An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

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DECLARATION OF AUTHENTICITY

I, Rosa Galante, declare that the Doctor of Psychology (Clinical Psychology) thesis entitled "An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers" is no more than 40,000 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:

Submitted with emendations: February 2012

DEDICATION

This thesis is dedicated to the memory of my grandfather, Angelo Galante – a proud and loving man who inspired me to achieve my aspirations.

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TABLE OF CONTENTS

LIST OF ABBREVIATIONS

ANOVA	One-Way Analysis of Variance
AHI	Apnoea-Hypopnoea Index
BDI	Beck Depression Inventory
BMI	Body Mass Index
CPAP	Continuous Positive Airway Pressure
EDS	Excessive Daytime Sleepiness
EEG	Electroencephalogram
EMG	Electromyogram
EOG	Electrooculogram
ESS	Epworth Sleepiness Scale
Hz	Hertz
IR	Infrared
JDS	Johns Drowsiness Scale
KSS	Karolinska Sleepiness Scale
LED	Light Emitting Diode
MAPQ	Multivariate Apnoea Prediction Questionnaire
MMPI	Minnesota Multiphasic Personality Inventory
MSLT	Multiple Sleep Latency Test
MWT	Maintenance of Wakefulness Test
NREM	Non Rapid Eye Movement
ODMS	Optalert Drowsiness Measurement System
OSA	Obstructive Sleep Apnoea
Osler	Oxford Sleep Resistance Test
POMS	Profile of Mood States
PVT	Psychomotor Vigilance Task
REM	Rapid Eye Movement
RT	Reaction Time
SCN	Suprachiasmatic Nucleus

SCL	Symptom Distress Checklist
SDQ	Stop Driving Questionnaire
SPSS	Statistical Package for Social Sciences
SSQ	Sleepiness Symptoms Questionnaire
STAI	State-Trait Anxiety Inventory
SWD	Shift Work Disorder
SWS	Slow-wave sleep

ABSTRACT

Regardless of aetiology, constant sleep disruption results in wide-ranging negative consequences in sleepiness, psychomotor and driving performance, and mood. Individuals with obstructive sleep apnoea (OSA) and those engaged in the tumultuous routine of shift-work experience reduced sleep and poorer sleep quality which is likely to render these consequences more pronounced.

This study aimed to compare subjective and objective sleepiness, psychomotor performance, simulated driving performance and mood disturbances in OSA patients, shift-workers and control participants. This study also aimed to determine what relationships exist between subjective and objective sleepiness, performance and mood for the three groups and to obtain the correlates of accident risk based on a history of reported accidents in the preceding three years.

Forty-seven participants were recruited to form an untreated moderate to severe OSA patient group (15 males and 2 females aged between 37 and 75 years), a rotating shift-work group (12 males and 3 females aged between 35 and 52 years) and a control group (6 males and 9 females aged between 34 and 62 years). Participants attended the sleep laboratory and participated in a sleep latency task, a driving simulator task, and a reaction time task. Objective sleepiness was measured by examining blink duration data and by overnight polysomnography. Participants also completed a series of questionnaires related to their level of sleepiness and their mood.

Group differences were analysed using One-Way Analysis of Variance procedures or Kruskal-Wallis H tests for non-parametric statistics, and relationships between subjective and objective sleepiness, performance, mood, and accident history were determined using correlational analyses. The findings of this study indicated that patients with OSA and shift-workers were differentially affected across measures of subjective and objective sleepiness,

performance and mood as a result of reduced sleep and poor sleep quality. However, relationships between subjective and objective sleepiness, performance and mood revealed that shift-workers exhibited equivalent or greater disturbances in alertness to patients with OSA. In addition, accident history was related to subjective sleepiness for OSA patients, and objective sleepiness and psychomotor functioning among shift-workers.

The findings of the present study lend support to the importance of identifying impairments related to alertness, psychomotor functioning, driving performance and mood for the minimisation of accidental injury to patients with OSA, shift-workers and the community at large. Whilst treatment options are likely to improve these negative effects for OSA patients to a considerable extent, there is a large burden of undiagnosed and untreated OSA. There are also challenges for management to devise more fitting shift schedules and potential intervention strategies that support health-enhancing work environments for shift-workers.

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

INTRODUCTION

Conceptualisations of sleep have evolved considerably from early theories which regarded it as a passive and near-death state. Aristotle, one of the first to devote systematic thought to sleep in his manuscript *'On Sleep and Sleeplessness'*, described sleep as the antithesis of waking (Aristotle, & translated by Beare, 2007). His account of the process of falling asleep, albeit picturesque, posited that the digestion of food created vapours which rose from the stomach because of their higher temperature and accumulated in the brain. The subsequent cooling of the brain caused the vapours to condense, descend and cool the heart, causing sleep (Lavie, 1996). Greek philosopher's expressed slight variations to Aristotle's notion, instead believing that the brain was the body's centre of sensation (Borbely, 1986). Later schools of thought in the eighteenth and nineteenth centuries proposed that sleep was the result of a lack of blood supply to the brain, or conversely, that sleep was caused by an excess of blood in the brain (Lavie, 1996). Theories related to hypnotoxins also gained support, and purported that toxins built up during the day, poisoning the brain, and upon reaching critical levels, sleep was initiated (Lavie, 1996).

The notion of sleep as a passive state remained relatively unchanged until the development of the electroencephalogram (EEG) in 1929 by Hans Berger which allowed the examination of brain activity during sleep and the documentation of changing patterns of sleep and wakefulness (Borbely, 1986). With this advance in sleep research, the notion of sleep as a passive process was disregarded. Although behavioural activity ceases during sleep, one's ability to recall the content of dreams upon awakening suggests that mental activity does occur during sleep (Hirshkowitz, Moore & Minhoto, 1997). Sleep is a universal experience that is essential for survival; however poor quality, insufficient quantity and poorly regulated timing of sleep exert negative effects on wakeful mental and physical functioning. As Borbely (1986) eloquently posited "many people take sleep so much for

granted they hardly ever stop to reflect on its origin and meaning...only when it is disturbed does sleep become a problem" (p. 4).

1.1. Physiology of Sleep

The objective classification of sleep evolved as a consequence of polysomnography which provides a continuous, all night tracing of brain waves (electroencephalogram; EEG), eye movements (electro-oculogram; EOG) and muscle activity (electromyogram; EMG) (Hirshkowitz, Moore & Minhoto., 1997). Sleep alternates cyclically between two intrinsically separate states termed rapid-eye-movement (REM) and non-rapid-eye movement (NREM) in a 90 to 100 minute cycle which is repeated up to five or more times during the night (Rosenthal, 1998). Generally, NREM sleep dominates the first portion of each sleep cycle and governs the transition from a state of wakefulness to deep sleep. Conversely, REM sleep is distributed towards the latter part of the sleep cycle (Rosenthal, 1998).

There is no sleep stage subdivision of REM sleep, but NREM sleep consists of four distinct stages (1, 2, 3 and 4) according to the traditional Rechtshaffen and Kales (1968) scoring manual. The recent American Academy of Sleep Medicine scoring manual proposed that stages three and four of sleep be combined into a single stage (N3) since these are collectively characterised by EEG slow waves (Shulz, 2008). The transition from wakefulness to *stage 1* sleep is characterised by a reduction in muscle activity and slow and predominantly horizontal eye movements (Hirshkowitz et al., 1997). Alpha activity is substituted by relatively low voltage waves with a prominence of activity in the theta range (4-7 Hz) (Monti & Monti, 2008). The occurrence of sleep spindles (brief bursts of rhythmic waves with a frequency of 12-14 Hz) and K-complexes (high amplitude potentials with a negative sharp wave followed by a positive component and a total duration exceeding 0.5 seconds) characterises *stage 2* sleep (Monti & Monti, 1998). Deep sleep or slow wave sleep (SWS) is characterised by the occurrence of high-voltage and slow wave activity (delta

waves with a frequency of 1-2 Hz or slower) in 20 to 50% of the EEG (stage 3), or greater than 50% of the EEG (stage 4) (Monti & Monti, 1998). Conversely, REM sleep is characterised by bursts of rapid eye movements under closed eyelids, desynchronised EEG activity, and muscle atonia (Rosenthal, 1998). Figure 1.01 presents an EEG recording of sleep stages 1, 2, 3 and 4 and REM sleep.

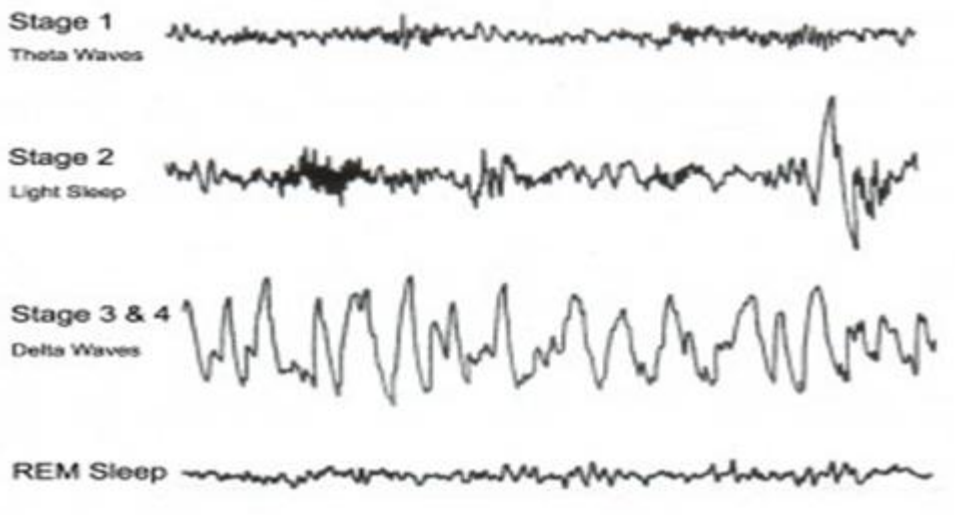

Figure 1.01: EEG recording of sleep stages 1, 2 3, and 4 and REM sleep

Although recordings from the EEG, EOG and EMG collectively determine problems in sleep architecture associated with sleepiness, Lal and Craig (2001) posited that the EEG signal provided the most predictive and reliable measure of fatigue. Increased delta and theta activity can be found in the waking EEG of sleep deprived individuals (Walsh & Lindblom, 1997). Deteriorations in performance occur during a brief period (2-20s) of EEG slowing termed micro-sleeps (Anch, Browman, Mitler & Walsh, 1988). Over a 48 hour period of sleep deprivation, micro-sleeps become increasingly more common, attesting to the strength of the sleep drive (Walsh & Lindblom, 1997).

1.2. Regulation of Sleep

The timing, duration and intensity of sleep are governed by two independent, yet inter-related processes: a *homeostatic* sleep drive and *circadian* rhythms.

1.2.1. Homeostatic sleep drive. The homeostatic sleep drive is regulated as a function of the duration of prior wakefulness (Carskadon & Dement, 1981). Conceptually, this is achieved by the accumulation of a 'pressure' for sleep, during wakefulness, which augments sleep propensity and is dissipated when sleep is initiated (Achermann, 2004). Sleep deprivation increases the homeostatic need for sleep, rendering sleep more likely to occur at inappropriate or unusual times. Priest et al (2001) demonstrated that periods of micro-sleeps (episodes of sleep ≥ 3s duration) occurred after only a single night of sleep deprivation. This finding suggests that while sleep can be briefly delayed, it cannot be averted indefinitely. Conversely, when excess sleep is obtained, the homeostatic mechanism decreases sleep propensity (Roth & Roehrs, 2000).

Sleep was largely conceptualised within the framework of homeostatic principles until the 1960s (Lavie, 2001). Despite this, the paucity of research examining the homeostatic mechanism and sleep loss effects has led to the assumption that this mechanism is a weak component of sleep regulation. However, the role of this sleep drive appears to have been grossly underestimated. Akerstedt and Gillberg (1986) curtailed subjects sleep across four conditions (0, 2, 4 and 8 hours), while minimising confounding circadian factors. Subjects' total sleep time revealed marked dose-dependent increases in the sleep drive with increasing prior sleep deprivation. This finding held true even when the 0 hour condition was disregarded. The substantial responses to sleep loss in this study support the contention that the homeostatic mechanism(s) which partly govern sleep have a strong influence on its overall regulation.

In addition to sleep latency measurements, EEG slow-wave activity during sleep indicates the presence of a homeostatic need for sleep (Akerstedt & Gillberg, 1986; Cajochen & Dijk, 2003; Daan, Beersma & Borbely, 1984; Roth & Roehrs, 2000). Achermann (2004) proposed that slow wave activity was greatest during the initial sleep episode when the need

for sleep was high, but that this gradually reduced when sleep was initiated. Not surprisingly, napping reversed this rise in slow-wave sleep, leading to a reduction in slow-wave activity in the subsequent night's sleep (Achermann, 2004). Slow-wave activity appears to represent an important marker of the homeostatic process; however circadian influences also have a strong effect on the regulation of sleep.

1.2.2. Circadian rhythms. In contrast to the paucity of research examining the homeostatic regulation of sleep, the circadian system has been well documented. Most living organisms exhibit daily rhythms in their physiology and behaviour. The circadian rhythm is among the most predominant of these biological rhythms and functions to maintain normal sleep and wake cycles (Richardson, 2005). A circadian rhythm is dependent upon a system which oscillates with a periodicity of approximately 24 hours in phase with the daily alternation of light and darkness (Toh, 2008). Anatomically, the suprachiastmatic nucleus (SCN) in the hypothalamus has been identified as the circadian pacemaker or 'biological clock' in most vertebrate species (Roth & Roehrs, 2000). The SCN imposes temporal order through the generation of output signals that transmit information pertaining to time of day (Zisapel, 2001). Lesions to the SCN were shown to eliminate circadian rhythmicity in many physiological and behavioural variables, including sleep and wakefulness (Moore-Ede, Sulzman & Fuller, 1982).

Since body temperature is characterised by a robust 24-hour variation, circadian rhythms are typically documented in this way (Campbell, 1997). Studies of the circadian cycle have demonstrated that sleep propensity in humans, measured by the speed of falling asleep, corresponds to the temperature nadir (Roth & Roehrs, 2000). Maximum alertness occurs near the peak, such that when temperature decreases, drowsiness ensues and upon reaching the nadir, sleepiness becomes overpowering. An increase in temperature

corresponds to a decrease in sleepiness. The cycle is initiated again when temperature has reached its maximum level (Hirshkowitz et al., 1997).

Limited synchrony between the circadian rhythm and the scheduled bedtime can impair nocturnal sleep and daytime alertness (Richardson, 2005). Although initially regarded as monophasic in nature, sleep is more appropriately viewed as a polyphasic system with at least two phase positions for the occurrence of sleep within the 24-hour day (Campbell, 1997). Sleep periods initiated at the middle of the falling slope will continue for an average of eight hours and under entrained conditions of daily life, correspond to nocturnal sleep (Campbell, 1997). Sleep periods occurring near the maximum temperature are terminated approximately two hours later, and correspond to an afternoon 'nap' (Campbell, 1997).

1.2.3. Interaction of homeostatic and circadian systems. Although generated by separate mechanisms, circadian and homeostatic drives collectively exert an influence on sleep and vigilance (Achermann, 2004). The concept of a two process model of sleep regulation was initially formulated in 1984, and has since been supported by numerous empirical studies (Daan et al., 1984). The model postulates that sleep propensity or the 'ability to initiate sleep' is determined by a homeostatic process and a circadian process. The homeostatic process, derived from the time course of slow wave activity, is represented by a linear rise during wakefulness and a linear decline during sleep (Achermann, 2004). Derived from sleep duration data, the circadian process is depicted by a distorted sine wave which describes the average partition of a circadian day into 16 hours of wakefulness and 8 hours of sleep (Refinetti, 2006). Figure 1.02 presents a diagram of the interaction between homeostatic and circadian drives under ideal conditions in the absence of sleep deprivation.

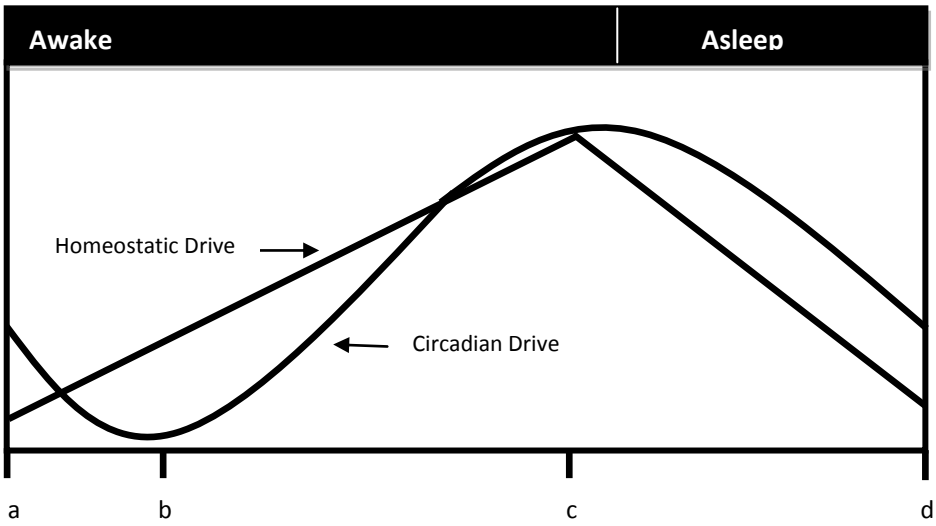

Figure 1.02: The interaction of the homeostatic sleep drive and the circadian sleep drive under ideal conditions in the absence of sleep deprivation (adapted from Refinetti, 2006).

Daan and colleagues (1984) extended this model to incorporate an upper and lower threshold to predict the timing of sleep. In essence, time point *a* represents the waking period, and exhibits no homeostatic sleep drive. The circadian drive continues to fall, accounting for the usual drowsiness that occurs upon awakening. At time point *b*, both drives rise steadily, after which point, the circadian and homeostatic sleep drives reach the maximum level and progression to sleep occurs (*c*). Throughout the sleep period, the circadian and homeostatic drives continue to fall until awakening (*d*) (Refinetti, 2006).

External conditions are assumed to affect the upper and lower thresholds. For example, sleep deprivation conditions create a suspension in the upper threshold, allowing the homeostatic sleep drive to increase further. Refinetti (2006) proposed that little or no sleep over a 48 hour period created conditions for a 'conflict' between the two sleep drives. Alternatively, bed rest, warmth, darkness, or the absence of social stimulation lowers the upper threshold so that sleep is precipitated. A transitory decline of the upper threshold can be evidenced after a brief nap (Daan et al., 1984). The circadian drive modulates the two thresholds which determine the onset and termination of a sleep episode respectively (Achermann, 2004).

Forced desynchrony paradigms provide convincing evidence that sleep propensity is controlled by a single circadian pacemaker that interacts with sleep-wake dependant processes (Lavie, 2001). In line with this, Dijk and Czeisler (1995) conducted an experiment where subjects followed a 28 hour sleep-wake cycle. During one third of the cycle, lights were switched off and subjects were encouraged to sleep. Waking hours were evidenced at different phases of the body temperature cycle, and the findings revealed that maximal sleep propensity coincided strongly with the nadir of the temperature rhythm. This rise in body temperature was paralleled by a steady decrease in sleep propensity (Dijk & Czeisler, 1995).

1.2.4. Four process model of sleep and wakefulness. A four process model of sleep and wakefulness presented by Johns (1993) extends the work of Daan and colleagues (1984). Consistent with the two process model, Johns (1993) proposed that sleep or wakefulness at a particular time was dependent upon the relative strength of two mutually inhibiting drives (wake and sleep), not the absolute strength of either drive independently. However, the addition of primary and secondary components to the sleep and wake drives is incorporated in the model to account for the powerful influence of sleep propensity (Johns, 1993).

The primary wake drive is equivalent to the circadian process C of the two process model; however a secondary wake drive is influenced by posture, behaviour, physical activity, feelings and mental activity. In contrast, the primary sleep drive is due to an intrinsic central nervous system process which varies with a circadian rhythm with a peak between 10pm to 12 midnight and a trough between 6am and 8am. The secondary sleep drive corresponds to the homeostatic process of the two process model (Johns, 1993; Johns, 1998)

The additive effects of the primary and secondary sleep and wake drives collectively constitute the total sleep and wake drive. They mutually inhibit one another (Is and Iw) to produce an oscillator, such that wakefulness is evident when the total wake drive exceeds the

total sleep drive, and sleep occurs when the sleep drive exceeds the wake drive (Johns, 1993). Figure 1.03 presents a diagram of the four process model with sleep and wake drives, and primary and secondary components each inhibiting the other.

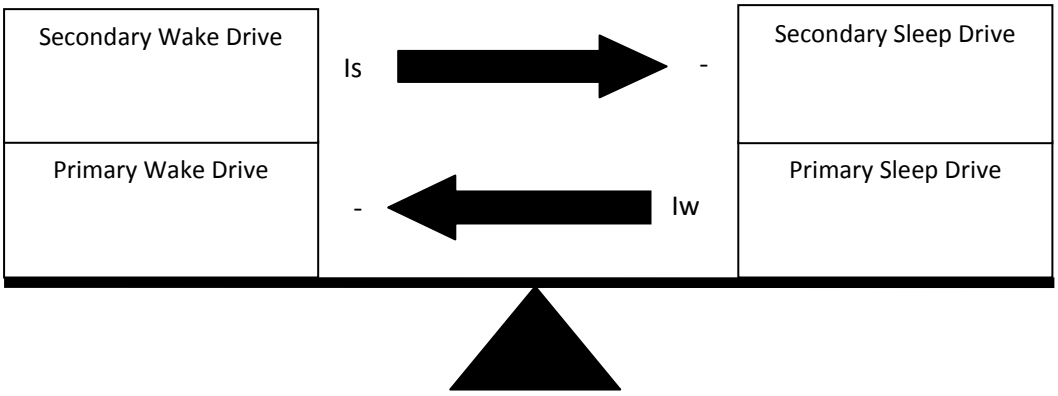

Figure 1.03: Johns (1993) four process model of sleep and wakefulness with sleep and wake drives, and primary and secondary components and each inhibiting the other.

The magnitude of the secondary sleep drive is considered to be the single most important determinant of sleep propensity since changes in this drive are under voluntary control (Johns, 1998). Despite this, sleep propensity may be influenced by a multitude of factors that affect the four sleep and wake drives collectively, and is predominantly related to the circumstances in which it is measured (Johns, 1993). Factors implicated in sleep propensity include time of day, previous sleep deprivation, effects of drugs on the central nervous system, age, physical, cognitive and affective state, irregular work hours such as shift work, or the presence of a sleep disorder (Johns, 1993). Despite the variance in sleep propensity throughout the day, different subjects will have an average sleep propensity (ASP) over prolonged periods about which fluctuations occur (Johns, 1993). For example, the ASP is reported to have an effect on an individual with obstructive sleep apnoea as it increases long-term the total sleep drive, but not the wake drive (Johns, 1993).

1.3. The Function of Sleep

Despite the absence of a unified understanding of sleep function, several theories have been proposed to explain why sleep is necessary. The restorative theory of sleep posits that sleep promotes physiological processes to rejuvenate the body and mind and both REM and NREM sleep are implicated in this hypothesis for body and brain tissue restoration respectively (Anch, Browman, Mitler & Walsh., 1988). Functionally, sleep has also been implicated as a means of energy conservation (Hirshkowitz et al., 1997). Adaptive theories have also been proposed suggesting that sleep is a behaviour which enhances survival (Rechtschaffen, 1998). Experimental protocols that restrict sleep chronically provide an appropriate mechanism for the study of sleep function, and typically reveal that the most reliable outcome of a lack of sleep is sleepiness (Van Dongen, Rogers & Dinges, 2003).

1.4. Sleepiness and Sleep Loss

1.4.1. The construct of sleepiness. Similarly to hunger and thirst, sleepiness represents an underlying physiological need state which is satisfied by sleeping and is integral to the survival of individuals (Mathis & Hess, 2009). The need for clarification of the concept of sleepiness is evidenced when considering that a variety of words are often used interchangeably to denote this construct, such as fatigue or tiredness (Johns, 1998). Fatigue refers to a physiological phenomenon characterised by 'time-on-task performance decrements' which is often relieved by changing the task (Mathis & Hess, 2009). Conversely, tiredness denotes a lack of energy and initiative which is often improved by rest, rather than sleep (Mathis & Hess, 2009). The subjective feeling of sleepiness is characterised by difficulty keeping eyes open, tendency to stay at rest and slowness (Nau, 1997).

Physiological sleepiness increases during wakefulness and in accordance with the two process model of sleep regulation, underlies a circadian rhythm (Daan et al., 1984). Although the expression of sleepiness is typically introspective, behavioural and physiological

manifestations of sleepiness are also evident. In a polysomnographic study assessing prolonged wakefulness, EEG delta and theta activity increased steeply during the hours when subjects would have usually slept (Aeschbach et al., 1997). Although sleepiness remained at a constant level during the day, theta activity increased in the afternoon and in the evening (Aeschbach et al., 1997).

1.4.2. The assessment of sleepiness. The measurement of sleepiness is generally divided into subjective and objective categories. Objective assessments provide a measure of sleep latency in the sleep laboratory during the day (Johns, 1998). Often referred to as the 'gold standard', the Multiple Sleep Latency Test (MSLT) provides an indication of sleep propensity against the polysomnogram by asking subjects to fall asleep whilst lying down (Balkin, Rupp, Picchioni & Wesensten, 2008). In contrast, the Maintenance of Wakefulness Test (MWT) instructs subjects to remain awake rather than fall asleep (Mathis & Hess, 2009). Bennett and colleagues (1997) purported that these measures are labour intensive as they require continual inspection of the EEG. Similarly, although the MSLT and MWT collectively relate to physiological sleepiness, experimental findings suggest that they tap into different behavioural components of sleepiness and differ in their sensitivity for the detection of sleepiness (Bennett et al., 1997; Harma et al., 1998; Johns, 2000).

Another measure of sleep latency, the Oxford Sleep Resistance Test (Osler) reproduces many of the characteristics of the MWT; however its utility for the discrimination between normal and sleepy individuals has been specified (Bennett et al., 1997; Priest et al., 2001). Objective psychomotor performance tests that assess reaction time, such as the psychomotor vigilance task (PVT), have also been extensively implicated in sleepiness assessments (Balkin et al., 2008; Franzen, Siegle & Buysse, 2008; Jewett, Dijk, Kronauer & Dinges, 1999; Johns, 1998). In addition, consideration of the implications of sleepiness with regard to safety has rendered task simulations, such as driving simulators, an important tool

for the assessment of sleepiness (Arand et al., 2005; Nau, 1997). Eye closure measures have also been implicated as a valid indicator of driver fatigue, and a measure which can be directly related to accident risk (Haworth & Vulcan, 1991). In a series of ten studies related to sleepiness, Skipper and Wierwille (1986) found that the position of the eyelid was the cleanest and most stable signal of the physiological and physical variables measured.

Subjective tests of sleepiness typically include sleep logs and various rating scales that provide estimations of general levels of sleepiness (Nau, 1997). The Epworth Sleepiness Scale (ESS) is a simple, widely used self-administered questionnaire that asks subjects to rate their usual chance of dozing in eight situations commonly encountered in daily life (Johns, 1998). In an initial validity study, a significant correlation was demonstrated between ESS scores and sleep latency during daytime sleepiness testing and nocturnal polysomnography (Johns, 1991). The Karolinska Sleepiness Scale (KSS) is another subjective rating scale that assesses sleepiness at the time of administration (Akerstedt & Gillberg, 1990). The KSS reflects physiological and behavioural changes when sleepiness ratings are relatively high, and thus, the clinical utility of this measure as an indicator of sleepiness has been documented (Gillberg, Kecklund & Akerstedt, 1996; Kaida et al., 2006). Indeed, Gillberg et al (1994) demonstrated that self-reported sleepiness with the KSS increased dramatically across the night and early morning between the hours of 9pm and 7am. However, relationships between subjective sleepiness and objective indices become tenuous in individuals with chronic sleep disorders such as obstructive sleep apnoea (Balkin et al., 2008). Despite reporting normal subjective alertness, objective evidence of pathological sleepiness may exist.

1.4.3. Physiological and performance effects of sleep loss. After sleep loss, dose-related increases in sleepiness have been demonstrated in the sleep laboratory across physiological measures, subjective reports and standardised behavioural tests that mirror operationally significant aspects of function (Rosa, 2006). Across physiological measures,

sleep latency has been reported to decrease after total and partial sleep loss, sleep fragmentation and a shift in waking activities to overnight hours (Arand et al., 2005). Sleep latency values were reduced by 50-60% after one night of total sleep deprivation, after five to seven consecutive days of reduced sleep (2-3 hours), and after fragmented sleep induced by forced arousals every minute (Bonnett & Arand, 2003). In line with this finding, Carskadon and Dement (1981) also reported a progressive increase in sleepiness that persisted over a seven day sleep restriction protocol.

Standardised performance tests that assess perceptual-motor responsiveness (e.g., reaction time tasks) also show degradation with sleep deprivation (Rosa, 2006). Belenky and colleagues (2003) revealed dose related patterns of progressively slower reaction times and lapses on the PVT when subjects sleep was curtailed to either three, five, seven or nine hours daily time in bed for seven consecutive days. Numerous studies have supported this dose related trend in well controlled laboratory conditions (Bonnett & Arand, 2003; Franzen, Siegle & Buysse, 2008; Howard, Gora, Swann & Pierce, 2002; Jewett et al 1999; Loh, Lamond, Dorrian, Roach & Dawson, 2004).

1.4.4. Driving performance and sleep loss. The combination of sleep loss and driving poses a major risk factor for road accidents. Studies of performance decrements during simulated driving tasks after sleep loss have been well documented (Anund et al., 2008; Arnedt, Wilde, Munt & Maclean., 2000; Howard et al., 2002). In a sample of professional drivers, Howard and colleagues (2002) demonstrated a decline in driving simulator performance after one night of sleep deprivation. Eye movement data as an indicator of sleepiness whilst driving revealed episodes of slow-eye-closure that were related to speed variation and steering variation which progressively increased after 17 hours of wakefulness (Howard et al., 2002). Arnedt et al (2000) reported a greater number of off road accidents, increased tracking variability and speed variability after prolonged wakefulness,

however these findings were not statistically significant. A confound between prolonged wakefulness and time of night may explain this finding given that driving performance was assessed near the nadir of the circadian rhythm (Arnedt et al., 2000).

Determining the point at which decrements in simulated driving become a risk to the driver and others has been equivocal (MacLean, Davies & Thiele, 2002). Legislations governing permitted levels of alcohol can provide a benchmark for investigations of driver sleepiness (MacLean, Davies & Thiele, 2002). Williamson and Feyer (2000) revealed that after 17-19 hours of sleep deprivation, performance on a variety of tests was equivalent or worse than that at a blood alcohol concentration (BAC) of 0.05%. These findings reinforce that sleep deprivation compromises performance of speed and accuracy required to maintain safety on the road and in other industrialised settings (Williamson & Feyer, 2000). A study of the general population by Nordbakke and Sagberg (2007) revealed that despite good knowledge of the various factors influencing driver sleepiness and effective measures to counteract sleepiness, most drivers continued to drive when recognising their sleepiness levels.

In addition to sleep deprivation, mood has been implicated as a factor that impairs driving performance, since judgement may be compromised under conditions of sleep deprivation, leading to risky behaviours. Indeed, a study assessing the relationship between mood states and driving behaviours in a sample of 163 drivers revealed that the mood states of depression, anger, fatigue and anxiety were negatively related to cautiousness whilst driving (Garrity & Demick, 2001). Conversely, another study revealed that mood variables (i.e., depression and anxiety) were not related to driving behaviours and accident history (Barbe et al., 1998).

1.4.5. Mood and sleep loss. Relative to physiological and performance effects, studies of the effects of mood disturbances after sleep deprivation have been less consistent

(Carskadon & Dement, 1981; Ford & Cooper-Patrick, 2001; Franzen et al., 2008). In an earlier sleep deprivation study by Carskadon and Dement (1979), a mood scale sensitive to sleep loss revealed fewer positive responses throughout the deprivation period. However, complete recovery to baseline levels were attained for mood variables after a single night of recovery sleep (Carskadon & Dement, 1979).

Franzen et al (2008) concluded that increased negative mood and decreased positive mood after one night of sleep deprivation was associated with subjective and objective sleepiness. Caldwell and LeDuc (1998) also reported significant decreases in mood, as measured by the Profile of Mood States (POMS) questionnaire after a 40-hour period of sustained wakefulness. Similarly, after 48 hours of sleep deprivation, Zhang and colleagues (2002) found that vigour was significantly reduced to a lower level after 20 hours of sleep loss. States of tension, fatigue and confusion increased significantly after 24 hours of sleep deprivation, and reached a significantly high level at 48 hours of sleep deprivation. Although anger and depression had a tendency to increase, this was not to a significant extent (Zhang et al., 2002). Wanner and Bader (2000) reported a decrease in happiness from night to morning. This finding lends support to the notion that sleep serves a mood regulatory function.

In contrast, research conducted by Scott, McNaughton and Polman (2006) found that subjective tension and anger on the POMS were unaffected by sleep deprivation, though sleep deprivation was associated with greater negative disturbances to subjective vigour, fatigue and depression (Scott et al., 2006). The discrepancy in findings across studies is likely to reflect the various instruments used and mood states assessed for measurement of affective disturbances after sleep loss. Furthermore, although sleepiness and mood are two independent constructs with negative consequences, they may be difficult to differentiate due to fluctuating time courses, which may complicate the subjective assessment of mood. Furthermore, it remains unclear from cross-sectional studies whether sleep loss influences

mood or anxiety levels or vice versa (Colton et al., 2006). Despite this, a meta-analysis revealed that mood was significantly altered after partial sleep deprivation to a greater degree than cognitive or motor performance (Pilcher & Huffcutt, 1996).

1.4.6. Summary of sleep loss. Research on the effects of sleep loss on physiology, performance, driving and affective states has led to better recognition of the negative health and safety consequences (Walsh & Lindblom, 1997). Generally, an increase in sleep tendency after sleep deprivation has led to the assumption that excessive daytime sleepiness (EDS) is the cardinal indicator of sleep loss. Although the causes of sleep loss are multi-factorial, two major categories have been implicated in the aetiology of sleep loss, including sleep disorders (e.g., obstructive sleep apnoea or OSA) and occupational factors (e.g., shift work) (Colton et al, 2006). While the significance of sleep loss has not been fully delineated in experimental studies, research on OSA patients and shift-workers has typically revealed that sleep disruption is likely to have profound effects on the lives of these individuals (Walsh & Lindblom, 1997).

1.5. Obstructive Sleep Apnoea (OSA)

Excessive daytime sleepiness has specifically been regarded as a primary complaint in OSA patients (Black, 2003; Vgontzas, 2008). Obstructive sleep apnoea is a disorder of neural respiratory control and upper airway dysfunction during sleep (Orr, 1997). The resulting reduction or cessation of airflow respectively referred to as hypopnoeas and apnoeas produce increases in inspiratory efforts against the obstructed airway, resulting in sleep fragmentation (Piper & Stewart, 1999). Pertinent symptoms of OSA typically fall into daytime and nocturnal categories. Snoring, witnessed apnoeas and nocturnal choking represent disrupted sleep features, whereas EDS represents a daytime symptom of OSA (Schlosshan & Elliott, 2004). Excessive daytime sleepiness manifests as a tendency to inadvertently fall asleep during quiet or passive activities, to take intentional naps, or to experience short, albeit

repetitive lapses while undertaking monotonous tasks (Pradeep Kumar, Bhatia, Tripathi, Srivastava & Jain, 2003).

