2.4 Mixed strategy interventions.

While not being an actual intervention style, a mixed strategy catering to the unique, complex, and multifaceted nature of patients, the condition (OSA) and treatment (CPAP) is likely to be most effective in improving and promoting CPAP adherence (Weaver & Sawyer, 2010). Studies exploring a combined intervention strategy involving support, education, and the concept of self-efficacy promotion through cognitive behavioural intervention have reported statistically significant findings and high CPAP adherence rates. Such strategies are likely to include verbal explanation for CPAP treatment (involving partners), viewing of educational videos, CPAP acclimatisation training, one-night CPAP titration in the laboratory, and regular telephone follow-up. This mode of intervention strategy is somewhat limited due to it being labour- and time-intensive as well as costly. Nevertheless, the positive results validating such an approach support the notion that a multidimensional perspective is needed to get the best results for improving and promoting CPAP adherence.

4.4 Limitations, Strengths and Future Direction

As with many studies there are limitations that need to be noted. Importantly, the current study was limited by the sample size, which appeared to be a commonly reported limitation in literature on CPAP adherence. To increase participation, conditions surrounding the recruitment of patients were somewhat relaxed—for example, by including patients with co-morbidities. However, co-morbidities are generally characteristic and not uncommon within the OSA population. In addition, another limiting factor was that patients were likely to use differing CPAP devices

during the study. As such, collection of data on CPAP usage may have differed from person to person according to the make, model, maintenance, and age of CPAP device. It was reported in Chapter 3 that faulty meters were the main factor limiting the availability of CPAP usage data. An imputation procedure was employed to estimate CPAP usage. While imputation is a widely used method for dealing with missing data it is acknowledged that this procedure may overestimate the model fit as well as correlation estimates, thus weakening variance. It is likely this procedure may have inflated CPAP usage figures. Nonetheless, other objective sleep-related data was collected at the implementation phase that displayed overall improvement following CPAP use, which suggests that adherence to CPAP use was achieved in the final sample. The use of self-reported measures for the collection of data pertaining to the psychological factors may have also represented another limitation although the use of valid measures would have overcome some of the subjectivity associated with this approach.

The strengths associated with the current study are also worth noting. While the findings contribute to the literature surrounding CPAP adherence, the study also explores a range of psychological factors that have had limited attention in previous research. Furthermore, the use of a personality questionnaire that was not intended to measure psychopathology was an important strength, as previous research has predominantly relied upon the use of a single psychometric tool, the MMPI, to measure mood and personality. This study also makes a valuable contribution to the limited literature on CPAP adherence by using predictive statistics. Previous studies have predominant used “surrogate” means of determining whether a predictive relationship between predictors and CPAP adherence existed—for example,

establishing predictive value from data, which showed that patients reporting depressive symptoms did not follow through with CPAP use or displayed little to no change in sleep-related variables when re-assessed post-CPAP implementation. In addition, the finding that fatigue was likely to play an important role as a predictive factor in CPAP adherence was an exciting one given that sleepiness has received much of the attention since the inception of OSA and CPAP adherence research.

Future studies including fatigue as a predictive factor in conjunction with sleepiness are likely to provide valuable insights. This focus is especially important, since OSA patients may present as fatigued but not necessarily have symptoms associated with daytime sleepiness. The implementation of a control group in the current study is also likely to be beneficial for comparative reasons. While this is not a novel approach and researchers have employed such designs in CPAP adherence studies in the past, having a comprehensive set of psychological factors to be explored as potential predictors will add value and power to the validity of these experimental designs. Future research would also benefit from the utilisation of a specific self-efficacy measure such as the SEMSA and the design of a valid and reliable OSA measure for the constructs of the HBM. Finally, more research is needed to establishing accurate pre-screening or identification of potential non-adherers and explore the efficacy of various intervention strategies to improve and promote CPAP adherence.

4.5 Conclusion

Given that CPAP use is widely acknowledged as the primary treatment option for OSA sufferers, adherence to treatment remains the core of much research within this domain. It has been widely cited that CPAP use decreases daytime sleepiness and improves mood and cognitive functioning, thus contributing to a better quality of life. Nonetheless, adherence to CPAP use remains a significant problem in the OSA population.

Historically, researchers have attempted to identify predictors of CPAP adherence, which have commonly included demographic information and sleep-related variables. Theoretical approaches, grounded in psychology and social learning theory, have been employed to study patient adherence with various other chronic illnesses and diseases. Of interest in the present study was the predictive value of mood, self-efficacy, health locus of control, and health belief in determining CPAP adherence.

While literature has recently emerged that focuses on the role psychological factors play in CPAP adherence, most of the attention has been on mood and personality traits, which have received varying results. Such research has also been limited by its focus on “surrogate” predictions of adherence determined by positive changes in psychological wellbeing as a result of CPAP adherence or as a consequence of non-adherence. The present study has contributed to existing OSA literature by providing a comprehensive investigation of the “psychological predictors” of CPAP adherence utilising predictive analysis. This study’s results have

supported the findings of previous OSA and general adherence research by exploring positive changes in mood and sleep-related variables stemming from effective CPAP use, and predictors associated with CPAP adherence were shown to include mood, self-efficacy, health locus of control, and health belief.

Overall, this study found high rates of adherence to CPAP use among moderate to severe OSA sufferers who completed the diagnostic and implementation phases. Given the prevalence of OSA in society and risks associated with non-adherence, there is an urgent need to develop screening strategies for non-adherence as well as adherence interventions tailored to the specific needs of OSA sufferers. This study demonstrated that psychological factors are likely play an important role in the understanding of adherence behaviour among OSA patients, and therefore screening and interventions that address identified psychological constructs should be developed and tested for efficacy in this population.

While it was not the intention of this study to validate current interventions to improve CPAP adherence, it is noteworthy that holistic intervention strategies involving a range of health care professionals and supports are likely to provide good anticipated results when implemented consistently, potentially leading to lifestyle modification and a better quality of life. Part of this holistic approach needs to pay attention to interventions aimed at treating mental health issues separate to OSA, where needed. While there are strong links between OSA and depression, there is limited information to indicate whether OSA may or may not precede depression. Once such psychological conditions are established they require specialised psychological/psychiatric treatment, as CPAP is not a treatment for depression.

Nonetheless, understanding patient non-adherence to CPAP use is crucial, especially as there are many psychological/psychiatric and medical consequences associated with OSA. As such, non-adherence to CPAP may lead to OSA becoming severely debilitating while decreasing quality of life.

This study has also provided initial insights into the potential role that fatigue plays in CPAP adherence as an alternative to sleepiness, as well as the novel psychometric measures that may be used to measure personality and constructs of the HBM. More importantly, the current study demonstrates that CPAP use continues to remain an effective treatment option for OSA sufferers, with significant improvements noted in sleep-related variables. While it is widely acknowledged that to date there is no single factor that is solely predictive of CPAP adherence, this study has produced positive results that identify psychological predictors of CPAP adherence and therefore paves the way for future research.

REFERENCES

- Access Economics. (2004). *Wake Up Australia: the Value of Healthy Sleep*. Retrieved from <http://www.accesseconomics.com.au/publicationsreports/Getreport.php?report=22&id=26>
- Agresti, A. (2002). *Categorical Data Analysis*. New Jersey: John Wiley & Sons Inc.
- Aleixo, P., & Norris, C. (2000). Personality and moral reasoning in young offenders. *Personality and Individual Differences*, 28(3), 609-623. doi:10.1016/S0191-8869(99)00124-5
- Aloia, M., Arnedt, T., Stepnowsky, C., Hecht, J., & Borrelli, B. (2005). Predicting treatment adherence in Obstructive Sleep Apnoea using principles of Behavior Change. *Journal of Clinical Sleep Medicine*, 1(4), 346-353.
- Aloia, M., Arnedt, J., Stanchina, M., & Millman, R. (2007). How early in treatment is PAP adherence established? Revisiting night-to-night variability. *Behavioural Sleep Medicine*, 5(3), 229-240. doi:10.1080/15402000701264005
- American Academy of Sleep Medicine. (2001). *International classification of sleep disorders, revised: Diagnostic and coding manual*. Chicago, Illinois: American Academy of Sleep Medicine. Archived from the original on 2007-09-27. Retrieved from <http://web.archive.org/web/20070927034423/http://www.absm.org/PDF/ICSD.pdf>

- American Academy of Sleep Medicine. (1999). Sleep-related breathing disorders in adults: recommendations for Syndrome definition and measurement techniques in clinical research. *Sleep*, 2(5), 667-689.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Amodio, D., Master, S., Yee, C., & Taylor, S. (2008). Neurocognitive components of the behavioral inhibition and activation systems: implications for theories of self-regulation. *Psychophysiology*, 45(1), 11-19. doi:10.1111/j.1469-8986.2007.00609.x
- Ancoli-Israel S., Kripke D., Klauber M., Mason W., Fell R., & Kaplan O. (1991). Sleep-disordered breathing in community-dwelling elderly. *Sleep*, 14(6), 486–495.
- Antonescu-Turcu, A., & Parthasarathy, S. (2010). CPAP and bi-level PAP therapy: new and established roles. *Respiratory Care*, 55(9), 1216-1229.
- Aronson, J. (2007). Compliance, concordance, adherence. *Journal of Clinical Pharmacology*, 63(4), 383–384. doi:10.1111/j.1365-2125.2007.02893.x
- Bailes, S., Libman, E., Baltzan, M., Grad, R., Kassissia, I., Creti, L., Rizzo, D., Amsel, R., & Fichten, C. (2011). Fatigue: The forgotten symptom of sleep apnoea. *Journal of Psychosomatic Research*, 70(4), 346–354. doi:10.

