Sleep inertia and alcohol impairment in young adults: Neurocognitive effects and interactions

Implications for fire escape behaviours

Melanie Joy Tokley, BPsyc(Hons)

Submitted in partial fulfilment of the requirements of the degree of

Doctor of Psychology (Clinical Neuropsychology)

School of Social Sciences & Psychology

Victoria University, Melbourne AUSTRALIA

June 2009

ABSTRACT

Alcohol intoxication is known to considerably increase the probability of death from fire across the lifespan, to the extent that it has been isolated as the single most significant risk factor. The study investigated the combined effects of sleep inertia and alcohol impairment on fire emergency-relevant cognitive performance indicators in a young adult population. Mental tracking, visual scanning, psychomotor speed, working memory and sustained, selective, and divided attention functions were assessed for performance decrements and reference to speed-accuracy trade-off effects. Participants were 24 young adults (18-26 years) who participated in a repeated-measures study over 2 non-consecutive nights; 1 night with alcohol administration and 1 'sober' night. During the alcohol administration night, 10-minute testing blocks occurred under (1) baseline sober and (2) baseline 0.05 blood alcohol concentration (BAC) conditions. Subsequently, subjects were awoken from stage 4 sleep and assessed in two consecutive 10-minute blocks (3) and (4). Self-reports of sleepiness and clearheadedness were also taken. The same procedure was used during the sober night (with condition (2) excluded). All cognitive functions assessed showed an alcohol effect (i.e., decrements between sober baseline (1) and conditions of alcohol (2)), and an even larger sleep inertia effect (i.e., greater decrements between sober baseline (1) and conditions of sleep inertia alone (3) and (4)). Sleep inertia selectively affected performance *speed* on the working memory task, whilst performance *accuracy* on this task was affected by conditions of alcohol impairment only. When the sober and alcohol nights were compared, there was no combined or synergistic interaction between sleep inertia and alcohol impairment on the cognitive tasks or subjective measures during the first 10 minutes of sleep inertia. Conditions of combined sleep inertia and alcohol impairment produced cognitive performance decrements that were greater than those produced by alcohol administration alone, but not those produced by sleep inertia alone. Indeed, sleep inertia effects appeared to 'override' alcohol effects. At 10-20 minutes post-awakening, however, sleep inertia effects began to dissipate, thus 'unmasking' alcohol effects on some tasks, including accuracy of working memory performance, selective/sustained attention and subjective sleepiness. Divided attention performance demonstrated a complex and

unpredictable pattern at 10-20 minutes post-awakening whereby performance under conditions of sleep inertia and alcohol intoxication combined became significantly worse than either condition alone, indicating that alcohol effects were also possibly being unmasked for this measure. Other measures (speed of working memory performance and subjective clearheadedness) showed no difference between conditions at 10-20 minutes post-awakening. The results suggest that (1) moderate alcohol impairment and sleep inertia do not combine to produce further decrements in neurocognitive functioning than those caused by the effects of alcohol or sleep inertia alone and (2) sleep inertia poses a greater risk to fire emergency escape than moderate alcohol impairment. Moreover this data suggests that when awoken abruptly in an emergency situation, prior alcohol consumption to 0.05 BAC will not further impede cognitive functioning that is already compromised by a state of sleep inertia. The study considers the importance of the arousing effects of task complexity, the BAC curve (particularly the descending limb), and speed-accuracy trade-off effects in predicting the effects of sleep inertia and alcohol on cognitive performance.

DECLARATION

I, Melanie Tokley, declare that the Doctor of Psychology (Clinical Neuropsychology) thesis entitled "Sleep inertia and alcohol impairment in young adults: Neurocognitive effects and interactions" is no more than 40,000 words in length, exclusive of tables, figures, appendices, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

Signature:  Date: 9th September 2009

ACKNOWLEDGEMENTS

This thesis would not have been possible without the support and guidance of my wonderful supervisors Professor Dorothy Bruck and Dr. Michelle Ball. It has been an incredible privilege to work with both of you, thank you for your endless encouragement.

I would also like to thank my patient and precious family. Particularly my mum, Louella, who shared in every triumph and tribulation of the writing process and provided much needed optimism when mine almost failed me. Thank you to my Dad, Michael, for the ongoing support and unflagging belief in my abilities.

Thank you to my colleagues in the 2006 neuropsychology doctoral intake whose friendship and support has been one of the highlights of my journey through post-graduate study. A special thank you to our course co-ordinator, Dr. Alan Tucker, whose incredible wisdom and mentorship will never be forgotten.