In a sample of 602 employed men and women aged 30 to 60 years, Young and colleagues (1993) found that 9% of women and 24% of men had undiagnosed sleep disordered breathing. In an extension of this, 2% of women and 4% of men in the middle aged work force met the minimal diagnostic criteria for OSA. Similarly, in an Australian sample, Bearpark and colleagues (1993) reported that sleep disordered breathing affected approximately 4.7% of women and 8.5% of men, with incidence increasing after 40 years of age. Although male gender has been implicated as a risk factor in OSA, Shahar and colleagues (2003) reported that the prevalence of women experiencing OSA after menopause was also high relative to premenopausal women. Anatomically, a small airway size has also been suggested to determine the likelihood of OSA (Shochat & Pillar, 2003). An increase in body mass index (BMI) and neck circumference also increases the risk of OSA (Young, Peppard & Gottlieb, 2002). Evidence of a hereditable component to OSA also exists, with a substantially increased risk when a primary family member is affected (Guilleminault, Partinen, Hollman, Powell & Stoochs, 1995).

Although initially regarded as a medical phenomenon with wide ranging sequelae including hypertension, cardiovascular disease and cerebrovascular disease (Douglas & Polo, 1994), the clinical manifestations of OSA may be more appropriately regarded as psychological in nature and are characterised by personality and mood changes, impaired daytime concentration, decreased performance, impaired driving ability and a greater risk of road accidents (Orr, 1997). The combination of high prevalence and wide ranging consequences renders OSA a major public health hazard (Phillipson, 1993).

1.5.1. Pathophysiology of OSA. Among healthy individuals, inspiratory effort is associated with a collapsing negative pressure and a simultaneous burst of activity from a variety of upper airway muscles that function to inhibit collapse (Orr, 1997). In contrast, models of the pathogenesis of OSA have been based on pharyngeal imaging during wakefulness which show reduced upper airway size relative to age and weight matched controls, causing obstruction to the upper airway (Malhotra & White, 2002). The muscles that keep the airway open during the waking state become blunted in their response during sleep allowing the narrowed airway to collapse (Cartwright, 2001). Negative pressure builds up in the lungs as a result of increased effort of the chest wall muscles, and an arousal occurs in order to break this cycle (Cartwright, 2001).

The cessation of airflow at the nose and mouth lasting at least 10 seconds is defined as an apnoea (Kwentus, Schulz, Fairman & Isrow, 1985). In contrast, partial obstruction is referred to as a hypopnoea, and is defined by decreased air flow (Leavy Holman, 2005). Most OSA patients report hundreds of repetitive apnoeas in a single night. At average of at least five apnoeas or hypopnoeas per hour of sleep is required to make a diagnosis of OSA, with a repetitive sequence of events evident in NREM or REM sleep (Cartwright, 2001; Guilleminault et al., 1976). The combination of apnoeas and hypopnoeas recurring during nocturnal sleep causes a significant disruption to normal sleeping patterns and contributes to EDS (Cartwright, 2001).

1.5.2. OSA and hypoxemia. Two earlier schools of thought emerged to explain EDS in OSA patients. The first proposes that EDS is the product of accumulating sleep disruption from respiratory difficulties, while the second suggests that EDS results from the effects of inadequate oxygenation of the blood or hypoxemia on cerebral function (Guilleminault et al., 1976). Bedard and colleagues (1991) investigated the relative contribution of sleep disruption and nocturnal hypoxemia in OSA patients. Their findings

revealed that in moderate and severe OSA patients, both nocturnal hypoxemia and sleep disruption contributed to impairments in vigilance. Daytime alertness, as measured by a psychomotor task was found to be sensitive to both of these pathogenic factors, however sleepiness in OSA patients was exclusively related to hypoxemia (Bedard et al., 1991).

1.5.3. Diagnosis of OSA. A diagnosis of OSA is dependent upon the integration of clinical information and polysomnography (Olsen et al., 2003), which determines the frequency and severity of respiratory pauses and their subsequent effect on normal sleep and waking function (Cartwright, 2001). The apnoea-hypopnoea index (AHI) delineates the number of apnoea and hypopnoea events per hour of sleep to determine the frequency and severity of OSA (Cartwright, 2001). Although considerable debate exists in the literature regarding accurate AHI specifications, Cartwright (2001) suggested that individuals with mild OSA typically report an AHI between 5 to 15 events, those with moderate OSA report an AHI between 15 and 30 events, and those with severe OSA exhibit an AHI greater than 30. Overnight polysomnography also determines the duration of apnoeic and hypopneic events, the relationship to body position and sleep stages, the level of oxygen desaturation, and the existence of arrhythmic episodes (Silverberg, Iaina & Oksenberg, 2002). Despite the relative simplicity in objectively diagnosing OSA, approximately 80 to 90% of individuals are undiagnosed, such that when a diagnosis is confirmed, associated signs and symptoms may have been present for an average of seven years (Silverberg et al., 2002). A study conducted by Ronald and colleagues (1998) supports this view, suggesting that OSA patients heavily utilised health care resources (e.g., physician claims and hospital admissions) 10 years prior to their diagnosis. Early diagnosis and treatment serves to improve the health and well-being of OSA patients.

1.5.4. Treatment of OSA. Treatments for OSA aim to establish normal nocturnal oxygenation and ventilation, abolish snoring, and eliminate sleep disruption as a result of

upper airway closure (Strollo & Rogers, 1996). Decisions regarding treatment are typically based on the effect that OSA has on daytime symptoms and cardiopulmonary function, rather than the absolute number of apnoeic and hypopnoeic events (Strollo & Rogers, 1996). Since the majority of patients with OSA are overweight, weight reduction has been suggested to lead to improvements (Saskin, 1997). Similarly, the avoidance of factors that increase the severity of upper airway obstruction such as sleep deprivation, alcohol, sedative medications and narcotics may prevent the progression of OSA (Leavy Holman, 2005). The role of surgery in the treatment of OSA remains controversial, since the application of surgical procedures to OSA patients is limited by a shortage of randomised, controlled trials to support its use (Caples, Gami & Somers, 2005).

The current gold standard treatment of OSA is nasal continuous positive airway pressure (CPAP). This treatment method therapeutically delivers constant airway pressure through the nostrils in order to maintain a patent airway (Leavy Holman, 2005). The infusion of positive air through a mask that the patient wears over the nose during sleep creates a pneumatic splint, to maintain upper airway patency and prevent collapse (Piper & Stewart, 1999). Improvements in daytime function result from the abolishment of most apnoea episodes, the elimination of oxygen desaturation, and the consolidation of sleep state architecture (Saskin, 1997). Although patients often report adherence difficulties characterised by nasal irritation, claustrophobic responses and difficulty tolerating pressure (Olsen et al., 2003), the effectiveness of CPAP in alleviating a variety of symptoms associated with OSA has been well documented (Ballester et al., 1999; Engleman et al.,1999; Sin, Mayers, Man, Ghahary & Pawluk., 2002).

A randomised placebo controlled trial conducted by Engleman and colleagues (1999) demonstrated that cognitive function, psychological wellbeing and quality of life were improved with CPAP treatment. However, their findings were limited to the assessment of

patients with mild OSA. Barnes and colleagues (2002) described an uncertainty about the need for, and the effectiveness of treatment in mild cases of OSA. Their findings revealed that although CPAP improved self-reported symptoms of OSA (e.g., snoring, restless sleep, daytime sleepiness and irritability), objective and subjective measures of daytime sleepiness were not observed with CPAP treatment. Furthermore, quality of life and mood scores were not improved after treatment. In a study of the effectiveness of CPAP in moderate to severe OSA, Ballester et al (1999) reported that relief of sleepiness and improvements in perceived health status were greater in a group receiving a combination of CPAP treatment and a weight loss program as opposed to a weight loss program in isolation from CPAP. Although these findings suggest that CPAP treatment in moderate to severe cases is adequately supported, further research is needed to clarify this matter.

1.5.5. Excessive daytime sleepiness (EDS) in OSA. Sleepiness refers to a physiological and behavioural need state marked by a tendency to fall asleep, and like sleep; sleepiness represents a universal daily experience (Nau, 1997). Although EDS has been consistently implicated as the cardinal symptom in OSA patients, it is not a universal feature in patients with OSA because not all patients report EDS even when the condition is moderate to severe (Black, 2003; Vgontzas, 2008). Young and colleagues (1993) revealed in a seminal epidemiological study that only 15.5% of males and 22.6% of females with an AHI of 5 events per hour or more reported sleepiness across 3 subjective sleepiness items. This lack of consistency is evidenced when considering that many patients may report or experience other symptoms of daytime dysfunction and may not relate their experience to the specific complaint of ‘sleepiness’ (Black, 2003). In a sample of 190 polysomnographically confirmed OSA patients, Chervin (2000) demonstrated that patients often selected different words to describe their problems. Surprisingly, the complaint of sleepiness was endorsed

least frequently, whereas lack of energy was selected as the term that patients used to describe their daytime experience (Chervin, 2000).

Although several factors have been purported to explain the causes of OSA related sleepiness, the contribution of these factors remains unclear. The presence of EDS has been ascribed to alterations in sleep structure (Gonsalves, Paiva, Ramos & Guilleminault, 2004). Specifically, reductions in slow wave sleep (stages 3 and 4) and rapid eye movement percentages, with corresponding increases in lighter sleep characterise these alterations (Black, 2003; Heinzer et al., 2001; Sauter et al., 2000). Heinzer and colleagues (2001) demonstrated that CPAP restored a more physiologic pattern of slow wave sleep across the night. Other investigations have not supported the association between EDS and sleep architecture (Guilleminault et al., 1988).

The potential association between EDS and nocturnal oxygenation has also yielded inconsistent findings (Bedard et al., 1991). Mediano et al (2007) investigated the relationship between EDS and polysomnographic variables in OSA patients with and without EDS. Their findings revealed that patients with EDS slept more efficiently, exhibited shorter sleep latencies as measured by the MSLT, and had lower nocturnal oxygenation levels than those without EDS (Mediano et al., 2007). The authors concluded that the severity of nocturnal hypoxemia could represent a major determinant of EDS in OSA patients. The remaining nocturnal variables (i.e., AHI, arousal index and sleep architecture) did not discriminate between the two groups. Other investigations have supported this finding revealing that neither the arousal index nor AHI were independent predictors of sleep latency (Banks et al., 2004). Conversely, a study conducted by Chesire and colleagues (1992) revealed that daytime performance in OSA patients was significantly associated with the frequency of apnoeas and hypopnoeas, the resulting sleep disturbance and the extent of nocturnal hypoxemia.

In a replication of the Mediano et al (2007) study, Roure et al (2008) investigated polysomnographic determinants of EDS in a larger cohort of OSA patients (*N*=2882), revealing that OSA patients with EDS slept longer and more efficiently than patients without EDS. Furthermore, although patients demonstrated slightly worse respiratory disturbances, sleep fragmentation and nocturnal oxygenation, the results suggested that OSA and sleep disruption are not the primary determinants of EDS in all of these patients. Utilising a different objective measure, Caffier, Erdmann and Ullsperger (2005) investigated the value of spontaneous blink parameters for the assessment of EDS in OSA patients before and after one night of CPAP therapy. The authors demonstrated that a decrease in EDS occurring after one night of CPAP therapy was accompanied by corresponding changes in eye-blink measures. The EDS patient group exhibited expected increases in blink frequency, shortening of blink duration and a decrease in the proportion of long-closure duration blinks (Caffier et al., 2005). These findings have important implications for research and demonstrate the importance of the spontaneous eye blink in the assessment of EDS.

In a comparison of the MWT and a modified behavioural Osler test in the evaluation of EDS, a strong association was reported for these two measures in a sample of patients presenting at a sleep disorders centre (Krieger et al., 2004). Sleep latency measured by the Osler, and EEG simultaneously were also strongly correlated (Krieger et al., 2004). However, the specific patient population was not defined with respect to diagnosis in the Krieger et al (2004) study, suggesting that this strong correlation may not be observed in OSA patients. Despite this, the authors concluded that the Osler represents a reliable tool for the evaluation of EDS (Krieger et al., 2004).

Studies of subjective measures in the investigation of EDS in OSA patients have also reported inconsistent results. Furata et al (1999) reported no relationship between the ESS and sleep latency, however sleep latency was related to the number of awakenings and the

AHI. Sauter et al (2000) supported this observation, finding that sleepiness in the ESS did not differ between moderate and severe OSA patients. However, Johns (1993) reported that ESS scores were associated with OSA severity. Specifically, primary snorers endorsed lower ESS scores relative to patients with mild, moderate and severe OSA. Similarly, the KSS is a frequently used tool for evaluating EDS at the time of administration (Akerstedt & Gillberg, 1990). In a validation study, Kaida et al (2006) reported that the KSS was closely associated with EEG and behavioural variables (i.e., PVT), demonstrating the utility of the KSS in the assessment of EDS. The weak to moderate correlation between subjective and objective measures of sleepiness (Black, 2003) has led to subsequent investigations of factors that may contribute to EDS in OSA patients. Other factors that have been purported to explain subjective sleepiness in OSA patients include gender, depression, smoking, alcohol use, heart disease, stroke, metabolic factors and personality features (Bixler et al., 2005; Hayashida et al., 2007; Koutsourelakis et al., 2008; Orr, 1997; Vgontzas, 2008). Age has also been suggested to strongly predict subjective reports of sleepiness, with the presence of EDS linearly declining with increasing age (Bixler et al., 2005). Although the causes of EDS remains poorly understood, the consequences of EDS in OSA patients appear to have wide ranging implications for psychomotor and driving performance and for mood.

1.5.6. Vigilance and psychomotor performance in OSA. Sleep fragmentation and nocturnal hypoxemia in OSA patients not only augments daytime sleepiness the following day, but also negatively affects reaction time and vigilance (Adams, Strauss, Schluchter & Redline, 2001). At the lower level of cognitive processes, vigilance refers to the ability to maintain an active state of wakefulness (Mazza et al., 2005). Reaction time tasks provide a useful mechanism for the assessment of vigilance impairment. Many studies examining vigilance in OSA patients have described impairments in psychomotor performance (Beebe, Groesz, Wells, Nichols & McGee., 2003; Sforza, Haba-Rubio, De Bilbao, Rochat & Ibamez.,

2004; Verstraeten, Cluydts, Pervernagie & Hoffman., 2004). In a comprehensive meta-analysis of norm-referenced and case-controlled data, Beebe et al (2003) proposed that despite study differences in the specific vigilance test utilised and in sample acquisition and composition, studies which met the inclusion criteria and utilised a measure of vigilance yielded at least a moderately negative effect.

Specifically, Sforza et al (2004) reported that relative to controls, patients with OSA performed more poorly on the PVT in terms of accuracy and exhibited a greater number of lapses and false responses. This was significantly more evident in patients with greater levels of objectively defined sleepiness and a higher AHI. The authors proposed that subjective sleepiness, sleep fragmentation, nocturnal hypoxemia and AHI collectively influenced PVT accuracy (Sforza et al., 2004). Mazza and colleagues (2005) employed 3 different measures of vigilance across 3 testing protocols, and revealed impaired performance in 95% of OSA patients on all 3 tests, relative to control subjects. In two studies assessing the effect of CPAP therapy on psychomotor performance, findings were consistent (Bedard et al., 1993; Sanchez et al., 2004). Though different measures were employed to assess vigilance, both studies revealed that CPAP treatment produced a clear improvement in reaction time performance at one month and at three months of treatment. Although improvements in reaction time were observed after treatment, Bedard et al (1993) demonstrated that some degree of vigilance impairment persisted relative to controls.

Relative to core intellectual and verbal abilities assessed under the rubric of neuropsychological functioning that are typically unaffected in OSA patients, vigilance has been shown to be significantly impaired (Beebe et al., 2003; El-Ad & Lavie, 2005). The magnitude of this finding lends support to the significance of vigilance screening in patients with untreated OSA. Maintaining accurate performance in tasks performed over time, especially those considered more monotonous in nature, such as driving (Adams, Strauss,

Schluchter & Redline., 2001) renders vigilance an important co-factor in the increased risk of road traffic accidents in patients with OSA (Engleman & Douglas, 2004).

1.5.7. Sleepiness, motor vehicle accidents and OSA. Among other variables such as inexperience, excessive speed, alcohol consumption and inattention (Maycock, 1997), a growing body of evidence has implicated sleepiness as a contributory factor in road accidents. Motor vehicle accidents are a major public health concern and are a leading cause of injury and death. Despite considerable reduction in the Victorian road toll in Australia since 1989, the Transport Accident Commission recorded that over 200 fatalities occurred in 2010 as a result of motor vehicle accidents (TAC, 2010). Within Australia, approximately 20% of fatal road accidents are attributable to driver sleepiness (TAC, 2010). Sleepiness accounted for 10% of accident involvement in a sample of 4621 car drivers, and on motorways this figure was increased to 20% (Maycock, 1997). Horne and Reyner (1995) reported two accident studies and demonstrated that sleepiness was likely to be a causative factor in between 16 and 23% of all accidents. Interestingly, across 679 sleep related vehicle accidents, a collision was reported to be higher during the night and in the mid-afternoon than any other time of the day (Horne & Reyner, 1995), emphasising the involvement of the circadian timing system in driver sleepiness.

The safe operation of a motor vehicle requires alertness, accurate perception, judgement and action (Lyznicki et al., 1998). Such requirements whilst driving are compromised under conditions of sleepiness (MacLean et al., 2003), rendering falling asleep at the wheel or loss of attention and slowing of reactions a likely outcome (Lyznicki et al., 1998). Maycock (1997) revealed that in a population of car drivers, 30% had felt close to falling asleep at the wheel during the year preceding the survey. In an account of perceptions of sleepiness among 1,249 truck drivers in Australia, Arnold and colleagues (1997) reported that 14% had admitted to falling asleep at the wheel over a nine month period and these

events were more pronounced in drivers who reported having slept less than six hours before commencing the drive. An additional complicating factor in the assessment of driver sleepiness is that many drivers deny knowledge of having fallen asleep at the wheel or feelings of sleepiness beforehand (Horne & Baulk, 2004).

The EDS inherent in patients with OSA poses a significant risk for driving performance in these individuals. Although much of the literature has consistently implicated OSA as a risk factor for driving performance (Bearpark et al., 1990; Findley et al., 1995; George, Boudreau & Smiley, 1996; Teran-Santos, Jimenez-Gomez & Cordero-Guevara., 1999; Turkington, Sircar, Allgar & Elliott., 2001), only one study did not corroborate these findings revealing no differences between OSA patients and control subjects in relation to traffic accidents (Carter, Ulfberg, Nystrom & Edling, 2003). Relative to the large sample of professional drivers employed (N=4000), only a small proportion (N=160) underwent a polysomnographic sleep study. Since polysomnographic markers commonly define OSA, the paucity of professional drivers who participated in the sleep study may have rendered differences between the groups difficult to ascertain.

In a consultant report examining the role of OSA in road safety and pathological sleepiness, OSA patients were more likely to have had at least one motor vehicle accident attributable to sleepiness and to experience behaviours associated with severe sleepiness, such as, falling asleep at the wheel, falling asleep at traffic lights, and pulling off to the side of the road as a result of sleepiness (Bearpark et al., 1990). In a large population based sample, Young et al (1997) reported that men with an AHI greater than 15 were 3.4 times more likely to be involved in at least 1 motor vehicle accident over a 5 year period. With the addition of females, this figure increased to a likelihood of 7.3 times. However, even among individuals with an AHI between 5 and 10, the likelihood of a motor vehicle accident is also increased (Teran-Santos et al., 1999).

1.5.8. Driving simulator performance in patients with OSA. The development of driving simulators has enabled the assessment of driving impairment in patients with OSA and provides a measure of an individual's ability to track and maintain attention (Turkington et al., 2001). Arguably, driving simulators are unable to provide all of the visual, vestibular and proprioceptive changes that occur whilst operating a steering wheel and changing courses. However, this method is considered cost effective and safer than in-vehicle or on road testing, and provides a greater degree of experimental control and precision of performance measures in the absence of interference of uncontrolled variables that operate in real life settings (George et al, 1996). Despite the varying complexity of the simulation employed, investigations of driving impairment in OSA patients have generally revealed poorer performance relative to controls (Findley et al., 1995; George et al., 1996; Turkington et al., 2001). Even control subjects who are investigated under conditions of alcohol intoxication perform better on simulated driving tasks than patients with OSA (George et al., 1996), highlighting the magnitude of driving impairment inherent in OSA patients. Adding to the magnitude of driving impairment is the OSA patient typically under-reporting their level of sleepiness. This may suggest the loss of a normal frame of reference following prolonged EDS (Engleman, Hirst & Douglas, 1997).

In a simulation of a monotonous highway drive which presented 780 obstacles over a 30 minute period, 62 patients with OSA hit a higher percentage of obstacles in comparison with control subjects (Findley et al., 1995). Similarly, in a study which employed two simulators to investigate driving performance (i.e., a monotonous highway drive and a city drive), patients with OSA hit a greater number of road obstacles and were generally more impaired than control subjects on measures of speed (Findley et al., 1989). Turkington and colleagues (2001) revealed that in a group of patients with OSA, performance on a driving simulator was influenced by a number of factors that were indirectly related to on road

driving, such as age, female sex, and admitted alcohol consumption. Surprisingly, no correlation was observed between AHI, ESS scores and simulator performance (Turkington et al., 2001). Despite this, a history of near miss accidents was an independent predictor of poor tracking error and number of off road events on the driving simulator (Turkington et al., 2001). After treatment with CPAP, significant improvements in tracking error, reaction time and off road events per hour have been observed, and have been suggested to persist for seven days following discontinuation of treatment in OSA patients (Turkington, Sircar, Saralaya & Elliott, 2004). Similarly, marked reductions in objective reports of motor vehicle accidents over a two year period has also been demonstrated in OSA patients after treatment, in comparison to patients not accepting of treatment (Findley, Smith, Hooper, Dineen & Suratt., 2000).

1.5.9. Disturbances of mood in patients with OSA. The construct of mood is broadly defined as a mild, albeit transient emotional state (Corsini, 2002). Difficulties in conceptualising this construct become evident when considering that a myriad of states, both positive and negative, exist within the rubric of mood. Adding to the complexity is that mood disturbances exist along a continuum from transient and mild forms (e.g., irritability), to clinically diagnosed mood disorders (e.g., depression). The interplay of mood and sleep has been characterised by negative outcomes when sleep is deficient, such as impaired quality of life. First, the magnitude of the relationship between sleep and mood is highlighted upon consideration that sleep related difficulties are often implicated as a symptom in a variety of mood disturbances, such as depression (Szuba, 2001). Second, while patients may report symptoms related to sleep disturbances such as lack of energy or fatigue, they may be hesitant to disclose disturbances in mood, rendering these individuals less likely to seek assistance for their concerns (Ford & Cooper-Patrick, 2001). Finally, the variety in well

established and standardised instruments that have been used to assess mood in patients with OSA may make comparisons across studies difficult.

Measures employed for the assessment of emotional functioning, among others, typically include the Profile of Mood States (POMS), the Hospital Anxiety and Depression Scale (HADS), the Beck Depression Inventory (BDI), the Minnesota Multiphasic Personality Inventory (MMPI), the Zung Rating Depression Scale Inventory, and the Symptom Distress Checklist (SCL) (Brown, 2006; Weaver, 2001). The POMS presents a series of adjectives such as 'tense', 'blue' and 'furious', and incorporates a Likert scale (not at all to extremely), and respondents are asked to identify the extent of their agreement with each statement (Shacham, 1983). Outcome variables from the POMS include five subscales (i.e., tension-anxiety, depression-dejection, confusion-bewilderment, vigour, anger-hostility, and fatigue) and a total disturbance score (Shacham, 1983). The BDI represents the 'gold standard' for the assessment of depression (Weaver, 2001) and consists of 21 statements depicting a particular aspect of depressive symptomatology (e.g., appetite, sense of failure) accompanied by a Likert scale of graded severity (Beck, 1961). The POMS and the BDI were incorporated in the present study to assess mood states and depressive symptomatology respectively.

The association between psychiatric disorders and OSA has been long debated in the literature. Although some studies have reported a relationship between psychiatric disorders and OSA (Aikens et al., 1999; Akashiba et al., 2002; Haynes, 2005; Kjelsberg, Ruud & Stavem, 2005; Sharafkhaneh et al., 2005), others have not supported this finding (Mosko et al., 1989; Pillar & Lavie, 1998; Sforza et al., 2002). In addition, the exact mechanisms underlying this possible association have not been fully elucidated. A mood disturbance may represent a biological or psychosocial consequence of OSA (Sharafkhaneh et al., 2005). On another level, mood disturbances and OSA may relate to similar underlying mechanisms, such as hypoxemia and sleep fragmentation, which may serve to promote depressive

symptoms (El-Ad & Lavie, 2005). A further contention is that mood disturbances may simply be a product of a chronic medical condition, such as OSA (Sharafkhaneh et al., 2005).

In a large cohort of over one hundred and eighteen thousand patients with OSA, the prevalence of a psychiatric co-morbid diagnosis was reported to be 21.8% for depression (compared to 9.43% for the non-apnoea group), 16.7% for anxiety (compared to 8.39% for the non-apnoea group), 11.9% for post-traumatic stress disorder (compared to 4.74% for the non-apnoea group), and 3.3% for bipolar disorder (compared to 1.88% for the non-apnoea group) (Sharafkhaneh et al., 2005).

The large prevalence of depression in patients with OSA has led to a burgeoning interest in this co-morbid condition. Indeed, investigations of depression have been subjected to rigorous experimentation and are often more widely examined than virtually all other mood disturbances (Szuba, 2001). Bardwell and colleagues (2003) examined the relationship between depressive symptoms and fatigue in patients with OSA. Interestingly, this investigation revealed that depressive symptoms accounted for a significantly greater proportion of variance in fatigue than did the severity of OSA as measured by AHI (Bardwell et al., 2003). Even among simple snorers, 31% experienced some form of depression relative to OSA patients (Vandeputte & de Weerd, 2003). In addition, some psychological symptoms have been suggested to persist even after treatment (Munoz et al., 2000). In essence, these finding lend support to the importance of assessing mood symptoms in patients with OSA, since mood may be likely to affect treatment uptake or compliance with CPAP therapy.

Despite knowledge that patients with OSA experience fragmented sleep and hypoxemia, the determinant factors of these psychological impairments remain inconclusive (Yue et al., 2003). Contrary to expectations, Moore, Bardwell, Ancoli-Israel and Dimsdale (2001) found that the degree of sleep fragmentation was not clearly associated with mood changes, until gender and age were controlled for. Similarly, Sforza et al (2002) revealed that

the presence of OSA and its severity were not the primary determinants of psychological changes (i.e., depression and anxiety) in OSA patients. In a study examining the potential explanatory variables and symptoms of anxiety and depression using the HAD scale in patients with OSA, Kjelsberg et al (2005) reported that daytime sleepiness and low compliance to CPAP therapy were associated with scores corresponding to a clinical diagnosis of depression. Low compliance to CPAP therapy was the only variable associated with a high level of anxiety, most likely due in part to a decreased tolerance of the equipment (Kjelsberg et al., 2005). In a study by Aloia and colleagues (2005), apnoea severity and obesity differentially contributed to depressive symptoms in patients with moderate to severe OSA, and gender appeared to moderate this relationship. Borak et al (1996) reported that sleep fragmentation rather than hypoxemia contributed to increased levels of anxiety, depression and mental stress. Conversely, others have related hypoxemia and time spent in REM sleep and deep sleep to psychological disturbance (Borak et al., 1994; Chesire et al., 1992).

Although not consistently documented (Ferini-Strambi et al., 2003; Mosko et al., 1989; Pillar & Lavie, 1998; Sforza et al., 2002), the co-morbidity of depression in patients with OSA is considered to be frequent even when a variety of different instruments are employed. Less attention appears to be given to other classes of psychopathology and mood states (e.g., anxiety, anger, confusion, somatic hyper-vigilance), and while such dimensions may be less marked than depressive symptoms, they may produce meaningful correlations with respiratory indices (Aikens et al., 1999). Aikens and colleagues (1999) demonstrated this trend in a sample of 178 OSA patients utilising the MMPI, revealing that while elevations on the depression subscale were elevated for one third of the sample, two thirds of this elevation were combined with other elevations (i.e., hypochondriasis, hysteria, social introversion, and psychasthenia). In a study conducted by Yue et al (2003), patients with

OSA endorsed higher scores across a broad array of psychological symptoms including somatisation, obsession-compulsion, anxiety, hostility, and depression. Similarly, Borak and colleagues (1996) reported that in addition to depression, the emotional status of 20 male patients with OSA was also characterised by anxiety and elevated mental stress. An investigation of the psychological correlates of OSA conducted by Bardwell, Berry, Ancoli-Israel and Dimsdale (2003) which utilised several mood measures revealed that patients with OSA experienced more anger than control subjects when controlling for age, BMI and hypertension. Not surprisingly, Bardwell et al (1999) found that with more sleep, vigour was increased among OSA patients. These findings lend support to the presence of other psychological disturbances present in OSA patients (Aikens et al., 1999; El-Ad & Lavie, 2005).

In contrast to investigations of depression, there is a paucity of research examining the relationship between OSA and anxiety. In fact, only one study revealed a correlation between anxiety and AHI in a small group of patients with OSA (Borak et al., 1996). Surprisingly, treatment with CPAP did not improve anxiety scores (Borak et al., 1996). It may be possible that the variability in the questionnaires and scales used to assess anxiety symptoms are not sensitive to the alternations in affect observed in OSA patients, rendering comparisons difficult. Few studies have utilised measures that tap into both state and trait anxiety. An example of this two-dimensionality in anxiety assessments is the State-Trait Anxiety Inventory (STAI) which is sensitive to transitory periods of anxiety as well as more stable personality features that predispose the respondent to more chronic levels of anxiety (Speilberger, 1983). The utilisation of anxiety measures which incorporate both transient and stable aspects of anxiety may yield different findings. The present study incorporated the STAI for the assessment of state and trait anxiety.

Although several investigations have reported an association between depression and OSA, this trend remains to be elucidated for anxiety. Methodological considerations may render comparisons between investigations a complex task since differences in sample size, study populations, gender distribution, age, AHI cut off points, and variability in measures of mood contribute to mixed findings (Shroder & O'Hara, 2005). However, despite these inconsistencies, OSA seems to play a substantial role in the development of mood disturbances and a reduced quality of life (Akashiba et al., 2002).

1.5.10. Summary of OSA. Obstructive sleep apnoea is a chronic condition characterised by repetitive episodes of hypoxemia and subsequent sleep fragmentation leading to EDS (Bedard et al., 1991; Guilleminault et al., 1976; Orr, 1997; Piper & Stewart, 1999). Wide-ranging and often psychologically oriented sequelae are observed in OSA patients, such as impaired vigilance, reduced psychomotor performance, impaired driving performance and increases in mood disturbances. Due to its medical nature, OSA is well understood and treatment is readily available, although many people remain undiagnosed and untreated. In contrast, there exists limited research examining individuals with poor sleep quality and quantity attributable to occupational factors such as rotating shift-work and irregular work schedules.

1.6. Shift-Work

Despite their varying aetiologies, OSA patients report similar sleep disturbances to those observed in people doing rotating shift-work. Although standard work schedules traditionally involve 40 hour weeks worked between the hours of 0900 and 1700 hr, the demand for services outside this 'window', particularly night work, has become common place. Shift-work and irregular work hours have been implicated as interchangeable terms to denote a work schedule in which different teams work in succession to cover most or all of a 24 hour period (Costa, 1997). Shift-work facilitates the management of around the clock

activities in relation to technological requirements (e.g., chemical and steel industries, power plants), social services (e.g., hospitals, transportation, telecommunications, security), economic industries (e.g., textiles, paper, food, mechanical services), and entertainment (Costa, 1997). Australian statistics indicate that 16% of the Australian workforce is employed in shift-work, with approximately 42% of workers engaged specifically in rotating shift-work (Australian Bureau of Statistics, 2009).

The variability inherent in shift-work schedules becomes evident when considering aspects such as the type of shift (morning/evening/night), the speed of rotation (number of shifts worked consecutively), the direction of rotation (forward or backward), and the length of the shift (Akerstedt, 2003). The multitude of working time arrangements among the shift-work population is likely to render comparisons across studies complex (Knutsson, 2004). Traditionally, shift rotations typically consisted of six or seven consecutive shifts of eight hour duration with one full weekend off every four to six weeks (Duchon, Keran & Smith, 1994), however recent conversion to a 'compressed work week' where shift duration is extended results in fewer work days and more consecutive days off (Purnell, Feyer & Herbison, 2002).

Shift-work, like OSA, represents a major public health hazard. The mismatch between the endogenous circadian system and environmental time cues interferes with biological functions and social life, resulting in immediate sleep disturbances, negative influences on performance efficiency, social relations and health (Costa, 1997). Despite inadequate evidence linking shift-work with increased mortality (Knutsson, 2003), shift-work has been associated with an increased risk of several health problems including gastrointestinal disturbances, cardiovascular disease, diabetes and metabolic disturbances, specific forms of cancer, and reproductive health issues in women (Costa, 1997; Culpepper, 2010; Garbarino et al., 2002a; Keller, 2009; Knutsson, 2003). In addition, oscillatory fluctuations in sleepiness

and alertness are likely to contribute significantly to 'human error', rendering work accidents, motor vehicle accidents and injuries more likely (Costa, 2003; Culpepper, 2010; Garbarino et al., 2002a). Greater absenteeism, greater employment dissatisfaction, lower morale, and increased familial and social difficulties are intrinsic among shift-workers (Costa, 1997; Costa, 2003; Culpepper, 2010; Nakata et al., 2004) which can negatively affect their quality of life. Mood disturbances, such as depression have also been implicated among the shift-work population (Akinnawo, 1988; Orton & Gruzelier, 1989; Scott, Monk & Brink, 1997). Despite the benefits of shift-work for the maintenance of continuous operations, there are wide-ranging implications across psychological, health and safety domains.

1.6.1. Shift-work and sleepiness. A considerable number of questionnaire studies have linked shift-work with increased levels of sleepiness, disturbances in sleep duration and sleep quality (Akerstedt, Ingre, Browman & Kecklund., 2008; Fido & Ghali, 2007; Gold et al., 1992; Paim et al., 2008; Santos et al., 2004). Specifically, working the night-shift has been associated with increased reported sleepiness (Akerstedt, Peters, Annund & Kecklund, 2005; Axelsson, Akerstedt, Kecklund & Lowden, 2004; Folkard & Tucker, 2003; Harma et al., 2002). Furthermore, clear intrusions of sleep-like EEG patterns have also been observed whilst working at night (Akerstedt, Peters, Anund & Kecklund., 2005). Morning shifts also exert a considerable influence on sleepiness (Akerstedt, 2003). Harma and colleagues (2002) demonstrated that the risk of severe sleepiness doubled in the morning-shift, relative to the evening-shift. Despite this, the night-shift remained the single most significant factor, leading to a six to 14 fold risk for severe sleepiness (Harma et al., 2002). However, one study found that on a measure of chronic sleepiness (i.e., the ESS), shift-workers did not score higher relative to non-shift-workers (Garbarino et al., 2002b).

Among a subject pool of nurses, Gold and colleagues (1992) revealed that those working a rotating shift schedule or a permanent night-shift reported fewer hours of sleep

relative to day and evening nurses. Additionally, night-shift nurses and rotating nurses were respectively 1.8 and 2.8 times more likely to report poor sleep quality (Gold et al., 1992). Falling asleep during the night-shift occurred at least weekly in 36% of rotating shift workers, 32% of permanent night workers and 21% of day and evening nurses working an occasional night-shift (Gold et al., 1992). These findings lend support to the notion that even among individuals working a permanent night-shift; the circadian clock fails to undergo sufficient adjustment (Folkard, 2008). In addition to shift type, associations between sleepiness and greater variability in workload have also been demonstrated among shift-workers (Takahashi et al., 2006). The age of the shift-worker has also been implicated as a factor in subjective sleepiness, such that increasing age may render the worker less tolerant of shift-work (Orr, 1997). In addition, Pati et al (2001) revealed a gender component in the assessment of subjective sleepiness, whereby females reported greater sleep disturbances compared to males.