1016/j.jpsychores.2010.09.009

- Baldwin, C., Griffith, K., Nieto, F., O'Connor, G., Walsleben, J., & Redline, S. (2001). The association of sleep-disordered breathing and sleep symptoms with quality of life in the Sleep Heart Health Study. *Sleep*, 24(1), 96–105.
- Ballard, R. (2008). Management of patients with obstructive sleep apnoea. *Journal of Family Practice*, 57(8), S24-S30.
- Bandolier. (2003). *Dosing and compliance?*. Retrieved from <http://www.medicine.ox.ac.uk/bandolier/band117/b117-8.html>
- Bandolier. (2004). *Patient Compliance with statins*. Retrieved from <http://medicine.ox.ac.uk/bandolier/booth/cardiac/patcomp.html>
- Bandura, A. (1997). *Self-efficacy: the exercise of control*. New York: Freeman.
- Barbe, F., Mayoralas, L., Duran, J., Masa, J., Maimo, A., Montserrat, J., Monasterio, C., Bosch, M., Lادarta, M., Rubio, M., Rubio, R., Medinas, M., Hernandez, L., Vidal, S., Douglas, N., & Agusti, A. (2001). Treatment with continuous positive airway pressure is not effective in patients with sleep apnoea but no daytime sleepiness. a randomized, controlled trial. *Annals of Internal Medicine*, 134(11), 1015-1023. doi:10.7326/0003-4819-134-11-200106050-00007

- Bardwell, W., Berry, C., Ancoli-Isreal, S., & Dimsdale, J. (1999). Psychological correlates of sleep apnoea. *Journal of Psychosomatic Research*, 47(6), 583-596.
- Bardwell, W., Ancoli-Israel, S., Dimsdale J. (2001). Neuropsychological effects of continuous positive airway pressure treatment in obstructive sleep apnoea patients. *Psychosomatic Medicine*, 63(4), 579–584.
- Bardwell, W., Moore, P., Ancoli-Israel, S., & Dimsdale, J. (2003). Fatigue in obstructive sleep apnoea: driven by depressive symptoms instead of apnoea severity? *The American Journal of Psychiatry*, 160(2), 350-355. doi:10.1176/appi.ajp.160.2.350
- Barnes, B. (2007). ResMed Origins: *Nasal CPAP*. Retrieved from https://www.researchgate.net/publication/17074605_Reversal_of_obstructive_sleep_apnoea_by_continuous_positive_airway_pressure_applied_through_the_nare
- Barnes, M., Houston, D., Worsnop, C., Neill, A., Mykityn, I., Kay, A., Trinder, J, Saunders, N., Douglas McEvoy, R., & Pierce, R. (2002). A randomized controlled trial of continuous positive airway pressure in mild obstructive sleep apnoea. *American Journal of Respiratory Critical Care Medicine*, 165(6), 773-780. doi:10.1164/ajrccm.165.6.2003166

- Baron, K., Berg, C., Czajkowski, L., Smith, T., Gunn, H., & Jones, C. (2011). Self-efficacy contributes to individual differences in subjective improvements using CPAP. *Sleep and Breathing*, *15*(3), 599–606. doi: 10.1007/s11325-010-0409-5
- Belcher, L., Sternberg, M., Wolitski, R., Halkitis, P., & Hoff, C. (2005). Condom use and perceived risk of HIV transmission among sexually active HIV-positive men who have sex with men. *AIDS Education and Prevention*, *17*(1), 79-89. doi: 10.1521/aeap.17.1.79.58690
- Bell, J., Airaksinen, M., Lyles, A., Chen, T., & Aslani, P. (2007). Concordance is not synonymous with compliance or adherence. *British Journal of Clinical Pharmacology*, *64*(5), 710-711. doi:10.1111/j.1365-2125.2007.02971_1.x
- Berlowitz¹, D., Ayas, N., Barnes, M., Brown, D., Cistulli, P., Geraghty, T., Graham, A., Lee, B., Morris, M., O'Donoghue, F., Rochford, P., Ross, J., Singhal¹, B, Spong, J., Wadsworth, B., & Pierce, R. (2012). Auto-titrating continuous positive airway pressure treatment for obstructive sleep apnoea after acute quadriplegia (COSAQ): study protocol for a randomized controlled trial. *Trials*, *14*(181), 1-10. doi: 10.1186/1745-6215-14-181
- Berry, R., Parish, J. & Hartse, K. (2002). The use of auto-titrating continuous positive airway pressure for treatment of adult obstructive sleep apnoea. An American Academy of Sleep Medicine review. *Sleep*, *25*(2), 148-173.

- Bixler, E., Vgontzas, A., Ten Have, T., Tyson, K., & Kales, A. (1998). Effects of age on sleep apnoea in men. *American Journal of Respiratory and Critical Care Medicine*; 157(1), 144–148. doi: 10.1164/ajrccm.157.1.9706079
- Bliwise., D., Yesavage, J., Sink, J., Widrow, L., & Dement, W. (1986). Depressive symptoms and impaired respiration in sleep. *Journal of Consulting and Clinical Psychology*, 54(5), 734-735.
- Blagrove, M., & Akehurst, L. (2001). Personality and the modulation of effects of sleep loss on mood and cognition. *Personality and Individual Differences*, 30(5), 819–828. doi.org/10.1016/S0191-8869(00)00075-1
- Bland, J., & Altman, D. (1997). Statistics notes: Cronbach's alpha. *British Medical Journal*, 314(7080), 570-572. doi: <http://dx.doi.org/10.1136/bmj.314.7080.572>
- Borak, J., Cieslicki, X., Koziej, N., Nlatuszewski, A., & Zielinski J. (1996). Effects of CPAP treatment on psychological status in patients with severe obstructive sleep apnoea. *Journal of Sleep Research*, 5(2), 123-127. DOI: 10.1046/j.1365-2869.1996.d01-60.x
- Bosely, C., Fosbury, J., & Cochrane, G. (1995). Psychological factors associated with poor compliance with treatment in asthma. *European Respiratory Journal*, 8(6), 899–904.

- Brickman, A., Yount, S., Blaney, N., Rothberg, S., & De-Nour, A. (1996). Personality traits and long-term health status: The influence of neuroticism and conscientiousness on renal deterioration in type-1 diabetes. *Psychosomatics* 37(5), 459–468. doi.org/10.1016/S0033-3182(96)71534-7
- Brostrom, A., Stromberg, A., Martensson, J., Ulander, M., Harder, L., Svanborg, E., Brostrom, A., Stromberg, A., Martensson, J., Ulander, M., Harder, L., & Svanborg, E. (2007). Association of type D personality to perceived side effects and adherence in CPAP- treated patients with OSAS. *Journal of Sleep Research*, 16(4), 439–447. doi:10.1111/j.1365-2869.2007.00620.x
- Ritchie E. Brown, R., Basheer, R., McKenna, J., Strecker, R., & McCarley, R. (2012). Control of Sleep and Wakefulness. *Physiological Reviews*, 92(3), 1087-1187. doi: 10.1152/physrev.00032.2011
- Budhiraja, R., Parthasarathy, S., Drake, C., Roth, T., Sharief, I., Budhiraja, P., Saunders, V., & Hudgel, D. (2007). Early CPAP Use Identifies Subsequent Adherence to CPAP Therapy. *Sleep*, 30(3), 320-324.
- Burke, L., & Ockene, I. (2001). *Compliance in Healthcare and Research*. Armonk New York: Futura Publishing Co.
- Carskadon, M., Dement, W., Mitler, M., Roth, T., Westbrook, P., & Keenan, S. (1986). Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. *Sleep*, 9(4), 519-524.

- Casale, M., Rinaldi, V., Bressi, F., Di Peco, V., Baptista, P., Sadun, B., Urrestarazu, E., Trivelli, M., & Salvinelli, F. (2008). A suitable test for identifying high risk adult patients of moderate-severe obstructive sleep apnoea syndrome. *European Review for Medical and Pharmacological Sciences*, 12(4), 275-80. doi: 10.2174/138920209787846998
- Center for Disease Control and Prevention. (2004). *Program Operations Guidelines for STD Prevention: Community and Individual Behaviour Change Interventions*. Retrieved from [http://www.cdc.gov/std/program/ community .pdf](http://www.cdc.gov/std/program/community.pdf)
- Chan, R., & Joseph, S. (2000). Dimensions of personality, domains of aspiration, and subjective wellbeing. *Personality and Individual Differences*. 28(2), 347–354. doi.org/10.1016/S0191-8869(99)00103-8
- Chen, J., Fox, S., Cantrell, C., Stockdale, S., & Kagawa-Singer, M. (2007). Health disparities and prevention: racial/ethnic barriers to flu vaccinations. *Journal of Community Health*, 32(1), 5-20.
- Chervin, R. (2000). Sleepiness, fatigue, tiredness, and lack of energy in obstructive sleep apnoea. *Chest*, 118(2), 372–379. doi:10.1378/chest.118.2.372
- Chesson, A., Ferber, R., Fry, J., Grigg-Damberger, M., Hartse, K., Hurwitz, T., Johnson, S., Littner, M., Kader, G., Rosen, G., Sangal, R., Schmidt-Nowara,

- W., & Sher, A. (1997). Practice Parameters for the Indications for Polysomnography and Related Procedures. *Sleep*, 20(6), 406-422.
- Chivers, L., & Blagrove, M. (1999). Nightmare frequency, personality and acute psychopathology. *Personality and Individual Differences*, 27(5), 843–851.
doi.org/10.1016/S0191-8869(99)00033-1
- Collard, T., Pieters, G., Aubert, P., Delguste, D., & Rodenstein, O. (1997). Review Article: Compliance with Nasal CPAP in Obstructive Sleep Apnoea Patients. *Sleep Medicine Review*, 1(1), 33-44.
- Cohen, N., Ross, E., Bagby, R., Farvolden, P., & Kennedy, S. (2004). The 5-factor model of personality and antidepressant medication compliance. *The Canadian Journal of Psychiatry*, 49(2), 106–113.
- Courneya, K., Friedenreich, C., Sela, R., Quinney, H., & Rhodes, R. (2002). Correlates of adherence and contamination in a randomized controlled trial of exercise in cancer survivors: an application of the theory of planned behavior and the five factor model of personality. *Annals of Behavioral Medicine*, 24(4), 257-268. doi.org/10.1207/S15324796ABM2404_02%I
- Creed, P., Muller, J., & Machin, M. (2001). The role of satisfaction with occupational status, neuroticism, financial strain and categories of experience in predicting mental health in the unemployed. *Personality and Individual Differences*, 30(3), 435–447.