Thank you to the participants of this study for their co-operation and enthusiasm, and to the sleep technicians for their assistance and diligence.

This research was financially supported by the National Fire Protection Association's Fire Protection Research Foundation via a grant from the US Fire Administration. The study formed part of a larger research project that was a collaboration between the School of Social Sciences & Psychology and the Centre for Environmental Safety and Risk Engineering (CESARE) at Victoria University.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

CHAPTER ONE: INTRODUCTION

Sleep inertia is a psychophysiological phenomenon experienced upon awakening from sleep. It is characterised by a period of hypovigilance and impaired cognitive and behavioural functioning, and is subjectively experienced as grogginess and disorientation. The decrements in performance associated with sleep inertia are especially prominent when sleep is interrupted and therefore awakening is abrupt (Wertz, Wright, Ronda, & Czeisler, 2006). This has significant ramifications for people who are required to make important decisions soon after awakening, including on-call emergency workers and other persons who are required to fight for their immediate safety and survival in an emergency situation (Scheer, Shea, Hilton, & Shea, 2008). For example, the sleep inertia phenomenon has relevance to a person's ability to function in an emergency fire situation where arousal from sleep is typically sudden and unexpected. Numerous studies show that fatal residential fires are most likely to occur during the nocturnal period, approximately 11pm to 7am (Barillo & Goode, 1996; Runyan, Bangdiwala, Linzer, Sacks, & Butts, 1992). Indeed, Australian statistics show that over two-thirds of fire victims were sleeping at the time of the fire and that 86% of victims who died in a house fire between 8pm and 8am were reported to be asleep (Brennan, 1998). Further, over 30% of victims were asleep when fires occurred during the day (8am to 8pm; Brennan, 1998). In an international context, research from around the developed world demonstrates that being asleep in a residential home is a significant risk factor for fire fatality (Barillo & Goode, 1996; Karter, 1986; Runyan et al., 1992)

Other factors that are known to increase one's risk for fire fatality include age (i.e., being very young or very old; Australasian Fire Authorities Council, 2005; Barillo & Goode, 1996; Brennan, 1998; Karter, 1986; Marshall et al., 1998; Runyan et al.,1992; Sekizawa, 1991) and being under the influence of alcohol. Statistical data from the UK, USA, and Australia has consistently shown that alcohol is typically implicated in over 50% of fire fatalities. This is particularly true for persons in the young and middle aged adult groups (i.e., 18 to 65 year olds), significantly elevating the risk for fire fatality in otherwise unimpaired persons (Marshall et al., 1998). Further to this, research has shown that

alcohol has also contributed to the number of deaths that occur outside of this age group, extending to both children and the elderly. For example, adult carers and guardians have been reported to be alcohol-affected in up to 15% of juvenile fire-related deaths (Marshall et al., 1998). To that end, alcohol intoxication is known to considerably increase the probability of death from fire across the lifespan to the extent that it has been isolated as the single most significant risk factor (Runyan et al., 1992).

1.1 Alcohol and fire

Research has identified alcohol use as a key factor in fire fatalities since the 1970s. Upon examination of all deaths due to 'rapid' fire in the state of Maryland, USA between 1972 and 1977, Berl and Halpin (1978) found that 50% of victims aged over 20 years, and approximately 70% of victims aged 30-60 years were substantially intoxicated (i.e., blood alcohol concentration (BAC) > 0.10) at the time of death. Similarly, in a study examining coronial data, Paetta and Cole (1990) retrospectively found that 56% of fire victims in North Carolina, USA in the year 1985 tested for the presence of alcohol had BACs at or exceeding 0.10. For fire deaths in this region during the period 1988 to 1999, medical records revealed that 53% of those tested for the presence of alcohol showed legal levels of intoxication (i.e., BAC > 0.10; Marshall et al., 1998). Alcohol was detected in the systems of 29.5% of New Jersey fire victims between 1985 and 1991 (Barillo & Goode, 1997), and 62% of fire victims from Scotland, UK in the decade spanning 1980 to 1990 (Squires & Busuttil, 1997). Between 1992 and 1996, 41% of all fire deaths in Sweden were associated with alcohol use (Sjogren, Eriksson, & Ahm, 2000). Locally, Watts-Hampton, Bruck and Ball (2007) examined Victorian coronial data for the period February 1998 to June 2005 to investigate the role of mental illness and alcohol use in accidental fires. They found that 71.2% of fire victims with a pre-existing or current probable or definite diagnosis of mental illness had a BAC of 0.05 or greater. Similar levels of alcohol intoxication were found in 35% of fire victims without a psychological diagnosis.