Although less abundant, evidence of increased sleepiness through physiological markers have also been demonstrated among shift-workers (Akerstedt, 2003; Porcu, Bellatreccia, Ferrara & Casagrande., 1998; Torsvall, Akerstedt, Gillander & Knutsson., 1989). Torsvall and colleagues (1989) utilised tape recorders to record EEG and EOG changes among 25 paper mill workers over 24-hour periods including morning, afternoon or night-shifts. Their findings revealed that sleep after the night-shift was curtailed by two hours relative to the evening-shift, and predominantly affected stage 2 and REM sleep, with no change observed in SWS (Torsvall et al., 1989). In addition, approximately 20% of the sample exhibited sleep episodes during night work (Torsvall et al., 1989). In addition to disruptions in sleep, disturbances in wakefulness are also evident among shift-workers to the extent that sleepiness reached levels where wakefulness could not be maintained during the night-shift (Torsvall et al., 1989). Blink duration measures have also provided a useful means

of assessing objective sleepiness. In a study conducted by Hakkanen et al (1999), professional bus drivers exhibited an increase in the amount of time that the eyes remained closed on a sleep latency measure. Compensatory mechanisms such as those that allow sleep time or napping during the night-shift have been suggested to partially counteract the insufficient duration of sleep at home among night workers relative to day workers (Ribeiro-Silva et al., 2006). Purnell and colleages (2002) found that nap times as short as 20 minutes during the night-shift significantly improved performance without affecting the main sleep period of shift-workers (Purnell et al., 2002). In contrast, a study examining the effects of a 30-minute nap during an actual night-shift among shift-workers revealed that this period of time was not sufficient to overcome sleep loss and circadian effects on performance during the first night-shift, with no prior daytime sleep (Howard, Radford, Jackson, Swann & Kennedy., 2010).

The contribution of the number of shifts worked consecutively (speed of rotation) on sleepiness is complex given the multitude of patterns that exist in this form. However, given that night workers tend to sleep somewhat less than their day working counterparts (Akerstedt et al, 2005; Axelsson et al, 2004; Folkard & Tucker, 2003; Gold et al., 1992 Harma et al., 2002), it seems acceptable to conclude that fewer consecutive night-shifts would create fewer disturbances of circadian physiological functions. The direction of rotation (e.g., forward or backward) represents another consideration in shift-work schedules. In the absence of controlled longitudinal studies comparing performance in forward or backward shift rotations, Knauth (1995) reported that a forward rotation was likely to be associated with fewer problems. Lastly, considerable data exists associating the duration of the shift with sleepiness. In a comprehensive review of the literature on extended work hours comparing eight and 12 hour shifts, inconsistent findings have been yielded, with some studies reporting negative effects attributable to longer shifts (Son et al., 2008) and others

specifying positive results or no differences among the two work durations (Bendak, 2003). In an investigation of extended work shifts and sleepiness, Rosa (1995) suggested that over-time that was unscheduled was disruptive to shift-workers as it exacerbated the difficulty in managing sleep time and recovery.

The magnitude of the sleepiness inherent in shift-workers is highlighted by several factors. First, fatigue severity scores have been demonstrated to approach the severity of those reported by individuals with multiple sclerosis, systematic lupus erythematosus, and sleep disorders (Shen et al., 2006). Second, even among prior shift-workers who return to daytime work schedules, sleep difficulties attributable to shift-work have been suggested to persist (Marquie & Foret, 1999). Third, the disturbances in sleep associated with shift-work are often implicated as the cause for leaving shift-work for daytime work hours (Akerstedt et al., 2008). Among other factors (e.g., age, personality traits, good physical fitness and adequate family support), the notion of the 'healthy worker effect' may function to explain the high inter-individual variability in tolerance to shift-work (Akerstedt et al., 2008; Costa, 2003). Indeed, a study conducted by Forberg et al (2010) revealed that in a sample of tunnel workers, few experienced problems with sleep, adapted easily to their work and reported a high level of sleep efficiency. Since shift-workers represent a self-selected group, those that may be more likely to experience difficulties with shift-work may be more likely to cease employment, rendering only 'healthy workers' engaged in shift-work (Knutsson & Akerstedt, 1992).

Fourth, as a result of insufficient sleep, shift-workers, particularly those engaged in night work may be more vulnerable to experiencing a sleep disorder such as insomnia, OSA, or shift-work disorder (SWD) (Ohayon et al., 2002). Shift-work disorder is distinct from, and more severe than the sleep disturbances associated with shift-work, representing a clinical entity under the category of 'circadian rhythm sleep disorder' in the Diagnostic and Statistical

Manual of Mental Disorders, Fourth Edition (American Psychiatric Association, 2000). Reflected in the current nosologic system, SWD manifests as extreme difficulty maintaining adequate sleep and wake function whilst engaged in a shift-work schedule, with the minimal criteria including a primary symptom of either insomnia or EDS (Drake et al., 2004). In a large prevalence study, Drake and colleagues (2004) separated the criteria of SWD into symptoms of insomnia or EDS, revealing 32% and 26% in night workers and rotating shift-workers respectively. Lastly and in line with the aforementioned factors, shift-workers rarely attend sleep disorder clinics for their sleep disturbances, suggesting an inherent acceptance of the suboptimal sleep quality among these individuals which appear to be attributable to the fault of the worker or a disorder rather than a natural consequence of the work schedule (Regestein & Monk, 1991).

1.6.2. Causes of shift-work related sleepiness. Circadian rhythm disruption and sleep loss represent the main causes that induce sleepiness among shift-workers (Akerstedt, 1995). In relation to the former, Monk (1997) posited that human beings are a "diurnal creature endowed with biological processes that work under the assumption that the night will be taken up with sleep and the daytime with activity" (p.249). This temporal orientation is accomplished by the generation of circadian rhythms which function to maintain normal sleep and wake cycles that are essential for the maintenance of a healthy organism (Richardson, 2005). Physiological and behavioural rhythms remain internally and externally synchronised under constant environmental light and dark cycles (Toh, 2008). However, among night workers and rotating shift-workers, circadian rhythms are disrupted by shift schedules that conflict with the circadian timing system (Monk, 1997) and require realignment to a routine that is more fitting for the shift-worker.

Several factors function to highlight the de-synchrony inherent among shift-workers and the resultant sleepiness experienced during night work. First, both spontaneous

desynchrony and forced desynchrony protocols have demonstrated that alertness and performance exhibit time of day patterns with a maximum in the late afternoon and a nadir in the early morning around 0500 hr (Akerstedt & Folkard, 1997). Hence, conflict arises when shift-workers are engaged in work at the lowest point of physiological arousal. Second, although the amount of prior wakefulness preceding a morning-shift is typically one to two hours, the night-shift is associated with an extended period of wakefulness, generally between 10 and 16 hours (Akerstedt & Folkard, 1997). Therefore, sleepiness would be more likely in reference to a night-shift as opposed to an afternoon-shift. Lastly, sleepiness increases with respect to the amount of sleep prior to the night-shift. Although environmental influences, such as noise may explain the truncated day sleep after night work, there exists a strong circadian influence on sleep latency (Akerstedt & Folkard, 1997). This is highlighted by the fact that sleep at the maximum body temperature rhythm is difficult to initiate, whereas sleep at the nadir is easily instigated (Akerstedt & Folkard, 1997). In addition, laboratory and field studies have provided support for the notion that the process of adjustment among shift-workers is slow (Monk, 1997). Indeed, even under normal working conditions, complete phase adjustment to night-shift-work rarely occurs (Folkard, 2008).

In reference to the latter, shift-workers experience significant sleep loss, which is predominantly dependent upon the type of shift system worked (Akerstedt & Folkard, 1997; Monk, 1997). Investigations of the different durations of sleep obtained pertaining to various shift systems generally reveal that those engaged in night work and early morning work demonstrate the most decrements relative to their evening-shift counterparts (Akerstedt, 2003; Akerstedt et al., 2005; Akerstedt & Folkard, 1997; Axelsson et al., 2004; Gold et al., 1992; Harma et al., 2002; Folkard & Tucker, 2003).

In a description of the interplay between homeostatic sleep pressure and circadian wake pressure, Van Dongen (2006) described that the two were not adequately synchronised

among shift-workers. In his account, the homeostatic pressure for sleep increases with the gradual maintenance of wakefulness during the night-shift, however the circadian pressure for wakefulness decreases to produce a net result of steady increased sleepiness. Conversely, the opposite occurs during daytime hours subsequent to the night-shift, whereby the homeostatic sleep pressure decreases across the daytime sleep episode and the circadian wake pressure increases. The consequence is a pressure for wakefulness that exceeds the pressure for sleep, and the resulting sleep debt contributes further to sleepiness and performance decrements during the night. Among rotating and night-shift-workers who revert to a nocturnal sleep schedule during days off, the circadian adjustment does not occur rapidly enough, contributing to an endogenously enhanced level of sleepiness (Van Dongen, 2006).

Research conducted by Totterdell and colleagues (1995) examined the question of whether increasing the number of rest days between spans of work shifts improved performance on subsequent work days. A sample of shift-working nurses completed self-ratings, cognitive performance tasks (e.g., reaction time tasks), and a sleep diary using a hand-held computer for 28 days. Surprisingly, across many measures, capabilities were worse on rest days which followed a night shift and tended to be worse on the first rest day compared with subsequent rest days (Totterdell et al., 1995). Alertness levels were lowest for shift-workers on the first rest day following a night shift. Similarly, Akerstedt and colleagues (2000) found that for individuals working long shifts in long sequences, three days were needed for normalization. The authors also noted that sleepiness was at its peak during the first day of recovery (Akerstedt et al., 2000). Elevated sleepiness and impaired alertness in turn have been shown to cause immediate and unfavourable consequences such as reduced efficiency, injuries, accidents and reduced quality of life (Akerstedt et al., 2005; Takahashi et al., 2006).

1.6.3. Adaptation to shift-work. Circadian rhythm disturbances have been implicated as the contributory factor for the medical and psychological problems among shift workers (Moore-Ede et al., 1982). Whilst medical conditions such as OSA typically produce the need for appropriate treatments that can alleviate decrements associated with the condition, the occupational nature of shift-work renders treatment a questionable outcome. Although the use of hypnotics and alertness enhancing medications have been broadly considered in the literature (Akerstedt & Ficca, 1997; Stone & Turner, 1997), the systematic use of such agents in an occupational setting particularly when considering adverse effects, renders this an unlikely possibility. To counteract this, behavioural and physiological approaches have typically been employed to facilitate 'adaptation' to shift-work and are typically based on the notion that an acceleration of the circadian system can adapt to a phase-shift, thus reducing the degree to which workers are required to sleep at inappropriate phases of the circadian cycle (Dawson, Encel & Lushington, 1995). This has typically been demonstrated by appropriately timed exposure to bright light and exogenous melatonin, referred to as the 'darkness hormone' (Arnedt & Deacon, 1997; Dawson et al., 1995). Dawson and colleagues (1995) examined adaptation to night work in three groups of subjects receiving either timed exposure to bright light, exogenous melatonin by capsule, or dim red light and placebo capsules. Their findings revealed that the bright light condition showed the greatest shift and a more direct energising effect, relative to the melatonin and placebo groups. The authors concluded that bright light was superior in its ability to phase shift the circadian system and improve sleep and performance. In a study of the effects of melatonin administration on daytime sleep following night-shift-work, Sharkey, Fogg and Eastman (2001) found that although melatonin increased sleep time in subjects who demonstrated difficulty in sleeping during the day, it exhibited no effect on alertness using the MSLT, or performance or mood during the night-shift. Therefore, although the beneficial effects of

melatonin can be observed during day sleep, shift-workers may face difficulties working at night due to circadian misalignment (Sharkey et al., 2001). In contrast, melatonin administration may be preferable in rapidly rotating shift schedules as workers would not be required to reverse the large phase shift induced by bright light (Dawson et al., 1995).

The use of dark sunglasses, referred to as 'blue blockers' has also been implicated as a compensatory mechanism among shift-workers (Knauth & Hornsberger, 2003; Sasseville et al., 2009). The sensitivity of the circadian clock to the blue portion of the visual spectrum has been demonstrated (Brainard et al., 2001; Thapan, Arnedt & Skene, 2001). A study conducted by Sasseville and colleagues (2009) aimed to induce a circadian darkness period of approximately eight hours during the day to improve adaptation to night work in an uncommonly high light intensity indoor environment. The findings revealed that the experimental condition slept for longer periods of time, demonstrated increased sleep efficiency, and lowered their sleep fragmentation with the use of blue blockers. The authors summarised that the use of dark glasses created improvements in the daytime sleep of permanent night-shift-workers. Despite this, the study by Sasseville et al (2009) employed only a small sample of permanent night-shift-workers, and the utility of blue blockers among rotating shift-workers and other shift schedules requires further consideration.

1.6.4. Vigilance and psychomotor performance in shift-workers. A tacit assumption in studies assessing vigilance in shift-workers is that the former is impaired due to the combined effects of circadian rhythm disruption and sleep loss. Such decrements appear to be predominantly related to night-shift-work, and in particular, the first night-shift in the sequence, most likely in part due to the reduction in sleep duration caused by abrupt changes in the work schedule (Axelsson et al., 2004; Hart et al., 2003). This notion was supported in a study conducted by Axelsson and colleagues (2004) which revealed deterioration in reaction times and an increase in lapses throughout the night-shift relative to

day and evening-shifts. In addition to shift type, the findings presented by Bonnefond and colleagues (2006) also revealed a significant main effect for age in relation to psychomotor performance utilising the PVT. Although the percentage of lapses and median reaction times did not vary during morning and evening-shifts, a three-fold increase was observed during the night-shift among middle and senior aged groups (Bonnefond et al., 2006). The authors concluded that age was more distinctly associated with the steep increase of performance lapses to a greater extent than subjective sleepiness utilising the KSS (Bonnefond et al., 2006).

Purnell, Feyer and Herbison (2002) revealed changes in reaction time performance during the first night of a night-shift in a group of 24 male aircraft maintenance engineers. However no performance decrements were observed on a simple reaction time task at any other point of the shift rotation. Task duration has been suggested to represent a powerful determinant of performance decrements in sleep deprived individuals (Dinges & Kribbs, 1991), such that tasks of a longer duration induce a greater likelihood for performance impairments (Purnell et al., 2002). Earlier investigations provided support for performance fluctuations occurring systematically during sustained and prolonged work (Akerstedt, 1988). In line with this, work pace has been demonstrated to affect performance, such that the maintenance of vigilance is dependent upon an activity-provoking work schedule (Sallinen et al., 2004). For example, in a study examining the effects of sleep debt and monotonous work on performance, administration of a reaction time task revealed that average reaction times were 26 ms longer whilst engaged in monotonous work as opposed to busy workdays (Sallinen et al., 2004). In addition to a small sample size, the study conducted by Sallinen investigated performance during a 12-hour day-shift. Hence, it may be likely that the performance decrements among evening and night-shift-workers reveal different trends.

Similarly, Porcu et al (1998) examined performance during a simulated night-shift and concluded that deteriorations in vigilance only affected performance where a high attentive load was required for a continued period of time. However, the validity of this finding seems questionable to the shift-work population since non-shift-workers were employed for the study. Given that night-shift-workers and rotating shift-workers often self-select into shift-work, it is uncertain whether similar effects would be observed during a laboratory simulation among non-shift-workers. Indeed, Van Dongen (2006) provided support for this view, proposing that the performance variability inherent among shift-workers was much greater in the laboratory than would be observed in operational shift-work settings, since shift-workers vulnerable to decrements in vigilance could opt out of shift-work.

Even with the administration of wake promoting medications such as Modafinil, inconsistent findings have been yielded with respect to psychomotor performance among shift-workers. Walsh and colleagues (2004) revealed that accidental declines in performance (i.e., lapses) per night among the placebo group was between 40% and 200% higher than in the treatment group. In addition, significant increases in the frequency of lapses became evident as the night progressed. Specifically, the placebo group exhibited significantly greater lapses than the treatment group across three simulated night-shifts across the time points of 0315 hrs and 0600 hrs (Walsh et al., 2004). Again, this study employed non-shift-workers in the placebo and treatment groups, and it is uncertain whether the effects of Modafinil on vigilance would be observed among actual shift-workers.

Conversely, in a randomised placebo controlled study among individuals with SWD, high levels of impaired performance at night persisted despite treatment with Modafinil (Czeisler et al., 2005). The applicability of this treatment method for vigilance impairment seems uncertain given that these studies employed participants at two distinct levels of

severity (i.e., non-shift-workers as opposed to those with a diagnosable shift-related condition). With regard to other compensatory mechanisms, Sasseville et al (2009) reported that blue blockers did not alter subjective or objective vigilance during simulated night-shift-work.

1.6.5. Motor vehicle accidents and driving simulator performance in shift-workers. Decrements in vigilance and psychomotor performance have long been associated with an increased propensity for motor vehicle accidents (Garbarino et al., 2002c). Among shift-workers contending with circadian rhythm disruption and sleep loss, the prevalence of sleep-related motor vehicle crashes is increased, particularly when commuting home from the night-shift (Akerstedt et al., 2005; Keller, 2009: Scott et al., 2007). Since sleep propensity and the circadian drive for sleep is greatest during the early morning, the increased likelihood for a motor vehicle accident after a night-shift, particularly between the hours of 0200 and 0600 hrs seems expected (Scott et al., 2007).

In a sample of nurses, Gold and colleagues (1992) revealed that rotating shift-workers and night workers were respectively 3.9 and 3.6 times more likely to fall asleep at the wheel whilst driving to and from work in the year preceding the study, relative to day and evening nurses. Despite this, no information was collected pertaining to shift duration or the effects of shift length on driving performance. The importance of obtaining data related to shift length becomes evident when considering that shifts of 12-hour duration or greater increase the risk of drowsy driving and the potential for motor vehicle accidents (Scott et al., 2007). Sleep duration also appears to be an important factor for the increased likelihood of motor vehicle accidents. In a separate population of nurses, Scott et al (2007) revealed that shorter durations were associated with an increased risk of struggling to remain awake whilst driving home, potentially leading to a motor vehicle accident. Episodes of cognitive disengagement whilst

driving has also been suggested to dramatically increase accident risk (Dorrian, Roach, Fletcher & Dawson, 2007).

In a questionnaire study of police shift-workers, Garbarino and colleagues (2002c) revealed no difference among shift-workers and non-shift-workers in relation to accidents. Although motor vehicle accidents were reported in 51.5% of the sample, they were related solely to sleepiness rather than shift type (Garbarino et al., 2002c). In a random sample of over 2000 Australian commercial vehicle drivers, Howard and colleagues (2004) reported a two-fold increase in accident risk in the sleepiest five percent of drivers, with an even stronger relationship for multiple accidents using the ESS and Functional Outcomes of Sleep Questionnaire. Interestingly, a lower accident rate was reported for country and interstate drivers, and those driving on the night-shift (Howard et al., 2004). The authors proposed that the lower traffic density inherent in country, interstate and night drives may account for lower accident risk among these drivers (Howard et al., 2004).

There is limited research examining driving simulator performance in shift-workers. Philip and colleagues (2005) examined fatigue, sleepiness and performance in 12 healthy males on simulated driving tasks versus real driving conditions. Their findings revealed that driving, as measured by line crossings, was unaffected by 12 hours of real and simulated driving, although strongly influenced by partial sleep restriction combined with extended wakefulness (Philip et al., 2005). Arguably, the latter two factors are implicated in shift-workers, and may yield important considerations for this population with regard to driving performance. Despite this, virtually no studies have investigated real driving conditions in shift-workers. Consideration of the performance impairments in driving should be determined utilising instrumented cars in real-life settings (Akerstedt et al., 2005).

Akerstedt and colleagues (2005) investigated impairments during a simulated drive home from a regular night-shift. Outcome variables included simulator parameters closely

related to real driving (i.e., incidents and accidents), as well as continuous measures of driving performance (i.e., the standard deviation of the lateral position of the vehicle, and eye closure duration) (Akerstedt et al., 2005). Incidents, decreased time to accidents, increased variability in lateral position, increased eye closure durations and increased subjective sleepiness were observed commuting home from the night-shift (Akerstedt et al., 2005). However, this study employed only a small sample size of non-shift-workers exposed to a period of simulated night-shifts. It remains uncertain as to whether similar driving impairments would be observed among actual shift-workers. Ingre and colleagues (2006) examined subject-specific estimates of the relation between subjective sleepiness (measured with the KSS), blink duration, and lane drifting on a driving simulator. Findings revealed that with higher KSS scores, the standard deviation of the lateral position on the driving simulator increased. Driving impairments associated with shift-work have to date received little attention in the literature with reasons for this remaining uncertain. In light of the view that shift-work represents the 'fault of the worker' (Regestein & Monk, 1991), relative to chronic medical conditions such as OSA, investigations of driving decrements among this population may not receive adequate attention.

1.6.6. Mood disturbances in shift-workers. The sleep loss and circadian rhythm disruption inherent among shift-workers has been indicated to cause mood disturbances, particularly depressive symptoms (Akinnawo, 1988; Orton & Gruzelier, 1989; Scott et al., 1997) and has been implicated as a cause for absenteeism (Nakata et al., 2004). Indeed, the adverse consequences on mood in shift-workers are highlighted when considering that these effects may be even greater than those on alertness and cognitive performance *(*Pilcher & Huffcutt, 1996*)*. However, despite the suggested magnitude of mood disturbances among shift-workers, psychological and psychiatric disturbances remain relatively under-investigated in the literature (Goodrich & Weaver, 1998). The fact that shift-work represents

an occupational matter as opposed to a medical phenomenon, may function to explain the paucity of research examining associations between mood and shift-work. Furthermore, of the studies assessing mood among shift-workers, inconsistent findings have been yielded.

In an earlier investigation examining 463 nurses, Skipper, Jung and Coffey (1990) found no association between shift-work and measures of depression. The absence of an association between shift-work and depression may in part be attributable to the nature of this work and differing requirements and job tasks for each shift. Since the work of nurses is different, direct comparisons among the shifts may not be valid since it is possible that the work itself also influences depression (Skipper et al., 1990). Goodrich and Weaver (1998) employed a diverse sample of industrial bakers, physical plant workers, police officers and safety workers, and found no association between depression and shift-work. Interestingly, shift-workers fared better overall with respect to depressive symptomatology as measured by the BDI relative to traditional workers, and no gender differences were found (Goodrich & Weaver, 1998).

Conversely, in a pilot study examining shift-work as a risk factor for depression, Scott et al (1997) found a high prevalence of major depressive disorder during or after the shift-work experience, with a higher rate for women than for men. Orton and Gruzelier (1989) investigated the effects of long work hours and reduced sleep on mood in a sample of house officers towards the end of a normal working day and after working for up to 31 continuous hours with reduced sleep. The authors demonstrated significant deleterious changes in mood occurring after the night duty across all mood scales in the POMS questionnaire (Orton & Gruzelier, 1989). Sharkey and colleagues (2001) examined mood during the night-shift and revealed better mood during the second night-shift compared to the first, with mood gradually declining across the night. However, this study simulated night-shift-work in normal subjects as opposed to employing actual shift-workers for the study protocol, and hence the

applicability of this finding is questionable to the shift-work population. In addition, mood was assessed with a general neurobehavioral assessment battery as opposed to a specific mood inventory, and it remains uncertain whether this test battery is sensitive to disturbances in mood.

In a study examining psychopathology among shift-workers, Akinnawo (1988) revealed that the shift-work sample experienced higher levels of psychopathology relative to their non-shift working counterparts. In fact, mood disturbances were indicated as the most prominent consequence resulting from shift-work (Akinnawo, 1988). However, the applicability of these findings remains uncertain to the shift-work population at large since psychopathology was operationalised as a cluster of general symptoms including intellect, mood, perception, speech, motor behaviours and general somatic symptoms which were not entirely specific to mood disturbances.

Despite suggested associations between shift-work and anxiety (Costa, 2003), few studies have examined anxiety symptoms in different shift-work schedules. A recent study conducted by Almondes and Arauyo (2009) examined the impact of different work schedules on levels of anxiety among workers in a petrochemicals company. A total of 239 workers were classified into either (a) a fixed daytime-shift (b) a 12-hour rotating day and night-shift, (c) a 12-hour rotating day-shift and (d) a 9.5-hour rotating day-shift, and completed the STAI. The authors concluded that shift-workers exhibited higher STAI scores relative to fixed daytime workers, although no significant differences were observed between the three shift categories (Almondes & Arauyo, 2009). Further research is needed to examine possible links between anxiety symptoms in shift-workers.

1.6.7. Summary of shift-work. The provision of irregular work hours beyond that of standard work schedules has become prevalent in society. Shift-work is associated with widespread consequences that are similar to those observed in patients with OSA, including

sleep loss, circadian rhythm disruption, impairments in vigilance, decreased psychomotor performance, increased propensity for motor vehicle accidents and adverse changes in mood. The impact of behavioural and physiological approaches to counteract the deleterious effects of shift-work has yielded inconsistent findings with regard to adequately adapting to shift-work (Dawson et al., 1995). In addition, the sequelae associated with shift-work remain significantly under-studied in the literature most likely due to the occupational nature of shift-work and the notion that shift-work remains the choice of the worker, relative to medical conditions such as OSA.

1.7. Rationale for the Present Study

The effects of sleepiness and sleep loss have wide-ranging implications across a variety of domains including alertness and vigilance, psychomotor performance, driving ability and changes in mood. Despite aetiological differences, patients with the chronic medical condition of OSA, and those engaged in the tumultuous routine of shift-work experience constant sleep disruption which is likely to render the above consequences more pronounced in these populations. Although inconsistencies exist in the literature with regard to decrements in OSA patients, the condition remains well documented, most likely due to its medical nature.

In contrast, there exists limited research examining these consequences in shift-workers. Research on shift-work seems to have been largely biased by an inherent belief that can be fittingly characterised by the notion of ‘let sleeping dogs lie’. Indeed, given the intrinsic level of acceptance that accompanies occupational shift-work, it seems likely that this population has been somewhat isolated and has not received due attention in the literature. The importance of challenging this assumption, and ‘waking the sleeping dog’ becomes apparent when considering that the sleepiness experienced by shift-workers has been demonstrated to approach the severity reported by patients with sleep disorders, such as

OSA (Shen et al., 2006). Therefore, if the consequences of OSA continue to be well documented, it seems reasonable to enhance our understanding of the disturbances experienced by non-clinical populations, such as shift-workers who may report equivalent disturbances. Furthermore, unlike the treatment options that can be offered to patients with OSA, the impact of behavioural and physiological approaches to counter the adverse effects of shift-work have been under-investigated and have yielded inconsistent findings. Therefore, there is a need for research that will enhance our understanding of the profile of disturbances for the shift-worker to facilitate the implementation of more fitting shift schedules and potential intervention policies and procedures.

Specifically, subjective and objective sleepiness and psychomotor performance have been suggested to mediate an increased risk for motor vehicle accidents in patients with OSA. However, there are a limited number of investigations examining these consequences among shift-workers. In addition, few studies have explored associations between simulated driving performance in shift-work populations. Since the relationship between subjective and objective sleepiness, psychomotor performance and accident risk is unclear, examining these associations may assist in identifying individuals at risk of motor vehicle accidents. In addition, the adverse consequences on mood in shift-workers have been suggested to be greater than those on alertness and cognitive performance (Pilcher & Huffcutt, 1996). However, mood disturbances in shift-workers are under-investigated, particularly anxiety symptoms. Examining disturbances in mood in a non-clinical sample, such as shift-workers, may facilitate the application of intervention strategies for this population.

1.7.1. Aims and hypotheses. The present study was investigative and aimed to compare subjective sleepiness, objective sleepiness, psychomotor performance, simulated driving performance, and mood disturbances (i.e., depressive symptomatology and anxiety symptoms) in patients with OSA and shift-workers in comparison to a control group. A

further aim of this study was to determine whether significant relationships existed between these measures for the three groups. Finally, this study aimed to investigate possible associations between subjective sleepiness, objective sleepiness, psychomotor performance, simulated driving performance and mood with the number of accidents experienced in the preceding three years in an attempt to determine accident risk. The following hypotheses were central to the present study.

1. Scores for subjective sleepiness would be greater among OSA patients relative to shift-workers, and shift-workers would have higher scores for subjective sleepiness relative to control subjects.
2. Scores for objective sleepiness would be greater among OSA patients relative to shift-workers, and shift-workers would have higher scores for objective sleepiness relative to control subjects.
3. OSA patients would perform more poorly on a test of psychomotor vigilance and driving simulator performance compared to shift-workers, and shift-workers would perform more poorly on these tasks relative to control subjects.
4. Patients with OSA would demonstrate greater disturbances in mood (as evidenced by higher scores across the three mood measures) relative to shift-workers, and shift-workers would demonstrate greater mood disturbances compared to control participants.
5. Subjective sleepiness measures would be associated with measures of objective sleepiness between OSA patients, shift-workers and control participants.
6. Measures of subjective sleepiness would be associated with poorer performance on a reaction time task and a simulated driving task between OSA patients, shift-workers and control participants.

7. Subjective sleepiness measures would be associated with mood disturbances among OSA patients, shift-workers and control participants.
8. Measures of objective sleepiness would be associated with poorer performance on a reaction time task and a simulated driving task between OSA patients, shift-workers and control participants.
9. Objective sleepiness measures would be associated with mood disturbances among OSA patients, shift-workers and control participants.
10. Performance on a reaction time task and a simulated driving task would be associated with mood disturbances among OSA patients, shift-workers and control participants.
11. Measures of subjective sleepiness, objective sleepiness, performance (i.e., psychomotor and driving simulator performance) and mood would be associated with the number of accidents in the preceding three years.

2. METHOD

2.1. Participants

The total sample was 47 participants (33 men and 14 women) who were recruited to form the following groups: an untreated moderate to severe OSA patient group, a rotating shift-work group, and a control group which comprised of participants who had never worked shifts and did not have OSA. In the untreated OSA group, there were 15 men and 2 women aged between 37 and 75 years old (M = 58.24, SD = 10.73). In the rotating shift-worker group there were 12 men and 3 women aged between 35 and 52 years old (M = 45.33, SD = 7.89). The control group consisted of 6 men and 9 women aged between 34 and 62 years old (M = 47.47, SD = 7.25).

The participants were all required to be 18 years of age or older, hold a current Australian driver's licence, be capable of providing informed consent, and also be capable of speaking, reading and writing English at a functional level. General and psychiatric health was examined by means of a screening questionnaire and individuals with conditions that may have affected driving performance, including chronic neurological illnesses (e.g., epilepsy, traumatic brain injury), significant medical co-morbidity (e.g., cardiovascular disease, diabetes), chronic psychiatric illnesses, visual acuity problems not correctable with glasses, and regular use of sedating medication/s were excluded. Participant selection was performed to allow each group to be matched as closely as possible for age, gender and educational level. The following section outlines the recruitment process for each of the three participant groups.

2.1.1. Obstructive sleep apnoea patients. Obstructive sleep apnoea patients were recruited via Physicians at Austin Health Sleep Clinics following a diagnosis of moderate to severe OSA verified by an overnight polysomnographic sleep study. Only patients with an AHI greater than 20 events per hour were selected to participate as they presented to the sleep

clinic. To be included, OSA participants were required to be advancing to treatment with CPAP. However, at the time of the study, none of the OSA participants had undergone CPAP therapy.

2.1.2. Rotating shift-workers. Rotating shift-work participants were recruited from the Melbourne Metropolitan area via advertisements placed in local newspapers, Austin Health newsletters, Trade Union publications, police stations, fire stations, ambulance stations, and local supermarkets. In addition, letters detailing the study were also mailed out to security companies, freight/shipping companies, gaming services, and courier services in the Melbourne Metropolitan area.

To be recruited for the study, workers were required to have done regular rotational shifts (day, afternoon, night) for the preceding three months. Rotating shift-work was operationalised as the alternation between a night shift and either a day or afternoon shift worked on a weekly basis, with no more than four days recovery time between the previously worked night-shift and the next rostered shift. Participants who worked only a permanent night-shift, but attended to other responsibilities on their non-working days were able to participate in the study, as this was likely to disrupt circadian sleep-wake patterns to a similar extent to working rotating shifts. A further inclusion criterion for shift-workers was that they were required to score less than 0.5 on the Multivariate Apnoea Prediction Questionnaire (MAPQ), indicating a low probability of OSA (Maislin et al., 1995). High scores on the Epworth Sleepiness Scale (ESS) were not used to exclude shift-workers given that shift-work can cause fatigue and sleepiness. Shift-workers with OSA, respiratory disorders, and/or a history of neurological disorders were excluded from the study. Rotating shift-workers with mild psychiatric conditions such as depression and anxiety (as measured by several mood questionnaires employed for the study) were permitted to participate. Shift-work participants were financially reimbursed with a cheque for $150 for their participation.

2.1.3. Control participants. Control participants were recruited via advertisement in the local paper, hospital newsletter, and university campus and were eligible for inclusion if they had not worked a rotating shift roster for a period of three months preceding the study, or worked nights as part of their usual work schedule. Potential control participants with OSA, chronic sleepiness, respiratory disorders, current psychiatric illness, and a history of major neuropathology were excluded. Control participants were required to have a score of less than 0.5 on the MAPQ and were excluded if they scored greater than 10 on the ESS and/or had an AHI greater than 20. Control participants who were currently unemployed or retired were permitted to participate in the study. Control participants were financially reimbursed with a cheque for $150 for their participation.

2.2. Apparatus

2.2.1. Screening questionnaires. The Initial Contact Screening Questionnaire was developed for the purpose of screening participants for inclusion or exclusion to the study. It consisted of a range of questions to elicit basic demographic information as well as information about the potential participants' medical history (see Appendix 1). The Epworth Sleepiness Scale (ESS) and the Multivariate Apnoea Prediction Questionnaire (MAPQ) were also included to screen respectively for excessive daytime sleepiness and OSA.

2.2.1.1. Epworth Sleepiness Scale (ESS) (Johns, 1993). The Epworth Sleepiness Scale (ESS) is a subjective rating scale used to measure levels of daytime sleepiness. It was used to identify participants with excessive daytime sleepiness and/or disordered sleep (see Appendix 2). The ESS required participants to rate the likelihood of falling asleep or 'dozing off' in eight situations commonly encountered in daily life on a scale ranging from 0 (0 = never) to 3 (3 = high chance of dozing). Possible scores range from 0 to 24, with higher scores reflecting a greater likelihood of sleepiness and disordered sleep. Johns and Hocking

(1997) have shown that in the absence of any sleep disorder, normal sleepers reported ESS scores that ranged between 0 and 10.

The ESS is a widely used method for measuring daytime somnolence in adult populations, is reliable under test-retest conditions, and has a high level of internal consistency (Johns, 1992). In an initial validation study, ESS scores were demonstrated to significantly correlate with sleep latency measured during the day with the MSLT and during overnight polysomnography (Johns, 1991). In addition, patients with sleep disorders showed significantly higher scores on the ESS in comparison with control participants (Johns, 1991). In a later study, scores on the ESS were able to distinguish between patients with simple snoring and patients with OSA, even it if was of only mild severity (Johns, 1993).

2.2.1.2. Multivariate Apnoea Prediction Questionnaire (MAPQ) (Maislin et al., 1995). The Multivariate Apnoea Prediction Questionnaire (MAPQ) is an instrument that can be used to predict sleep apnoea risk (see Appendix 3). The MAPQ consists of three questions that tap into symptoms of sleep disordered breathing and 10 questions that enquire about other symptoms of EDS. Participants are asked to identify whether they have experienced or have been told that they experienced symptoms of sleep apnoea during the last month. The frequency with which these symptoms occur are rated on a 6-point Likert scale (0 = never, 1 = rarely, less than once a week, 2 = 1-2 times a week, 3 = 3-4 times a week, 4 = 5-7 times a week, 5 = don't know). Symptom frequency is only scored for questions rated 1 to 3. Possible scores range from 0 to 1, with 0 representing a low risk of sleep apnoea and 1 representing a high risk. MAPQ scores were derived by detailing the age, weight, height, and gender of the participant, and the participants endorsed responses to the first 3 items of the questionnaire (1 = snorting or gasping, 2 = loud snoring, 3 = breathing stops/choke or struggle for breath).