Curran, S., Andrykowski, M., & Studts, J. (1995). Short form of the Profile of mood states (POMS-SF): Psychometric information. *Psychological Assessment*, 7(1), 80-83. doi:10.1037/1040-3590.7.1.80

Davies, R., & Stradling, J. (1996). The epidemiology of sleep apnoea. *Thorax*, 5(S2), S65-S70

de Wit, J., Vet, R., Schutten, M., & van Steenberghe, J. (2005). Social-cognitive determinants of vaccination behavior against hepatitis B: an assessment among men who have sex with men. *Preventive Medicine*, 40(6), 795-802. doi.org/10.1016/j.ypmed.2004.09.026

de Zeeuw, J., Baberg, H., Duchna, H., Kempkens, D., Walther, J., Schultze-Werninghaus, G., Rache, K., & Orth, M. (2007). Locus of control belief is a predictor of CPAP-compliance in patients with obstructive sleep apnoea syndrome. *Pneumologie*, 61(5), 283-290. doi:10.1055/s-2007-959162

Deloitte Access Economics (2011). *Re-awakening Australia. The economic cost of sleep disorders in Australia, 2010*. Retrieved from <http://www.sleephealthfoundation.org.au/pdfs/news/Reawakening%20Australia.pdf>

Dempsey, J., Veasey, S., Morgan, B., & O'Donnell, C. (2010). Pathophysiology of Sleep Apnoea. *Physiological Reviews*, 1(90), 47-112. doi: 10.1152/Physrev.00043.2008.

- Denollet, J. (2000). Type D personality: a potential risk factor refined. *Journal of Psychosomatic Research*, 49(4), 255–266.
- Derderian, S., Bridenbaugh, R., & Rajagopal, K. (1988). Neuropsychologic symptoms in obstructive sleep apnoea improve after treatment with nasal continuous positive airway pressure. *Chest*, 94(5), 1023-1027. doi:10.1378/chest.94.5.1023
- DeVellis, R., DeVellis, B., Wallston, B., & Wallston, K. (1980). Epilepsy and learned helplessness. *Basic and Applied Social Psychology*, 1(3), 241-253. doi:10.1207/s15324834basp0103_4
- DeVon, H., Block, M., Moyle-Wright, P., Ernst, D., Hayden, S., Lazzara, D., Savoy, S., & Kostas-Polston, E. (2007). A psychometric toolbox for testing validity and reliability. *Journal of Nursing Scholarship*, 39(2):155-64. doi: 10.1111/j.1547-5069.2007.00161.x
- Dickens, C. (1837). *The Posthumous Papers of the Pickwick Club*. London: Chapman and Hall.
- DiMatteo, M., Lepper, H., & Croghan, T. (2000). Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Archives Internal Medicine*, 160(14), 2101-2107. doi:10.1001/archinte.160.14.2101.

- Doherty, L., Kiely, J., Lawless, G., & McNicholas WT. (2003). Impact of nasal continuous positive airway pressure therapy on the quality of life of bed partners of patients with obstructive sleep apnoea syndrome. *Chest*, 124(6), 2209-14. doi:10.1378/chest.124.6.2209
- Donnellan, M., Oswald, F., Baird, B., & Lucas, R. (2006). The mini-IPIP scales: Tiny-yet-effective measures of the Big Five factors of personality. *Psychological Assessment*, 18(2), 192-203. doi:10.1037/1040-3590.18.2.192
- Durán, J., Esnaola, S., Rubio, R., & Iztueta, A. (2001). Obstructive sleep apnoea–hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 yr. *American Journal of Respiratory Critical Care Medicine*, 163(3), 685–689. doi:10.1164/ajrccm.163.3.2005065
- Drake, C., Day, R., Hudgel, D., Stefadu, Y., Parks, M., Syron, M., et al. (2003). Sleep during titration predicts Continuous Positive Airway Pressure compliance. *Sleep*, 26(3), 308-311.
- Dwight, M., Kowdley, K., Russo, J., Ciechanowski, P., Larson, A., & Katon, W. (2000). Depression, fatigue, and functional disability in patients with chronic hepatitis C. *Journal of Psychosomatic Research*, 49(5), 311-317.

- Edinger, J., Carwile, S., Miller, P., Hope, V., & Mayti, C. (1994). Psychological status, syndromatic measures, and compliance with nasal CPAP therapy for sleep apnoea. *Perceptual Motor Skills*, 78(3), 1116–1118.
- Elliott, R., & Marriott, J. (2009). Standardised assessment of patients' capacity to manage medications: a systematic review of published instruments. *BMC Geriatrics*, 9(27), 1-10. doi:10.1186/1471-2318-9-27
- Engleman, H., Martin, S., Deary, I., & Douglas, N. (1994). Effect of continuous positive airway pressure treatment on daytime function in sleep apnoea/hypopnoea syndrome. *Lancet*, 343(8897), 572-575.
doi:10.1016/S0140-6736(94)91522-9
- Engleman, H., Asgari-Jirhandeh, N., McLeod, A., Ramsay, C., Deary, I., & Douglas, J. (1996). Self-reported use of CPAP and benefits of CPAP therapy : a patient survey. *Chest*, 109(6), 1470-1476. doi:10.1378/chest.109.6.1470
- Engleman, H., Martin, S., Deary, I. & Douglas, N. (1997). Effect of CPAP therapy on daytime function in patients with mild sleep apnoea/hypopnoea syndrome. *Thorax*, 52(2), 114–119.
- Engleman, H., Martin, S., Kingshott, R., Mackay, T., Deary, I., & Douglas, N. (1998). Randomised, placebo-controlled trial of daytime function after continuous positive airway pressure therapy for the sleep apnoea/hypoponea syndrome. *Thorax*, 53(5), 341–343.

Engleman, H., Kingshott, R., Wraith, P., Mackay, T., Deary, I., & Douglas, N. (1999).

Randomized placebo-controlled crossover trial of continuous positive airway pressure for mild sleep Apnoea/Hypopnea syndrome. *American Journal of Respiratory and Critical Care Medicine*, 159(2), 461-467.

doi: 10.1164/ajrccm.159.2.9803121

Engleman, H., Matt, R., & Wild (2003). Improving CPAP use by patients with the

sleep apnoea/hypopnoea syndrome (SAHS). *Sleep Medicine Reviews*,

7(1), 81–99. doi:10.1053/smr.2001.0197

American Pharmacist Association (2004). *Enhancing Patient Adherence: Proceedings*

of the Pinnacle Roundtable Discussion. Retrieved from

[http://www.pharmacist.com/AM/Template.cfm?Section=Home2&](http://www.pharmacist.com/AM/Template.cfm?Section=Home2&TEMPLATE=/CM/ContentDisplay.cfm&CONTENTID=11174)

[TEMPLATE=/CM/ContentDisplay.cfm&CONTENTID=11174](http://www.pharmacist.com/AM/Template.cfm?Section=Home2&TEMPLATE=/CM/ContentDisplay.cfm&CONTENTID=11174)

Eysenck, S., Eysenck, H., & Barrett, P. (1985). A revised version of the

psychoticism scale. *Personality and Individual Differences*, 6(1), 21-29.

doi.org/10.1016/0191-8869(85)90026-1

Fischer, M., Stedman, M., Lii, J., Vogeli, C., Shrank, W., Brookhart, A., &

Weissman, J. (2010). Primary Medication Non-Adherence: Analysis of 195,930 Electronic Prescriptions. *Journal of General Internal Medicine*,

25(4), 284-290. doi:10.1007/s11606-010-1253-9

- Flemons, W., & Tsai, W. (1997). Quality of life consequences of sleep-disordered breathing. *Journal of Allergy and Clinical Immunology*, 99(2), S750-S756.
- Fletcher, E., Lesske, J., Qian, W., Miller, C., & Unger T. (1992). Repetitive, episodic hypoxia causes diurnal elevation of blood pressure in rats. *Hypertension*, 19(6), 555–561. doi: 10.1161/01.HYP.19.6.555
- Francis, L. (1999). Happiness is a thing called stable extraversion: A further examination of the relationship between the Oxford Happiness Inventory and Eysenck's dimensional model of personality and gender. *Personality and Individual Differences*, 26(1), 5-11. doi:10.1016/S0191-8869(98)00185-8
- Francis, L., & Wilcox. C. (1998). The relationship between Eysenck's personality dimensions and Bem's masculinity and femininity scales revisited. *Personality and Individual Differences*, 25(4), 683–687. doi.org/10.1016/S0191-8869(98)00085-3
- García-Río, F., Racionero, M., Pino, J., Martínez, I., Ortuño, F., Villasante, C., & Villamor, J. (2000). Sleep apnoea and hypertension: the role of peripheral chemoreceptors and the sympathetic system. *Chest*, 117(5), 1417-1425.
- Giles, T., Lasserson, T., Smith, B., White, J., Wright, J., & Cates, C. (2006). Continuous positive airways pressure for obstructive sleep apnoea in adults. *Cochrane Database Systematic Reviews*, 25(1), 1-80. doi: 10.1002/14651858.CD001106.pub2

- Glanz, K., Rimer, B., & Lewis, F. (2002). *Health Behavior and Health Education: Theory, Research and Practice* (3rd ed.). Jossey-Bass: San Francisco, CA.
- Glicksohn, J., & Bozna, M. (2000). Developing a personality profile of the bomb-disposal expert: The role of sensation seeking and field dependence-independence. *Personality and Individual Differences*, 28(1), 85–92.
doi.org/10.1016/S0191-8869(99)00083-5
- Glicksohn, J., & Golan, H.(2001). Personality, cognitive style and assortive mating. *Personality and Individual Differences*, 30(7), 1199–1209. doi.org/10.1016/S0191-8869(00)00103
- Greenham-Conway, B. (2000). *Compliance with continuous positive airway pressure therapy improves mood and psychological functioning in sleep-apnoea-hypopnea syndrome*. Adler School of Professional Psychology: US
- Guilleminault, C., Eldridge, F., Tilkian, A., Simmons, F., & Dement, W. (1977). Sleep apnoea syndrome due to upper airway obstruction: a review of 25 cases. *Archives of Internal Medicine*, 137(3), 296-300. doi:10.1001/archinte.1977.03630150020008.
- Halamandaris, K., & Power, K. (1999). Individual differences, social support and coping with the examination stress: A study of the psychosocial and academic