These sobering statistics are testament to the significant role alcohol plays in fire fatalities. The exact *nature* of the role that alcohol plays in increasing a person's

vulnerability to fire death is still unknown however. There are a number of ways in which alcohol intoxication can interfere with one's successful escape from an emergency fire situation. Does alcohol intoxication affect a person's ability to awaken to their smoke alarm? Does it impair their ability to correctly interpret a smoke alarm? Does it impair their ability to heed the warning provided by their smoke alarm? Does it impair their ability to perform behaviours congruent with safe evacuation? Does it lead to more unsafe behaviours that result in a victim being intimate with the fire from its onset? A recent Australian study, using a sample of young adults, found that alcohol intoxication significantly reduced the ability to awaken to an auditory alarm (Ball & Bruck, 2004). The role of alcohol in impairing the performance of effective escape behaviours following awakening to a smoke alarm is as yet uninvestigated.

1.2 Aims

The current study aimed to further contribute to understanding fire death vulnerability afforded by alcohol intoxication. Specifically, it investigated people's ability to perform effective escape behaviours during conditions of acute sleep inertia whilst under the influence of moderate levels of alcohol. The extent to which alcohol impairment interacts with sleep inertia is unknown. It is possible that alcohol synergistically interacts with sleep inertia to produce performance deficits greater than the sum of those afforded by alcohol impairment and sleep inertia alone.

Commensurate with Ball and Bruck (2004), the current study targeted young adults, a population of somewhat less experienced drinkers than older persons and whose lifestyle typically affords an increased opportunity for alcohol consumption. Young adults are also a population whose alcohol use significantly elevates their risk for death in a fire (Marshall et al., 1998). The current study specifically selected performance tasks to accurately capture the cognitive functions that underpin the performance of an effective evacuation during an emergency fire situation.

1.3 Problem summary

In an attempt to offset the social and economic burden of fires and fire fatalities, considerable research has been dedicated to isolating the factors that contribute to risk of death in a fire. Alcohol has emerged as the single most significant risk factor. The current research aims to contribute to this important body of literature by investigating the interactive effects of alcohol impairment and sleep inertia on cognitive functions that underpin effective emergency fire escape behaviours. The wider Australian community was reminded of the importance of effective emergency fire escape behaviours in the wake of the devastating February 2009 Victorian bushfires.

CHAPTER TWO: LITERATURE REVIEW

2.1 Sleep inertia

Sleep inertia is a ubiquitous phenomenon and a significant aspect of human sleep/wake behaviour (Dinges, 1990). It is a transient state of decreased arousal and disorientation occurring immediately after wakening from sleep. Noting a significant behavioural slowness following sleep interruption, Broughton (1968) first referred to the sleep inertia phenomenon as 'sleep drunkenness'. The same state has also been named 'post-sleep disorientation', again referring to behavioural decrements, before Lubin and colleagues (1976) coined the term 'sleep inertia' in reference to the comparatively lengthier sleep-wake than wake-sleep transition. Sleep inertia has been described as a paradoxical phenomenon in reference to the observation that performance is more impaired immediately following waking from sleep than prior to sleep, despite one having fully dissipated their sleep need (Folkard & Åkerstedt, 1992). By exploring the theoretical hypotheses of sleep inertia in a chronological sequence, it is possible to fully appreciate the current understanding and conceptualisations of this complex and paradoxical process. The following sections discuss the major theoretical models of sleep inertia.

2.1.1 Arousal hypothesis

Sleep inertia was, retrospectively, first described in the context of arousal theories. The arousal hypothesis dictates that sleep inertia can be conceptualised as a period of lowered arousal, where levels of cerebral activation and subcortical arousal represent an incomplete neurophysiological disengagement from the sleep process. It is proposed that this causes a general cognitive slowing that is responsible for the behavioural changes noted during the sleep inertia period (Tassi & Muzet, 2000).