Maislin et al (1995) demonstrated the ability of the MAPQ to predict sleep apnoea items relative to a clinical diagnosis of sleep apnoea, whereby the prevalence of sleep apnoea

ranged from 20% in patients with Index 1 values, to 74% in patients endorsing the full range of sleep apnoea items. Furthermore, the MAPQ has been demonstrated to be reliable under test-retest conditions in a sample of 1,071 patients with various sleep disorders (r = 0.92).

2.2.2. Preliminary questionnaires

2.2.2.1. Demographic Questionnaire. The Demographic Questionnaire was developed for the purpose of the present study (see Appendix 4). It was used to obtain basic demographic information about the participants including, age, gender, height, weight, current occupation, and highest level of education completed.

2.2.2.2. Driving Information Questionnaire. This questionnaire was developed for the current study to ascertain information regarding participants' driving practices and the nature of their current employment if this was applicable (see Appendix 5). Participants were asked to identify whether they drove at work, the type of shift/s they worked, whether they rotated shifts, and where they drove (i.e., metropolitan, country or interstate). Specific questions relating to employment (i.e., duration of longest shift, days worked per week, and hours worked per week) were also ascertained. Questions pertaining to driving patterns, included number of hours driven per week (at work and not work related), and kilometres driven per year (at work and not work related). In addition, specific questions related to sleeping habits were also incorporated, including number of hours slept per night (on work days and days off), number of alcoholic beverages consumed on a daily basis and number of other beverages (i.e., tea, coffee, cola) consumed on a daily basis. An estimate of the number of accidents participants experienced (utilising a method designed by Austin Health) was also incorporated into the Driving Information Questionnaire. This information was gathered from a history of accident reports from the preceding three years, and included all motor vehicle accidents that occurred regardless of fault, as well as home and work related accidents.

2.2.3. Measures of subjective sleepiness

2.2.3.1. Karolinska Sleepiness Scale (KSS) (Akerstedt & Gillberg, 1990). The KSS is a commonly used measure of sleepiness at a particular point in time (see Appendix 6). Participants are required to rate their level of sleepiness at the time of completing the questionnaire on a scale ranging from 1 to 9 (1 = extremely alert, 3 = alert, 5 = neither alert nor sleepy, 7 = sleepy, but no difficulty remaining awake, to 9 = extremely sleepy, fighting sleep). Even items endorsed have a scale value despite having no verbal label. Higher scores are indicative of greater subjective sleepiness. KSS ratings have been demonstrated to be closely correlated with EEG and EOG variables, and other behavioural variables, suggesting a high level of validity in measuring sleepiness (Kaida et al., 2006).

2.2.3.2. Stop Driving Questionnaire (SDQ). The SDQ is a self-rating questionnaire that was developed at the Sleep Disorders Unit of Austin Health, Melbourne, Australia to identify when drivers felt they would stop driving after completing the simulated driving task (see Appendix 7). Participants were required to identify whether they would continue to drive in two real life situations, namely, a city drive and a prolonged country drive. Participants rated their level of alertness on a scale from 1 to 4 (1 = I would continue driving, 2 = I would continue driving only if pressured to do so, 3 = I would stop driving now even if under pressure to continue, to 4 = I would have stopped driving some time ago).

2.2.3.3. Sleepiness Symptoms Questionnaire (SSQ). The SSQ is a self-rating scale developed at the Sleep Disorders Unit of Austin Health, Melbourne, Australia which asked participants to identify how often they noticed eight sleepiness symptoms during the simulated drive on a 7-point Likert scale (1 = not at all, 3 = occasionally, 5 = frequently, and 7 = most of the time) (see Appendix 8). Even items endorsed have a scale value despite having no verbal label. Sleepiness symptoms included: ‘struggling to keep eyes open’, ‘vision becoming blurred’, ‘nodding off to sleep’, ‘difficulty keeping to the middle of the road’,

'difficulty maintaining the correct speed', 'head dropping down', 'mind wandering to other things', and 'reactions slow'. Possible scores ranged from 1 to 7 for each item, as well as a total summed score of all eight symptoms between 8 and 56. Higher scores were suggestive of increased sleepiness. For the current study, a total score for all items was recorded. Internal consistency has been documented for the SSQ, with a Cronbach's alpha of 0.95 (Radford, 2001).

2.2.4. Instruments for assessing objective sleepiness

2.2.4.1. Optalert Drowsiness Measurement System (ODMS; Sleep Diagnostics Pty Ltd) (Johns, Tucker, Chapman, Crowley & Michael., 2007). The ODMS is a system used to assist research on the impairment of performance caused by drowsiness. This system provides a continual measure of drowsiness by continuously recording detailed eye and eyelid movements. The system is made up of several components including non-optical spectacle frames and a computer which runs the ODMS software. The spectacle frames house an infrared (IR) emitter and detector assembly attached to the frame that allows for the objective measurement of eye and eyelid movements at over 500 times per second, based on reflectance IR light directed at the eyes. Prescription lenses can also be fitted to the glasses. The following image shows the Optalert glasses with IR emitters and light pulses directed towards the eye.

Figure 2.01: Optalert glasses (without prescription lenses) with IR emitters in the arm attached to the frame, showing the direction of the IR light pulses directed up at the eye (adapted from Johns, 2007 with permission).

The ODMS measures several parameters including the ratio of the amplitude to the velocity of each movement, the binocular synchronisation between the movements of each eye, and the duration of all movements. Collectively, these parameters provide a continuous and objective measure of drowsiness every minute, termed the Johns Drowsiness Scale (JDS). The JDS ranges from 0 to 10, and an increase in this scale is indicative of greater levels of drowsiness. The ODMS defines drowsiness as either cautionary (JDS level of 4.5), or critical (JDS level above 5.0). Subjects with a JDS score below 4 are considered to be alert.

For the current study, performance indicators analysed for the Optalert included an average JDS score and the amount of time that the eyelids remained closed per minute, termed 'percent long closure'. Eyelid closure, as measured by the Optalert has been found to correlate strongly with driving performance in sleepy participants (Johns et al., 2007). In addition, the scale of drowsiness, as measured by the Optalert has been validated against several objective and subjective measures including driving simulator performance, psychomotor vigilance performance tests, reaction time, EEG, EOG, driver hazard perception and self-reported sleepiness assessments (Optalert ®, 2011).

2.2.4.2. Oxford Sleep Resistance Test (Osler; Stowood Scientific Instruments Ltd) (Bennett, Stradling & Davies, 1997). The Osler is an objective measure of sleep latency, or the duration of time taken to fall asleep. This test utilises a computerised, non-assisted method for monitoring wakefulness and detecting sleep onset. Relative to other standard measures of daytime sleepiness, the Osler is simple to use, low cost, and provides an automatic reading at its completion. The participant's dominant hand is placed on a box held in their lap. Their index finger is positioned on a non-recoil proximity sensor, with a sensing distance of approximately 1 to 2mm, and transmits finger contact to a computer. A light emitting diode (LED) is positioned 4 feet away at the participant's eye level in the frontal

visual field. The red light illuminates regularly for 1 second every 3 seconds, and participants are instructed to tap the sensor when the LED is illuminated. Both the LED and the hand device are connected to a computer which records the response data. The Osler defines sleep onset when there is no response to 7 consecutive illuminations. Performance indicators employed for assessing sleepiness included an average sleep latency (measured in minutes), and the number of omissions that occurred in response to an illumination. The following photograph shows the Osler unit and the hand held device.

Figure 2.02: Oxford Sleep Resistance Test (Osler) unit and hand-held sensor device (picture taken with permission from Stowood Scientific Instruments Ltd).

The simplistic nature of administration of the Osler, coupled with its ability to automatically and objectively define sleep onset, provides a robust method for quantifying daytime sleepiness. The Osler has been shown to distinguish normal subjects from sleep apnoea patients, has good concurrent validity with another widely used measure of sleep latency, termed the Maintenance of Wakefulness Test (MWT), and 'lapses' on the Osler have been validated against polysomnographically defined micro-sleeps (Priest et al., 2001).

2.2.5. Instruments for assessing driving performance and reaction time

2.2.5.1. Aus Ed Driving Simulator (© Grunstein et al., 1998) (Desai et al., 2007).

The AusEd Driving Simulator was utilised to assess participants' driving performance and tapped into several cognitive abilities deemed necessary for driving. These include: reaction

time, vigilance, tracking ability and divided attention. The simulator was controlled using an accelerator and brake pedal and a steering wheel was attached to a table in front of the screen. For the current study, performance markers for the driving simulator included: (1) mean variation in speed from the optimum 60 to 80km/h speed range, (2) mean variation in speed from the 70km/h speed range, (3) mean variation in lane position during the drive, (4) mean reaction time for braking episodes, and (5) crash incidence. The following image displays the driver's view of the graphics displayed on the monitor.

Figure 2.03: Drivers view of the driving simulator (photo taken with permission from Grunstein).

The AusEd Driving Simulator has been demonstrated to correlate with a sleep latency measure, and is sensitive to performance decrements including sleep deprivation, circadian influences, time-on-task effects and sleep disorders (Desai et al., 2007). For example, patients with OSA performed more poorly on measures of steering deviation, mean reaction time, and crash frequency relative to 'intermediate' and control groups (Desai et al., 2007). The AusEd Driving Simulator may have widespread utility for the detection and management of driver fatigue.

2.2.5.2. Psychomotor Vigilance Task (PVT) (Ardmore, Dinges, Kribbs & Powell, 2000). The PVT is a simple hand-held reaction time and vigilance task that requires continuous attention to identify and respond to randomly occurring stimuli over a period of 10 minutes. The task is sensitive to performance variations caused by sleepiness. The PVT is a purpose built microcomputer enclosed in a small plastic box with two response buttons at

the bottom of the unit (right and left), and one display screen situated at the top of the unit. Two response buttons function to correspond with the dominant hand of the participant.

Participants are instructed to observe the LED display window closely and respond as quickly as possible to the appearance of numbers presented at variable intervals (2000-10,000msec) by pressing the appropriate response button with the thumb of their dominant hand. For correct responses, the LED displayed the reaction time in milliseconds. If the incorrect button was pressed, participants received an error message (ERR). A response in the absence of a numeric stimulus appeared as a 'false start' (FS). Outcome measures for the PVT which were employed for the current study included: (1) mean reaction time, (2) slowest 10% of reaction times, and (3) lapses (number of reaction times greater than 500 milliseconds). The photograph below shows an image of the PVT unit.

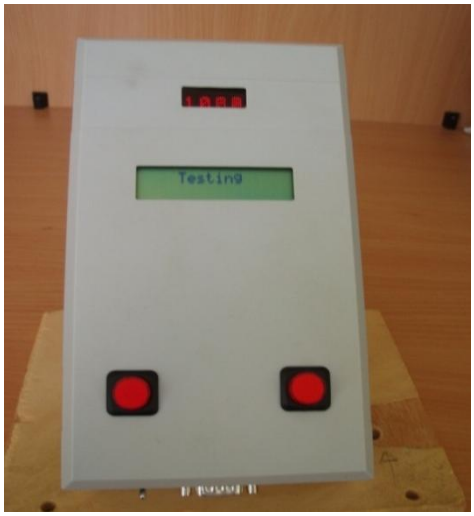

Figure 2.04: PVT Microcomputer displaying LED window display and response buttons (picture taken with permission from Ambulatory Monitoring Inc).

The PVT has been demonstrated to be sensitive to sleep deprivation effects, partial sleep loss effects, and circadian variation in performance (Dinges, Orne, Whitehouse & Carota Orne, 1987).

2.2.6. Mood measures

2.2.6.1. Beck Depression Inventory (BDI) (Beck et al., 1961). The Beck Depression Inventory (BDI) is a 21-item questionnaire presented in multiple choice format for the purpose of ascertaining the presence and degree of depression in adults (see Appendix 9). Each of the 21-items on the BDI assesses a symptom or attitude specific to depressed individuals and is consistent with descriptions of depression in the psychiatric literature. The items contained within the BDI consist of a graded series of four evaluative statements which are rank ordered and weighted to reflect the range of severity of the symptom from neutral to maximum severity. Numerical values of 0, 1, 2 and 3 are assigned to each statement to indicate the degree of severity. Possible scores range from 0 to 63 and respondents are classified as experiencing minimal, mild, moderate, and severe depression. Higher scores are indicative of greater levels of depression. Respondents' total BDI scores were analysed for the current study.

The BDI is a widely utilised measure for assessing the presence of depression in adults, and has been reported to have high internal consistency in psychiatric and non-psychiatric samples, high content validity, sensitivity to change, and high convergent validity with other depression rating scales (Ritcher et al., 1998).

2.2.6.2. State-Trait Anxiety Inventory (STAI) (Spielberger, 1983). This inventory is a 40-item self report questionnaire used to measure anxiety in adults (see Appendix 10). The STAI is sensitive to transitory periods of anxiety (state) as well as more stable personality features that may predispose the respondent to experiencing more chronic levels of anxiety (trait). The STAI consists of 20 items for trait anxiety and 20 items for state anxiety. Each STAI item is given a weighted score of 1 to 4 (1 = not at all, 2 = somewhat, 3 = moderately so, 4 = very much so). A rating of 4 indicates the presence of a high level of anxiety for 10 state anxiety items and 11 trait anxiety items. A high rating is indicative of an

absence of anxiety in the remaining 10 state anxiety items and 9 trait anxiety items, as the scoring weights for anxiety-absent items are reversed (i.e., responses endorsed as 1, 2, 3, or 4 are scored as 4, 3, 2 or 1). For state anxiety, anxiety-absent items are 1, 2, 5, 8, 10, 11, 15, 19, and 20, and for trait anxiety, anxiety-absent items are 21, 23, 26, 27, 30, 33, 34, 36, and 39. Anxiety present items are not reversed. The STAI is scored by considering both the variable itself and the relative magnitude of the respondents' score. Possible scores range from 20 to 80 for state anxiety items and trait anxiety items. Respondents' total score for state anxiety and trait anxiety were employed as outcome measures.

Estimates of reliability coefficients for trait anxiety have been reported to be between 0.65 and 0.86; however coefficients for state anxiety are much lower (between 0.16 and 0.62). This low stability level would be expected since responses to state anxiety items are considered to reflect the influence of transient situational factors at the time of test completion (Spielberger, 1983). In addition, correlation coefficients presented by Spielberger (1983) suggest that the STAI is a valid measure of anxiety and is correlated with other anxiety scales (between 0.75 and 0.8).

2.2.6.3. Profile of Mood States – Short Form (POMS-SF) (Shacham, 1983). The POMS-SF is an adaptation to the original 65-item POMS which is a self-report inventory used to assess mood state changes in psychiatric populations as well as healthy samples (see Appendix 11). The POMS-SF consists of 37 items which describe mood on a 5-point Likert scale (0 = not at all, 1 = a little, 2 = moderately, 3 = quite a bit, 4 = extremely). Respondents rate six mood subscales, including Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigour, Fatigue-Inertia and Confusion-Bewilderment. The POMS-SF also provides a measure of total mood disturbance when all factors are summed (the Vigour scale is negatively weighted). Participants are asked to rate the inventory according to how they have been feeling during the past week, inclusive of the day of completion. Standard scores for all

six mood factors and a total mood disturbance score were analysed as outcome measures for the current study.

A study examining five clinical populations and one sample of healthy adults found that estimates of internal consistency for the POMS-SF total mood disturbance score and individual subscale scores ranged from 0.76 to 0.95, and were generally similar to, or exceeded, those of the original 65-item POMS (Curran, Andrykowski & Studts, 1995).

2.2.7. Overnight polysomnography (sleep study). The polysomnogram is a non-invasive procedure that is performed by a sleep technician. During this procedure, a machine consisting of a series of amplifiers converts the body's physiological signals into a graphical representation to determine what occurs during sleep (Castronovo, 2006). Data from these amplifiers are then stored onto a computer which converts analogue signals to digital signals for subsequent analysis (Castronovo, 2006).

Several physiological outcome measures are recorded while the participant sleeps, including EEG, EOG and EMG data, blood oxygen levels ($Sa0_2$), respiration, abdominal and thoracic movement, airflow nasal pressure, body position, and leg movements. Small electrodes are attached to the participant's scalp, face, chest and legs with self-adhesive tape, and two bands are strapped around the chest and abdomen to monitor breathing. In addition, an airflow detector is attached to the nose and mouth, and an oxygen sensor is attached to the finger. The results of the sleep study are later analysed by a sleep technician to determine the presence of any sleep disordered breathing. Outcome measures for the polysomnography included: (1) total number of apnoeas and hypopnoea events per hour, (2) lowest oxygen saturation level reached overnight, (3) total number of arousals per hour of sleep, (4) sleep efficiency (in minutes), and (5) amount of time spent in REM sleep.

2.3. Procedure

Ethics proposals were submitted to the Victoria University Department of Psychology Research Ethics Committee and the Austin Health Human Research Ethics Committee for consideration, and approval was obtained to undertake the study. Testing was undertaken between 2007 and 2010.

Obstructive sleep apnoea participants were referred to the researcher by a Sleep Physician at Austin Health following a diagnosis of moderate to severe OSA which was confirmed by overnight polysomnography. Shift-work and control participants responded to advertisements (see Appendix 12) via email or telephone contact to the researcher. A Participant Information Letter detailing the project and a consent form was mailed out to potential participants (see Appendix 13). Consenting individuals made contact with the researcher to indicate their willingness to participate in the study, and an initial consultation was arranged to obtain informed consent, confirm eligibility against the exclusion criteria and arrange a suitable date for testing.

All participants were instructed not to consume any caffeine, stimulant medication or alcohol on the day of testing, and female participants were informed not to wear mascara as this greatly affected and interfered with the overall quality of eyelid movements whilst wearing the Optalert glasses. On the day of testing and overnight sleep study, participants were asked to bring comfortable sleeping attire that they could change into so that the placement of electrodes would not greatly interfere with them during the set-up of the polysomnography.

Most testing sessions were held during the week (between Monday and Friday). However, arrangements were made for shift-workers to undergo testing on a given day of the weekend if there were conflicts with their regular weekly work schedules. All participants attended one testing session only and testing times were identical for each participant to

control for circadian variations in performance. Control and OSA participants were tested on a day of their convenience. Testing sessions for shift-workers were performed at least 24 hours after the end of their last night shift to allow adequate recovery time.

Participants were required to attend the Austin Health Sleep Laboratory at 12:30pm on the day of testing. Upon arrival at the laboratory, informed consent was obtained and participants were familiarised with the laboratory, testing equipment (i.e., driving simulator and PVT) and the testing schedule. The demographic questionnaire and driving information questionnaire were completed by participants as preliminary measures prior to commencing the testing protocol. The entire test battery ran for approximately four hours, after which control and shift-work participants were set up for the overnight polysomnography. Obstructive sleep apnoea participants were free to leave after administration of the test battery, after having already undergone a sleep study at an earlier date.

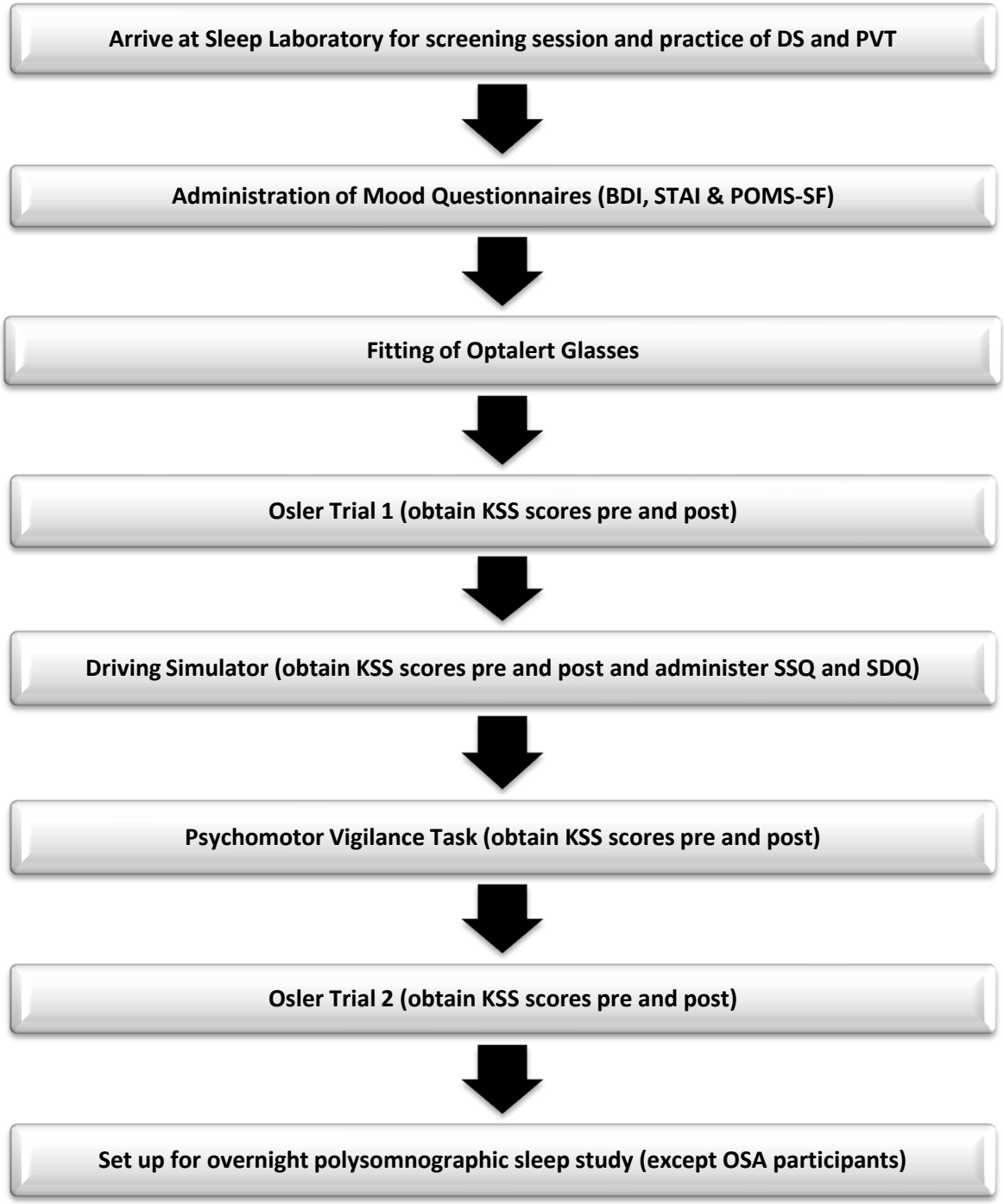

Figure 2.05: Testing Protocol for OSA, Shift Workers and Control Participants

After familiarisation with the testing equipment, participants were seated at a desk in the testing room and were requested to complete a series of mood questionnaires, consisting of the BDI, STAI and the POMS-SF. Participants were provided with instructions regarding the specific task requirements pertaining to each questionnaire. The mood questionnaires took approximately 15 to 20 minutes to complete. Participants were instructed to inform the researcher upon completion of the questionnaires.

After completion of the mood questionnaires, participants were fitted with the Optalert glasses to assess eyelid movements while completing the two Osler trials, the driving simulator task and the PVT. Adequate fitment of the glasses ensured that the IR pulses were directed up in a 30 degree beam and were centered on the lower edge of the upper eyelid. The time taken for fitment of the glasses varied for each participant and achieving eye blinks of optimal quality was dependent on the length of the eyelashes (longer eyelashes could interfere with the sensor arm), and a wider bridge of the nose (which could alter the position of the glasses).

Participants were left alone in a room to complete each of the tasks, and lights were turned off to facilitate a monotonous environment conducive to sleepiness. Prior to beginning, and at the completion of each task, participants were asked to rate their level of sleepiness according the KSS. An overhead camera was fitted in the testing room and participants could be viewed from a separate computer in the sleep laboratory across all tests. Participants were instructed to press the intercom system (situated beside them during each of the tests) upon completion of each test, and the researcher entered the room to store the data and begin set-up procedures for the next test. A five minute break at the completion of each test was permitted to avoid participants becoming fatigued.

For the administration of the two Osler trials (beginning and end of the testing protocol), participants were instructed to sit in a chair approximately four feet away from a table where the Osler was located, and were informed about the task requirements. Drowsiness was monitored using the ODMS which was initiated at the commencement of the two 40 minute Osler trials.

Participants were called back into the testing room for the commencement of a standardised 30-minute course of the driving simulator. Room lights were switched off in order to simulate a monotonous night-time drive on a series of curved and straight roads on a

dual highway resembling a rural road. A 45-centimetre screen was mounted to a table from the perspective of the participant seated in the driver's seat and looking out of the windscreen. Forward vision was limited to 'low beam' lights. A small speedometer was fixed in the upper left hand corner of the screen, and speeds ranging between 60 and 80 kilometres per hour were illuminated in green. Participants were informed to remain in the centre of the left lane while maintaining a speed range between 60 and 80km/h. The speedometer illuminated in red if participants deviated from this range.

During the task, ten random trucks emerged intermittently at a gradual pace and participants were instructed to apply the brakes firmly the moment they identified the truck in front of them. Upon participants applying the brakes firmly, the truck would disappear and participants could resume driving. No other stimulating protocols were apparent during the drive (e.g., cars, pedestrians, traffic signs), apart from regularly placed reflective markers on both edges and in the middle of the road. In this respect, the simulated driving environment was designed to be conducive to driver sleepiness. If participants' veered off to the side of the road or 'crashed', they were instructed to pull a lever at the back of the steering wheel towards them to resume driving.

Participants were instructed to sit comfortably in a chair adjacent to the computer screen located on a desk and fitted with a steering wheel, while a brake and accelerator pad was mounted below them on the floor. Prior to commencing the driving simulator task, a set of instructions appeared on the monitor and the researcher verbally reiterated these to the participant. Participants were instructed to remove their shoes and any other belongings (e.g., watch), which may have obstructed their ability to operate the steering wheel or floor mounted pedals. The ODMS was set up for the driving simulator and lights were turned off to simulate a night-time drive. After completion of the driving simulator task, participants were requested to complete the SDQ and SSQ and were permitted to have a short break.

For the administration of the ten minute PVT, participants were again seated at a computer desk where the PVT was located. The unit was programmed prior to commencing the test in order to ensure that the response buttons (right or left) corresponded to the participant's dominant hand. Participants were provided with instruction regarding the task requirements. The ODMS was set up for the PVT to monitor eyelid movement and lights were turned off while completing the task.

Participants were given a short break prior to commencing the final trial of the Osler. OSA participants were able to leave post completion of the testing protocol after having already undertaken an overnight sleep study as part of their diagnosis. All control and shift-work participants proceeded to an overnight polysomnography immediately following administration of the testing protocol. Participants were set-up by a sleep technician in the sleep laboratory and were informed that they were free to roam around the laboratory, watch television or read a book until their usual bed-time. Participants were woken at 6am the following morning, and after having removed all electrodes, they were free to leave. Two participants (one shift-worker and one control) did not undergo a sleep study due to work commitments and scheduling difficulties.

2.4. Data Analysis

2.4.1. Power Analysis. A power analysis was conducted to calculate the sample size necessary to achieve a given level of power for a One-Way Analysis of Variance (between-subjects) procedure. Based on guidelines provided by Cohen (1992) a clinically important difference of 2 with a corresponding power level of .80 was adhered to. Based on three groups (OSA, Shift Work and Control participants), with a large effect size (.50) and a power of .80, an estimated 42 participants were needed to detect an effect with alpha set at .05. For bivariate correlations (2-tailed) with a large effect size and a power level of .95, an estimated 46 participants were needed to detect an effect with alpha set at .05.

2.4.2. Statistical Analysis. Data obtained from the PVT, driving simulator, Osler and Optalert were downloaded and relevant outcome measures were transferred onto a participant score profile sheet (refer to Appendix 14). Raw data obtained from questionnaires were number coded to ensure participant confidentiality, and were scored according to their relevant scoring procedures outlined in the measures section. Questionnaire data was also transposed onto a participant score profile sheet for each participant. All outcome measures were then entered into SPSS Version 17.0 for subsequent analysis.

3. RESULTS

3.1. Preliminary Analyses

Prior to conducting the main analyses, descriptive statistics were computed for each dependent variable to check for accuracy and ensure that all data was within the specified ranges. Missing values were inspected by examining frequency tables, histograms and box plots, and appeared to be missing at random. Cases with missing values were retained wherever possible and were managed by implementing analysis-by-analysis exclusion for each statistical procedure.

Univariate outliers were identified by converting raw scores to *z* scores, and cases with standardised *z* scores above 3 or below -3 were considered to be outliers (Field, 2009). Histograms and box-plots were also examined to inspect outliers. A total of 8 outliers were identified in the entire dataset. Six cases (4 shift-work participants and 2 OSA patients) were found to have extreme scores on objective sleepiness measures (PVT % long closure, Driving Simulator Average JDS, Driving Simulator % long closure, Osler 1 % long closure, Osler 2 % long closure, and Osler total misses). One case (OSA patient) yielded an extreme score on a performance measure (driving simulator crash number), and one case (OSA patient) was found to have an extreme score on a mood measure (BDI Depression-Dejection). A procedure was implemented for the 8 univariate outliers whereby original raw scores were truncated to yield a value within the standardised range. However, all 8 cases were found to be extreme outliers with standardised residual scores of above 3 on the dependent variables of objective sleepiness, performance, and mood. After inspection of the outliers, it appeared that the values were relevant to the population studied and they were retained in the dataset for subsequent analysis. The Mahalanobis distance statistic was used to test for additional multivariate outliers. No outliers were identified, since all values were below the chi-square cut-off value for all cases (Tabachnick & Fidell, 2007).

The normality of data distribution was examined through inspection of histograms, probability plots and skewness/kurtosis statistics. However, given that the sample size was small, skewness and kurtosis of some variables was expected. The normality of data distribution was also tested by the Shapiro-Wilks statistic for small sample sizes. Where the assumption of normal distribution was met, a One-Way Analysis of Variance (ANOVA) procedure was used. Group (OSA, shift-work and control) was entered as a fixed factor and each measure was entered as a dependent variable. The assumption of homogeneity of variance was assessed with Levene's test. The F-statistic was utilised in cases where this assumption was met. Where homogeneity of variance was violated, Field (2009) suggested reporting an alternative version of the F-statistic (Welch's F ratio). Post hoc comparisons were computed for all significant ANOVA analyses using Gabriel's procedure. Field (2009) suggested that Gabriel's procedure should be utilised when sample sizes are slightly different, and offers greater power relative to other post hoc procedures. Although attempts were made to match groups as closely as possible based on age, gender, and education level, the three groups differed significantly on a number of demographic characteristics (refer to section 3.2). Effect sizes for ANOVA analyses were calculated using eta squared.

Where the assumption of normal distribution was violated, the use of non-parametric statistics was required. Furthermore, the number of participants in each of the three groups was unequal. Where analyses required the use of non-parametric statistics, the Kruskal-Wallis H test was utilised. This statistic allows for the comparison of more than two groups and was thus appropriate for the present study (Pallant, 2007). In addition, the Kruskal-Wallis H test has been suggested to have greater power than other non-parametric counterparts when sample sizes are less than 25 cases per group (Pallant, 2007) which was the case in the present study. Post hoc comparisons were computed for all significant non-parametric analyses using the Mann-Whitney U test. In order to control for type 1 error, a Bonferroni

adjustment was applied to the alpha value of .05 in order to compare each of the groups with one another. Effect sizes for analyses involving non-parametric tests were calculated by dividing the Mann Whitney U test value (*z*) with the square root of the total number of cases (Green & Salkind, 2008).

Given that a large proportion of variables were not normally distributed, bivariate correlational analyses were conducted using a non-parametric alternative (Kendall's *tau*). Field (2009) suggested that Kendall's *tau* should be used rather than Spearman's coefficient with a small sample size.

3.2. Demographic Characteristics of the Sample

Seventeen male and female OSA patients (15 males and 2 females with a mean age of 58.24 years; *SD* = 10.73), 15 male and female shift-workers (12 males and 3 females with a mean age of 45.33 years; *SD* = 7.89) and 15 control participants (6 males and 9 females with a mean age of 47.47 years; *SD* = 7.25) participated in the study.

Normally distributed variables were assessed using One-Way (between subjects) ANOVA (Table 3.01). Results revealed a statistically significant difference in age for the three groups, $F(2,44) = 9.92$, $p = .00$. Post hoc comparisons using Gabriel's procedure indicated that OSA patients were significantly older relative to shift-work and control participants. Control participants and shift-work participants did not differ significantly as a function of age. Body mass index (BMI) also differed between groups, $F(2,44) = 18.30$, $p = .00$. Specifically, OSA patients had a higher BMI compared to shift-work and control participants. Controls and shift-work participants did not differ significantly in their BMI. The groups also differed significantly according to the longest shift worked in hours, $F(2,44) = 4.29$, $p = .02$. Shift-work participants differed significantly from OSA patients in their longest shift worked in hours. OSA patients did not differ significantly from controls in terms

of the longest shift worked in hours, and shift-work participants did not differ significantly from control participants. The three groups did not differ according to height, $p = < .05$.

Kruskal-Wallis H tests for variables not normally distributed revealed a significant difference in weight for the three groups, $H(2) = 18.58$, $p = .00$. OSA patients weighed significantly more than shift-work and control participants, although shift-work and control participants did not differ significantly according to weight. The groups also differed significantly on MAPQ scores, $H(2) = 25.02$, p = .00. OSA patients endorsed higher scores on the MAPQ relative to shift-work and control participants; however, shift-work and control participants did not differ in their scores on the MAPQ. There was also a significant difference between the groups on a measure of chronic/trait sleepiness (i.e., the ESS), $H(2) = 9.83$, $p = .01$. Specifically, OSA patients reported higher ESS scores compared with control participants. Shift-work and control participants did not differ significantly in their ESS scores, and OSA patients and shift-workers did not significantly differ in ESS scores. Hours of sleep achieved per night on work days were also significantly different between the groups. Control participants achieved a greater number of hours of sleep per night on work days relative to shift-work participants and OSA patients. OSA patients did not differ from shift-workers in terms of the amount of sleep obtained per night on work days.

The three groups did not differ on the amount of days worked per week, hours worked per week, kilometres driven per year (at work and not at work), hours of sleep achieved on days off, hours driven per week (at work and not work related), glasses of alcohol (on work days and days off), beverages per day (coffee, tea and cola) and total accidents in the past three years (all p = < .05) (refer to Table 3.01).