- adjustment of first year home students. *Personality and Individual Differences*, 26(4), 665–685. doi.org/10.1016/S0191-8869(98)00172-X
- Hardinge, F., Pitson, D., & Stradling, J. (1995). Use of the Epworth Sleepiness Scale to demonstrate response to treatment with nasal continuous positive airways pressure in patients with obstructive sleep apnoea. *Respiratory Medicine*, 89(9), 617-620. doi.org/10.1016/0954-6111(95)90230-9
- Hassaballa, H., Tulaimat, A., Herdegen, J., & Mokhlesi, B. (2005). The effect of continuous positive airway pressure on glucose control in diabetic patients with severe obstructive sleep apnoea. *Sleep and Breathing*, 9(4), 176-80. doi.org/10.1007/s11325-005-0033-y
- Hossain, J., Ahmad, P., Reinish, L., Kayumov, L., Hossain, N., & Shapiro, C. (2005). Subjective fatigue and subjective sleepiness: Two independent consequences of sleep disorders? *Journal of Sleep Research*, 14(3), 245–253.
- Howell, D. (2007). The analysis of missing data. In Outhwaite, W. & Turner, S. *Handbook of Social Science Methodology*. London: Sage.
- Hussain, A. (1996). Obstructive sleep apnoea syndrome. *Sound Asleep*, 19, 1-3.
Retrieved from <http://www.britishsnoring.co.uk/pdf/j6.pdf>
- Hoy, C., Vennelle, M., Kingshott, R., Engleman, H., & Douglas, N. (1999). Can Intensive Support Improve Continuous Positive Airway Pressure Use in

Patients with the Sleep Apnoea/Hypopnea Syndrome? *American Journal of Respiratory and Critical Care Medicine*, 159(4), 1096–1100. doi: 10.1164/ajrccm.159.4.9808008

Hui, D., Chan, J., Choy, D., Ko, F., Li, T., Leung, R., & Lai, C. (2000). Effects of Augmented Continuous Positive Airway Pressure Education and Support on Compliance and Outcome in a Chinese Population. *Chest*, 117(5): 1411-1416. doi:10.1378/chest.117.5.1410

Iber, C., Ancoli-Israel, S., Chesson, A. & Quan, S. (2007). American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications: American Academy of Sleep Medicine.

ICSD-2. (2005). *The International Classification of Sleep Disorders: Diagnostic and Coding Manual* (2nd ed.). Westchester: American Academy of Sleep Medicine.

Janson, C., Nages, E., Svedberg-Randt, S., & Lindberg, E. (2000). What characterizes patients who are unable to tolerate continuous positive airway pressure (CPAP) treatment? *Respiratory Medicine*, 94(2), 145-149. doi:10.1053/rmed.1999.0703

Janz, N., & Becker, M. (1984). The Health Belief Model: A Decade Later. *Health Education Behavior*, 11(1), 1-47. doi: 10.1177/109019818401100101

- Javaheri, S., Parker, T., Liming, J., Corbett, W., Nishiyama, B., Wexler, L., & Roselle, G. (1998). Sleep apnoea in 81 ambulatory male patients with stable heart failure: types and their prevalences, consequences, and presentations. *Circulation* 97, 2154-2159. doi: 10.1161/01.CIR.97.21.2154
- Johns, M. (1993). Daytime sleepiness, snoring, and obstructive sleep apnoea. The Epworth Sleepiness Scale. *Chest*, 103(1), 30-36. doi:10.1378/chest.103.1.30
- Johns, M. (1991). A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*, 14(6), 50-55.
- Johns, M. (2002). Sleep propensity varies with behaviour and the situation in which it is measured: the concept of somnificity. *Journal of Sleep Research*, 11(1), 61-67. doi:10.1046/j.1365-2869.2002.00274.x
- Joo, M., & Herdegen, J. (2007). Sleep apnoea in an urban public hospital: assessment of severity and treatment adherence. *Journal of Clinical Sleep Medicine*, 3(3), 285-288.
- Kaminska, M., Jobin, V., Mayer, P., Amyot, R., Perraton-Brillon, M., & Bellemare, F., (2010). The Epworth Sleepiness Scale: Self-administration versus administration by the physician, and validation of a French version. *Canadian Respiratory Journal*, 17(2), 27-34.

- Katon, W., & Ciechanowski, P. (2002). Impact of major depression on chronic medical illness. *Journal of Psychosomatic Research*, 53(4), 859-63.
- Katon, W., Sullivan, M., & Walker, E. (2001). Medical symptoms without identified pathology: relationship to psychiatric disorders, childhood and adult trauma, and personality traits. *Annals of Internal Medicine*, 134(9), 917-25.
doi:10.7326/0003-4819-134-9_Part_2-200105011-00017
- Kavanagh, D., Gooley, S., & Wilson, P. (1993). Prediction of adherence in diabetes. *Journal of Behavioural Medicine*, 16(5), 509–522. doi:10.1007/BF00844820
- Kjelsberg, F., Ruud, E., & Stavem, K. (2005). Predictors of symptoms of anxiety and depression in obstructive sleep apnoea. *Sleep Medicine*, 6(4), 341-346. doi:10.1016/j.sleep.2005.02.004
- Kline, C., Crowley, E., Ewing, G., Burch, J., Blair, S., Durstine, J., Davis, J. & Youngstedt, S. (2011). The effect of exercise training on obstructive sleep apnea and sleep quality: a randomized controlled trial. *Sleep*, 34(12), 1631-1640. doi: 10.5665/sleep.1422
- Kribbs, N., Pack, A., Kline, L., Getsy, J., Schuett, J., Henry, J., Maislin, G. & Dinges, D. (1993). Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnoea. *American Review of Respiratory Disease*, 147(5), 1162-1168. doi:10.1164/ajrccm/147.5.1162

- Kripke, D., Ancoli-Israel, S., Klauber, M., Wingard, D., Mason, W., & Mullaney, D. (1997). Prevalence of sleep-disordered breathing in ages 40-64 years: a population-based survey. *Sleep*, 20(1), 65-76.
- Krishnan, V., & Collop, N. (2006). Gender differences in sleep disorders. *Current Opinion in Pulmonary Medicine*, 12(6), 383-389.
- Laerd statistics (n.d.). *Testing for Normality using SPSS*. Retrieved from <https://statistics.laerd.com/spss-tutorials/testing-for-normality-using-spss-statistics.php>
- Lanfranchi, P., Braghiroli, A., Bosimini, E., Mazzuero, G., Colombo, R., Donner, C., Giannuzzi, P. (1999). Prognostic value of nocturnal Cheyne-Stokes respiration in chronic heart failure. *Circulation* 99,1435-1440. doi:10.1161/ 01.CIR.99.11.1435
- Lavie, P. (2003). *Restless Nights: Understanding Snoring and Sleep Apnoea*. New Haven, CT: Yale University Press.
- Lave, P. (2008). Who was the first to use the term Pickwickian in connection with sleepy patients? History of sleep apnoea syndrome. *Sleep Medicine Reviews*. 12(1), 5-17. doi:10.1016/j.smr.2007.07.008
- Lavie, P., Pillar, G., & Malhotra, A. (2002). *Sleep Disorders: Diagnosis, management and treatment. A handbook for clinicians*. United Kingdom: Martin Dunitz.

- Leganger, A., Kraft, P., & Roysamb, E. (2000). Perceived self-efficacy in health behaviour research: Conceptualisation, measurement and correlates. *Psychology and Health, 15*(1), 51–69. doi:10.1080/08870440008400288
- Lewis, D. (2007). *Winsorisation for estimates of change*. Papers presented at the ICES-III, June 18-21, 2007, Montreal, Quebec, Canada. Retrieved from <http://www.amstat.org/meetings/ices/2007/proceedings/ICES2007-000113.PDF>
- Lewis, K., Seale, L., Bartle, I., Watkins, A., & Ebdon, P. (2004). Early predictors of CPAP use for the treatment of obstructive sleep apnoea. *Sleep, 27*(1), 134-138.
- Lindberg, E., & Gislason, T. (2000). CLINICAL REVIEW ARTICLE: Epidemiology of sleep-related obstructive breathing. *Sleep Medicine Reviews, 4*(5), 411-33. doi:10.1053/smr.v.2000.0118
- Linton, D., & Wiener, N. (2001). Personality and potential conceptions: Mating success in a modern Western male sample. *Personality and Individual Differences, 31*(5), 675–688. doi.org/10.1016/S0191-8869(00)00170-7
- Lovas, I. (2013). *BLDC motor control for respirators*. Freescale Semiconductor - February 12, 2013. Retrieved from <http://www.edn.com/design/medical/4406849/BLDC-motor-control-for-respirators>

- Luszczynska, A., Scholz, U., & Schwarzer, R. (2005). The general self-efficacy scale: Multicultural validation studies. *Journal of Psychology, 139*(5), 439-457.
- Mahtani, K., Heneghan, C., Glasziou, P., & Perera, R. (2011). Reminder packaging for improving adherence to self-administered long-term medications. *Cochrane database of systematic reviews, 7*(9), 1-62. doi:10.1002/14651858.CD005025.
- Malcarne, V. L., Drahota, A., & Hamilton, N. A. (2005). Children's health-related locus of control beliefs: Ethnicity, gender, and family income. *Children's Health Care, 34*(1), 47-59. doi:10.1207/s15326888chc3401_4
- Malhotra, A., & White, D. (2002). Obstructive sleep apnoea. *Lancet, 360*(9328), 237-245. doi:10.1016/S0140-6736(02)09464-3
- Malhotra, A., Ayas, N., & Epstein, L. (2000). The art of science of continuous positive airway pressure therapy in obstructive sleep apnoea. *Current Opinion in Pulmonary Medicine, 6*(6), 490-495.
- Manni, R., Politini, L., Ratti, M., & Tartara, A. (1999). Sleepiness in obstructive sleep apnoea syndrome and simple snoring evaluated by the Epworth Sleepiness Scale. *Journal of Sleep Research, 8*(4), 319-320. doi:10.1046/j.1365-2869.1999.00166.x