Lindsley's neurophysiological investigations into activation, described in Malmo's (1959) paper, were amongst the first to use electroencephalogram (EEG) recordings to identify that "light sleep and drowsy states" (Malmo, 1959, p. 367) were characterised by electroencephalographic frequencies that were not as low as those found during deep sleep, yet lower than the frequency waves found in awake states. Lindsley was also the

first to link lesions in the ascending reticular activating system (a portion of the reticular formation found in the brainstem) with "a behavioural picture of lethargy and somnolence" (Malmo, 1959, p. 368). This was one of the earliest documentations of a relationship between neurophysiologically measured intensity of arousal and activation, and level of performance. Shortly following this, the transition between sleep and wake was identified as a distinct state, and researchers began to attempt to define it in neurophysiological terms.

In 1968, Broughton showed that the amplitude and latency of visual evoked potentials (VEP) recorded on awakening were more approximated with those recorded during sleep than to baseline waking recordings. Further to this, when participants were awoken from slow wave sleep, VEP recordings showed polysomnographic carry-over of typical slow wave sleep components, and even in the absence of these components, decreased amplitude and increased latency of 100-300 ms VEPs were consistently observed. These changes in VEP were not observed following waking from rapid eye movement (REM) sleep. Broughton concluded that these results represented changes in cerebral responsiveness, due to a functional deafferentation of the cerebral cortex occurring during slow wave sleep and after slow wave sleep awakenings. He postulated that this observed impairment in cerebral responsiveness may be the source of the confusion that had been anecdotally reported following awakening from deep sleep. Although these early findings represent a relatively primitive model of sleep inertia, Broughton was the first to theoretically account for a greater behavioural impairment following slow wave sleep than REM sleep. Indeed, consistent with the arousal hypothesis, Broughton showed polysomnographic evidence that lower levels of brain activation in slow wave sleep (*cf.* light sleep or REM sleep) affected the subsequent severity of sleep inertia, due, it was assumed, to an incomplete disengagement from the prior sleep state.

As measuring techniques became more sophisticated and knowledge of sleep parameters more advanced, EEG investigations further illuminated the neurophysiological processes associated with sleep inertia. Ogilvie and Simons (1992) were surprised to find that the sharp changes in EEG noted during the transition from wake to sleep did not occur as

rapidly during the transition from sleep to wake. They measured electroencephalographic activity in 12 adults who were asked to respond to auditory tones heard at anytime throughout the night, so that spontaneous sleep-wake transitions could be investigated. They found an unpredicted gradual and continued drop of theta and delta power, associated with deep slow wave sleep, occurring well into the first few minutes of wakefulness, as determined by behavioural responses to the tones. They noted that although theta and delta power had decreased by 50% at the first behavioural response following spontaneous awakening, it was still evident in participants' EEG recordings well into the wakeful state. Only at 70 seconds following the behavioural response had delta frequencies diminished to the extent that they were now statistically distinguishable from delta frequencies evident during sleep. The results for theta power showed a similar trend. Ogilvie and Simons concluded that the slower changes in EEG power observed during sleep to wake transitions (*cf.* wake to sleep transitions) were evidence that the "EEG substrate of sleep continues into wakefulness" (p. 85). They regarded these findings as a physiological explanation for the phenomenon of sleep inertia, known then as 'sleep drunkenness'. The electroencephalographic pattern of increased EEG power in the delta-theta band and decreased power in the beta range is now considered the "spectral EEG signature" (Ferrara et al., 2006, p. 237) of sleep inertia.

Cerebral blood flow was another physiological substratum investigated to further delineate the neurophysiological parameters of sleep inertia. In the context of the arousal hypothesis, studies on cerebral blood flow (e.g., Meyer, Ishikawa, Hata, & Karacan, 1987) and cerebral blood flow velocities (e.g., Kuboyama et al., 1997) during sleep were used as indicators of underlying neuronal metabolism and activity. Consistent with the basic tenet of the arousal hypothesis, studies demonstrated that blood flow characteristics following nighttime and morning awakenings were different to those found during the day. Further, Hajak and colleagues (1994), who simultaneously measured cerebral haemodynamics and polysomnography, found that on morning awakening it took up to 30 minutes for cerebral blood flow velocities to reach baseline levels as recorded during wake time the previous evening. They also discovered an uncoupling of electrical activity and cerebral perfusion during the sleep-wake transition. Hajak and colleagues

concluded that this dissociation was testament to the slow and complex nature of the sleep-wake transition.