Table 3.01

One-Way (between subjects) ANOVA tests and Kruskal-Wallis H tests for differences between demographic characteristics of OSA patients, shift-workers and control participants

	OSA patients (n = 17)		Shift workers (n = 15)		Control participants (n = 15)					
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*H*	*p*	*r*
Age	58.24	10.73	45.33	7.89	47.47	7.25	9.92	-	.00	.31
Height (m)	1.75	0.07	1.74	0.09	1.70	0.12	1.41	-	.26	.06
Weight (kg)	103.53	13.36	77.93	13.58	74.93	20.90	-	18.58	.00	.40
Body Mass Index	33.93	4.94	25.91	3.60	25.71	4.54	18.30	-	.00	.45
MAPQ Score	0.69	0.17	0.30	0.15	0.23	0.23	-	25.02	.00	.54
ESS Score	11.59	5.83	6.87	5.05	5.42	4.80	-	9.83	.01	.21
Longest shift (hours)	8.15	2.77	11.03	1.86	9.69	2.70	4.29	-	.02	.20
Days worked per week	4.70	1.09	4.77	0.94	4.69	1.49	-	0.20	.91	.00
Hrs worked per week	36.56	14.69	43.40	7.30	39.27	16.64	-	0.44	.80	.01
Kms driven/yr (at work)	11700.00	17981.78	10832.00	5297.17	18572.73	19798.19	-	2.32	.31	.05
Kms driven/year (no work)	10562.50	9858.79	11733.33	6963.85	11914.29	13900.85	-	1.10	.58	.02
Hrs of sleep (work days)	6.50	0.87	6.30	1.16	7.19	0.90	-	6.82	.03	.15
Hrs of sleep (days off)	7.24	1.19	7.43	1.76	8.03	0.77	-	5.71	.06	.12
Accident History (in 3 yrs)	0.27	0.47	0.13	0.35	0.20	0.56	-	0.01	.93	.00
Hrs driven/week (work)	11.50	10.66	9.65	11.36	4.50	4.98	-	4.77	.09	.32
Hrs driven/week (no work)	5.17	3.68	7.13	5.13	6.23	5.16	-	1.17	.56	.03
Alcohol (on work days)	0.13	0.34	0.60	0.91	0.67	1.40	-	2.44	.30	.17
Alcohol (on days off)	2.88	4.81	2.60	3.74	2.13	2.26	-	0.01	.99	.00
Coffee per day	2.65	1.69	2.33	1.76	2.07	1.98	-	0.64	.73	.06
Tea per day	1.59	1.70	1.00	1.31	0.80	1.26	-	2.69	.26	.23
Cola per day	0.53	0.94	0.93	1.44	0.64	1.28	-	0.69	.71	.00

MAPQ = Multivariate Apnoea Prediction Questionnaire; ESS = Epworth Sleepiness Scale

3.2.1. Education levels and occupation levels of the entire sample. Demographic information regarding participants' education levels and occupation levels were obtained. Figure 3.01 displays a breakdown of the education levels of the entire sample.

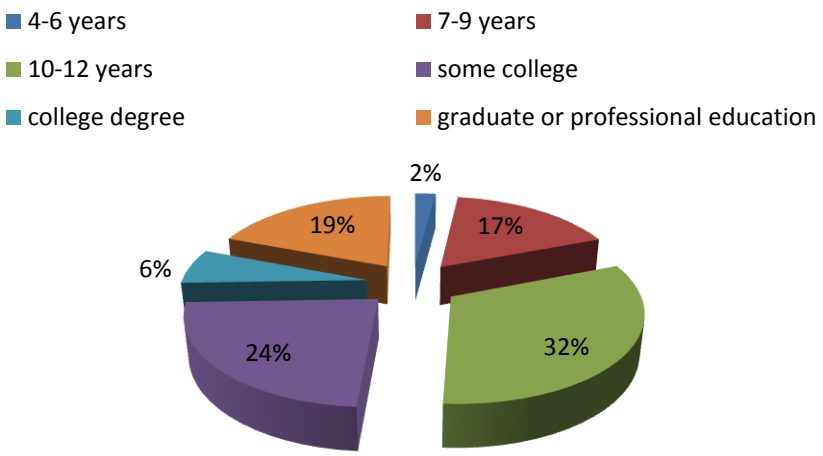

Figure 3.01: Education levels of the sample

A total of 8 participants reported completing year 7 to year 9 of secondary education, with a greater proportion (n = 15) completing years 10 to 12. Eleven participants indicated that they attended some college, and three participants reported having a college degree. Nine of the participants undertook graduate or professional education. Only one participant reported having undertaken primary education only (i.e., 4 to 6 years of education).

Figure 3.02 displays a breakdown of the occupation levels of the entire sample.

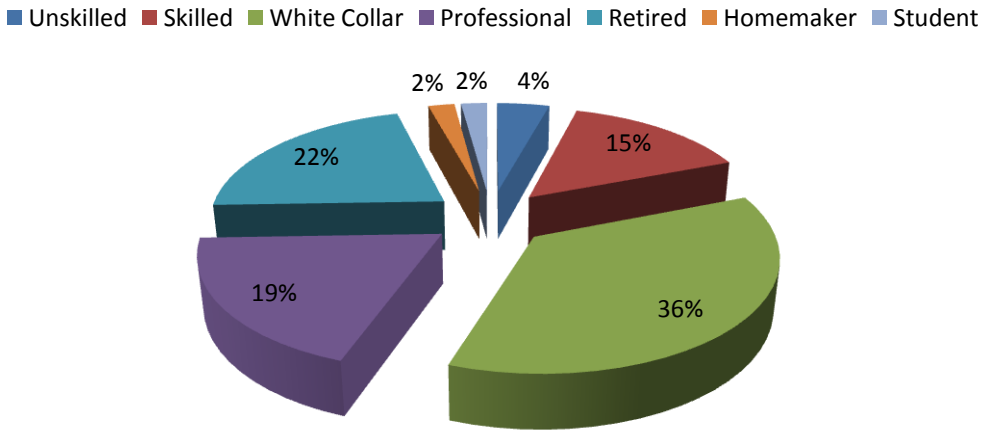

Figure 3.02: Occupation Levels of the Sample

A large percentage of the sample was employed in white collar work. Occupations included bank officers, business analysts, financial planners, information technology

specialists, production agents, and sales managers. Retired participants comprised 22% of the sample. Professional employment incorporated occupations such as registered nurses, ambulance paramedics and bank managers and comprised 19% of the sample. Fifteen percent of participants were engaged in skilled work, including occupations such as fire-fighters, computer operators, stevedores, and surveillance officers. A small percentage of the sample was engaged in unskilled work. Homemakers and students collectively comprised 4% of the entire sample.

3.3. Group Differences

3.3.1. Subjective sleepiness. Table 3.02 presents Kruskal-Wallis H tests for differences between subjective sleepiness variables (i.e., state sleepiness) for OSA patients, shift-work participants and control participants. There were no significant differences between the groups for mean KSS scores, the Stop Driving Questionnaire (suburban or long distance items) or the Sleepiness Symptoms Questionnaire total score.

Table 3.02

Kruskal-Wallis H tests for differences between subjective sleepiness variables of OSA patients, shift-workers and control participants

	OSA patients (n = 17)		Shift workers (n = 15)		Control participants (n = 15)				
	M	*SD*	*M*	*SD*	*M*	*SD*	*H*	*p*	*r*
KSS Mean	4.89	1.64	5.72	1.51	4.64	1.16	5.15	.08	.11
SDQ Suburban	1.00	0.00	1.27	0.59	1.33	0.72	3.80	.15	.08
SDQ Long Distance	1.82	1.01	1.93	1.03	1.67	0.82	0.42	.81	.01
SSQ Total Score	17.94	5.88	22.33	8.39	19.53	6.58	2.91	.23	.06

KSS = Karolinska Sleepiness Scale, SDQ = Stop Driving Questionnaire, SSQ = Sleepiness Symptoms Questionnaire

3.3.2. Objective sleepiness (Optalert). Kruskal-Wallis H tests comparing Optalert measures for the PVT, Driving Simulator and Osler trials are displayed in table 3.03 and revealed that shift-work participants had a greater amount of time that the eyelids remained closed per minute on the Osler 1 trial relative to OSA patients, $H(2) = 7.29$, $p = .03$. OSA patients did not differ significantly from control participants, and shift-work participants did

not differ from control participants. No other significant differences were found for the Optalert between the groups.

Table 3.03

Kruskal-Wallis H tests for differences between objective sleepiness measures (Optalert) of OSA patients, shift-workers and control participants

	OSA patients (n = 17)		Shift workers (n = 15)		Control participants (n = 15)				
	M	*SD*	*M*	*SD*	*M*	*SD*	*H*	*p*	*r*
PVT Average JDS *	2.09	1.72	1.93	1.68	1.22	0.92	1.41	.49	.03
PVT % Long Closure **	0.01	0.04	0.07	0.14	0.003	0.006	0.32	.85	.01
Driving Simulator Average JDS ***	1.48	1.08	1.64	1.88	0.63	0.62	3.96	.14	.09
Driving Simulator % Long Closure ****	0.02	0.04	0.14	0.49	0.02	0.04	0.56	.76	.01
Osler 1 Average JDS *****	2.23	1.18	3.47	2.72	2.23	1.97	2.24	.33	.05
Osler 1 % Long Closure ^	0.02	0.04	0.69	0.93	0.12	0.39	7.29	.03	.16
Osler 2 Average JDS ^^	2.83	1.71	3.12	2.29	2.49	2.02	0.47	.79	.01
Osler 2 % Long Closure #	0.30	0.75	0.29	0.63	0.27	0.57	2.85	.24	.06

PVT – Psychomotor Vigilance Task, JDS = Johns Drowsiness Scale

* = OSA n = 16, controls n = 14; ** OSA n = 16, *** OSA n = 14, shift workers n = 13, controls n = 11, **** OSA n = 15, *****OSA n = 14, SW n = 13, ^ OSA n = 14, ^^ OSA n = 14, SW n = 14, control n = 13, # OSA n = 16, controls n = 14.

3.3.3. Objective sleepiness (Osler). Table 3.04 displays Kruskal-Wallis H tests for differences between Osler variables (objective sleepiness) for OSA patients, shift-workers and control participants. There were no significant differences between the three groups on measures of objective sleepiness (average sleep latency and Osler total misses) assessed by the Osler.

Table 3.04

Kruskal-Wallis H tests for differences between objective sleepiness measures (Osler) of OSA patients, shift-workers and control participants

	OSA patients (n = 17)		Shift workers (n = 14)		Control participants (n = 15)				
	M	*SD*	*M*	*SD*	*M*	*SD*	*H*	*p*	*r*
Average Sleep Latency	18.17	5.16	19.50	1.94	20.00	0.00	1.88	.39	.04
Total Osler Misses	6.40	8.64	3.71	4.87	3.60	4.45	1.42	.49	.03

3.3.4. Objective sleepiness (polysomnography). Table 3.05 displays differences between OSA patients, shift-workers and control participants on polysomnographic

measures. A One-Way ANOVA revealed a statistically significant difference in the number of apnoea/hypopnoea events experienced per hour of sleep between the three groups $F(2,41) = 23.77$, $p = .02$. A large effect size of $r = .61$ was found. Post hoc comparisons using Gabriel's procedure indicated that OSA patients experienced a significantly greater number of apnoea and hypopnoea events per hour of sleep relative to shift-workers and control subjects. No significant differences were demonstrated between shift-workers and control participants. Additionally, the lowest level that oxygen saturation dropped to overnight ($MinSp0_2$) differed significantly between the groups $F(2,38) = 4.31$, $p = .03$. A post hoc procedure revealed that shift-workers experienced lower oxygen saturation levels during the night compared with OSA patients and control participants. No significant differences were found between shift-workers and control participants. The amount of time spent in REM sleep did not differ significantly between the groups.

Kruskal-Wallis H tests revealed a significant difference in the total number of arousals experienced per hour of sleep between the three groups, $H(2) = 9.81$, $p = .01$. Specifically, OSA patients experienced a greater number of arousals per hour of sleep compared to control and shift-work participants. Shift-workers did not differ from control participants in the total number of arousals experienced per hour of sleep. Sleep efficiency also differed between the groups, with OSA patients reporting decreased sleep efficiency relative to shift-workers. There were no significant differences between OSA patients and control participants, and between shift-work and control participants on this measure of objective sleepiness.

Table 3.05

One Way (between subjects) ANOVA tests and Kruskal-Wallis H tests for differences between objective sleepiness measures (polysomnography) of OSA patients, shift-workers and control participants

	OSA patients (n = 17)		Shift workers (n = 15)		Control participants (n = 15)					
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*H*	*p*	*r*
AHI events per hour *	46.22	20.27	13.18	9.51	9.99	6.20	23.77	-	.00	.61
Minimum $Sp0_2$ **	81.76	7.71	87.92	3.60	87.58	4.06	4.31	-	.03	.22
Arousals total per hour ***	30.16	23.35	11.17	4.58	11.52	4.62	-	9.81	.01	.21
Sleep Efficiency (%) ^	68.75	19.93	92.87	36.61	78.57	16.36	-	7.93	.02	.17
Time in REM sleep #	53.52	36.29	81.50	29.53	82.61	35.59	3.49	-	.06	.15

AHI = Apnoea-Hypopnoea Index, Minimum $Sp0_2$ = percentage of total sleep time where oxygen saturation is <90%

* = shift workers n = 13, controls n = 14; ** shift workers n = 12, controls n = 12; *** OSA n = 16, shift workers n = 12, controls n = 13, ^ shift workers n = 13, controls n = 14, # shift workers n = 11, controls n = 14.

3.3.5. Performance measures (PVT). Table 3.06 presents differences between OSA patients, shift-workers and control participants on measures of psychomotor vigilance. A One-Way ANOVA revealed a statistically significant difference between the groups in median reaction time on the PVT, $F(2,42) = 5.19$, $p = .01$. Post hoc tests revealed that OSA patients had a higher median reaction time compared to shift-workers and control participants. No significant differences were found between shift-workers and control participants. In addition, an increase in the duration of responses as measured by the slowest 10% on the PVT differed between the groups, $F(2,42) = 4.29$, $p = .02$. Specifically, OSA patients had an increase in the duration of responses as measured by the slowest 10% compared with shift-workers and control participants. There were no significant differences found between shift-workers and control participants. A Kruskal-Wallis H test revealed no significant differences between the groups for the number of lapses on the PVT.

Table 3.06

One Way (Between Subjects) Analysis of Variance (ANOVA) tests and Kruskal-Wallis H tests for differences between psychomotor vigilance task measures of OSA patients, shift-workers and control participants

	OSA patients (n = 16)		Shift-workers (n = 15)		Control participants (n = 14)					
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*H*	*p*	*r*
PVT Median RT *	254.06	53.07	208.47	22.80	226.29	38.66	5.19	-	.013	.19
PVT Slowest 10% **	2.19	0.70	2.85	0.52	2.59	0.67	4.29	-	.020	.17
PVT Lapses ***	6.94	6.91	2.60	2.10	3.93	3.87	-	4.56	.102	.10

PVT = Psychomotor Vigilance Task

3.3.6. Performance measures (Driving Simulator). Kruskal-Wallis H tests comparing OSA patients, shift-workers and control participants performance on the driving simulator task revealed a statistically significant difference between the groups on the mean variation in speed from the 70km per hour speed range, $H(2) = 8.11$, $p = .02$ and the mean variation in speed from the optimum 60 to 80km speed range. Specifically, OSA patients had a greater mean variation in speed from the 70km per hour speed range when compared with shift-workers. Control subjects had a greater mean variation in speed from the 70km per hour speed range compared with shift-workers. There was no significant difference between OSA patients and control participants. Mean variation in lane position during the drive, mean reaction time for braking episodes, and crash incidence were not significantly different between the three groups.

Table 3.07

Kruskal-Wallis H tests for differences between driving simulator measures of OSA patients, shift-workers and control participants

	OSA patients (n = 16)		Shift-workers (n = 15)		Control participants (n = 15)				
	M	*SD*	*M*	*SD*	*M*	*SD*	*H*	*p*	*r*
Position Median Average	75.62	29.66	58.36	33.26	64.59	32.06	4.14	.13	.09
Speed 70 Average	10.10	5.72	6.03	2.41	8.48	3.87	8.11	.02	.18
Speed 60-80 Average	4.53	4.25	1.40	1.51	3.34	3.26	7.48	.02	.17
RT Median	1258.62	403.11	1254.66	466.99	1360.28	346.52	1.53	.47	.03
Crash Number	1.94	3.21	0.40	1.06	0.27	0.46	5.04	.08	.11

3.3.7. Mood measures. Normally distributed variables assessed using One-Way ANOVA revealed a statistically significant difference between OSA patients, shift-workers and control participants on the POMS Vigour scale, $F(2,43) = 4.77$, $p = .01$. Post hoc comparisons using Gabriel's procedure indicated that control subjects scored significantly higher on POMS Vigour relative to shift-workers and OSA patients. There were no significant differences between OSA patients and shift-workers. State Anxiety on the STAI and the POMS Confusion-Bewilderment scale did not differ between the three groups.

Kruskal-Wallis H tests for variables that were not normally distributed revealed a statistically significant difference between groups on the total BDI score, $H(2) = 6.60$, $p = .04$. Specifically, OSA patients endorsed higher scores on the BDI relative to control subjects. There were no significant differences between OSA patients and shift-workers, and between shift-workers and control subjects. A significant difference was also found between groups on the total POMS score, $H(2) = 10.18$, $p = .01$. OSA patients endorsed a higher total score on the POMS compared to control subjects. In addition, shift-workers endorsed a higher total score on the POMS relative to control subjects. There was no significant difference between OSA patients and shift-workers. The Trait Anxiety scale of the STAI, POMS Tension-Anxiety, POMS Depression-Dejection, POMS Anger-Hostility and POMS Fatigue scores did not differ among the three groups.

Table 3.08

One Way (Between Subjects) ANOVA tests and Kruskal-Wallis H tests for differences between mood measures (BDI, STAI and POMS) of OSA patients, shift-workers and control participants

	OSA patients (n = 17)		Shift-workers (n = 15)		Control participants (n = 14)					
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*H*	*p*	*r*
BDI Total Score	11.65	5.40	9.53	6.38	6.43	4.75	-	6.60	.037	.15
STAI State Anxiety	35.18	10.07	31.27	7.69	28.64	7.49	2.28	-	.115	.10
STAI Trait Anxiety	35.47	11.06	34.27	7.66	31.86	9.42	-	0.85	.655	.02
POMS Tension-Anxiety	5.41	4.70	7.47	6.55	3.14	2.96	-	5.63	.060	.13
POMS Depression-Dejection	4.29	4.90	3.47	3.54	1.79	2.48	-	3.59	.166	.08
POMS Confusion-Bewilderment	3.47	2.90	4.13	3.02	1.79	1.76	3.01	-	.060	.12
POMS Vigour	8.35	3.46	8.47	4.56	12.64	4.85	4.77	-	.013	.18
POMS Anger-Hostility	4.24	4.31	6.20	5.60	1.71	2.13	-	5.26	.072	.12
POMS Fatigue	7.94	4.26	8.13	5.17	5.00	3.68	-	5.64	.060	.13
POMS Total Score	17.00	17.81	20.93	22.14	0.79	14.16	-	10.18	.006	.23

BDI = Beck Depression Inventory, STAI = State-Trait Anxiety Inventory, POMS = Profile of Mood States

3.4. Correlational Analyses between variables

3.4.1. Subjective sleepiness and objective sleepiness. Table 3.09 presents correlations (Kendall's *tau*) between subjective and objective sleepiness measures for OSA patients, shift-workers and control participants. Patients with OSA demonstrated a significant negative correlation between average sleep latency scores and the SDQ (suburban item). Shift-work participants demonstrated a significant positive correlation between KSS scores and the Average JDS on the PVT and the first Osler trial, and the percentage of time that the eyes remained closed on the PVT and the driving simulator. The SDQ (long distance item) was positively correlated with the percentage of time that the eyes remained closed per minute on the PVT. Positive correlations were also found for scores on the SSQ and the Average JDS on the Osler 1 and 2 trials. SSQ total scores were also significantly negatively correlated with the total number of misses on the Osler (trials 1 and 2).

For polysomnography measures, only shift-workers demonstrated a significant negative correlation between the number of AHI events per hour and the total number of arousals during a night's sleep and the SSQ total score. There were no significant

relationships between subjective sleepiness measures and objective measures among control participants.

Table 3.09

Correlations (Kendall's tau) between subjective sleepiness measures and objective sleepiness measures for OSA patients, shift-workers and control participants

OSA	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
PVT Average JDS	-.37	-.16	-.05	-.05	-.01
PVT % LC	.26	.15	.18	.18	.00
DS Average JDS	-.23	-.37	-.36	-.36	-.17
DS % LC	-.01	.10	-.15	-.15	.28
Osler 1 Average JDS	-.07	.21	-.13	-.13	.17
Osler 1 % LC	.28	.19	-.05	-.05	.26
Osler 2 Average JDS	-.10	-.16	.11	.11	-.23
Osler 2 % LC	-.12	-.09	.16	.16	-.17
Sleep Latency	.36	.13	.30	.30	.28
Osler Total Misses	-.20	.06	-.05	-.05	-.14
AHI Events/hour	-.05	-.21	-.13	-.13	-.36
$MinSp0_2$	-.19	.08	-.02	-.02	.08
Arousals Total	.11	-.09	-.15	-.15	-.29
Sleep Efficiency	.19	-.07	.12	.12	-.02
Time in REM	.13	.01	.12	.12	.08

SW	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
PVT Average JDS	.04	**.39***	-.05	.11	.27
PVT % LC	-.13	**.61****	.12	**.49***	.38
DS Average JDS	-.21	.37	.07	.30	.22
DS % LC	-.05	**.52***	.23	.26	.33
Osler 1 Average JDS	-.01	**.43***	.26	.20	**.43***
Osler 1 % LC	-.13	.38	-.03	.26	.16
Osler 2 Average JDS	.09	.27	.05	.22	**.40***
Osler 2 % LC	-.09	.11	.10	.19	.25
Sleep Latency	.27	-.26	.13	-.28	-.37
Osler Total Misses	-.12	-.36	-.15	-.17	**-.47***
AHI Events/hour	-.33	-.37	-.28	-.04	**-.49***
$MinSp0_2$	.32	-.21	.09	-.27	.11
Arousals Total	-.27	-.32	-.21	.02	**-.52***
Sleep Efficiency	.17	.37	.20	.13	.34
Time in REM	-.19	.29	-.03	-.02	.28

CONTROL	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
PVT Average JDS	-.07	.07	-.13	.07	.02
PVT % LC	.07	.15	.05	.05	.07
DS Average JDS	-.13	-.28	.22	-.24	-.32
DS % LC	-.40	-.31	.16	.21	.06
Osler 1 Average JDS	.23	-.16	.22	.01	.10
Osler 1 % LC	.01	.05	.43	.32	.36
Osler 2 Average JDS	.26	.04	.12	-.19	.00
Osler 2 % LC	.31	.08	-.04	-.26	.09
Sleep Latency	-.32	.11	-.34	-.39	-.35
Osler Total Misses	.00	.07	.17	-.21	-.19
AHI Events/hour	.25	-.11	.37	.10	.16
Min$Sp0_2$	-.29	.09	-.23	-.06	-.18
Arousals Total	.34	.20	.08	-.06	-.01
Sleep Efficiency	-.09	.18	.23	.24	.09
Time in REM	.11	.24	.27	.18	.14

ESS = Epworth Sleepiness Scale; KSS = Karolinska Sleepiness Scale; SDQ = Stop Driving Questionnaire; SSQ = Sleepiness Symptoms Questionnaire; PVT = Psychomotor Vigilance Task; DS = Driving Simulator; JDS = Johns Drowsiness Scale; % LC = Percent Long Closure; AHI = Apnoea-Hypopnoea Index, Minimum $Sp0_2$ = percentage of total sleep time where oxygen saturation is <90%

NB: N's for each of the groups differed between cells

** Significant at .05 level (2-tailed)

3.4.2. Subjective sleepiness and performance. Table 3.10 displays correlations (Kendall's *tau*) between subjective sleepiness measures and performance variables for the three groups. For OSA patients, there was a significant negative correlation between scores on the ESS and median reaction time on the driving simulator. For shift-workers, a significant negative correlation was found between total scores on the SSQ and the median reaction time on the PVT. Significant positive correlations were also observed between the position median average of the driving simulator and the KSS, SDQ (suburban item) and the SSQ total score, with a large effect size found for SSQ total score. For control subjects, only a significant positive relationship was found for ESS scores and the average 70km per hour speed range on the driving simulator. All other variables were not associated.

Table 3.10

Correlations (Kendall's tau) between subjective sleepiness measures and performance measures for OSA patients, shift-workers and control participants

OSA (n=16)	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
PVT Median RT	-.11	.00	.14	.14	-.15
PVT Slowest 10%	.19	-.03	.07	.07	.00
PVT Lapses	-.13	.11	-.01	-.01	.04
DS Position Median Average	-.25	-.08	-.06	-.06	.12
DS Speed 70 Average	-.07	.03	.12	.12	.05
DS Speed 60-80 Average	-.02	.09	.15	.15	.14
DS RT Median	**-.44***	-.22	-.25	-.25	-.24
DS Crash Number	-.11	-.12	-.07	-.07	.05

SW (n=15)	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
PVT Median RT	.23	-.31	-.26	-.14	**-.48***
PVT Slowest 10%	.13	.16	-.10	.05	.22
PVT Lapses	-.23	.14	.07	.19	-.09
DS Position Median Average	.21	**.38***	**.44***	.28	**.59****
DS Speed 70 Average	.09	-.02	.29	.12	.20
DS Speed 60-80 Average	.15	.02	.16	.17	.18
DS RT Median	-.11	.02	.29	.35	.09
DS Crash Number	-.41	.16	-.24	-.09	.26

CONTROL (n=14)	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
PVT Median RT	.23	.13	.38	.13	-.09
PVT Slowest 10%	-.06	-.08	-.15	-.32	-.08
PVT Lapses	-.07	.10	.22	.36	.04
DS Position Median Average	.21	-.06	.06	.10	.31
DS Speed 70 Average	**.48***	.04	.06	.00	.33
DS Speed 60-80 Average	.38	-.06	-.03	.02	.37
DS RT Median	-.21	.04	.16	.21	.02
DS Crash Number	-.05	-.41	.49	.40	.18

ESS = Epworth Sleepiness Scale; KSS = Karolinska Sleepiness Scale; SDQ = Stop Driving Questionnaire; SSQ = Sleepiness Symptoms Questionnaire; PVT = Psychomotor Vigilance Task; DS = Driving Simulator; * = significant at .05 level (2-tailed); RT = Reaction Time

** Significant at .05 level (2-tailed)

3.4.3. Subjective sleepiness and mood. Table 3.11 presents correlations (Kendall's *tau*) between subjective sleepiness measures and mood variables for each of the three groups. A significant positive correlation was demonstrated between mean scores on the KSS and the confusion-bewilderment scale of the POMS for OSA patients. The POMS Vigour scale was

significantly negatively correlated with the ESS, the KSS mean score and the SDQ (suburban and long distance items) for OSA patients.

Among shift-workers, there was a significant positive correlation between mean scores on the KSS and the total score on the BDI. The STAI – Trait Anxiety was also correlated with the KSS mean score.

For control participants, significant positive correlations were found between the ESS and BDI total, the POMS Confusion-Bewilderment scale, and the POMS total score. The ESS was positively correlated with the POMS Tension-Anxiety at the .01 level. The KSS mean score was significantly negatively correlated with the STAI- State Anxiety and Trait Anxiety scales and the POMS total score. A significant negative correlation was also found for the KSS mean score and the POMS Vigour scale.

Table 3.11

Correlations (Kendall's tau) between subjective sleepiness measures and mood measures for OSA patients, shift-workers and control participants

OSA (n=17)	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
BDI Total	.21	.25	.25	.25	.07
STAI – State Anxiety	.08	.27	.22	.22	.23
STAI – Trait Anxiety	.04	.25	.16	.16	.11
POMS Tension-Anxiety	.06	.29	.15	.15	.18
POMS Depression-Dejection	-.05	-.03	.10	.10	.02
POMS Confusion-Bewilderment	.14	**.37***	.31	.31	.13
POMS Anger-Hostility	-.20	-.03	-.08	-.08	.13
POMS Vigour	**.39***	**-.52****	**-.52***	**-.52***	-.36
POMS Fatigue	.27	.17	.32	.32	.02
POMS Total Score	.18	.27	.33	.33	.11

SW (n=15)	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
BDI Total	-.03	**.42***	.02	.21	.29
STAI – State Anxiety	-.01	.08	-.07	-.02	.22
STAI – Trait Anxiety	.01	**.43***	.08	.32	.34
POMS Tension-Anxiety	-.13	.22	-.17	.13	.17
POMS Depression-Dejection	.12	.36	.02	.24	.31
POMS Confusion-Bewilderment	-.14	.21	-.05	.32	.15
POMS Anger-Hostility	-.04	.36	-.15	.26	.27
POMS Vigour	-.28	-.10	.15	.32	-.12
POMS Fatigue	.04	.23	-.21	.08	.25
POMS Total Score	.02	.26	-.18	.14	.23

CONTROL (n=14)	ESS	KSS	SDQ Part 1	SDQ Part 2	SSQ Total
BDI Total	**.47***	.33	-.28	-.15	.21
STAI – State Anxiety	.31	**.43***	-.11	-.07	.15
STAI – Trait Anxiety	.37	**.44***	-.09	.15	.37
POMS Tension-Anxiety	**.57****	.40	-.09	-.20	.02
POMS Depression-Dejection	.24	.22	-.30	-.34	-.16
POMS Confusion-Bewilderment	**.43***	.08	.12	.06	.12
POMS Anger-Hostility	.19	-.12	-.02	-.42	-.35
POMS Vigour	-.28	**-.47***	.13	.01	-.07
POMS Fatigue	.38	.18	.02	.18	.27
POMS Total Score	**.50***	**.45***	-.04	-.03	.10

ESS = Epworth Sleepiness Scale; KSS = Karolinska Sleepiness Scale; SDQ = Stop Driving Questionnaire; SSQ = Sleepiness Symptoms Questionnaire; BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; POMS = Profile of Mood States

* Significant at .05 level (2-tailed); ** Significant at .01 level (2-tailed)

3.4.4. Objective sleepiness and performance. Table 3.12 displays correlations (Kendall's *tau*) between objective sleepiness measures and performance variables for OSA patients, shift-workers and control participants. For OSA patients, a significant positive correlation was found between the average median position on the driving simulator and the percentage of time that the eyes remained closed on the Osler 2. There was also a significant positive association between median reaction time on the driving simulator and the average JDS on the PVT. Median reaction time on the driving simulator was also positively correlated with the percentage of time that the eyes remained closed per minute on the Osler 2. A significant negative correlation was found between median reaction time on the driving simulator and average sleep latency. For polysomnography measures, there were significant

negative correlations between the amount of time spent in REM sleep and Speed 70 and 60-80km/hour averages on the driving simulator.

For shift-workers, there was a significant negative correlation between the median reaction time on the PVT and the Average JDS of the Osler 1. Median reaction time on the PVT was also positively correlated with the total number of misses on the Osler at the .01 level. The slowest 10% of reaction times on the PVT was positively correlated with the average JDS scores on the PVT. There was also a significant negative correlation between the number of crashes on the driving simulator and average sleep latency. For polysomnography measures, there were positive correlations found between the median reaction time on the PVT and the number of AHI events per hour, as well as the total number of arousals per night. There was also a significant negative association between the number of lapses on the PVT and the lowest level that oxygen saturation dropped to overnight.

For control participants, a positive correlation was found between the slowest 10% of reaction times on the PVT and the average JDS on the driving simulator. There was also a positive relationship between the 60-80km/hour speed range average on the driving simulator and the average JDS on the Osler 2. For polysomnography variables, there was a significant negative relationship between the median reaction time on the PVT and the number of AHI events per hour. The average median position on the driving simulator was also positively correlated with the number of AHI events per hour at the .01 level. In addition, both the 70km and 60 to 80km per hour speed range averages on the driving simulator were positively correlated with the number of AHI events per hour.

The lowest level that oxygen saturation dropped to overnight was negatively correlated with the average median position on the driving simulator. In addition, both the 70km/hour and the 60 to 80km/hour speed range averages on the driving simulator were

negatively correlated with the lowest level that oxygen saturation dropped to overnight at the .01 level (2-tailed).

Table 3.12

Correlations (Kendall's tau) between objective sleepiness measures and performance measures for OSA patients, shift-workers and control participants

OSA	PVT Median RT	PVT Slowest10%	PVT Lapses	DS Pos Median Av	DS Sp 70 Av	DS Sp 60-80 Av	DS RT Median	DS Crash Number
PVT Av JDS	.20	-.35	.35	.25	.12	.08	**.39***	.13
PVT % LC	-.07	-.01	.05	-.23	-.13	-.13	-.18	-.24
DS Av JDS	-.06	.09	-.20	.04	.09	.02	-.02	.06
DS % LC	-.23	.11	-.06	-.16	.05	.05	-.18	.01
Osler 1 Av JDS	-.13	-.01	.12	-.02	.02	.07	.20	.17
Osler 1 % LC	-.25	.08	-.13	-.05	-.32	-.29	-.16	-.14
Osler 2 Av JDS	.24	-.25	.26	.10	.10	.08	.21	.03
Osler 2 % LC	.31	-.24	.21	**.47***	.10	.08	**.41***	.31
Sleep Latency	-.21	.21	-.22	-.09	-.05	.02	**-.42***	-.04
Osler Misses	.24	-.30	.27	.23	-.03	-.01	.20	.06
AHI Events/hr	.15	.03	-.04	-.13	.08	.06	.14	.21
$MinSp0_2$	.05	-.26	.25	.19	.02	.03	.01	-.03
Arousals total	.15	.01	-.06	-.03	.16	.16	.24	.28
Sleep Efficiency	-.08	.20	-.28	-.05	-.23	-.28	-.28	-.27
Time in REM	-.31	.27	-.35	-.18	**-.40***	**-.42***	-.35	-.37

SW	PVT Median RT	PVT Slowest10%	PVT Lapses	DS Pos Median Av	DS Sp 70 Av	DS Sp 60-80 Av	DS RT Median	DS Crash Number
PVT Av JDS	-.19	**.48***	-.11	.00	-.17	.33	.02	.11
PVT % LC	.13	.01	.12	.08	.00	.19	-.08	.14
DS Av JDS	-.28	.08	.28	-.03	-.15	-.10	-.03	.08
DS % LC	-.20	.10	.33	.13	-.22	-.13	.22	-.06
Osler 1 Av JDS	**-.43***	.21	-.11	.21	.10	.13	-.19	.16
Osler 1 % LC	-.03	.05	.32	.07	.03	.03	.15	.27
Osler 2 Av JDS	-.34	.30	-.32	.32	.01	-.06	.21	.16
Osler 2 % LC	-.17	-.02	-.09	.36	.33	.17	.12	.38
Sleep Latency	-.21	.08	.00	-.37	-.37	-.37	-.21	**-.61***
Osler Misses	**.64****	-.37	-.04	-.37	-.01	-.14	-.01	.05
AHI Events/hr	**.47***	-.32	.14	.23	-.13	-.18	.08	-.07
$MinSp0_2$	-.27	.21	**-.59***	.06	.19	.06	-.13	.00
Arousals total	**.55***	-.35	.25	-.15	-.06	-.03	.24	-.03
Sleep Efficiency	-.23	.27	-.17	.03	.08	.18	-.33	-.02
Time in REM	-.33	.44	-.41	.02	.09	.27	-.27	.09

CONTROL	PVT Median RT	PVT Slowest10%	PVT Lapses	DS Pos Median Av	DS Sp 70 Av	DS Sp 60-80 Av	DS RT Median	DS Crash Number
PVT Av JDS	-.22	.00	-.03	.24	-.11	.13	.07	-.06
PVT % LC	-.14	.19	-.23	.13	.01	.10	-.13	-.07
DS Av JDS	-.07	**.54***	-.46	.04	-.07	-.04	-.26	.39
DS % LC	-.21	.14	-.09	-.06	-.17	-.08	.19	.30
Osler 1 Av JDS	.17	-.31	.30	.24	.11	.28	.26	.25
Osler 1 % LC	-.09	.16	-.10	-.06	-.01	-.04	.06	.00
Osler 2 Av JDS	.15	-.09	.09	.33	.33	**.44***	.15	-.08
Osler 2 % LC	-.16	.22	.25	.12	.23	.25	-.01	-.18
Sleep Latency	-.38	.32	-.33	-.37	-.21	-.31	-.10	-.44
Osler Misses	-.12	.16	-.12	-.02	-.06	-.10	.10	-.14
AHI Events/hr	**.47***	-.36	.36	**.54****	**.47***	**.52***	.14	.43
MinSp0$_2$	-.27	-.13	-.06	**-.47***	**-.63****	**-.63****	-.03	-.40
Arousals total	.38	-.09	-.02	.10	.23	.23	.00	.15
Sleep Efficiency	.00	.23	-.15	-.08	-.14	-.14	-.17	.03
Time in REM	-.03	.21	-.23	-.19	-.12	-.08	-.23	-.07

PVT = Psychomotor Vigilance Task; DS = Driving Simulator; JDS = Johns Drowsiness Scale; % LC = Percent Long Closure; RT = Reaction Time; AHI = Apnoea-Hypopnoea Index, Minimum Sp0$_2$ = percentage of total sleep time where oxygen saturation is <90%

* Significant at .05 level (2-tailed); ** Significant at .01 level (2-tailed)

NB: N's for each of the groups differed between cells

3.4.5. Objective sleepiness and mood. Table 3.13 presents correlations (Kendall's *tau*) between objective sleepiness measures and mood variables for the three groups. A significant positive correlation was found between the total number of misses on the Osler and OSA patients total BDI scores, and the Confusion-Bewilderment scale of the POMS. The percentage of time that the eyes remained closed per minute on the PVT was negatively correlated with OSA patients' scores on the Tension-Anxiety and Depression-Dejection scale of the POMS. There was also a positive correlation between the average JDS score on the driving simulator and OSA patients' scores on the POMS Vigour scale. For polysomnography variables, there was a significant negative correlation between the amount of time spent in REM sleep and OSA patients' scores on the POMS fatigue scale.