- Marinker, J., & Shaw, J. (2003). Not to be taken as directed. *British Medical Journal*, 326(7385), 348-349. doi: <http://dx.doi.org/10.1136/bmj.326.7385.348>
- Martin, T., & Kirkaldy, B. (1998). Gender differences on the EPQ-R and attitudes to work. *Personality and Individual Differences*, 24(1), 1-5. doi.org/10.1016/S0191-8869(97)00143-8
- Masa, J., Rubio, M., & Findley, L. (2000). Habitually sleepy drivers have a high frequency of automobile crashes associated with respiratory disorders during sleep. *American Journal of Respiratory and Critical Care Medicine*, 162(4), 1407-1412. doi:10.1164/ajrccm.162.4.9907019
- Massie, C., Hart, R., Peralez, K., & Richards, G. (1999). Effects of humidification on nasal symptoms and compliance in sleep apnoea patients using continuous positive airway pressure. *Chest*, 116(2), 403-408. doi:10.1378/chest.116.2.403
- Mathur, R., & Douglas, N. (1995). Family studies in patients with the sleep apnoea-hypopnea syndrome. *Annals of Internal Medicine*, 122(3), 174-178. doi:10.7326/0003-4819-122-3-199502010-00003
- McArdle, N., Devereux, G., Heidarnejad, H., Engleman, H., Mackay, T., & Douglas, N. (1999). Long-term use of CPAP therapy for sleep apnoea/hypopnea syndrome. *American Journal of Respiratory and Critical Care Medicine*, 159(4), 1108-1114. doi: 10.1164/ajrccm.159.4.9807111

- McCormick-Brown, K. (1999). *Health Belief Model*. Retrieved, from http://hsc.usf.edu/~kmbrown/Health_Belief_Model_Overview.htm
- McDaid, C., Griffin, S., Weatherly, H., Durée, K., van der Burgt, M., van Hout, S., Akers, J., Davies, R., Sculpher, M., & Westwood, M. (2009). Continuous positive airway pressure devices for the treatment of obstructive sleep apnoea-hypopnoea syndrome: a systematic review and economic analysis. *Health Technology Assessment*, 13(4), 1- 119. doi:10.3310/hta13040
- McFadyen, T., Espie, C., McArdle, N., Douglas, N., & Engleman, H. (2001). Controlled, prospective trial of psychosocial function before and after continuous positive airway pressure therapy. *European Respiratory Journal*, 18(6), 996-1002. doi:10.1183/09031936.01.00209301
- Means, M., Lichstein, K., Edinger, J., Taylor, D., Durrence, H., Husain, A., Aguillard, N., & Radtke, R. (2003). Changes in Depressive Symptoms after Continuous Positive Airway Pressure Treatment for Obstructive Sleep Apnoea. *Sleep and Breathing*, 7(1), 31-42. Doi:10.1007/s11325-003-0031-x
- Meurice, J., Dore, P., Paquereau, J., Neau, J., Ingrand, P., Chavagnat, J., & Patte, F. (1994). Predictive factors of long-term compliance with nasal continuous positive airway pressure treatment in sleep apnoea syndrome. *Chest*, 105(2), 429-433. doi:10.1378/chest.105.2.429

- Mezzanotte, W., Tangel, D., & White, D. (1992). Waking genioglossal electromyogram in sleep apnoea patients versus normal controls (a neuromuscular compensatory mechanism). *Journal of Clinical Investigation*, 89(5), 1571-1579. doi:10.1172/JCI115751
- Millman, R., Fogel, B., McNamara, M., & Carlisle, C. (1989). Depression as a manifestation of obstructive sleep apnoea: reversal with nasal continuous positive airway pressure. *Journal of Clinical Psychiatry*, 50(9), 348-51.
- Moran, A., Everhart, D., Davis, C., Wuensch, K., Lee, D., & Demaree, H. (2011). Personality correlates of adherence with continuous positive airway pressure (CPAP). *Sleep and Breathing*, 15(4), 687–694. doi10.1007/s11325-010-0422-8
- Muñoz, A., Mayoralas, L., Barbé, F., Pericás, J., & Agustí, A. (2000). Long-term effects of CPAP on daytime functioning in patients with sleep apnoea syndrome. *European Respiratory Journal*, 15(4), 676-81.
- National Institutes of Health. (2010). Retrieved from http://obssr.od.nih.gov/scientific_areas/health_behaviour/adherence/adherenceresearchnetwork.aspx
- Ngoh, L. (2003). Health literacy: a barrier to pharmacist-patient communication and medication adherence. *Journal of the American Pharmacists Association*, 49(5), 132-146. doi:10.1331/JAPhA.2009.07075

- Norman, P., Bennett, P. (1995). "Health Locus of Control". In Conner, M., Norman, P. *Predicting Health Behaviour*. Buckingham: Open University Press.
- Norman, P., Bennett, P. Smith, C., & Murphy, S. (1998). Health Locus of Control and Health Behaviour. *Journal of Health Psychology*, 3(2), 171-180. doi:10.1177/135910539800300202
- Noymer, A. (2008). Alpha, Significance Level of Test. In Lavrakas, P. (2nd ed.). *Encyclopedia of Survey Research Methods* (p19.). London. SAGE Publications, Inc. doi: <http://dx.doi.org/10.4135/9781412963947.n13>
- Nunnally, J., & Bernstein, I. (1994). *Psychometric theory*. *Journal of Psychoeducational Assessment*, 17(3), 275-280. doi: 10.1177/073428299901700307
- Ohayon, M., Caulet, M., Philip, P., Guilleminault, C., & Priest, R. (1997). How sleep and mental disorders are related to complaints of daytime sleepiness. *Archives of Internal Medicine*, 157(22), 2645-2652. doi:10.1001/archinte.1997.00440430127015.
- Olsen, S., Smith, S., Oei, T., & Douglas, J. (2008). Health belief model predicts adherence to CPAP before experience with CPAP. *European Respiratory Journal*, 32(3), 710-17. doi: 10.1183/09031936.00127507

- Olsen, S., Smith, S., & Oei, T. (2008). Adherence to continuous positive airway pressure therapy in obstructive sleep apnoea suffers : a theoretical approach to treatment adherence and intervention. *Clinical Psychology Review*, 28(8), 1355-1371. doi:10.1016/j.cpr.2008.07.004
- Osterberg, L., & Blaschke, T. (2005). Adherence to Medication. *New England Journal of Medicine*, 353(5), 487-497. doi:10.1056/NEJMr050100
- Paiva, T., Farinha, A., Martins, A., Batista, A., & Guilleminault, C. (1997). Chronic headaches and sleep disorders. *Archives of Internal Medicine*, 157(15), 1701-1705. doi:10.1001/archinte.1997.00440360117014
- Patrick. (2013). *Benefits of a Polysomnography*. Sleep Solution. Retrieved from <http://sleepsasolutions.com/benefits-of-a-polysomnography>
- Penedo, F., Gonzalez, J., Dahn, J., Antoni, M., Malow, R., Costa, P., & Schneiderman, N. (2003). Personality, quality of life and HAART adherence among men and women living with HIV/AIDS. *Journal of Psychosomatic Research*, 54(3), 271-278.
- Pepin, J., Krieger, J., Rodenstein, D., Cornette, A., Sforza, E., Delguste, P., Deschaux, C., Grillier, V., & Levy, P. (1999). Effective Compliance during the first 3 Months of Continuous Positive Airway Pressure: A European Prospective Study of 121 Patients. *American Journal of Critical Care Medicine*, 160(4), 1124-1129. doi: 10.1164/ajrcm.160.4.9802027

- Peppard, P., Young, T., Palta, M., Dempsey, J., & Skatrud, J. (2000). Longitudinal study of moderate weight change and sleep-disordered breathing. *Journal of the American Medical Association*, 284(23), 3015-3021. doi:10.1001/jama.284.23.3015.
- Pillar, G., & Lavie, P. (1998). Psychiatric Symptoms in Sleep Apnoea Syndrome: Effects of Gender and Respiratory Disturbance Index. *Chest*, 114(3), 697-703. doi:10.1378/chest.114.3.697
- Popescu, G., Latham, M., Allgar, V., & Elliott, M. (2001). Continuous positive airway pressure for sleep apnoea/hyopnoea syndrome: Usefulness of a 2 week trial to identify factors associated with long term use. *Thorax*, 56(9), 727-735. doi:10.1136/thorax.56.9.727
- Positive Airway Pressure. (n.d.). In Wikipedia, n.d., Retrieved May 29, 2014, from http://en.wikipedia.org/wiki/Positive_airway_pressure.
- Poulet, C., Veale, D., Arnol, N., Lévy, P., Pepin, J., & Tyrrell, J. (2009). Psychological variables as predictors of adherence to treatment by continuous positive airway pressure. *Sleep Medicine*, 10(9), 993–999. doi: 10.1016/j.sleep.2009.01.007.
- Price, I. (2000). *PESS202 Research Methods and Statistics*. Retrieved from http://www.une.edu.au/WebStat/unit_materials/index.htm

- Redline, S., & Tishler, P. (2000). The genetics of sleep apnoea. *Sleep Medicine Reviews*, 4(6), 583-602. doi:10.1053/smr.2000.0120
- Redline, S., Tishler, P., Tosteson, T., Williamson, J., Kump, K., Browner, I., Ferrette, V., & Krejci, P. (1995). The familial aggregation of obstructive sleep apnoea. *American Journal of Respiratory and Critical Care Medicine*, 151(3), 682-687. doi:10.1164/ajrccm/151.3_Pt_1.682
- Reeves-Hoche, M., Meck, R., & Zwillich, C. (1994). Nasal CPAP: an objective evaluation of patient compliance. *American Journal of Respiratory and Critical Care Medicine*, 149(1), 149-54. doi:10.1164/ajrccm.149.1.8111574
- Reynolds, C., Coble, P., Kupfer, D., & Holzer, B. (1982). Application of the multiple sleep latency test in disorders of excessive sleepiness. *Journal of Electroencephalography and Clinical Neurophysiology*, 53(4), 43-52. doi.org/10.1016/0013-4694(82)90009-8
- Reynolds, C., Kupfer, D., McEachran, A., Taska, L., Sewitch, D., & Coble, A. (1984). Depressive psychopathology in male sleep apnoeics. *Journal of Clinical Psychiatry*, 45(7), 287-290.
- Rimm, H., & Jerusalem, M. (1999). Adaptation and validation of an Estonian version of the General Self-Efficacy Scale (ESES). *Anxiety, Stress, and Coping: An International Journal*, 12(3), 329-345. doi:10.1080/10615809908250481

- Robbins, M., Francis, L., & Rutledge, C. (1997). The personality characteristics of Anglican stipendiary parochial clergy in England: Gender differences revisited. *Personality and Individual Differences*, 23(2), 199–204. doi:10.1016/S0191-8869(97)00042-1
- Roehrs, T., Zorick, F., Wittig, R., Conway, W., & Roth, T. (1989). Predictors of objective level of daytime sleepiness in patients with sleep-related breathing disorders. *Chest*, 95(6), 1202-1206. doi:10.1378/chest.95.6.1202
- Rolfe, I., Olson, L. & Saunders, N. (1991). Long-term acceptance of continuous positive airway pressure in obstructive sleep apnoea. *American Review of Respiratory Disease*, 144(5), 1130-1133. doi:10.1164/ajrccm/144.5.1130
- Rosenthal, L., Gerhardstein, R., Lumley, A., Guido, P., Day, R., Syron, M., & Roth, T. (2000). CPAP therapy in patients with mild OSA: implementation and treatment outcome. *Sleep Medicine*, 1(3), 215–220.
- Rotter, J. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs*, 80(1), 1-28. doi: 10.1037/h0092976
- Sage, C., Southcott, A., & Brown, S. (2001). The Health Belief Model and compliance with CPAP treatment for Obstructive Sleep Apnoea. *Behavior Change*, 18(3), 177-185. doi: 10.1375/beh.18.3.177

- Sánchez, A., & Buela-Casal, G. (1999). Assessment of anxiety levels and personality in patients with obstructive sleep apnoea syndrome before and after a month of treatment with continuous positive airway pressure CPAP. *Ansiedad y Estrés*, 5(1), 37-45
- Schafer, J. (1997). *Analysis of Incomplete Multivariate Data*. London: Chapman & Hall.
- Schafer, J. (1999). Multiple imputation: a primer. *Statistical Methods in Medical Research*, 8(1), 3-15. doi:10.1177/096228029900800102
- Schafer, J. & Olsen, M. (1998) Multiple imputation for multivariate missing-data problems: a data analyst's perspective. *Multivariate Behavioral Research*, 33(4), 545-571. doi:10.1207/s15327906mbr3304_5
- Schwarzer, R., Mueller, J., & Greenglass, E. (1999). Assessment of perceived general self-efficacy on the Internet: Data collection in cyberspace. *Anxiety, Stress, and Coping: An International Journal*, 12(2), 145-161. doi:10.1080/10615809908248327
- Shadish, W.R., Clark, M.H., & Steiner, P.M. (2008). Can non-randomized experiments yield accurate answers? A randomized experiment comparing random and non-random assignments. *Journal of the American Statistical Association*, 103(484), 1334–1356. doi: 10.1198/016214508000000733

- Shah, N. Hirsch, A., Zacker, C., Wood, C., Schoenthaler, A., Ogedegbe, G., & Stewart, W, (2009). Predictors of First-Fill Adherence for Patients With Hypertension. *American Journal of Hypertension*, 22(4), 392–396. doi: 10.1038/ajh.2008.367
- Shahrabani, S., Benzion, U., & Yom Din, G. (2009). Factors affecting nurses' decision to get the flu vaccine. *European Journal of Health Economics*, 10(2), 227-31. doi: 10.1007/s10198-008-0124-3
- Sherbourne, C., Meredith, L., Rogers, W., & Ware, J. (1992). Social support and stressful life events: age differences in their effects on health-related quality of life among the chronically ill. *Quality of Life Research*, 1(4), 235-46. doi:10.1007/BF00435632
- Shrank, W., Avorn, J., Rolon, C., & Shekelle, P. (2007). Effect of content and format of prescription drug labels on readability, understanding, and medication use: a systematic review. *Annals of Pharmacotherapy*, 41(5), 783-801. doi:10.1345/aph.1H582
- Smith, S., Lang, C., Sullivan, K., & Warren, J. (2004). A preliminary investigation of the effectiveness of a sleep apnoea education program. *Journal of Psychosomatic Research*, 56(2), 245-249. doi:10.1016/S0022-3999(03)00545-2

- Smith, S., Oei, T., Douglas, J., Brown, I., Jorgensen, G., & Andrews, J. (2008). Confirmatory factor analysis of the Epworth Sleepiness Scale (ESS) in patients with obstructive sleep apnoea. *Sleep Medicine*, 9(7), 739-744. doi:10.1016/j.sleep.2007.08.004
- Smolley, L., Ivey, C., Farkas, M., Faucette, E., & Murphy, S. (1993). Epworth Sleepiness Scale is useful for monitoring daytime sleepiness. *Sleep Research*, 22, 389.
- Somers, V., White, D., Amin r., Abraham, W., Costa, F., Culebras, A., Daniels, S., Floras, J., Hunt, C., Olson, L., Pickering, T., Russell, R., Woo, M., & Young, T. (2008). Sleep Apnoea and Cardiovascular Disease: An American Heart Association/American College of Cardiology Foundation Scientific Statement From the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing In Collaboration With the National Heart, Lung, and Blood Institute National Center on Sleep Disorders Research (National Institutes of Health). *Circulation*, 118(10), 1080-1111, doi: 10.1161/CIRCULATIONAHA.107.189420
- Stammnitz, A., Jerrentrup, A., Penzel, T., Peter, J., Vogelmeier, C., & Becker, H. (2004). Automatic CPAP titration with different self-setting devices in patients with obstructive sleep apnoea. *European Respiratory Journal*, 24(2), 273-278. doi: 10.1183/09031936.04.00074304

Stepnowsky, C., Mao, W., Bardwell, W., Lored, J., & Dimsdale, J. (2012). Mood Predicts Response to Placebo CPAP. *Sleep Disorders, 2012*, 1-6. doi:10.1155/2012/404196.

Stepnowsky, C., & Moore, P. (2003). Nasal CPAP treatment for obstructive sleep apnoea. Developing a new perspective on dosing strategies and compliance. *Journal of Psychosomatic Research, 54*(6), 599-605. doi.org/10.1016/S0022-3999(03)00038-2

Stepnowsky, C., Bardwell, W., Moore, P., Ancoli-Israel, S., & Dimsdale, J. (2002). Psychologic correlates of compliance with Continuous Positive Airway Pressure. *Sleep, 25*(7), 758-762.

Stepnowsky, C., Marler, M., & Ancoli-Israel, S. (2002). Determinants of nasal CPAP compliance. *Sleep Medicine, 3*(3), 239-247. doi.org/10.1016/S1389-9457(01)00162-9

Stradling, J., & Davies, R. (2000). Is more NCPAP better? *Sleep, 23*(4), S150-S153.

Stradling, J., Hardinge, M., & Smith, D. (2004). A novel, simplified approach to starting nasal CPAP therapy in OSA. *Respiratory Medicine, 98*(2), 155-158. doi.org/10.1016/j.rmed.2003.09.010

- Strohl K, & Redline S. (1996). Recognition of obstructive sleep apnoea. *American Journal of Respiratory and Critical Care Medicine*, 154(2), 279-289. doi: 10.1164/ajrccm.154.2.8756795
- Strohl, K., Saunders, N., Feldman, N., & Hallett, M. (1978). Obstructive sleep apnoea in family members. *New England Journal of Medicine*, 299(18), 969-973. doi:10.1056/NEJM197811022991801
- Sullivan, C., Berthon-Jones, M., Issa, F., & Eves, L. (1981). Reversal of Obstructive Sleep Apnoea by Continuous Positive Airway Pressure Applied through the Nares. *Lancet*, 1(8225), 862-865. doi:10.1016/S0140-6736
- Tabachnick, B., & Fidell, L. (2006). *Using Multivariate Statistics* (5th ed.). Boston: Allyn & Bacon.
- Sharma, S., Khanna, A., & Sharma, A. (n.d.). Sleep Disordered Breathing Disorders. Retrieved from The Association of Physicians of India: http://www.apiindia.org/medicine_update_2013/chap104.pdf
- Thorpy, M. (1992). The clinical use of the Multiple Sleep Latency Test. *Sleep*, 15(3), 268-276.
- Tilson, H. (2004). Adherence or compliance? Changes in terminology. *Annals of Pharmacotherapy*, 38(1), 161-162. doi: 10.1345/aph.1D207

- Trinder, J., Padula, M., Berlowitz, D., Kleiman, J., Breen, S., Rochford, P., Worsnop, C., Thompson, B., & Pierce, R. (2001). Cardiac and respiratory activity at arousal from sleep under controlled ventilation conditions. *Journal of Applied Physiology*, 90(4), 1455-1463.
- Trupp, R., Corwin, E., Ahijevych, K., & Nygren, T. (2011). The impact of educational message framing on adherence to continuous positive airway pressure therapy. *Behavioral Sleep Medicine*, 9(1), 38-52. doi: 10.1080/15402002.2011.533993.
- Tzischinsky, O., Shahrabani, S., & Peled, R. (2011). Factors Affecting the Decision to be Treated with Continuous Positive Airway Pressure for Obstructive Sleep Apnoea Syndrome. *Israel Medical Association Journal*, 13(7), 413-419.
- U.S. NIH Office of Behavior and Social Sciences Research. (2008). *Framework for adherence research and translation: a blueprint for the next ten years*. http://obssr.od.nih.gov/pdf/Workshop_final_report.pdf
- Valentin, A., Subramanian, S., Quan, S., Berry, B., & Parthasarathy, S. (2011). Air leak is associated with poor adherence to autoPAP therapy. *Sleep*, 34(6), 801-806. doi:10.5665/SLEEP.1054
- Victor, L. (1999). *Obstructive Sleep Apnoea*. Retrieved from <http://www.aafp.org/afp/1999/1115/p2279.html>