Among shift-workers', there was a significant positive relationship between BDI total scores and the percentage of time that the eyes remained closed per minute on the PVT. A high effect size was found for this correlation. A significant positive correlation was also observed between the percentage of time the eyes remained closed per minute on the PVT

and scores on the STAI – Trait Anxiety, the POMS Anger-Hostility scale and the POMS Confusion-Bewilderment scale. A significant positive correlation was found for the percentage of time that the eyes remained closed per minute during the Osler 2 and the STAI – State Anxiety and Trait Anxiety scales. There was also a positive correlation found for the Osler 2 Average JDS and the POMS Fatigue scale. Positive correlations were found for the percentage of time that the eyes remained closed on the Osler 2 and the POMS scales of Tension-Anxiety, Depression-Dejection, and Confusion-Bewilderment. For polysomnography variables, a positive association was demonstrated between sleep efficiency and the STAI – State Anxiety scale. In addition, time spent in REM sleep was positively correlated with shift-workers' total scores on the BDI.

For control subjects', a significant negative correlation was demonstrated between the percentage of time that eyes remained closed per minute on the driving simulator and scores on the STAI – State Anxiety scale. A large effect size was observed for this correlation. The percentage of time that the eyes remained closed per minute on the driving simulator was also negatively correlated with the Vigour scale of the POMS. Average JDS scores on the PVT were negatively correlated with the POMS Confusion-Bewilderment scale. Average JDS scores on the Osler 1 were positively correlated with the POMS Vigour scale. For polysomnography variables, only time spent in REM sleep was positively correlated with STAI – Trait Anxiety scores.

Table 3.13

Correlations (Kendall's tau) between objective sleepiness measures and mood measures for OSA patients, shift-workers and control participants

OSA	BDI Total	STAI – S	STAI – T	POMS T-A	POMS D-D	POMS C-B	POMS A-H	POMS Vigour	POMS Fatigue	POMS Total
PVT Av JDS	-.21	.25	-.03	-.29	-.22	-.12	.12	.18	-.07	-.19
PVT % LC	-.21	-.38	-.39	**-.42***	**-.42***	-.18	-.38	-.14	-.07	-.26
DS Av JDS	-.18	-.27	-.25	.02	.07	-.20	.05	**.47***	.09	-.13
DS % LC	.16	-.06	-.08	.14	-.02	.15	.09	-.08	.25	.19
Osler 1 Av JDS	.17	.12	.15	.11	.00	.06	.14	-.27	.02	.08
Osler 1 % LC	.24	.04	-.23	.11	-.06	-.03	-.04	-.17	-.24	.04
Osler 2 Av JDS	.00	-.19	-.03	-.25	-.13	-.02	.01	.22	.15	-.11
Osler 2 % LC	.18	.15	.05	.15	.00	.16	.24	.04	-.07	.09
Sleep Latency	.24	.08	.02	.10	.21	.13	.03	-.33	.24	.23
Osler Total Misses	**.39***	.28	.22	.21	.18	**.39***	-.24	-.08	.01	.26
AHI Events/hour	.06	-.11	-.13	-.06	-.05	-.05	-.23	.19	.18	-.09
$MinSp0_2$	.32	.13	-.01	.21	.22	.32	.33	-.13	.07	.25
Arousals total	.06	-.25	-.19	-.04	-.07	-.11	-.24	.25	.19	-.01
Sleep Efficiency	.02	-.04	.00	-.01	.06	.03	-.01	.11	-.14	.02
Time in REM	-.18	.02	-.13	-.02	-.05	-.19	.01	.05	**-.42***	-.16

SW	BDI Total	STAI – S	STAI – T	POMS T-A	POMS D-D	POMS C-B	POMS A-H	POMS Vigour	POMS Fatigue	POMS Total
PVT Av JDS	.18	.04	.23	.16	.22	-.03	.37	-.21	.14	.18
PVT % LC	**.58****	.20	**.54***	.42	.43	**.48***	**.57***	.07	.27	.39
DS Av JDS	.08	-.05	.07	.10	-.01	-.09	.11	-.01	.16	-.03
DS % LC	.22	-.10	.12	.04	.10	-.12	.19	-.22	.20	.05
Osler 1 Av JDS	.25	.08	.13	.04	.05	-.03	.14	-.25	.17	.03
Osler 1 % LC	.24	.02	.16	.16	.11	.17	.12	-.02	.19	.21
Osler 2 Av JDS	.18	.12	.33	.27	.38	.21	.31	-.07	**.49***	.35
Osler 2 % LC	.38	**.41***	**.45***	**.45***	**.44***	**.49***	.22	.13	.37	.38
Sleep Latency	-.32	-.38	-.37	-.38	-.31	-.38	-.38	-.27	-.19	-.31
Osler Total Misses	-.34	.10	-.11	-.13	-.20	-.01	-.20	.23	-.38	-.29
AHI Events/hour	-.16	-.37	-.16	-.12	-.16	.11	-.07	.34	-.13	-.07
$MinSp0_2$	-.19	.26	-.16	-.21	-.17	-.26	-.24	-.29	-.13	-.21
Arousals total	-.32	-.44	-.22	-.24	-.30	-.03	-.23	.32	-.29	-.26
Sleep Efficiency	.21	**.53***	.29	.26	.22	.19	.38	-.05	.03	.07
Time in REM	**.48***	.32	.25	.26	.23	.26	.41	.07	.18	.18

CONTROL	BDI Total	STAI – S	STAI – T	POMS T-A	POMS D-D	POMS C-B	POMS A-H	POMS Vigour	POMS Fatigue	POMS Total
PVT Av JDS	.13	-.01	.09	.04	.26	**-.45***	-.06	.03	-.13	-.10
PVT % LC	.20	.34	.23	.02	.11	-.35	-.04	-.40	-.19	.03
DS Av JDS	-.16	-.05	-.14	-.14	.18	-.27	.39	.18	-.34	-.14
DS % LC	-.37	**-.60****	-.34	-.43	-.21	-.27	-.33	**-.49***	-.03	-.44
Osler 1 Av JDS	.04	-.36	-.04	.20	.21	.20	-.04	**.47***	.39	.05
Osler 1 % LC	-.14	-.10	-.01	-.17	-.19	-.01	-.23	.15	-.01	-.17
Osler 2 Av JDS	.19	.00	-.03	.06	.30	-.27	-.02	.00	-.02	-.09
Osler 2 % LC	.23	.14	.08	.09	.14	-.25	-.13	-.09	-.03	-.06
Sleep Latency	-.21	-.09	-.26	-.24	.00	-.41	-.15	-.26	-.31	-.29
Osler Total Misses	-.15	-.23	-.21	.02	.33	-.32	-.03	.12	-.20	-.14
AHI Events/hour	.03	-.12	-.08	.08	.18	-.11	.07	.12	-.07	.08
$MinSp0_2$	.10	.31	.21	.00	.08	.10	.27	-.26	.04	.11
Arousals total	.26	.13	.14	.18	.02	.07	-.19	-.11	.14	.23
Sleep Efficiency	.24	.38	.41	.08	.10	.11	.27	-.31	-.10	.23
Time in REM	.29	.41	**.44***	.21	.13	.22	.21	-.38	.01	.31

PVT = Psychomotor Vigilance Task; DS = Driving Simulator; JDS = Johns Drowsiness Scale; % LC = Percent Long Closure; RT = Reaction Time; AHI = Apnoea-Hypopnoea Index, Minimum $Sp0_2$ = percentage of total sleep time where oxygen saturation is <90%; BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; POMS = Profile of Mood States, T-A = Tension-Anxiety; D-D: Depression-Dejection; C-B Confusion-Bewilderment; A-H = Anger-Hostility.

* Significant at .05 level (2-tailed); ** Significant at .01 level (2-tailed)

NB: N's for each of the groups differed between cells

3.4.6. Mood and performance. Table 3.14 displays correlations (Kendall's *tau*) between mood variables and performance measures for the three groups. Among OSA patients, there was a significant positive correlation at the .01 level between the Anger-Hostility scale of the POMS and the average median position on the driving simulator. There was also a significant positive relationship at the .01 level between the Fatigue scale of the POMS and the speed 70km/hour and 60 to 80km/hour speed range deviation on the driving simulator.

Among shift-workers, there was a significant positive correlation between the 70km/hour speed range deviation on the driving simulator and the STAI – State Anxiety scale. There were also significant correlations between the number of crashes on the driving simulator and scores on the STAI – State Anxiety and the Tension-Anxiety scale of the POMS.

For control participants, there was a significant negative correlation between the median reaction time on the driving simulator and scores on the STAI – State Anxiety. There was also a significant positive correlation between the median reaction time on the driving simulator and scores on the Vigour scale of the POMS.

Table 3.14

Correlations (Kendall's tau) between mood measures and performance variables for OSA patients, shift-workers and control participants

OSA (n =16)	PVT Median RT	PVT Slowest 10%	PVT Lapses	DS Position Median Average	DS Speed 70 Average	DS Speed 60-80 Average	DS RT Median	DS Crash Number
BDI Total	.20	-.09	.12	.23	.32	.37	-.01	.35
STAI – S	-.04	.03	.04	.20	-.05	.00	-.08	.06
STAI – T	.07	.03	.03	.23	.11	.13	.03	.15
POMS T-A	-.04	.10	-.04	.18	.08	.08	-.09	.11
POMS D-D	.06	.09	-.07	.28	.26	.26	.00	.24
POMS C-B	.37	-.24	.29	.28	.30	.33	-.02	.15
POMS A-H	-.01	-.02	.06	**.49****	.13	.08	.18	.24
POMS Vigour	.08	-.05	-.02	.09	-.07	-.16	.29	-.08
POMS Fatigue	.34	-.11	.17	.11	**.53****	**.55****	-.04	.34
POMS total	.12	.06	-.01	.18	.26	.28	-.13	.18

SW (n =15)	PVT Median RT	PVT Slowest 10%	PVT Lapses	DS Position Median Average	DS Speed 70 Average	DS Speed 60-80 Average	DS RT Median	DS Crash Number
BDI Total	-.08	-.02	-.01	.17	.23	.31	-.31	.16
STAI – S	.13	.09	-.05	.17	**.48***	.38	-.09	**.52***
STAI – T	.00	.00	-.02	.22	.16	.12	-.08	.24
POMS T-A	.11	-.07	.10	.05	.17	.11	-.07	**.45***
POMS D-D	-.01	.05	-.03	.21	.03	.07	-.15	.19
POMS C-B	.06	.01	-.09	.02	.28	.30	-.04	.07
POMS A-H	.05	.19	-.02	-.03	-.05	.17	-.27	.13
POMS Vigour	.25	-.12	.11	-.04	.12	-.02	.29	.02
POMS Fatigue	-.15	.15	-.07	.11	-.05	.07	-.20	.18
POMS total	-.02	.06	-.01	.10	.06	.13	-.12	.29

CONTROL (n =14)	PVT Median RT	PVT Slowest 10%	PVT Lapses	DS Position Median Average	DS Speed 70 Average	DS Speed 60-80 Average	DS RT Median	DS Crash Number
BDI Total	.16	-.16	.03	.18	.30	.40	-.30	-.17
STAI – S	.03	.21	-.21	.01	.12	.10	**-.46***	-.26
STAI – T	.09	-.04	.04	.10	.14	.21	-.32	-.13
POMS T-A	.16	-.19	.11	.07	.09	.18	.07	-.21
POMS D-D	.07	-.17	.12	.23	.06	.18	-.09	-.08
POMS C-B	.30	-.18	.13	-.17	-.02	.02	-.12	.00
POMS A-H	.20	.17	-.19	.12	-.02	.05	-.41	.20
POMS Vigour	.09	-.17	.23	.16	.00	.09	**.43***	.39
POMS Fatigue	.14	-.33	.32	-.02	.00	.12	.10	.00
POMS total	.41	-.20	.12	.17	.14	.19	-.21	-.07

PVT = Psychomotor Vigilance Task; DS = Driving Simulator; JDS = Johns Drowsiness Scale; % LC = Percent Long Closure; RT = Reaction Time; AHI = Apnoea-Hypopnoea Index, Minimum Sp0² = percentage of total sleep time where oxygen saturation is <90%

* Significant at .05 level (2-tailed)

3.5. Correlational Analyses between Subjective Sleepiness, Objective Sleepiness, Performance, Mood and Accident History

Patients with OSA and controls each reported a total of three accidents, and shift-workers reported two accidents over this period of time. For OSA patients, one accident implicated another vehicle (not at work), and two accidents involved no other vehicle (not work related). Shift-workers reported two accidents involving no other vehicle (not work related), and controls reported three accidents each involving another vehicle (at work).

3.5.1. Accident history and subjective sleepiness. Table 3.15 displays correlations (Kendall's *tau*) between subjective sleepiness measures and the total number of accidents over a 3 year period for OSA patients, shift-workers and control participants. Only OSA patients showed a significant positive correlation between total scores on the SSQ and accident history.

Table 3.15

Correlations (Kendall's tau) between subjective sleepiness measures and accident history for OSA patients, shift-workers and control participants

	ESS	KSS	SDQ Part1	SDQ Part2	SSQ Total
OSA (n = 17)					
Total Accidents	.28	.29	.48	.48	**.68***
SW (n = 15)					
Total Accidents	-.04	-.39	-.19	-.36	-.27
CONTROL (n = 15)					
Total Accidents	.04	.25	-.19	-.02	.21

ESS = Epworth Sleepiness Scale; KSS = Karolinska Sleepiness Scale; SDQ = Stop Driving Questionnaire; SSQ = Sleepiness Symptoms Questionnaire

* = significant at .05 level (2-tailed)

3.5.2. Accident history and objective sleepiness. Tables 3.16 and 3.17 present correlations (Kendall's *tau*) between objective sleepiness measures and accident history for the three groups. There was a significant negative correlation between total accidents and JDS score on the driving simulator for shift-workers. In addition, there was a positive correlation between accident history and the percentage of time that the eyes remained closed per minute on the PVT for control subjects. No other objective sleepiness measures were correlated with accident history.

Table 3.16

Correlations (Kendall's tau) between objective sleepiness measures (Optalert and Osler) and accident history for OSA patients, shift-workers and control participants

	PVT Average JDS	PVT % long closure	DS Average JDS ^	DS % long closure ^	Osler 1 Average JDS	Osler 1 % long closure	Osler 2 Average JDS	Osler 2 % long closure	Sleep Latency	Osler Total Misses
OSA (n = 17)										
Total Accidents	-.13	-.05	-.08	.13	-.03	.18	-.36	-.31	.28	-.17
SW (n= 15)										
Total Accidents	-.15	-.22	**-.48***	-.28	-.13	-.40	.03	-.02	.11	.32
CONTROL (n= 15)										
Total Accidents	.44	**.74****	-.06	-.22	-.34	-.02	.35	.38	.10	.06

PVT =Psychomotor Vigilance Task; DS = Driving Simulator; JDS = Johns Drowsiness Scale

* = significant at .05 level (2-tailed); ** = significant at .01 level (2-tailed) ^ = OSA n = 16

Table 3.17

Correlation (Kendall's tau) between objective sleepiness measures (polysomnography) and accident history for OSA patients, shift-workers and control participants

	AHI Events/hour	Minimum Sp0²	Arousals total/hour ^	Sleep Efficiency	Time in REM Sleep
OSA (n = 17)					
Total Accidents	-.28	-.11	-.44	.11	.28
SW (n = 13)					
Total Accidents	.39	.12	.11	-.19	.06
CONTROL (n = 15)					
Total Accidents	.20	.13	.20	.38	.38

^ OSA = 16

3.5.3. Accident history and performance. Table 3.18 displays correlations (Kendall's *tau*) between performance on the PVT and driving simulator and total accidents for the three groups. Only shift-work participants demonstrated a significant negative correlation between the number of lapses on the PVT and accident history. No other performance variables were correlated with accident history.

Table 3.18

Correlations (Kendall's tau) between performance measures and accident history for OSA patients, shift-workers and control participants

	PVT Median RT	PVT Slowest 10%	PVT Lapses	DS Position Median Average	DS Speed 70 Average	DS Speed 60-80 Average	DS Median RT	DS Crash Number
OSA (n = 17)								
Total Accidents	-.26	.10	-.07	-.17	-.17	.00	-.50	-.42
SW (n= 15)								
Total Accidents	.23	.00	**-.48***	-.27	-.23	-.27	-.15	-.19
CONTROL (n= 15)								
Total Accidents	-.02	.04	-.07	.21	.24	.24	-.13	-.23

PVT =Psychomotor Vigilance Task; RT = Reaction Time; DS = Driving Simulator

* = significant at .05 level (2-tailed)

^ = OSA n = 16

3.5.4. Accident history and mood. No significant correlations were found for OSA, shift-workers and control subjects for accident history and mood variables.

Table 3.19

Correlations (Kendall's tau) between mood measures and accident history for OSA patients, shift-workers and control participants

	BDI Total Score	STAI State Anxiety	STAI Trait Anxiety	POMS Tension-Anxiety	POMS Depression-Dejection	POMS Confusion-Bewilderment	POMS Anger-Hostility	POMS Vigour	POMS Fatigue	POMS Total
OSA (n =17)										
Total Accidents	-.20	.17	-.03	-.03	-.12	-.18	-.20	-.31	-.30	-.11
SW (n= 15)										
Total Accidents	-.19	-.24	-.18	-.18	-.12	-.06	-.08	.08	-.23	-.23
CONTROL (n=14)										
Total Accidents	.26	.43	.27	-.11	.05	-.45	-.12	-.43	-.36	.00

BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; POMS = Profile of Mood States

4. DISCUSSION

4.1. Review of Rationale and Aims of the Present Study

Regardless of the aetiology, constant sleep disruption results in wide-ranging negative consequences in sleepiness, psychomotor performance, driving ability and mood. People with OSA and those engaged in the often tumultuous routine of rotating shift-work experience reduced sleep duration and poorer sleep quality which is likely to render the above mentioned consequences more pronounced. The medical condition of OSA is well understood and treatment is readily available, although many people remain undiagnosed and untreated. In contrast, effects and health consequences associated with shift-work are less well understood and treatment is limited at the present time. The establishment of a profile of health implications for the shift-worker appears to be a complex task, and is often thwarted by the 'let sleeping dogs lie' notion that is propagated by both industry and trade unions. Given that the sleepiness experienced by shift-workers has been shown to be as severe as that experienced by patients with OSA (Shen et al., 2006), it would seem urgent that we 'wake the sleeping dog'. In addition, it is important that the effects of shift-work are better understood and that steps are taken to make changes to work policies and schedules to improve health and safety outcomes.

The aim of the current study was to compare subjective sleepiness, objective sleepiness, psychomotor performance, simulated driving performance and mood disturbances in patients with OSA, shift-workers and control participants. The study also aimed to determine whether significant relationships existed between these measures within each of the three groups of participants. Lastly, associations between accident history and the dependent variables subjective sleepiness, objective sleepiness, psychomotor performance, simulated driving performance, and mood were examined to determine accident risk.

4.2. Demographic Characteristics

Symptoms of OSA, such as EDS, typically become evident only well into the patient's adult years, with the average age of onset reported to be in the mid-forties (Bearpark et al., 1993; Orr, 1997). The mean age of the OSA patients in the present study was 58 years (SD = 10.73). Control participants and shift-workers were significantly younger, with mean ages of 47 years and 45 years respectively. Orr (1997) indicated that symptoms of OSA can be present for as long as ten years prior to diagnosis in many patients. Ronald and colleagues (1998) provided support for this notion, revealing that OSA patients utilised health care resources at approximately twice the rate of controls ten years prior to their diagnosis. Therefore, it may have been the case that the OSA patients in the present study were experiencing symptoms of OSA long before receiving confirmation of their diagnoses. It is likely that the quality of life of these patients may have been improved had the diagnosis and subsequent treatment of OSA occurred at an earlier stage. Given that many patients who are treated with CPAP experience a dramatic improvement in their quality of life (Ronald et al., 1998), early diagnosis and treatment is likely to enhance the overall health and well-being of OSA patients.

Patients with OSA weighed significantly more, and demonstrated a higher BMI and MAPQ scores in comparison to both shift-workers and control participants in the present study. Research has supported this weight trend, revealing that an increase in BMI and neck circumference significantly increases the risk of OSA (Young et al., 2002). In the current study, OSA patients had a mean BMI of 33.93, and thus could be classified as being in the obese range. Shift-workers and control participants fell in the slightly overweight range, with mean BMI's of 25.91 and 25.71 respectively. Not surprisingly, the OSA patients exhibited higher MAPQ scores (M = 0.69) in comparison to both shift-workers (M = 0.30) and control participants (M = 0.23). Multivariate Apnoea Prediction Questionnaire (MAPQ) scores that

were greater than 0.50 in the present study were deemed to be suggestive of a diagnosis of OSA. Thus, this finding adds further support for the clinical utility of the MAPQ in predicting OSA risk (Maislin et al., 1995).

Consistent with previous studies, OSA patients in the current study also endorsed significantly higher scores on the ESS (Akerstedt & Gillberg, 1990; Johns, 1993) relative to both shift-workers and control participants. Johns (1993) proposed that a clinically 'normal' range for ESS scores is between two and ten. The OSA patients in the present study exhibited a mean score well above the normal range (M = 11.59). Therefore, OSA patients were more likely to report dozing off in a variety of passive situations commonly encountered in daily life (Johns, 1993). In contrast, shift-workers and control participants endorsed similar mean scores within the normal range (M = 6.87 and 5.42 respectively). These findings support those of Garbarino and colleagues (2002b) who found that ESS scores were not significantly higher in shift-workers relative to non-shift-workers. Since the participants in the Garbarino et al (2002b) study were police officers, it may have been the case that stressful work and responsibility may have rendered this population more likely to under-estimate the possibility of falling asleep, or that the nature of this work may have been favourable in assisting them to maintain a high level of vigilance in many conditions.

Shift-work, particularly after many years, has been associated with significant disturbances in sleep duration (Akerstedt et al., 2008; Gold et al., 1992; Paim et al., 2008). Hence, it is not surprising that the shift-workers in the present study reported that they slept for a significantly reduced number of hours on work days and that they worked a significantly greater number of hours relative to OSA patients and control participants. In addition, since shift-work often necessitates continuous 24-hour operation, it could be argued that shift-workers have greater opportunities to work extra shifts or to engage in overtime work. In turn, this is likely to further compromise their sleep duration and performance. Rosa

(1995) provided support for this finding, indicating that unscheduled over-time was disruptive to the shift-worker as it exacerbated the difficulty in organising sleep and recovery time

4.3. Group Differences

4.3.1. Subjective sleepiness measures. Contrary to expectation, subjective sleepiness (i.e., state sleepiness measures) utilised in the present study revealed no differences between OSA patients, shift-workers and control participants (hypothesis 1). Surprisingly, the three groups reported comparable levels of daytime sleepiness and alertness. Since reports of EDS have long been recognised as the cardinal complaint in patients with OSA (Orr, 1997; Schlosshan & Elliott, 2004), and questionnaire studies have indicated increased levels of subjective sleepiness among shift-workers (Akerstedt et al., 2008; Fido & Ghali, 2007; Gold et al., 1992), this finding was unanticipated. However, Black (2003) and Vgontzas (2008) suggested that reports of EDS were not a universal feature in OSA patients since not all patients described this symptom even when the condition was in the moderate to severe range. The lack of difference between the groups in the present study may have been attributable to OSA patients reporting or experiencing other symptoms of daytime dysfunction, rather than relating their experience to the specific complaint of 'sleepiness'. Consistent with the findings obtained in the present study, Akerstedt and colleagues (2008) found no differences in subjective sleepiness ratings among shift-workers and control participants. The authors indicated that shift-workers may overlook their sleep disturbances as 'part of the job' which would lead to under-reporting of disturbed sleep, and suggested that objective indices are probably needed to more accurately assess sleepiness.

The minimisation of sleepiness-related symptoms by OSA patients has become widely acknowledged in clinical practice. As a phenomenon, under-reported sleepiness in untreated OSA patients has been attributed to a loss of a normal frame of reference for

alertness, because of prolonged EDS (Engleman et al., 1997). The lack of difference observed between the groups for measures of subjective sleepiness may have been due to OSA patient's under-reporting excessive sleepiness due to either a lack of awareness of their level of sleepiness, or a fear of consequences for driving and the possibility of losing their license. In addition, it may be the case that when the severity of OSA increases or the condition progressively worsens, OSA patients may compensate for their excessive sleepiness and thus report normal levels of alertness, despite feeling sleepy. In addition, conditions that induce chronic sleep impairment, such as OSA and shift-work, may result in a dissociation between subjective reports of sleepiness and objectively defined sleepiness. Therefore, despite reporting subjective alertness, objective evidence of pathological sleepiness may be present (Balkin et al., 2008).

The lack of significant differences between the groups across the various measures of subjective sleepiness may also be explained by various participant characteristics. Among OSA patients, Bixler et al (2005) revealed that age was a strong independent predictor of subjective sleepiness. Specifically, the prevalence of subjective sleepiness decreased in a linear fashion with increasing age between the ages of 30 and 75 years, such that only the young and the elderly reported increased subjective sleepiness. In the present study, the OSA participants had a mean age of 58 years placing them at middle-age; hence, it may have been that reports of subjective sleepiness were reduced rendering group differences difficult to detect. In addition, the OSA patients in the present study may have had reduced child-rearing responsibilities and lifestyle differences and may have reported reduced subjective sleepiness as a result of this.

Furthermore, several studies have documented that the most powerful predictor of subjective sleepiness is OSA severity (Bedard et al., 1991; Chesire et al., 1992). In the present study, the OSA patients were grouped according to whether or not they had a

diagnosis of OSA as opposed to the severity of the condition. On inspection of AHI values for OSA patients, a total of 76% were classified as having severe OSA according to specifications proposed by Cartwright (2001). Only 24% of the sample was classified as moderate and (by design) none of the OSA participants exhibited mild OSA. Given that a substantial proportion of OSA patients had severe OSA, this may have made it difficult to observe relationships between OSA measures of sleepiness within the OSA patient group.

Among OSA patients, other factors have been purported to explain subjective sleepiness that were not directly assessed in the present study, but which may have had an effect on ratings of subjective sleepiness, including smoking, snoring intensity, heart disease, stroke, metabolic factors and personality features such as hypochondriasis (Bixler et al., 2005; Hayashida et al., 2007; Koutsourelakis et al., 2008; Orr, 1997). Indeed, depression has been implicated as a factor causing subjective sleepiness among OSA patients as it may be related to disturbances in pathways involved in the regulation of sleep and wakefulness (Bixler et al., 2005). Future studies may wish to investigate the contribution of these factors in the assessment of subjective sleepiness for OSA patients.

In addition, it may have been the case that the shift-workers in the present study represented a group of workers who were relatively accepting of their work schedules and had adapted well to shift-work. Thus, the notion of the 'healthy worker effect' may explain why group differences were not observed in ratings of subjective sleepiness. These findings are consistent with those presented by Forberg and colleagues (2010) who found that tunnel workers experienced few problems, adapted easily to their work, and reported a high level of sleep efficiency.

Moreover, subjective reports of sleepiness have been suggested to be dependent on the length of the shift, such that shifts of 12-hour duration induce greater levels of sleepiness relative to shifts of 8-hour duration (Son et al., 2008). The shift-workers in the present study

worked an average of 11 hours per day. It may have been the case that shift lengths greater than 12 hours may have induced greater reports of subjective sleepiness. In addition, given that the shift-workers in the present study were required to obtain recovery time prior to testing, this may have led to reduced ratings of subjective sleepiness. It may have also been likely that if shift-workers were tested through the night, ratings of subjective sleepiness may have increased. This was not the case in the present study.

Orr (1997) indicated that age is likely to play a role in the sleepiness inherent among shift-workers, with workers in their late 40s and early 50s displaying a decreased tolerance for shift-work. It is noteworthy that the shift-workers in the present study had a mean age of 45 years. Gender has also been implicated as a factor in ratings of subjective sleepiness. Pati et al (2001) reported that female shift-workers reported greater sleep disturbances than men. However, in the present study, there were few female shift-workers relative to males, and this may have been likely to render group differences difficult to observe. Nonetheless, these findings lend support to the multi-dimensionality of subjective sleepiness in often-utilised self-report measures of sleepiness.

4.3.2. Objective sleepiness measures. The hypothesis that objective sleepiness scores would be higher in OSA patients in comparison to shift-workers and that shift-workers would have higher levels of objective sleepiness relative to control participants was supported in part (hypothesis 2). For Optalert variables, shift-workers showed higher levels of time with their eyes closed per minute (percent long closure) during the Osler task in comparison to OSA patients and control participants. This finding suggests that shift-workers experienced higher levels of objective sleepiness than OSA patients and shift-workers. This finding supports that of previous research which examined blink duration as an indicator of sleepiness in professional bus drivers that found an increase in the percentage of time that the eyes were closed among sleepy subjects on a sleep latency task (Hakkanen et al., 1999).

Among shift-workers, circadian rhythm disruption and sleep loss represent the main causes that induce sleepiness (Akerstedt, 1995). In combination, these factors reduce sleep duration and contribute to an endogenously enhanced level of sleepiness over time (Van Dongen, 2006). In contrast, patients with OSA experience sleep fragmentation and decreased sleep depth as a result of frequent awakenings (Piper & Stewart, 1999; Strollo & Rogers, 1996). Hence, the effects on sleep differ between OSA patients and shift-workers. It may be the case that circadian disruption, sleep loss, and shortened sleep duration renders shift-workers more likely to experience greater levels of objectively defined sleepiness compared to patients with OSA. Furthermore, given the variability in shift-schedules which may be constantly changing, shift-workers may experience greater difficulties in adjusting to their schedules, leading to greater levels of sleepiness. Perhaps patients with OSA compensate for their sleepiness over time in response to sleep quality and are better able to adapt to their sleepiness. Alternatively, it has been suggested that among shift-workers working a permanent night-shift, the circadian clock fails to sufficiently adjust (Folkard, 2008). This finding attests to the importance of further examining objective sleepiness in shift-workers.

Few studies have utilised the Optalert Drowsiness Measurement System (ODMS) in OSA patients, and this study may be one of the first to record these outcome measures among shift-workers. It may be that the non-significant differences found for the three groups on the PVT, driving simulator and Osler task reflect the lack of differences obtained on the ODMS. However, this finding may point to the importance of utilising objective indices, such as blink duration, for the assessment of sleepiness in shift-workers, since their sleep was more disturbed on measures of blink duration in comparison to that of OSA patients in the present study. Despite not reaching statistical significance, it is noteworthy that most JDS scores and the percentage of time that the eyes remained closed per minute on each of the tasks was increased among shift-workers compared to OSA patients and control participants. This

finding suggests that shift-workers may have been more likely to experience greater levels of objectively defined sleepiness. It may be that the small sample size also contributed to a lack of observed differences between the groups.

A JDS score of 4.5 has been suggested to indicate a cautionary level of drowsiness (Johns et al., 2007). In the present study, mean JDS scores for OSA patients, shift-workers and control participants for the PVT, driving simulator, and the Osler task ranged from 0.01 to 3.47, indicating relatively low levels of drowsiness. Circadian factors may explain the lack of differences found between the three groups since participants in the present study were not sleep-deprived and were tested during daytime hours. Time-on-task effects may also serve to explain objectively defined sleepiness. Each of the Osler trials ran for a period of 40 minutes. It is noteworthy that a significant difference was observed for the percentage of time that the eyelids remained closed on the first Osler trial for shift-work participants. Perhaps tasks of shorter duration (i.e., the PVT and driving simulator) were not sensitive enough to detect significant sleepiness differences between the groups using the ODMS. Furthermore, micro-sleeps have been suggested to be greater than three second duration (Priest et al., 2001). Given that the participants in the present study had their eyes closed for less than one second across each of the tasks, this may have made it difficult to detect group differences for ODMS measures.

On another measure of objective sleepiness (Osler), no significant differences were observed between the three groups. This finding was inconsistent with previous research which revealed that the Osler discriminated between normal and sleepy individuals (Bennett et al., 1997; Priest et al., 2001). However, it is notable that despite not reaching statistical significance, the group differences observed for the Osler were in the same direction of that specified by hypothesis 2. Patients with OSA exhibited shorter sleep latencies and also had a greater number of total misses on the Osler compared to shift-workers and control

participants respectively. Therefore, although the ODMS was sensitive to changes in objective sleepiness among shift-workers, the Osler revealed that OSA patients may have been more likely to experience increased objective sleepiness relative to shift-workers and control participants that could be detected with a larger sample size. The Osler measures sleep latency; hence it is possible that this measure taps more into the construct of 'sleepiness'. On the other hand, the ODMS provides a measure of performance impairment(s) caused by fatigue. Given that sleepiness and fatigue represent two independent constructs and shift-workers and OSA patients are differentially affected by sleep, it is possible that these measures of objective sleepiness affect OSA patients and shift-workers differently. In addition, it may be that the small sample size of the present study rendered group differences difficult to detect.

The lack of significant differences between the groups in the present study was surprising given that the duration of the Osler (i.e., 40 minutes) would be likely to detect differences in sleepiness, particularly when administered over two trials. The lack of difference may be attributable to several factors. First, it may be that the red LED light on the Osler acted as a small stimulus to staying awake and may have indicated a 'ceiling' effect in subjects who were only minimally sleepy (Bennett et al., 1997). Second, although a test termination time of 21s was chosen for the Osler, it is likely that participants may have demonstrated periods of sleep less than 21s, and that they may have missed less than 7 consecutive stimuli, which would still allow the test to proceed, in spite of indications of sleepiness. Lastly, it may also be conceivable that a sleepy subject could be asleep for 20s every 30s without ever missing seven stimuli in a row and still complete the 40 minute test (Priest et al., 2001).

Not surprisingly, for polysomnography variables, OSA patients experienced a greater number of apnoea and hypopnoea events per hour, a greater number of arousals per hour of

sleep and decreased sleep efficiency relative to shift-workers and control participants. A diagnosis of OSA is dependent upon the combination of the number of apnoea and hypopnoea events per hour of sleep to determine the frequency and severity of OSA. Despite extensive debate with regard to AHI specifications, severe OSA is generally defined as an AHI of greater than 30 events per hour (Cartwright, 2001). In the present study, OSA patients had a mean AHI of 46.22 (*SD* = 20.27). In contrast, shift-workers had a mean AHI of 13.18 (*SD* = 9.51), and control participants obtained a mean value of 9.99 (*SD* = 6.20). Although the values reported for shift-workers and control participants are likely to indicate the presence of some mild sleep-disordered breathing, an important consideration would be whether the shift-workers and control participants reported symptoms as a result of mild OSA. These findings are supported by those of Silverberg et al (2002) who found that approximately 80 to 90% of individuals have undiagnosed OSA, often for an average of seven years prior to diagnosis. The findings of the present study lend support to the importance of early diagnosis and treatment. Considering the high rates of undiagnosed OSA, it is somewhat anticipated that the shift-workers and control participants in the present study may have exhibited some degree of sleep-disordered breathing. However, shift-workers and control participants were screened to exclude participants with OSA, and it is unlikely that these values were commensurate with a clinical diagnosis of the condition.