- Waldhorn, R., Herrick, T., Nguyen, M., O'Donnell, A., Soderro, J., & Potolicchio, S. (1990). Long-term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnoea. *Chest*, 97(1), 33-38. doi:10.1378/chest.97.1.33
- Wallston, K. (n.d.). *Multidimensional Health Locus of Control (MHLC) Scales*. Retrieved from <http://www.nursing.vanderbilt.edu/faculty/kwallston/mhlcscales.htm>
- Wallston, K., Stein, M., & Smith, C. (1994). Form C of the MHLC Scales: A Condition-Specific Measure of Locus of Control. *Journal of Personality Assessment*, 63(3), 534-553. doi:10.1207/s15327752jpa6303_10
- Weaver, T., & Sawyer, A. (2010). Adherence to continuous positive airway pressure treatment for obstructive sleep apnoea: implications for future interventions. *Indian Journal of Medical Research*, 131(2), 245-258.
- Weaver, T., Maislin, G., Dinges, D., Younger, J., Cantor, C., McCloskey, S., & Pack, A. (2003). Self-Efficacy in Sleep Apnoea: Instrument Development and Patient Perceptions of Obstructive Sleep Apnoea Risk, Treatment Benefit, and Volition to Use Continuous Positive Airway Pressure. *Sleep*, 26(6), 727-732.
- Wells, R., Freedland, K., Carney, R., Duntley, S., & Stepanski, E. (2007). Adherence, reports of benefits, and depression among patients treated with continuous

positive airway pressure. *Psychosomatic Medicine*, 69(5), 449-454. doi:
10.1097/psy.0b013e318068b2f7

White, K. (2000). *Dickens' "Fat Boy:" A Classic Case of Sleep Apnoea*. Retrieved
from [http://www.talkaboutsleap.com/sleep-disorders/archives/Snoring
_apnoea_dickens.htm](http://www.talkaboutsleap.com/sleep-disorders/archives/Snoring_apnoea_dickens.htm)

Wiegand, L., & Zwillich, C. (1994). Obstructive sleep apnoea. *Disease-a-Month*,
40(4), 197-252.

Wiese, J., Boethel, C., Phillips, B., Wilson, J., Peters, J., & Viggiano, T. (2005).
CPAP compliance: video education may help! *Sleep Medicine*, 6(2), 171-174.
doi.org/10.1016/j.sleep.2004.08.006

Wild, M., Engleman, H., Douglas, N., & Espie, C. (2004). Can psychological factors
help us to determine adherence to CPAP: A prospective study. *European
Respiratory Journal*, 24(3), 461-465. doi:10.1183/09031936.04.00114603

World Health Organization. (2003). *Adherence to Long-Term Therapies: Evidence for
Action*. Geneva: World Health Organisation. Retrieved from [http://www.who
.int/chp/knowledge/publications/adherence_full_report.pdf](http://www.who.int/chp/knowledge/publications/adherence_full_report.pdf)

Wright, J., Johns, R., Watt, I., Melville, A., & Sheldon, T. (1997). Health effects of
obstructive sleep apnoea and the effectiveness of continuous positive airway

pressure: a systemic review of the research evidence. *British Medical Journal*, 314(7084), 851-860. doi: <http://dx.doi.org/10.1136/bmj.314.7084.851>

Wuensch, K. (2009). *Factor Analysis with SPSS*. Retrieved from www2.chass.ncsu.edu/garson/pa765/structur.htm

Yamamoto, H., Akashiba, T., Kosaka, N., Ito, D., & Horie, T. (2000). Long-term effects nasal continuous positive airway pressure on daytime sleepiness, mood and traffic accidents in patients with obstructive sleep apnoea. *Respiratory Medicine*, 94(1), 87-90. doi:10.1053/rmed.1999.0698

Young, T. (2009). Rationale, design and findings from the Wisconsin Sleep Cohort Study: Toward understanding the total societal burden of sleep disordered breathing. *Sleep Medicine Clinics*, 4(1), 37-46. doi:10.1016/j.jsmc.2008.11.003

Young, T., Evans, L., Finn, L., & Palta, M. (1997). Estimation of the clinically diagnosed proportion of sleep apnoea syndrome in middle-aged men and women. *Sleep*, 20(9), 705-706.

Young, T., & Peppard, P. (2000). Sleep-disordered breathing and cardiovascular disease: epidemiologic evidence for a relationship. *Sleep*, 23(4), S122-S126.

Young, T., Blustein, J., Finn, L., & Palta, M. (1997). Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults.

Sleep, 20(8), 608-613.

Young, T., Palta, M., Dempsey, J., Skatrud, J., Weber, S., & Badr, S. (1993). The occurrence of sleep-disordered breathing among middle-aged adults.

New England Journal of Medicine, 328(17), 1230-1235. doi: 10.1056/NEJM199304293281704

Young, T., Peppard, P., & Gottlieb, D. (2002). Epidemiology of Obstructive Sleep Apnoea. *American Journal of Respiratory Critical Care Medicine*, 165(9),

1217-1239. doi: 10.1164/rccm.2109080

Yu, B. Ancoli-Israel, S., Dimsdale, J. (1999). Effect of CPAP treatment on mood states in patients with sleep apnoea. *Journal of Psychiatric Research*,

33(5), 427-32. doi: 10.1016/S0022-3956(99)00020-5

Zammit, G. (2008). *Excessive Sleepiness Associated with Obstructive Sleep Apnoea.*

Touch Briefing. Retrieved from <http://www.touchbriefings.com/pdf/3237/zammit.pdf>

Appendix A

Austin Health consent form



Version: 1 Date: 22/02/2005
--

Consent Form to Participate in Research

Project Title:

Behavioural And Psychological Factors Affecting Treatment Compliance In
Patients With Obstructive Sleep Apnoea.

*I,have been invited to participate in the above
study which is being conducted under the direction of Dr. Gerard Kennedy.*

*I understand that while the study will be under his supervision, other relevant
and appropriate persons may assist or act on his behalf.*

My consent is based on the understanding that the study involves:

1. the administration of psychological tests
2. the use of Nasal CPAP
3. the completion of a sleep diary and a short follow up interview

The study may involve the following risks, inconvenience and discomforts, which
have been explained to me:

The use of nasal CPAP may cause some initial inconvenience and discomfort.

The main inconvenience is the time involved. This includes completing two 20 minute questionnaires – one before your initial sleep study and one in six month time.

I have received and read the attached 'Participant Information Sheet' and understand the general purposes, methods and demands of the study. All of my questions have been answered to my satisfaction. I understand that the project may not be of direct benefit to me.

I can withdraw or be withdrawn by the Principal Investigator from this study at any time, without prejudicing my further management.

I consent to the publishing of results from this study provided my identity is not revealed.

I hereby voluntarily consent and offer to take part in this study.

Signature (Participant)

Date:

Time:

Witness to signature

Date:

Time:

Signature (Investigator)

Date:

Time:

Appendix B

Victoria University consent form

Victoria University of Technology

Consent Form for Participants Involved in Research

INFORMATION TO PARTICIPANTS:

We would like to invite you to be a part of a study into factors affecting degrees compliance to treatment in obstructive sleep apnoea patients. The objective of the study is to investigate treatment compliance issues in patients advised to use CPAP. The study will examine the role of psychological and behavioural factors, such as knowledge, attitudes, personality, mood, sleepiness, self-efficacy, and locus of control in compliance to CPAP therapy. It is envisaged that the data and results collected from this study will contribute to the existing literature and provide for a solid basis for the development of intervention programs aimed at reducing non-compliance in the treatment of OSA.

CERTIFICATION BY PARTICIPANT

I,

of

certify that I am at least 17 years old* and that I am voluntarily giving my consent to participate in the study entitled: Factors affecting compliance to treatment in obstructive sleep apnoea patients, being conducted at Victoria University of Technology by: Simon Mamone and Dr. Gerard Kennedy.

I certify that the objectives of the study, together with any risks to me associated with the procedures listed below to be carried out in the study, have been fully explained to me by Simon Mamone and that I freely consent to participation involving the use of these procedures.

Procedures:

The study will be conducted in five phases in a staggered stage format (meaning that participants will be able to participate anytime within the designated time period).

Data will be gathered in three forms, participants' reports and objective reports (from the CPAP pump fitted with compliance meters). Participants will be tested twice during the six month study period. This will allow the measurement of medium-term compliance to CPAP treatment. Compliance will be described in terms of CPAP use as a percentage of hours prescribed by the patient's physician.

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this study at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that the information I provide will be kept confidential.

Signed:..... Date:

Witness other than the experimenter (as appropriate)

Signed:..... Date:

Any queries about your participation in this project may be directed to the researcher (Name: Dr Gerard Kennedy on 9365 2481 or Simon Mamone 9285 5338). If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University of Technology, PO Box 14428 MCMC, Melbourne, 8001 (telephone no: 03-9688 4710).

[*please note: where the subject/s is aged under 18, separate parental consent is required; where the subject is unable to answer for themselves due to mental illness or disability, parental or guardian consent may be required.]

Appendix C

Demographic questions

Please complete the following questions about yourself. Please remember that all information collected will be kept confidential, and will only be used in order to complete the proposed study. If you have any questions or need any further assistance, please do not hesitate to contact the investigators of this study.

Are you	Please circle your age range
Male <input type="checkbox"/>	18 – 28
Female <input type="checkbox"/>	28 – 38
	38 – 48
	48 – 58
	58 – 68
	68 +

Please tick the highest education level
you have achieved

Primary school	<input type="checkbox"/>
High school	<input type="checkbox"/>
TAFE	<input type="checkbox"/>
University degree	<input type="checkbox"/>
Post graduate degree	<input type="checkbox"/>
Private course/other courses	<input type="checkbox"/>

Do you suffer from any other illness apart
from Obstructive Sleep Apnoea (OSA)?

Yes ☐

No ☐

Do you suffer from any other illness apart
from Obstructive Sleep Apnoea (OSA)?

Yes ☐

No ☐

Do you believe you actually have OSA?