Patients with OSA experienced a significantly lower level of oxygen saturation overnight relative to control participants and shift-workers. Considerable debate exists in the literature regarding the role of nocturnal hypoxemia in EDS among OSA patients. While several studies have reported that nocturnal hypoxemia was a major determinant of EDS in OSA patients (Bedard et al., 1991; Mediano et al., 2007), others revealed that OSA and sleep disruption were not the primary determinants of EDS in OSA patients, despite nocturnal oxygenation (Roure et al., 2008). This finding points to the importance of further

investigating polysomnography indices among shift-workers in the assessment of objective sleepiness. In the present study, shift-workers worked a significantly greater amount of hours daily relative to control participants and OSA patients. It seems expected then, that the combination of working a greater number of hours and circadian rhythm disruption would contribute to an increase in EDS. Indeed, it is possible that after engaging in work of a difficult nature, subsequent sleep may not result in full recovery, rendering shift-workers sleepier relative to OSA patients and control participants, which was suggested by some of the objective sleepiness measures in the current study. Future research may wish to account for factors such as nature of work in the assessment of objective sleepiness. In addition, control participants and OSA patients were tested at a time of their convenience, however, shift-workers were required to attend for testing 24 hours after the conclusion of their last night-shift. This may have meant that shift-workers were sleepier compared to control participants and OSA patients on the night of their sleep study. No significant differences were observed between the three groups for the amount of time spent in REM sleep.

4.3.3. Performance measures. Vigilance represents an important co-factor in the increased risk of road traffic accidents in patients with OSA (Engleman & Douglas, 2004), and in shift-workers (Garbarino et al., 2002a). The present study partly supported the hypothesis that OSA patients would perform more poorly on a test of psychomotor vigilance and a driving simulator task compared to shift-workers, and that shift-workers would perform more poorly on these tasks relative to control participants (hypothesis 3). However, no significant differences were found between the groups for the number of lapses on the PVT. This finding differs from those reported by Sforza et al (2004) who revealed that relative to control participants, OSA patients exhibited a greater number of lapses on the PVT. It is noteworthy that in the Sforza study, a greater number of lapses were significantly more evident in patients with greater levels of objectively defined sleepiness. Overall, the OSA

patients in the present study did not experience significantly high levels of objective sleepiness, and hence, it may have been difficult for an effect to be observed between the groups.

With respect to shift-workers, Axelsson and colleagues (2004) revealed an increase in the number of lapses throughout the night-shift relative to day and evening-shifts. The shift-workers in the present study were tested 24 hours after the conclusion of their last night shift to allow recovery time. It may be that if shift-workers were tested immediately following a night-shift, differences between the groups may have been evident. However, the present study aimed to assess shift-workers at a time when they were not directly affected by sleep periods of acute sleep restriction in order to reflect the longer term effect of shift-work on sleepiness function and mood.

In addition, Bonnefond et al (2006) reported that older age was significantly related to the number of lapses on the PVT. The shift-workers in the present study had a mean age of 45 years compared to 47 years for control participants and 58 years for OSA patients, and this may have concealed differences between the groups, given that they were relatively young. It is also noteworthy to consider that tasks of a longer duration than the PVT (i.e., 10 minutes) (Dinges & Kribbs, 1991) may have produced group differences. Furthermore, the maintenance of vigilance is largely dependent upon work schedules that promote activity as opposed to monotony (Sallinen et al., 2004), and this was not accounted for in the present study. It is also possible that the low number of participants in each group may have contributed to the lack of differences observed for PVT lapses in the present study. Among other studies which have reported significant group differences, larger sample sizes have been employed. For example, Sforza et al (2004) employed a sample of 152 patients with OSA and 45 control participants and demonstrated that OSA patients had a greater number of lapses

than control subjects. Similarly, Axelsson et al (2004) demonstrated an increase in the number of lapses among 317 shift-workers.

OSA patients performed more poorly on median reaction time on the PVT relative to controls and shift-workers respectively, indicating that OSA patients experienced reduced levels of sustained attention. This finding supports previous research which indicates that vigilance is significantly impaired among OSA patients (Beebe et al., 2003; El-Ad & Lavie, 2005). Surprisingly, shift-workers performed better on median reaction time on the PVT relative to control participants in the present study. This finding may be attributable to the time of day that participants were tested. Although night-shift-work has been suggested to produce greater decrements in performance, the shift-workers in the present study obtained 24 hours of recovery time prior to their testing session which was conducted during daytime hours. Hence, it is likely that this may have concealed performance decrements among shift-workers.

With regard to the slowest 10% of reaction times, OSA patients performed the most poorly relative to control participants and shift-workers. In fact, shift-workers fared best on this measure relative to the other groups. Few studies have documented trends in the slowest 10% of reaction times for the PVT among shift-workers. However, previous research has suggested that the performance variability inherent among shift-workers may be greater in the laboratory or testing environment compared with actual operational shift-work settings (Van Dongen, 2006). It may be that this measure of PVT accuracy was more pronounced among shift-workers due to performance variability.

On the driving simulator task, OSA patients exhibited the greatest speed variation from the 70km per hour speed range and the optimal 60 to 80km per hour speed range compared with control participants and shift-workers respectively. This finding partly supported hypothesis 3 and was in line with previous research which indicated that OSA patients exhibit

a greater number of errors on measures of speed relative to controls (Findley et al., 1989). However, control participants performed more poorly on measures of speed variability relative to shift-workers. Despite the paucity of research examining driving simulator performance in shift-workers, simulations have been conducted with subjects driving home from the night-shift, and have generally revealed a greater number of incidents, decreased time to accidents, and variability in lateral position when commuting home from the night shift (Akerstedt et al., 2005). The shift-workers recruited for the present study were required to obtain recovery sleep post conclusion of their last night-shift prior to attending for testing. These results suggest that shift-workers are able to perform at the same level as healthy controls during the day after 24 hours of recovery from their night-shift. Moreover, sleep propensity and the circadian drive for sleep is increased during the early morning (Scott et al., 2007). Since participants' were tested during daytime hours, the likelihood of driving performance decrements among shift-workers was likely to be reduced. Furthermore, shifts of longer than 12-hour duration have been suggested to increase the risk of drowsy driving, rendering deteriorations in performance more likely (Scott et al., 2007). Given that the shift-workers in the present study worked an average of 11 hours per day, this may not have been of sufficient duration to observe effects of driving impairment amongst this population.

Average median position from the centre of the left lane, median reaction time and number of crashes did not differ significantly between the three groups. Although these findings differ considerably from previous research which demonstrates an increase in driving simulator impairment in OSA patients and shift-workers relative to control subjects (Akerstedt et al., 2005; Findley et al., 1995; George et al., 1996; Philip et al., 2005; Turkington et al., 2001), other characteristics not exclusive to OSA patients may explain these findings. Turkington and colleagues (2001) demonstrated that participant characteristics such as older age, female sex, previous driving experience, previous accident history, and admitted

alcohol consumption may influence driving performance. There were few females in the OSA patient group and shift-work group which may explain the inability of the present study to identify differences between the groups. In addition, prior driving experience was not thoroughly examined in the present study. However, the present study contributed to the literature by incorporating a measure of accident history for the preceding three years.

It is noteworthy that one participant was not able to participate in the driving simulator due to feeling disoriented and experiencing dizziness. With this in mind, it may be possible that prior experience or competence with video games may have led to a lack of differences between the groups. Although participants' were familiarised with the driving simulator prior to the 30 minute drive, this may not have been of adequate duration to overcome learning effects with the task. Task duration may also serve to explain the results, since tasks of shorter duration (i.e., 30 minutes on the driving simulator) may not have been sensitive enough to detect group differences. Furthermore, no significant differences were observed between the groups on measures of subjective sleepiness administered immediately after the driving simulator (i.e., the SDQ and SSQ) which indicated that OSA patients and shift-workers reported comparable levels of sleepiness to controls, and may not have been experiencing significant sleepiness during the driving simulator task.

4.3.4. Mood measures. Since the implications of negative mood are often adverse, understanding the nature and extent of such disturbances among patients with OSA and shift-workers seems essential. The present study hypothesised that patients with OSA would demonstrate greater disturbances in mood relative to shift-workers and that shift-workers would report greater mood disturbances compared to control participants and this was partly supported (hypothesis 4). On a specific measure of depression, the BDI, OSA patients endorsed higher scores relative to shift-workers, and shift-workers reported higher scores compared with controls. This finding suggests that OSA patients experienced greater levels of

depressive symptomatology compared with shift-workers, and is in line with previous research which suggests that the co-morbidity of depressive symptoms in OSA patients is a common feature of the syndrome (Bardwell et al., 2003; Ferini-Strambi et al., 2003; Pillar & Lavie, 1998; Vandeputte & de Weerd, 2003). Indeed, even when employing different measures to assess depressive symptoms, these symptoms continue to be observed among this population. In addition, shift-workers demonstrated greater disturbances in depressive symptomatology as measured by the BDI relative to control subjects in the present study. This finding supports that of previous research which suggests a high prevalence of depressive symptoms during the shift-work experience (Scott et al., 1997). Indeed, higher rates of depression have been indicated for women as opposed to men (Scott et al., 1997). Interestingly, although the percentage of males in the OSA patient group and shift-work group was much greater than females, differences between the groups were still detected. These findings suggest a strong gender component in the assessment of depression in OSA patients and shift-workers and add further support for the clinical utility of the BDI as a sensitive measure in the assessment of depressive symptomatology among these populations.

On the STAI, no differences were observed between the groups for both State-Anxiety and Trait-Anxiety. The present study attempted to utilise a measure of anxiety that tapped into transitory periods of anxiety as well as more stable personality features that may predispose individuals to more chronic levels of anxiety. Hence, the lack of differences observed between the groups was surprising. Although not statistically significant, inspection of State and Trait Anxiety scores across the three groups revealed that they were in the same direction to that specified in hypothesis 4. Patients with OSA demonstrated higher scores for both State Anxiety and Trait Anxiety relative to shift-workers and controls. It may be the case that the nature of the inventory which asked participants to rate on a Likert scale how they were feeling ‘right now’ may have been too limited a time frame to detect differences. Perhaps a

measure of anxiety that employed a more liberal time frame for participants to judge their anxiety levels against may have yielded different findings. In addition, it is likely that the small sample size in the present study may have compromised statistical power, rendering group differences difficult to observe.

Since few studies have focused on investigating anxiety in OSA patients and shift-workers, and the present study revealed no differences between the groups on the STAI, further research is needed to document these trends. However, a study conducted by Kjelsberg et al (2005) may explain the lack of differences observed in the present study. Low compliance with CPAP therapy was found to be the only variable associated with a high level of anxiety among OSA patients. However, OSA patients had only recently received a diagnosis of OSA, were untreated, and had not yet commenced therapy with CPAP. It may be that effects could not be observed between the groups because OSA patients were not receiving CPAP therapy. In addition, the present study was cross-sectional and did not assess changes in anxiety after CPAP had been implemented. Future studies may wish to investigate changes in anxiety levels over time after CPAP implementation. In a separate study conducted by Borak et al (1996), sleep fragmentation was the only contributing factor to anxiety levels. However, given that both OSA patients and shift-workers differentially experience fragmented sleep, this may have led to a lack of differences between the groups.

On the Profile of Mood States, only the Vigour scale and the Total Mood Disturbance score exhibited group differences. Although the scales of Anxiety, Depression, Confusion, Anger, and Fatigue revealed no differences between the groups, there was a trend towards higher scores in both OSA patients and shift-workers. Mosko et al (1989) revealed that the POMS scales did not distinguish patients with OSA from controls. However, it does not seem surprising that the Anxiety scale of the POMS revealed no group differences, since this was also the case for the more specific measure of anxiety, the STAI. Despite this, the lack of

differences between the groups for the Depression scale of the POMS was unexpected, given that differences were found for the BDI. Pillar and Lavie (1998) proposed that the POMS Depression scale was a relatively specific measure of depression relative to those more specific to depression, such as the BDI. In addition, the POMS evaluates more transitory moods and general affects when compared with the BDI, perhaps making it more difficult for group differences to be observed.

Although unexpected, no differences were found between the groups for the Confusion scale of the POMS. It could be argued that as a construct, confusion may be unrelated to sleep loss and circadian rhythm disruption. Unlike the constructs of depression and anxiety which appear to be related to poor sleep, the mood state of confusion may be more difficult to disentangle. Surprisingly, the Anger scale of the POMS also revealed no group differences. It could be the case that shift-workers in the present study represented a group of healthy workers who had self-selected into this type of work, and did not experience significant hostility. Similarly, a significant percentage of OSA patients in the present study were retired, and may have been content with their current situation, rendering feelings of anger less likely. As a result of these factors, differences between the groups may have been difficult to detect.

The POMS Fatigue scale also revealed no group differences. However, this finding seems expected since there were no significant differences found for the groups on subjective sleepiness measures. It may be the case that the Fatigue scale of the POMS is not a sensitive measure of fatigue relative to other measures, since it combines a variety of different mood states. On another level, although the terms fatigue and sleepiness are often used interchangeably to denote the same construct, they each represent a separate concept (Johns, 1998). Fatigue represents a physiological phenomenon characterised by time-on-task performance decrements which is relieved when the task is changed, however sleepiness refers to an underlying and persistent need state until which is only satisfied when sleep is

initiated (Mathis & Hess, 2009). Arguably, measures of subjective fatigue differ from those of subjective sleepiness, rendering group differences difficult to ascertain on the POMS Fatigue scale.

The POMS Vigour scale revealed differences between the groups, supporting hypothesis 4. Specifically and as expected, OSA participants reported the least amount of vigour relative to shift-workers, who in turn reported less vigour relative to controls. Few studies have been conducted examining vigour among OSA patients and shift-workers, however Bardwell et al (1999) found that patients with OSA experienced increased vigour the more they slept. Despite seeming intuitively obvious, this finding supports the notion that when sleep is disturbed, as is the case in OSA patients and shift-workers, vigour is decreased in these populations.

Lastly, the Total Mood Disturbance score of the POMS revealed significant differences between the groups, partially supporting hypothesis 4. Contrary to expectation, shift-workers demonstrated a greater total mood disturbance score relative to OSA patients and controls respectively. Orton and Gruzelier (1989) provided support for the findings obtained in the present study, suggesting that deleterious effects in mood occurred after the night-shift across all mood scales of the POMS relative to controls. Given that the shift-workers in the present study participated after their last night-shift, this may have rendered mood disturbances more pronounced. In terms of OSA patients, demographic characteristics revealed that they worked fewer hours relative to shift-workers and that a significant proportion of patients were retired at the time of the study. These factors may function to explain why shift-workers exhibited higher mood disturbance scores on the POMS relative to OSA patients.

4.4. Relationships between Variables

4.4.1. Subjective sleepiness and objective sleepiness. Based on the extensive research literature examining associations between measures of subjective sleepiness and

objective indicators of sleepiness (Balkin et al., 2008; Black, 2003, Johns, 1991), the present study hypothesised that subjective sleepiness would be correlated with objective sleepiness for OSA patients, shift-workers and control participants (hypothesis 5). Unexpectedly, and largely inconsistent with previous studies, the present findings revealed few correlations between subjective sleepiness measures and objectively defined sleepiness among OSA participants. Although Black (2003) reported weak to moderate correlations between these variables, Balkin et al (2008) revealed that relationships between these measures become tenuous in OSA patients because despite reporting subjective alertness, objective evidence of pathological sleepiness may be present. Adding to the complexity is the notion that EDS is not a universal feature of OSA, even in moderate to severe cases (Black, 2003), hence, relationships between objective and subjective sleepiness are likely to be vague in patients with OSA. No associations were demonstrated across measures of subjective and objective indices for control participants.

In contrast, relationships between subjective and objective indices were evident in shift-workers in the present study, in part supporting hypothesis 5. Most subjective measures were correlated with objective indices on the PVT, driving simulator, Osler 1 and 2, and polysomnographic variables, attesting to the importance of examining these associations further in shift-workers. Surprisingly, the ESS was the only subjective measure which did not demonstrate correlations with objective indicators of sleepiness. This finding was unexpected given that previous research has demonstrated relationships between the ESS and sleep latency measures during daytime sleepiness testing (Johns, 1991), and the participants' in the present study were also tested during daytime hours.

Previous literature appears to have largely under-estimated associations between scores endorsed on the KSS among shift-workers. It is likely that scores endorsed on the KSS reflect the level of sleep disruption experienced by this population as a result of conflicting

work and sleep schedules. Furthermore, the SDQ and SSQ were correlated with eyelid closure, average JDS scores, total misses on the Osler and some polysomnography variables, which is likely to indicate that reports of subjective sleepiness among shift-workers are consistent with evidence of pathological sleepiness. In addition, eyelid closure monitoring in the present study appeared to be a robust measure of objective sleepiness, as were polysomnography variables.

4.4.2. Subjective sleepiness and performance. Although less abundant, associations between subjective sleepiness and performance decrements have been documented in the literature (Findley et al., 1995; George et al., 1996; Ingre et al., 2006; Turkington et al., 2001). Based on these findings, the present study hypothesised that measures of subjective sleepiness would be associated with poorer performance on a reaction time task and a simulated driving task for OSA patients, shift-workers and control participants (hypothesis 6), and this was partially supported. Despite this, few relationships were observed for these measures between the three groups. Among OSA patients, only the median reaction time on the driving simulator was correlated with ESS scores. This finding is consistent with previous literature and supports the notion that the sleepiness experienced by OSA patients poses a significant risk for driving performance (Findley et al., 1995; George et al., 1996; Turkington et al., 2001). Surprisingly, control participants demonstrated a similar trend to OSA patients, whereby the ESS was positively correlated with the mean variation in speed from the 70km/h speed range. These findings may suggest that OSA patients demonstrate greater impairments in responding promptly to stimuli when sleepy, whereas control participants exhibit greater difficulty maintaining adequate speed when sleepy.

Unexpectedly, no relationships were observed between other performance measures and subjective reports of sleepiness. It may be the case that the OSA patients in the present study under-stated their level of sleepiness on the ESS, which may have rendered it difficult

to ascertain relationships between this subjective measure and performance indicators. In addition, Johns (1993) proposed that a clinically 'normal' range for ESS scores was between two and ten. In the present study 35% of OSA patients endorsed ESS scores less than eight. While this finding may be suggestive of under-reporting in OSA patients, it may also be the case that some OSA patients do not experience EDS or do not attribute their daytime impairments to sleepiness. The present findings seem fitting with that presented by Sforza and colleagues (2004) who revealed that a lack of clarity exists regarding whether performance is directly affected by sleepiness or whether performance and sleepiness co-vary.

Among shift-workers, the average median position on the driving simulator was positively correlated with KSS scores, the SDQ (suburban item) and the SSQ total score. These findings are consistent with those presented by Ingre et al (2006) who reported that with higher KSS levels, the standard deviation of the lateral position on the driving simulator increased. In the present study, negative correlations were observed between the median reaction time on the PVT and total SSQ scores. This finding seems somewhat surprising given that the total SSQ score pertains to sleepiness symptoms that participants' may have experienced during the driving simulator while the PVT assessed psychomotor performance. However, the present study was investigative in nature, and these findings suggest that as symptoms of sleepiness increase, shift-workers experience reduced psychomotor functioning. This seems expected given that shift-workers must contend with sleep loss and circadian rhythm disruption as a result of their irregular work schedules. However, the findings presented by Bonnefond et al (2006) revealed that median reaction times on the PVT showed no variation during the morning and afternoon-shifts. This is surprising since shift-workers in the present study were tested in the early afternoon, and yet, reaction times were decreased in response to sleepiness. The findings of the present study likely indicate that impairments in

performance represent a multi-factorial phenomenon involving several inter-related components that affect speed and accuracy differentially.

4.4.3. Subjective sleepiness and mood. Despite the few studies that have examined relations between subjective sleepiness and mood, sleep has been suggested to serve a mood regulatory function (Wanner & Bader, 2000). In line with this, the present study hypothesised that subjective sleepiness measures would be associated with mood disturbances among the three groups, and this hypothesis was partly supported (hypothesis 7).

Among OSA patients, the Confusion-Bewilderment scale of the POMS was positively associated with KSS scores, indicating that feelings of confusion-bewilderment increase with reports of subjective sleepiness. Interestingly, the POMS Vigour scale was positively correlated with the ESS and KSS, suggesting that increases in vigour are associated with increases in ratings of subjective sleepiness. This finding is largely inconsistent with previous findings which reveal decreases in vigour with increasing subjective sleepiness (Scott et al., 2006; Zhang et al., 2002). This finding is difficult to explain, however despite reporting feeling sleepy; OSA patients can still report feeling vigorous. Items incorporated within the POMS Vigour scale included feeling lively, cheerful and full of pep, and it is possible that these mood states can co-exist with reports of increased sleepiness. As expected, the POMS Vigour scale was negatively associated with the SDQ, suggesting that increases in vigour are associated with reports of increased sleepiness.

Among shift-workers, both the BDI and the STAI- trait anxiety scale were positively correlated with KSS scores, suggesting that increases in reported sleepiness are associated with increased depressive symptomatology and trait anxiety. Indeed, it seems expected that sleep loss would render disturbances in mood and increased subjective sleepiness more likely. These findings are consistent with those presented by Franzen et al (2008) who

revealed that increased negative mood was associated with subjective sleepiness. Hence, it is likely that these domains share a similar pathway.

Surprisingly, numerous correlations were observed for control participants in the present study across most mood measures and subjective sleepiness ratings. This finding was unanticipated since it was expected that OSA patients and shift-workers would report greater subjective sleepiness and mood disturbances relative to controls. However, it may be that OSA patients and shift-workers in the present study placed greater emphasis on their sleepiness rather than their mood, and this may have made associations between these variables less pronounced. Patients with OSA and shift-workers may find it difficult to differentiate perceptions of sleepiness from those of mood, since both represent an unpleasant experience when mood is negative.

4.4.4. Objective sleepiness and performance. Previous studies have provided support for associations between objective indicators of sleepiness and performance measures (Adams et al., 2001; Beebe et al., 2003; Sforza et al., 2004). In the present study, objective indicators included eyelid closure data, a measure of sleep latency and polysomnography variables. The present study sought to determine whether measures of objective sleepiness would be associated with reduced performance on a reaction time task and a simulated driving task for OSA patients, shift-workers and control participants (hypothesis 8), and for all groups, this hypothesis was partially supported.

For OSA patients, correlations were observed for eyelid closure data, sleep latency and time in REM sleep and driving simulator variables only. These findings lend support to the notion that although sleepiness can be under-stated subjectively amongst OSA patients; evidence of physiological sleepiness still exists, particularly in relation to performance. Control participants demonstrated similar correlations to OSA patients across measures of eyelid closure and polysomnographic variables (objective sleepiness) and the PVT and

driving simulator (performance measures). Therefore, although control participants may not report levels of sleepiness consistent with OSA patients or shift-workers, the present study revealed that relationships between objective indicators of sleepiness and performance decrements were still observed among controls.

Among shift-workers, numerous correlations were observed for most objective sleepiness measures (eyelid closure, sleep latency and polysomnographic variables) and performance variables. Interestingly, unlike OSA patients who demonstrated greater correlations between objective sleepiness and driving simulator variables, shift-workers exhibited a greater number of correlations between objective sleepiness and PVT variables. Whilst these findings seem to suggest that objective sleepiness renders driving impairments more likely among OSA patients and decrements in psychomotor functioning more prominent among shift-workers, a larger sample size would be required to confirm these findings.

Arguably, although the PVT was shorter in duration relative to the driving simulator, it may be that the repetitive and unchallenging nature of the PVT maximises sleepiness among shift-workers, and that longer task durations (i.e., driving simulator) maximised sleepiness among OSA patients who were required to maintain vigilance over a longer period of time. With this in mind, it would be more likely that associations between PVT performance and objective indicators would be observed. In addition, the substantial number of correlations observed among shift-workers relative to OSA patients and controls may be attributable to the fact that shift-workers in the present study worked a greater number of hours in their employment, which is likely to have led to greater objective sleepiness, which in turn may have led to a greater number of performance decrements, particularly psychomotor functioning.

4.4.5. Objective sleepiness and mood. Few studies have examined objective indicators of sleepiness and disturbances in mood. However, based on a study conducted by Franzen et al (2008), sleep loss exhibited strong effects on affective functioning, leading to a reduction in positive mood and an increase in negative mood. Since OSA patients and shift-workers are differentially affected in their sleep, the present study hypothesised that objective sleepiness measures would be associated with mood disturbances for the three groups, and this hypothesis was partially supported (hypothesis 9).

The present findings revealed that increased objective sleepiness was associated with feelings of anxiety, depression, and confusion as well as reports of decreased vigour among OSA patients. Of the studies that have examined associations between objective indicators of sleepiness and mood, only polysomnographic variables have been investigated. For example, Borak et al (1996) demonstrated a relationship between anxiety and the AHI, and Aloia and colleagues (2005) revealed an association between depressive symptomatology and the AHI. However, the findings of the present study point to the importance of further investigating other objective markers of sleepiness, such as eyelid closure data and sleep latency measures, since such objective indicators are likely to have an impact on mood among OSA patients.

Further research is needed to clarify possible associations between objective sleepiness and confusion for OSA patients. However, the present study offers preliminary support for a possible link between sleep latency (an objective indicator of sleepiness) and feelings of confusion. Decreased vigour was associated with increased eyelid closure for OSA patients in the present study. These findings support those of Bardwell et al (1999) who revealed that OSA patients experienced increased vigour the more they slept. Despite seeming intuitively obvious, this finding offers indirect support for previous findings that OSA patients experience greater sleepiness relative to control subjects. The present study

offers support for this contention, and revealed a relationship between the amount of time spent in REM sleep and the POMS scale of Fatigue.

Shift-workers demonstrated correlations between objective sleepiness measures and the scales of Depression, Confusion, Anger and Fatigue on the POMS, and State and Trait Anxiety on the STAI. Indeed, Orton and Gruzelier (1989) supported these findings in their study, demonstrating significant changes in mood occurring after the night duty across all mood scales in the POMS questionnaire. Although mood variables were similar among OSA patients and shift-workers; the latter group may have been likely to experience more anger relative to OSA patients. It is difficult to speculate as to why this was the case in the present study, however, it may be that as a result of working a greater number of hours, coupled with the sleep loss inherent in the lives of shift-workers, greater feelings of anger may result. The amount of time spent in REM sleep was correlated with the POMS Fatigue scale. Control subjects demonstrated relationships between objective sleepiness and the Confusion and Vigour scales of the POMS and State Anxiety on the STAI. These findings may indicate that control participants report fewer mood disturbances as a result of objective sleepiness.

It is noteworthy that time spent in REM sleep was the only polysomnographic variable that was associated with mood variables in the present study. However, given the exploratory nature of these analyses, the present findings should be considered preliminary and will need further replication. Relationships between subjective mood ratings and objective assessments of sleepiness may be affected by measurement sensitivity (Franzen et al., 2008), whereby physiological markers of sleepiness are likely to be measured more precisely and exhibit greater sensitivity relative to mood inventories.

4.4.6. Mood and performance. Despite the paucity of research examining associations between mood disturbances and performance decrements among OSA patients and shift-workers, mood has been suggested to affect sleepiness, which in turn, is likely to

influence performance (Garrity & Demick, 2001; Horne & Reyner, 1999). It was hypothesised that performance on a reaction time task and a simulated driving task would be associated with mood disturbances among OSA patients, shift-workers and control participants (hypothesis 10), and this was supported in part. Among patients with OSA, scores on the POMS Anger-Hostility scale were positively correlated with the average median position on the driving simulator. In addition, the POMS Fatigue scale was positively correlated with the mean variation in speed from the optimal 60 to 80km/h speed range and the 70km/h speed range. These findings are consistent with previous research which demonstrates that anger and fatigue are related to a lack of cautiousness whilst driving (Garrity & Demick, 2001). It seems expected that increased feelings of anger and greater levels of fatigue would render performance decrements on a driving simulator task more likely.

For shift-workers in the present study, a positive correlation was found for the mean variation in speed from the 70km/h speed range and crash number and the STAI- State Anxiety scale. The STAI – State Anxiety scale assessed how the participants' were feeling at the time of administration of the inventory, hence it is likely that increases in state anxiety were associated with greater variation in speed and an increase in the number of crashes on the driving simulator. Furthermore, the POMS Tension-Anxiety scale was positively correlated with the number of crashes on the driving simulator suggesting that greater anxiety levels are related to impaired driving performance and an increased number of accidents among shift-workers. Future research should further explore associations between mood and performance among these populations to ascertain relationships among these variables.

Among control subjects, State-Anxiety on the STAI was negatively correlated with median reaction times on the driving simulator, suggesting that greater state-anxiety was related with decreased reaction times on the simulator. In addition, the POMS Vigour scale

was positively correlated with median reaction times on the driving simulator, indicating that greater levels of vigour are associated with increased reaction times for braking episodes.

Patients with OSA, shift-workers and control participants demonstrated similar associations with one another in terms of disturbances in mood and performance decrements. However, it is noteworthy that in the present study, performance on the driving simulator was the only performance variable related to mood disturbances. These findings have important implications for road accidents, and point to the importance of further exploring mood disturbances in these populations.

4.5. Motor Vehicle Accident History

Although numerous factors have been suggested to contribute to sleepiness, knowledge about the associations between these factors and accidents continues to be under-investigated in the literature. The present study attempted to obtain information regarding whether significant relations existed for OSA patients, shift-workers and control participants between motor vehicle accident history (in the preceding three years), and measures of subjective and objective sleepiness, performance and mood. Although the hypothesis that these measures would be associated with accident history was partially supported (hypothesis 11), few of the measures were correlated with accident history.

In the present study, 17% of the entire sample reported experiencing an accident in the preceding three years. It is noteworthy that OSA patients and shift-workers reported a greater number of accidents involving no other vehicle (not work related). It could be argued that the increased sleepiness inherent in OSA patients and shift-workers may render them more likely to experience accidents in which they are at fault (i.e., those involving no other vehicle). In contrast, control participants only experienced accidents in which another vehicle may have been at fault. Although non-significant, it was surprising that controls reported a greater

number of kilometres driven per year at work and not-work related. Shift-workers and OSA patients were almost equivalent in terms of the amount of kilometres driven per year.

4.5.1. Accident history and subjective sleepiness. Indeed, the most common method for the detection of sleepiness is the self-report of the driver (MacLean et al., 2003). The decision to stop or to continue driving is dependent on a number of factors including whether the driver is aware of and able to detect their level of sleepiness and whether the detection of sleepiness is critical to the detection of driving impairment (MacLean et al., 2003). Relationships between daytime sleepiness (measured by the ESS) and accident risk have been reported in previous studies (Howard et al., 2004; Maycock, 1997). In the present study, only the SSQ was correlated with accident risk for OSA patients. This finding is consistent with that presented by Bearpark et al (1990) who reported that OSA patients were more likely to have had at least one accident attributable to sleepiness in the previous two years, and report symptoms of severe sleepiness (e.g., falling asleep at the wheel). The present study may be one of few studies that identified a relationship between the SSQ and accident history in OSA patients, and supports the utility of the SSQ in the assessment of driver sleepiness. In a study of the general population, specific symptoms of sleepiness were usually present prior to episodes of falling asleep (Nordbakke & Sagberg, 2007).

Surprisingly, despite the strong associations reported in previous studies between subjective sleepiness and accident history, no other subjective measures (ESS, KSS and SDQ) were correlated with number of accidents in the preceding three years in the present study. As previously stated it may be that the lack of association between these subjective sleepiness measures and accident history is attributable to the notion of possible under-reporting of sleepiness symptoms in patients with OSA (Engleman et al., 1997) as well as shift-workers for concerns related to the consequences for driving and work.

4.5.2. Accident history and objective sleepiness. Few studies have explored associations between accident history and objectively defined measures of sleepiness. Of these studies, no correlations have been observed between polysomnographic measures of sleep and accident history (Howard et al., 2004; Turkington et al., 2001). Although the presence of OSA is related to crash risk, there is not a clear relationship between OSA severity, measured by the AHI, and crash risk (Howard et al., 2004; Turkington et al., 2001). In addition, there is limited research investigating associations between objective sleepiness utilising eyelid closure measures, and accident history. In the present study, the average JDS on the driving simulator was related to accident history among shift-workers. Akerstedt et al (2005) found that increased eye closure durations were observed when commuting home from the night-shift; however the Akerstedt et al study did not explore relationships between objective sleepiness (i.e., eye closure data) and accident history. In addition, the percentage of time that the eyes remained closed on the PVT was correlated with accident history for control participants, although this was not the case for OSA patients and shift-workers.

All polysomnographic measures were not correlated with accident history for the three groups in the present study. Although this finding is likely to reflect the paucity of research examining these associations, Teran-Santos et al (1999) reported that among OSA patients, AHI's above 15 were 7.3 times more likely to be involved in at least 1 motor vehicle accident over a 5 year period. It is noteworthy that the OSA patients in the present study exhibited a mean AHI of 46.22, which would have been likely to detect a possible relationship with accident history. Howard and colleagues (2004) proposed that prior sleep patterns need to be controlled in order for objective measures of sleepiness to be utilised optimally. However, the present study was unable to control for sleep patterns prior to testing, since shift-workers experience irregularity in their work and sleep schedules. Future studies may wish to control

prior sleep patterns in the laboratory to determine if associations exist between objective sleepiness and accident history.

4.5.3. Accident history and performance. Impairments in vigilance and driving performance have been suggested to be a significant contributory factor in the risk of road traffic accidents (Engleman & Douglas, 2004). Since both OSA patients and shift-workers have been demonstrated to exhibit impairments in vigilance and driving (Akerstedt et al., 2005; Beebe et al., 2003; Findley et al., 1995; George et al., 1996; Purnell et al., 2002; Sforza et al., 2004), it seems expected that associations would exist between performance measures and accident history. Surprisingly, only the number of lapses on the PVT was correlated with accident risk among shift-workers. This trend was not observed for OSA patients and controls. Sforza et al (2004) reported that lapses on the PVT provided a more sensitive assessment of daytime impairment relative to other measures, such as reaction time. Since the shift-workers in the present study worked a greater number of hours than OSA patients and control participants and were tested following the conclusion of their last night-shift, this may have rendered them more impaired on the PVT. Indeed, previous studies have demonstrated that those engaged in night work demonstrate the most performance decrements relative to day and evening-shift counterparts (Akerstedt, 2003; Akerstedt et al., 2005; Akerstedt & Folkard, 1997; Folkard & Tucker, 2003).

The lack of associations between performance measures (i.e., PVT and driving simulator) and accident history in the present study may be attributable to the following factors. First, it may be that the potential danger of driving motivates sleepy individuals to put great effort into remaining awake. It seems reasonable to suggest that patients with OSA and shift-workers are likely to be more aware of the limits of their vigilance as a result of their inherent levels of sleepiness, and may take necessary precautions upon becoming concerned about their inability to drive. Second, it has been demonstrated that performance on a driving

simulator is affected by factors which are not directly related to driving, such as gender and age (Turkington et al., 2001) which may help to explain the lack of associations between driving performance and accident history.

4.5.4. Accident history and mood. Few studies have investigated possible associations between mood variables and accident history. The present study revealed no correlations between these variables among the three groups, which may suggest that mood disturbances are unrelated to the number of accidents experienced in the preceding three years. These findings corroborate those obtained by Barbe et al (1998), indicating that clinical variables (i.e., depression and anxiety as measured by the Beck Depression Inventory and Beck Anxiety Inventory respectively) were not related to accident history.