Yes ☐

No ☐

Do you THINK it will be difficult to use
CPAP?

Yes ☐

No ☐

Do you THINK it will be uncomfortable
to use CPAP?

Yes ☐

No ☐

Do you THINK it will be uncomfortable
to use CPAP?

Yes ☐

No ☐

Do you THINK CPAP treatment
alleviates OSA symptoms for you?

Yes ☐

No ☐

Appendix D

The Profile of Mood States

Below is a list of words that describe feelings people have. Please read each one carefully. Then fill in ONE circle 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	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	20. Discouraged	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
2. Angry	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	21. Resentful	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
3. Worn out	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	22. Nervous	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
4. Unhappy	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	23. Miserable	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
5. Lively	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	24. Cheerful	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
6. Confused	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	25. Bitter	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
7. Peeved	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	26. Exhausted	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
8. Sad	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	27. Anxious	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
9. Active	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	28. Helpless	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
10. On edge	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	29. Weary	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
11. Grouchy	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	30. Bewildered	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
12. Blue	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	31. Furious	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
13. Energetic	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	32. Full of pep	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
14. Hopeless	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	33. Worthless	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
15. Uneasy	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	34. Forgetful	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

- | | | | |
|---------------------------|---|----------------------------|---|
| 16. Restless | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> | 35. Vigourous | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> |
| 17. Unable to concentrate | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> | 36. Uncertain about things | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> |
| 18. Fatigued | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> | 37. Bushed | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> |
| 19. Annoyed | <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> <input type="radio"/> | | |

Appendix E

General Self-Efficacy Scale

Below is a list of statements. Please read each one carefully. Then fill in ONE circle that best describes you next to each statement.

	NOT AT ALL TRUE	HARDLY TRUE	MODERATELY TRUE	EXACTLY TRUE
1. I can always manage to solve difficult problems if I try hard enough.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
2. If someone opposes me, I can find the means and ways to get what I want.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
3. It is easy for me to stick to my aims and accomplish my goals.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
4. I am confident that I could deal efficiently with unexpected events.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
5. Thanks to my resourcefulness, I know how to handle unforeseen situations.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
6. I can solve most problems if I invest the necessary effort.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
7. I can remain calm when facing difficulties because I can rely on my coping abilities.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
8. When I am confronted with a problem, I can usually find several solutions.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

- | | | | | |
|---|-----------------------|-----------------------|-----------------------|-----------------------|
| 9. If I am in trouble, I can usually think of a solution. | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 10. I can usually handle whatever comes my way. | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

Appendix F

The Eysenck Personality Questionnaire

Please answer each question by filling in the 'YES' or 'NO' following the question. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the questions.

- PLEASE REMEMBER TO ANSWER EACH QUESTION

	YES	NC
1. Does your mood often go up and down?	<input type="radio"/>	<input type="radio"/>
2. Do you take much notice of what people think?	<input type="radio"/>	<input type="radio"/>
3. Are you a talkative person?	<input type="radio"/>	<input type="radio"/>
4. If you say you will do something, do you always keep your promise no matter how inconvenient it might be?	<input type="radio"/>	<input type="radio"/>
5. Do you ever feel 'just miserable' for no reason?	<input type="radio"/>	<input type="radio"/>
6. Would being in debt worry you?	<input type="radio"/>	<input type="radio"/>
7. Are you rather lively?	<input type="radio"/>	<input type="radio"/>
8. Were you ever greedy by helping yourself to more than your fair share of anything?	<input type="radio"/>	<input type="radio"/>
9. Are you an irritable person?	<input type="radio"/>	<input type="radio"/>
10. Would you take drugs which may have strange or dangerous effects?	<input type="radio"/>	<input type="radio"/>
11. Do you enjoy meeting new people?	<input type="radio"/>	<input type="radio"/>
12. Have you ever blamed someone for doing something you knew was really your fault?	<input type="radio"/>	<input type="radio"/>

13. Are your feelings easily hurt?	<input type="radio"/>	<input type="radio"/>
14. Do you prefer to go your own way rather than act by the rules?	<input type="radio"/>	<input type="radio"/>
15. Can you usually let yourself go and enjoy yourself at a lively party?	<input type="radio"/>	<input type="radio"/>
16. Are <i>all</i> your habits good and desirable ones?	<input type="radio"/>	<input type="radio"/>
17. Do you often feel 'fed-up'?	<input type="radio"/>	<input type="radio"/>
18. Do good manners and cleanliness matter much to you?	<input type="radio"/>	<input type="radio"/>
19. Do you usually take the initiative in making new friends?	<input type="radio"/>	<input type="radio"/>
20. Have you ever taken anything (even a pin or button) that belonged to someone else?	<input type="radio"/>	<input type="radio"/>
21. Would you call yourself a nervous person?	<input type="radio"/>	<input type="radio"/>
22. Do you think marriage is old-fashioned and should be done away with?	<input type="radio"/>	<input type="radio"/>
23. Can you easily get some life into a rather dull party?	<input type="radio"/>	<input type="radio"/>
24. Have you ever broken or lost something belonging to someone else?	<input type="radio"/>	<input type="radio"/>
25. Are you a worrier?	<input type="radio"/>	<input type="radio"/>
26. Do you enjoy cooperating with others?	<input type="radio"/>	<input type="radio"/>
27. Do you tend to keep in the background on social occasions?	<input type="radio"/>	<input type="radio"/>
28. Does it worry you if you know there are mistakes in your work?	<input type="radio"/>	<input type="radio"/>
29. Have you ever said anything bad or nasty about anyone?	<input type="radio"/>	<input type="radio"/>
30. Would you call yourself tense or 'highly-stung'?	<input type="radio"/>	<input type="radio"/>
31. Do you think people spend too much time safeguarding their future with savings and insurance?	<input type="radio"/>	<input type="radio"/>
32. Do you like mixing with people?	<input type="radio"/>	<input type="radio"/>
33. As a child were you ever cheeky to your parents?	<input type="radio"/>	<input type="radio"/>
34. Do you worry too long after an embarrassing experience?	<input type="radio"/>	<input type="radio"/>
35. Do you try not to be rude to people?	<input type="radio"/>	<input type="radio"/>

- | | | |
|---|-----------------------|-----------------------|
| 36. Do you like plenty of bustle and excitement around you? | <input type="radio"/> | <input type="radio"/> |
| 37. Have you ever cheated at a game? | <input type="radio"/> | <input type="radio"/> |
| 38. Do you suffer from 'nerves'? | <input type="radio"/> | <input type="radio"/> |
| 39. Would you like other people to be afraid of you? | <input type="radio"/> | <input type="radio"/> |
| 40. Have you ever taken advantage of someone? | <input type="radio"/> | <input type="radio"/> |
| 41. Are you mostly quiet when you are with other people? | <input type="radio"/> | <input type="radio"/> |
| 42. Do you often feel lonely? | <input type="radio"/> | <input type="radio"/> |
| 43. Is it better to follow society's rules than go your own way? | <input type="radio"/> | <input type="radio"/> |
| 44. Do other people think of you as being very lively? | <input type="radio"/> | <input type="radio"/> |
| 45. Do you always practise what you preach? | <input type="radio"/> | <input type="radio"/> |
| 46. Are you often troubled about feelings of guilt? | <input type="radio"/> | <input type="radio"/> |
| 47. Do you sometimes put off until tomorrow what you ought to do today? | <input type="radio"/> | <input type="radio"/> |
| 48. Can you get a party going? | <input type="radio"/> | <input type="radio"/> |

Appendix G

Multidimensional Health Locus of Control–C

Each item below is a belief statement about your medical condition with which you may agree or disagree. Beside each statement is a scale which ranges from strongly disagree to strongly agree. For each item we would like you to fill in the circle that represents the extent to which you agree or disagree with that statement.

- Please make sure that you answer EVERY ITEM and that you fill in ONLY ONE circle per item. This is a measure of your personal beliefs; obviously, there are no right or wrong answers.

[illegible]

Appendix H

Health Belief Scale

The following questions assess your beliefs about sleep apnoea. This measure surveys four domains of perceived beliefs relevant to having sleep apnoea: (a) susceptibility to sleep apnoea complications; (b) severity of sleep apnoea complications; (c) benefits of sleep apnoea control; and (d) barriers to executing therapeutic behaviours.

- Please read each statement carefully. Then fill in ONE circle that best describes you next to each statement.

	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR	AGREE	STRONGLY AGREE
<u>Susceptibility</u>					
1. Sleep apnoea can be a serious condition if you do not control it	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
2. My sleep apnoea would be worse if I did nothing about it	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
3. I believe that CPAP treatment will prevent any complications associated with my sleep apnoea	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
4. My sleep apnoea is well controlled	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<u>Severity</u>					
5. My sleep apnoea is no problem to me as long as I feel all right	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

- | | | | | | |
|---|-----------------------|-----------------------|-----------------------|-----------------------|-----------------------|
| 6. My sleep apnoea will have a bad effect on my future health | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 7. My sleep apnoea will cause me to be sick a lot | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 8. I believe I will always need CPAP treatment | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

Benefits

- | | | | | | |
|--|-----------------------|-----------------------|-----------------------|-----------------------|-----------------------|
| 9. I believe I can control my sleep apnoea | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 10. I believe that CPAP will control my sleep apnoea | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 11. CPAP will help me feel better | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

Barriers

- | | | | | | |
|--|-----------------------|-----------------------|-----------------------|-----------------------|-----------------------|
| 12. I would have to change too many habits to follow my CPAP treatment | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 13. It is difficult following the instructions for CPAP treatment prescribed to me | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 14. I can not understand what I've been told about my CPAP treatment | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 15. CPAP treatment interferes with my normal activities | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

Appendix I

Epworth Sleepiness Scale

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? Even if you haven't done some of these activities recently, think about how they would have affected you.

	would never doze	slight chance of dozing	moderate chance of dozing	high chance of dozing
Sitting and reading	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Watching television	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Sitting inactive in a public place--for example, a theater or meeting	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
As a passenger in a car for an hour without a break	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Lying down to rest in the afternoon	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Sitting and talking to someone	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Sitting quietly after lunch (when you've had no alcohol)	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
In a car while stopped in traffic	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>