4.5.5. Future considerations for accident history. Several methodological and clinical factors should be considered in view of the limited associations observed between subjective and objective sleepiness, performance, mood, and accident history. First, despite obtaining information regarding accident history, the present study did not ascertain the likely cause of these accidents. It may have been the case that such accidents were due to factors other than sleepiness. Future studies should enquire about the cause of accidents when obtaining accident data. Second, collisions have been reported to be higher during the night and in the mid-afternoon relative to other times of the day (Horne & Reyner, 1995), however, the participants in the present study may have experienced accidents at other points of the circadian timing system, rendering it difficult to establish associations between sleepiness and performance variables and accident history. Third, few studies have assessed accident history, and of the studies examining these associations, different methods have been employed to devise such measures, possibly leading to inconsistencies. For example, the present study examined accident history in the preceding 3 years; however other studies may have employed more stringent or liberal time frames in the assessment of accident history. Finally,

the analyses in the present study enquired about accidents that had previously occurred, leading to the assumption that past accidents may be repeated in the future. In the present study, OSA patients were about to commence CPAP therapy which may lead to a reduction in sleepiness symptoms, rendering performance decrements (i.e., accidents) less likely. The challenge then, seems to be for the shift-worker since the behavioural and physiological approaches to counter the adverse effects of shift-work have revealed inconsistent findings in terms of benefit (Dawson et al., 1995; Sharkey et al., 2001).

4.6. Limitations of the Present Study and Directions for Future Research

A number of statistical, methodological and clinical limitations should be considered when interpreting the findings of the present study. First, the violation of the assumption of normal distribution by a number of variables necessitated the use of non-parametric statistics. The limited sensitivity of non-parametric statistics, coupled with the small sample size may have made it difficult to detect differences between the groups and associations that may have actually existed (Pallant, 2007). Second, the present study utilised correlational analyses to determine possible associations between subjective and objective sleepiness, psychomotor performance, simulated driving performance and mood with accident history. To extend these findings, a regression analysis could have been conducted to determine which of these independent variables significantly predicts accident risk. Future research may wish to consider the variable/s that may mediate an increased risk for motor vehicle accidents in patients with OSA and shift-workers to assist in the implementation of intervention strategies for these populations.

Methodologically, the present study may be criticised for its small sample size *(N*=47*).* Additionally, multiple comparisons were conducted in this study, increasing the likelihood of detecting a significant result by chance alone. As a result, it may be difficult to ascertain the representativeness of OSA patients and shift-workers recruited for the study.

Additionally, the three groups differed significantly according to age and gender and it is uncertain as to how this may have affected the findings. In the OSA patient group and the shift-work group, there were a significantly greater number of males as opposed to females, whereas the control group employed a greater number of females. Previous research has documented male gender as a risk factor for OSA (Bearpark et al., 1993; Young et al., 1993), however it is unknown whether those employed in shift-work are predominantly male or female, and whether the sample in the present study reflected this trend. Furthermore, the OSA patient group was significantly older relative to shift-work and control participants despite attempts to match participants as closely as possible based on age.

In addition, the recruitment process may have introduced an element of selection bias as participants' were financially reimbursed for their time. It is also possible that the shift-workers may have represented a group of individuals who were dissatisfied with their working arrangements and participated in the study to obtain reassurance or solutions regarding their well-being in their current work environment. Furthermore, an often unfortunate consequence of utilising technological measures renders equipment failure more likely. As a result of this, it is uncertain how missing data may have affected the results obtained. Lastly, although shift-work participants were required to have been employed in a shift-working capacity for the preceding three months for inclusion in the study, it was not documented how long they had actually worked in shift-work overall. Future research may wish to explore whether the length of time employed in shift-work impacts upon measures of objective and subjective sleepiness, performance measures and mood.

In terms of clinical limitations, the present study investigated simulated driving performance which appears to be highly dependent on attentional ability. Attentional processes are largely neuropsychological in nature and the present study did not address neuropsychological functioning in relation to subjective and objective sleepiness,

performance and mood. Future studies may wish to address possible associations between these variables. Lastly, while the BDI was a specific measure of depressive symptomatology, and the STAI provided a measure of anxiety symptoms, the POMS-SF may not have been a sensitive measure of depression or anxiety, since it also tapped in to other components of mood. Future research could refine the use of self-report inventories to ascertain a sensitive measure of depression and anxiety.

4.7. Contributions of the Present Study

There are practical and theoretical benefits of the present research. First, the results of this study contribute to the body of literature on sleepiness, psychomotor performance, driving performance and mood disturbances in patients with OSA as well as shift-workers – two populations differentially affected by sleep loss. Most previous studies have tended to investigate the effects of these variables in OSA patients and shift-workers in isolation. However, upon consideration that the sleepiness experienced by shift-workers has been shown to be equally as severe as that experienced by patients with OSA (Shen et al., 2006), the present study also employed a non-clinical population (i.e., shift-workers) in addition to a comparative control group.

The present research also utilised a variety of objective indicators of sleepiness (i.e., drowsiness monitoring and eyelid closure data, sleep latency outcome measures, and a polysomnographic sleep study), in addition to examining subjective sleepiness. Of the studies examining objective sleepiness among OSA patients and shift-workers, few have employed a multitude of objective measures for a comprehensive assessment of objective sleepiness in these populations. Since the nature of sleepiness is multi-dimensional (Franzen et al., 2008), it seems important to reflect this multi-dimensionality in the measures employed for objective sleepiness.

In addition, a selection of mood scales were employed in the present study that tapped into depressive and anxiety symptoms, as well as other mood states that may be sensitive to sleep loss and disrupted sleep. Previous literature has tended to employ mood inventories pertaining solely to depressive symptoms, with fewer studies specifically examining anxiety symptoms. The inclusion of a range of mood inventories in the present study enabled for a wide-ranging account of the mood disturbances experienced by OSA patients and shift-workers.

This study may also be unique in its attempt to document accident history collectively among OSA patients, shift-workers and controls, and ascertain associations between history of accidents in the preceding three years and subjective and objective sleepiness, performance and mood. Although numerous factors have been suggested to mediate an increased risk for motor vehicle accidents in patients susceptible to sleep loss, knowledge about the associations between these factors and accidents has been largely under-investigated. Indeed, understanding the factors that may contribute to an increased risk of accidents may assist physicians and management to alert their patients and workers respectively of the dangers of driving while sleepy as well as any other associated risks.

4.8. Summary and Conclusion

Patients with OSA and shift-workers experience constant sleep disruption despite varying aetiologies, which is likely to produce wide-ranging consequences for alertness, psychomotor functioning, driving performance and mood. This study was designed to compare subjective and objective sleepiness, psychomotor performance, simulated driving performance, and mood in patients with OSA and shift-workers relative to a control group. A further aim of this study was to determine whether significant relationships existed between these measures for the three groups. Finally, this study aimed to investigate possible associations between subjective and objective sleepiness, psychomotor functioning, driving

performance and mood with the number of accidents experienced in the preceding three years in an attempt to determine accident risk.

The main findings of the present study were that OSA patient's demonstrated significant impairments across most polysomnographic indicators of sleepiness relative to shift-workers and controls, although shift-workers experienced levels of sleepiness greater than OSA patients and controls as evidenced by eyelid closure data. Surprisingly, no deficits were indicated for subjective sleepiness measures across the groups. These findings likely indicate that the subjective consequences of OSA and shift-work are more complex and less precise relative to objective indicators.

Regarding performance variables, OSA patients were generally more impaired in their reaction times and across measures of speed variation relative to shift-workers and controls. Perhaps a novel finding of the present study was that shift-workers tended to fare better overall across most performance measures. In terms of mood disturbances, the three groups were differentially affected. Patients with OSA reported greater levels of depressive symptoms; however shift-workers endorsed higher scores across the total mood disturbance scale of the POMS, likely suggesting that they experience a multitude of mood impairments, which may be related to the sleep disruption experienced as a result of sleep restriction and circadian disruption. Unsurprisingly, OSA patients reported decreased levels of vigour relative to shift-workers and controls respectively.

The present study also demonstrated significant relationships between measures of subjective and objective sleepiness, performance and mood for OSA patients, shift-workers and control participants. A novel finding of the present study was that most subjective measures were correlated with objective indices on the PVT, driving simulator, Osler 1 and 2, and polysomnographic variables for shift-workers but not among OSA patients and controls. A substantial number of correlations were also found for objective sleepiness and

performance and objective sleepiness and mood among shift-workers. These findings attest to the importance of examining these associations further in shift-workers since they are likely to report equivalent or greater decrements in alertness compared to OSA patients.

Since few studies have documented relationships between mood and performance variables, the present study explored possible associations between these measures. This study was unique in documenting that performance on the driving simulator was the only performance variable related to mood disturbances for the three groups. These findings have important implications for road accidents, and point to the importance of further exploring relationships between mood disturbances in these populations, since it might contribute to an increased risk of accidents among OSA patients and shift-workers.

Pertaining to accident history, few of the measures incorporated in the present study (subjective sleepiness, objective sleepiness, performance and mood) revealed associations with the former. However, the present study did reveal that subjective ratings of sleepiness are likely to be related to accident history for OSA patients, while eyelid closure data and psychomotor functioning may be likely to contribute to an increased risk of accidents among shift-workers.

In essence, the findings of the present study lend support to the importance of identifying impairments related to alertness, psychomotor functioning, driving performance and mood for the minimisation of accidental injury to patients with OSA, shift-workers and the community at large. Given that decrements in these areas render negative consequences more likely, continued research which examines these effects among these populations is essential. Since treatment options are likely to improve these negative effects for OSA patients to a considerable extent, the challenges then are for management to devise more fitting shift schedules and potential intervention strategies that support health-enhancing work

environments for shift-workers. Until these recommendations are translated into practice, ‘the sleeping dog will remain asleep’.

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Appendix 1: Initial Contact Screening Questionnaire

INITIAL CONTACT SCREENING QUESTIONNAIRE

Name:

Age:

Date of birth:

Phone:

Address:

E-mail:

Drivers License (please bold): Yes No

Vision problems not corrected with glasses (bold): Yes No

Regular use of sedating medication (bold): Yes No

If so, what is it? _____

Current medications: _____

Any chronic neurological illness or significant medical problems: Yes No

If yes, what?

_____

_____

_____

Appendix 2: Epworth Sleepiness Scale (ESS)

EPWORTH SLEEPINESS SCALE (ESS)

How likely are you to doze off or fall asleep in the situations described in the box below, in contrast to just feeling tired? This refers to your usual way of life in recent times. If you haven't done some of these things recently, try to work out how they would have affected you.

Use the following scale to choose the **most appropriate** number for each situation:

0 = would **never** doze

1 = **slight** chance of dozing

2 = **moderate** chance of dozing

3 = **high** chance of dozing

Situation	**Chance of Dozing**
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g., a theatre or meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after lunch without alcohol	
In a car, while stopped for a few minutes in traffic	

Total Score =

Appendix 3: Multivariate Apnoea Prediction Questionnaire (MAPQ)

MULTIVARIATE APNOEA PREDICTION QUESTIONNAIRE (MAPQ)

During the last month, have you had, or have you been told about the following symptoms:

(Indicate the frequency by placing a cross in one box only)

0 = never

1 = rarely, less than once a week

2 = 1-2 times a week

3 = 3-4 times a week

4 = 5-7 times a week

5 = don't know

Symptoms

	0	1	2	3	4	5
Snorting or gasping	☐	☐	☐	☐	☐	☐
Loud snoring	☐	☐	☐	☐	☐	☐
Breathing stops, choke or struggle for breath	☐	☐	☐	☐	☐	☐

Appendix 4: Demographic Questionnaire

DEMOGRAPHIC QUESTIONNAIRE

1. **What is your age?** _____

2. **What is your sex?** *(please circle the number of your answer)* 1 Male 2 Female

3. **What is your weight?** _____ in kilograms

4. **What is your height?** _____ in centimetres

5. **What is your current occupation?** _____
(Please also tick one of the categories listed below to indicate your answer)

_____ **(1) Unskilled work,** such as farm labour, food service, janitor, house cleaner, factory work

_____ **(2) Skilled work**, such as technician, carpenter, hairdresser, seamstress, plumber, electrician

_____ **(3) White collar (office) work** such as clerk, salesperson, secretary, small business

_____ **(4) Professional:** doctor, lawyer, teacher, business

_____ **(5) Not currently working** (check one:)

_____ **(6) Unemployed**

_____ **(7) Retired**

_____ **(8) Homemaker**

_____ **(9) Student**

_____ **(10) Other:** please specify _____

6. **What is your highest level of education you have completed?**
(Please tick one of the categories listed below to indicate your answer)

_____ **(1) None;** 0 years

_____ **(2) 1-3 years** (some primary school)

_____ **(3) 4-6 years** (completed primary school)

_____ **(4) 7-9 years** (some secondary school)

_____ **(5) 10-12 years** (completed secondary school)

_____ **(6) Some college;** no degree

_____ **(7) College degree**

_____ **(8) Graduate or professional education**

Appendix 5: Driving Information Questionnaire

DRIVING INFORMATION QUESSTIONNAIRE

We would like to ask you some questions about driving. For the following questions, please circle the appropriate response.

1. Do you drive at work?	Yes	No	
2. Which shifts do you work?	Days	Afternoons	Nights
3. Do you rotate shifts	Yes	No	
4. Where do you drive	Metropolitan	Country	Interstate

For the following questions, please write the appropriate number on the line provided

5. How many hours is your longest shift?	_____		
6. How many days do you work per week?	_____		
7. How many hours do you work per week?	_____		
8. How many kilometres do you drive per week?	At work _____	Not work related _____	
9. How many kilometres do you drive each year?	At work _____ 000kms	Not work related _____ 000kms	
10. How many hours of sleep do you have each night?	On work days _____	On days off _____	
11. How many glasses of alcohol do you normally have each day?	On work days _____	On days off _____	
12. How many cups do you have each day of the following beverages?	Tea _____	Coffee _____	Cola _____

Most drivers have had an accident at some time. We would like to ask you about any accidents in the last 3 years. Include any accident where someone was injured, the police were called or a vehicle was damaged and required repair.

13. Have you had any motor vehicle accidents in the last 3 years?	Yes	No
14. Number of accidents involving another vehicle:	At work _____	Not work related _____
15. Number of accidents with no other vehicle involved:	At work _____	Not work related _____

Appendix 6: Karolinska Sleepiness Scale (KSS)

KAROLINSKA SLEEPINESS SCALE (KSS)

The following is a 9 point scale to describe sleepiness. Put a cross in the box next to the point that describes how *sleepy* you feel *right now*

1. ☐ Extremely alert
2. ☐
3. ☐ Alert
4. ☐
5. ☐ Neither alert nor sleepy
6. ☐
7. ☐ Sleepy - but no difficulty remaining awake
8. ☐
9. ☐ Extremely sleepy - fighting sleep

Appendix 7: Stop Driving Questionnaire (SDQ)

STOP DRIVING QUESTIONNAIRE (SDQ)

PART 1. With regards to how alert you feel, which one of the following statements best describes how you feel about driving for a short period in *suburban traffic* right now.

(TICK ONE BOX)

1. ☐ I would continue driving
2. ☐ I would continue driving only if pressured to do so
3. ☐ I would stop driving now even if under pressure to continue
4. ☐ I would have stopped driving some time ago

PART 2. With regards to how alert you feel, which one of the following statements best describes how you feel about driving for a *continuous long distance* right now.

(TICK ONE BOX)

1. ☐ I would continue driving
2. ☐ I would continue driving only if pressured to do so
3. ☐ I would stop driving now even if under pressure to continue
4. ☐ I would have stopped driving some time ago

Appendix 8: Sleepiness Symptoms Questionnaire (SSQ)

SLEEPINESS SYMPTOMS QUESSTIONNAIRE (SSQ)

Did you notice any of the following during your driving session?

Please rate how often (TICK ONE BOX IN EACH ROW)

	Not at all		Occasionally		Frequently		Most of the time
	1	2	3	4	5	6	7
Struggling to keep eyes open	☐	☐	☐	☐	☐	☐	☐
Vision becoming blurred	☐	☐	☐	☐	☐	☐	☐
Nodding off to sleep	☐	☐	☐	☐	☐	☐	☐
Difficulty keeping to the middle of the road	☐	☐	☐	☐	☐	☐	☐
Difficulty maintaining the correct speed	☐	☐	☐	☐	☐	☐	☐
Mind wandering to other things	☐	☐	☐	☐	☐	☐	☐
Reactions were slow	☐	☐	☐	☐	☐	☐	☐
Head dropping down	☐	☐	☐	☐	☐	☐	☐

Appendix 9: Beck Depression Inventory (BD)

BDI-II

Date:

Name: _____ Marital Status: _____ Age: _____ Sex: _____

Occupation: _____ Education: _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

_____ Subtotal Page 1

Continued on Back

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11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater than usual.
3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

21 22 23 24 25 26 27 28 29 30 A B C D E

_____ Subtotal Page 2
_____ Subtotal Page 1
_____ Total Score

Appendix 10: State-Trait Anxiety Inventory (STAI)

mind garden

SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-1

Please provide the following information:

Name_____ Date_____ S_____

Age_____ Gender (*Circle*) **M** **F** T_____

DIRECTIONS:

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right* now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I feel strained	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel satisfied	1	2	3	4
9. I feel frightened	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel indecisive	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel confused	1	2	3	4
19. I feel steady	1	2	3	4
20. I feel pleasant	1	2	3	4

Published by Mind Garden, Inc., 1690 Woodside Rd, Suite 202, Redwood City, CA 94061

STAIP-AD Test Form Y
www.mindgarden.com

mind garden

SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-2

Name_____ Date_____

DIRECTIONS

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
21. I feel pleasant	1	2	3	4
22. I feel nervous and restless	1	2	3	4
23. I feel satisfied with myself	1	2	3	4
24. I wish I could be as happy as others seem to be	1	2	3	4
25. I feel like a failure	1	2	3	4
26. I feel rested	1	2	3	4
27. I am "calm, cool, and collected"	1	2	3	4
28. I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
29. I worry too much over something that really doesn't matter	1	2	3	4
30. I am happy	1	2	3	4
31. I have disturbing thoughts	1	2	3	4
32. I lack self-confidence	1	2	3	4
33. I feel secure	1	2	3	4
34. I make decisions easily	1	2	3	4
35. I feel inadequate	1	2	3	4
36. I am content	1	2	3	4
37. Some unimportant thought runs through my mind and bothers me	1	2	3	4
38. I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
39. I am a steady person	1	2	3	4
40. I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

STAIP-AD Test Form Y
www.mindgarden.com

Appendix 11: Profile of Mood States – Short Form (POMS-SF)

PROFILE OF MOOD STATES – SHORT FORM (POMS – SF)

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	O	O	O	O	O	20. Discouraged	O	O	O	O	O
2. Angry	O	O	O	O	O	21. Resentful	O	O	O	O	O
3. Worn out	O	O	O	O	O	22. Nervous	O	O	O	O	O
4. Unhappy	O	O	O	O	O	23. Miserable	O	O	O	O	O
5. Lively	O	O	O	O	O	24. Cheerful	O	O	O	O	O
6. Confused	O	O	O	O	O	25. Bitter	O	O	O	O	O
7. Peeved	O	O	O	O	O	26. Exhausted	O	O	O	O	O
8. Sad	O	O	O	O	O	27. Anxious	O	O	O	O	O
9. Active	O	O	O	O	O	28. Helpless	O	O	O	O	O
10. On edge	O	O	O	O	O	29. Weary	O	O	O	O	O
11. Grouchy	O	O	O	O	O	30. Bewildered	O	O	O	O	O
12. Blue	O	O	O	O	O	31. Furious	O	O	O	O	O
13. Energetic	O	O	O	O	O	32. Full of pep	O	O	O	O	O
14. Hopeless	O	O	O	O	O	33. Worthless	O	O	O	O	O
15. Uneasy	O	O	O	O	O	34. Forgetful	O	O	O	O	O
16. Restless	O	O	O	O	O	35. Vigorous	O	O	O	O	O
17. Unable to concentrate	O	O	O	O	O	36. Uncertain about things	O	O	O	O	O
18. Fatigued	O	O	O	O	O	37. Bushed	O	O	O	O	O
19. Annoyed	O	O	O	O	O						

Appendix 12: Recruitment Advertisement

THE EFFECTS OF OBSTRUCTIVE SLEEP APNOEA AND ROTATING SHIFT WORK

A study looking at sleepiness, performance and mood impairments in shift workers and obstructive sleep apnoea patients (in comparison to a control group) is being conducted by researchers in the Sleep Disorders Unit at Austin Health.

We are looking for people aged between 18 and 65 years to participate in the study.

If you are currently employed in rotating shift-work and have been for at least 3 months, you may be eligible to participate

OR

If you have not been employed in shift work for at least 3 months, then you may be eligible to participate in the control group

- You will complete a series of questionnaires about how you have been feeling lately
- You will participate in a driving simulation task and a reaction time task
- You will participate in an overnight sleep study

Please contact:

Ms Rosa Galante on 0438 ### ### or email rosa.galante@email.au for additional information about participating in this study.

Sleep Disorders Unit

Austin Hospital

Studley Road, Heidelberg, 3084

Appendix 13: Participant Information Sheet and Informed Consent for OSA Patients, Shift-Workers and Control Participants

Participant Information and Consent Form (Obstructive Sleep Apnoea Participants)

Version 4B Dated 09.02.2010

Site: Austin Health/Victoria University

Full Project Title: An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

Principal Researcher: Dr Mark Howard

Associate Researcher(s): Dr Gerard Kennedy, Dr Maree Barnes & Miss Justine Westlake

Student Researcher: Ms Rosa Galante

This Participant Information and Consent Form is **5** pages long. Please make sure you have all the pages.

1. Your Consent

You are invited to take part in this research project designed to investigate sleepiness, performance (psychomotor performance and driving performance) and mood in shift-workers and people with obstructive sleep apnoea. This is a student project for a Doctor of Psychology (Clinical Psychology).

This Participant Information Form contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this. We cannot guarantee or promise that you will receive any benefits from this project.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project. Participation is entirely voluntary. You may withdraw from the project for any reason and at any time without prejudice and without giving any reason.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

2. Purpose and Background

This project is designed to investigate the nature and extent of subjective and objective sleepiness, psychomotor functioning, driving simulator performance and mood in patients with obstructive sleep apnoea and in shift-workers compared to people without obstructive sleep apnoea and who do not do shift-work (control group). Sleep is likely to be disrupted in patients with sleep apnoea as well as shift-workers, and this can lead to a number of consequences such as impaired performance at work or while driving a vehicle, which can increase the risk of accidental injury.

This study aims to evaluate the effect of these conditions on sleepiness, performance and mood. In particular, we are looking at how you report your sleepiness, how sleepy you actually are, your driving ability and reaction time, and how you report your mood to be. In this study, a number of tasks that measure how sleepy you are, performance on a computer-based driving task and a reaction time task, and a series of questionnaires will be used to assess your sleepiness, performance and mood. It is also aimed to relate any impairment to estimates of accident history

3. Procedure

Your participation in the study will involve attendance at the Austin Hospital over one or two sessions.

1. First, any questions you or your family members may have will be answered, and the study will be fully explained to you. If you agree to participate, you will be asked to sign the Consent Form and will also have an opportunity to practice on some of the equipment that will be used in the study. This session will take about one hour to complete.
2. On the day of testing, you will be requested not to consume any caffeine or stimulant medication until completion of the study. You will be asked to arrive at approximately 12:30pm and the session will finish at around 5:00pm. During this session, you will be asked to complete a series of questionnaires about how you have been feeling lately and about your mood. After completing these questionnaires, your level of sleepiness will be assessed through a sleep latency measure. Your performance on a driving simulator task and a reaction time task will also be assessed. In addition, you will be asked to complete a questionnaire which asks about your motor vehicle accident history for the last three years. A series of questionnaires designed to help assess levels of sleepiness will also be administered. This session will take approximately four to five hours to complete.

4. Possible Benefits

Participating in this study may not benefit you directly; however, results of this study may offer benefits to patients with obstructive sleep apnoea and shift-workers who experience disrupted sleep for different reasons. Results of this study may contribute to a greater understanding of subjective and objective sleepiness, performance and mood disturbances in patients with obstructive sleep apnoea and in shift-workers.

5. Possible Risks

There are no significant physical or psychological risks associated with participating in the study. The main inconvenience will be the time commitment involved. There is no cost for being in this study. Travel costs will be reimbursed on production of a receipt.

6. Privacy, Confidentiality and Disclosure of Information

Any information obtained in connection with this project will remain confidential. The results of the study may be published, but your identity will not be revealed, nor will your results be shared with anyone else for any other purpose. Participant records may be inspected by authorised persons for the purpose of data audit (e.g. members of the Austin Health Human Research Ethics Committee), but no other people will be authorised to access them. The records dealing with this study will be kept in safe storage for 7 years, and will then be shredded.

7. Results of the Project

At the end of the study you will receive a copy of your results and these will be explained to you by one of the researchers.

8. Further Information or Any Problems

For the duration of the study the supervisors will be Dr Mark Howard and Dr. Gerard Kennedy. If you have any questions concerning the nature of the research or your rights as a participant, please contact:

Dr Mark Howard 9496 3688

Dr Gerard Kennedy 9919 2481

9. Other Issues

If you wish to contact someone, independent of the study, about ethical issues or your rights or to make a complaint, you may contact:

Name: **Dr Andrew Crowden**

Position: **Chairperson Austin Health Human Research Ethics Committee**

Phone: **(03) 9496 2901**

Or

Name: **the Secretary**

Position: **Secretary of Victoria University Human Research Ethics Committee**

Telephone: **(03) 9919 4710**

10. Participation is Voluntary

Participation in any research project is voluntary. If you do not wish to take part, you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Your decision whether to take part or not to take part, or to take part and then later withdraw, will not affect your relationship with Austin Health or Victoria University.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the consent form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw.

11. Ethical Guidelines

This project will be carried out according to the *National Statement on Ethical Conduct in Human Research (March 2007)* produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Austin Health Human Research Ethics Committee.

CONSENT FORM
VERSION 4B DATED 09.02.2010
SITE: AUSTIN HEALTH/VICTORIA UNIVERSITY

Full Project Title: An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

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I have read, and I understand the Participant Information version 4B dated 9/02/2010.

Please tick box when signing the consent form:

I freely agree to participate in this project according to the conditions in the Participant Information, including completion of questionnaire booklet ☐ Yes ☐ No

I will be given a copy of the Participant Information and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed).....

Signature Date

Name of Witness to Participant's Signature (printed).....

Signature Date

Declaration by researcher*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's Name (printed).....

Signature Date

*A senior member of the research team must provide the explanation and provision of information concerning the research project.

Note: All parties signing the Consent Form must date their own signature.

Revocation of Consent Form

Full Project Title: An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardize any treatment or my relationship with Austin Health or Victoria University.

Participant's Name (printed).....

Signature Date

Participant Information and Consent Form (Shift-Work Participants and Control Participants)

Version 4A Dated 09.02.2010

Site: Austin Health/Victoria University

Full Project Title: An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

Principal Researcher: Dr Mark Howard

Associate Researcher(s): Dr Gerard Kennedy, Dr Maree Barnes & Miss Justine Westlake

Student Researcher: Ms Rosa Galante

This Participant Information and Consent Form is **6** pages long. Please make sure you have all the pages.

1. Your Consent

You are invited to take part in this research project designed to investigate sleepiness, performance (psychomotor performance and driving performance) and mood in shift-workers and people with obstructive sleep apnoea. This is a student project for a Doctor of Psychology (Clinical Psychology).

This Participant Information Form contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this. We cannot guarantee or promise that you will receive any benefits from this project. You will be paid $150 to compensate you for your time in participating in this study.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project. Participation is entirely voluntary. You may withdraw from the project for any reason and at any time without prejudice and without giving any reason.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

2. Purpose and Background

This project is designed to investigate the nature and extent of subjective and objective sleepiness, psychomotor functioning, driving simulator performance and mood in patients with obstructive sleep apnoea and in shift-workers compared to people without obstructive sleep apnoea and who do not do shift-work (control group). Sleep is likely to be disrupted in patients with sleep apnoea as well as shift-workers, and this can lead to a number of consequences such as impaired performance at work or while driving a vehicle, which can increase the risk of accidental injury.

This study aims to evaluate the effect of these conditions on sleepiness, performance and mood. In particular, we are looking at how you report your sleepiness, how sleepy you actually are, your driving ability and reaction time, and how you report your mood to be. In this study, a number of tasks that measure how sleepy you are, performance on a computer-based driving task and a reaction time task, and a series of questionnaires will be

used to assess your sleepiness, performance and mood. It is also aimed to relate any impairment to estimates of accident history.

3. Procedure

Your participation in the study will involve attendance at the Austin Hospital over one or two sessions.

1. First, any questions you or your family members may have will be answered, and the study will be fully explained to you. If you agree to participate, you will be asked to sign the Consent Form and will also have an opportunity to practice on some of the equipment that will be used in the study. This session will take about one hour to complete.
2. On the day of testing, you will be requested not to consume any caffeine or stimulant medication until completion of the study. You will be asked to arrive at approximately 12:30pm and the session will finish at around 5:00pm. During this session, you will be asked to complete a series of questionnaires about how you have been feeling lately and about your mood. After completing these questionnaires, your level of sleepiness will be assessed through a sleep latency measure. Your performance on a driving simulator task and a reaction time task will also be assessed. In addition, you will be asked to complete a questionnaire which asks about your motor vehicle accident history for the last three years. A series of questionnaires designed to help assess levels of sleepiness will also be administered. This session will take approximately four to five hours to complete.
3. You will then stay for an overnight sleep study in the Austin Hospital Sleep Unit (see below).
4. At 6am the following morning, you will go home.

What does the overnight sleep study involve?

The overnight sleep study takes place in the sleep laboratory. When you arrive, you will be shown to your private room. Bathroom facilities are shared. There is a small lounge/television room for your uses, and microwave/fridge facilities are available. Bring night attire, toiletries, and something to read. You are welcome to bring your own pillow. You should bring all of your own medication and take any medication as you normally would. Since caffeine is a stimulant, you are asked to refrain from drinking any coffee, tea or coke from 7am on the morning of the overnight study. If you wish, you may bring non-caffeinated drinks with you to the hospital. Alcohol should be avoided all day on the day of this study.

The sleep technician is a trained scientist or nurse who is experienced in this area. After you complete the tests for the research study, he/she will explain the equipment and procedures to you, then will attach several electrodes to your head, face, chest and legs to monitor your heart and the activity of your brain, your eyes and the muscles of your face and legs. You will also have two bands strapped around your chest and abdomen to monitor your breathing, an airflow detector attached to your nose and mouth and an oxygen sensor attached to a finger. This may sound very uncomfortable and restrictive, however, you are able to walk around, read, watch television, eat and drink. You will be asked to go to bed at around 10:00-11:00pm, and the electrodes will be plugged into a board at the head of your bed. There is an infra-red camera in your room which allows the technician to see you during the night.

4. Possible Benefits

Participating in this study may not benefit you directly; however, results of this study may offer benefits to patients with obstructive sleep apnoea and shift-workers who experience disrupted sleep for different reasons. Results of this study may contribute to a greater understanding of subjective and objective sleepiness, performance and mood disturbances in patients with obstructive sleep apnoea and in shift-workers.

5. Possible Risks

There are no significant physical or psychological risks associated with participating in the study. The main inconvenience will be the time commitment involved. There is no cost for being in this study. Travel costs will be reimbursed on production of a receipt.

6. Privacy, Confidentiality and Disclosure of Information

Any information obtained in connection with this project will remain confidential. The results of the study may be published, but your identity will not be revealed, nor will your results be shared with anyone else for any other purpose. Participant records may be inspected by authorised persons for the purpose of data audit (e.g. members of the Austin Health Human Research Ethics Committee), but no other people will be authorised to access them. The records dealing with this study will be kept in safe storage for 7 years, and will then be shredded.

7. Results of the Project

At the end of the study you will receive a copy of your results and these will be explained to you by one of the researchers.

8. Further Information or Any Problems

For the duration of the study the supervisors will be Dr Mark Howard and Dr. Gerard Kennedy. If you have any questions concerning the nature of the research or your rights as a participant, please contact:

Dr Mark Howard 9496 3688

Dr Gerard Kennedy 9919 2481

9. Other Issues

If you wish to contact someone, independent of the study, about ethical issues or your rights or to make a complaint, you may contact:

Name: **Dr Andrew Crowden**

Position: **Chairperson Austin Health Human Research Ethics Committee**

Phone: **(03) 9496 2901**

Or

Name: **the Secretary**

Position: **Secretary of Victoria University Human Research Ethics Committee**

Telephone: **(03) 9919 4710**

10. Participation is Voluntary

Participation in any research project is voluntary. If you do not wish to take part, you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Your decision whether to take part or not to take part, or to take part and then later withdraw, will not affect your relationship with Austin Health or Victoria University.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the consent form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw.

11. Ethical Guidelines

This project will be carried out according to the *National Statement on Ethical Conduct in Human Research (March 2007)* produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Austin Health Human Research Ethics Committee.

CONSENT FORM
VERSION 4A DATED 09.02.2010
SITE: AUSTIN HEALTH/VICTORIA UNIVERSITY

Full Project Title: An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

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I have read, and I understand the Participant Information version 4B dated 9/02/2010.

Please tick box when signing the consent form:

I freely agree to participate in this project according to the conditions in the Participant Information, including completion of questionnaire booklet ☐ Yes ☐ No

I will be given a copy of the Participant Information and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed).....

Signature Date

Name of Witness to Participant's Signature (printed).....

Signature Date

Declaration by researcher*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's Name (printed).....

Signature Date

*A senior member of the research team must provide the explanation and provision of information concerning the research project.

Note: All parties signing the Consent Form must date their own signature.

Revocation of Consent Form

Full Project Title: An Investigation of Subjective and Objective Sleepiness, Performance and Mood in Patients with Obstructive Sleep Apnoea and Shift-Workers

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardize any treatment or my relationship with Austin Health or Victoria University.

Participant's Name (printed).....

Signature Date

Appendix 14: Participant Score Profile Sheet

PARTICIPANT SCORE PROFILE

Demographics		BDI		
Subject ID		Total Score		
Age		STAI		
Gender		STAI- State Anxiety		
Subject Type		STAI- Trait Anxiety		
Height		POMS-SF		
Weight/BMI		Tension-Anxiety		
Occupation		Depression-Dejection		
Occupation level		Confusion-Bewilderment		
Education level		Vigour		
ESS Score		Anger-Hostility		
KSS Scores		Fatigue		
KSS Mean		Total POMS		
PVT		SDQ		
Median RT		Suburban traffic		
Slowest 10%		Long distance		
Lapses		MAPQ		
Average JDS		Total MAPQ		
% Long Closure		Driving Information		
Driving Simulator		Drive at work – Y/N		
Pos Median Average		Shifts worked		
Speed 70 Average		Rotating shifts?		
Speed 60-80 Average		Driving locations		
RT Median		Longest shift (hrs)		
Crash Number		Days worked per week		
Average JDS		Hours worked per week		
% Long Closure		Hrs driven/week at work		
Osler		Hrs driven/week no work		
Sleep Latency		Km's driven/yr at work		
Osler total misses		Km's driven/yr no work		
Osler 1 Average JDS		Hrs sleep/night at work		
Osler 1 % Long Clos.		Hrs sleep/night days off		
Osler 2 Average JDS		Glasses alcohol –work		
Osler 2 % Long Clos.		Glasses alcohol – no work		
SSQ		Tea per day		
Total score		Coffee per day		
Polysomnography		Cola per day		
AHI Total per hour		Accident History		
Min Sp02		Accidents in last 3 years		
Arousals total events per hour		No. involving another vehicle	At work	Not at work
Sleep efficiency		No. Involving no other vehicle	At work	Not at work
Time in REM				