Balkin and colleagues (2002) noted a similar dissociation in a behavioural context. They observed that awakening from sleep involves a rapid return of consciousness, but a relatively slower re-establishment of alertness that can take 20-30 minutes. They used Positron Emission Tomography (PET) studies to investigate the regional patterns of brain activity mediating these dissociated processes. Their results demonstrated that following awakening from sleep, cerebral blood flow is most rapidly restored to central brain regions including the brainstem and thalamus, suggesting that reactivation in these areas is associated with the re-establishment of conscious awareness. Anterior cortical regions showed increases in perfusion up to 15 minutes post-awakening which led the authors to conclude that dissipation of the behavioural and alertness decrements associated with sleep inertia is underpinned by reactivation of these frontal brain regions. Functional imaging studies of regional cerebral blood flow during sleep inertia indicate that the brain regions subserving higher-order cognitive functions such as working memory, planning, sequencing, and executing goal-directed behaviours, are the slowest to achieve waking activity levels at 20 minutes post-awakening (Balkin et al., 2002).

According to the arousal hypothesis, the performance decrements observed during sleep inertia are due to a lowered level of physiological arousal (i.e., still more or less synchronised EEG, reduced blood perfusion, etc.) which causes a general slowing down of cognitive processing. The arousal hypothesis also accounts for the known amplification of performance decrements associated with arousal from slow wave sleep compared to REM, stage 1, or stage 2 sleep (see section *2.2.1* below), as EEG content and blood perfusion levels are still characteristic of sleep following awakening from slow wave sleep. The arousal hypothesis predicts that the more synchronised EEG content is, and the closer other neurophysiological measures are to sleep content, the more performance is likely to be deteriorated during the sleep inertia period. As shown by a number of electrophysiological and metabolic measures, sleep inertia is a slow and complex transitional process.

2.1.2 Dinges' (1990) pressure for sleep model

Similar to the arousal hypothesis, Dinges (1990) also viewed sleep inertia as a period of cognitive slowing, but felt it was owing to a number of factors that were the result of increased pressure for sleep. Dinges incorporated sleep inertia into a theoretical framework that posited sleep pressure as "a common process underlying hypnopompic, hypnagogic, and sleep-related waking reverie" (p. 161). Whilst he acknowledged the experimental evidence showing that sleep inertia performance decrements were greater following slow wave sleep than REM or light sleep (e.g., Bonnet, 1985; Feltin & Broughton, 1968; Wilkinson & Stretton, 1971; see section *2.2.1* below), Dinges believed that the extent of hypnopompic reverie and sleep inertia performance decrements was ultimately due to pre-awakening sleep *depth*, not pre-awakening *stage* of sleep. Because sleep depth is determined by prior sleep deprivation, which increases sleep pressure (i.e., the probability of transition from wakefulness to sleep), sleep pressure, he posited, is a key factor in determining sleep inertia and hypnopompic processes.

Specifically, sleep pressure enhances the physiological processes associated with sleep. That is, increased sleep pressure causes an increased pressure for thermoregulatory down-regulation, a known physiological response associated with the onset of sleep (Kräuchi, Cajochen, & Wirz-Justice, 2004), which in turn reduces cerebral metabolic activity. These physiological changes are directly correlated with increased sleep depth which also increases the amount of slow wave sleep obtained and the awakening threshold. The consequence of these factors is increased hypnopompic reverie and increased cognitive and performance decrements during sleep inertia. Indeed, research has shown a significant covariation between body temperature (measured orally) and performance upon awakening (Rosa & Bonnet, 1985). The global metabolic decline in cortical activity that covaries with thermoregulation and with pressure (and therefore with depth) of sleep makes it "exceedingly difficult for a person to perform well if aroused abruptly from sleep when basal metabolic levels are low" (Dinges, 1990, p. 170). Dinges hypothesised that this would be true for all types of neurocognitive processes, particularly those requiring memory and attentional functions. According to Dinges' model, the cognitive and behavioural effects of sleep inertia have a time course greater than that

detectable on EEG because metabolic brain activity is relatively slower to recover than electrical brain activity following deep sleep. This is commensurate with the findings of Balkin and colleagues (2002) and Hajak and colleagues (1994).

Dinges' (1990) sleep pressure model of sleep inertia and hypnopompic processes was consistent with the basic tenet of the arousal hypothesis, yet acknowledged and drew together a number of other sleep factors that contributed to the nature of sleep inertia. It was from here that models of sleep inertia became increasingly sophisticated to also incorporate the influences of circadian, ultradian and homeostatic processes.

2.1.3 Folkard and Åkerstedt's (1992) three-process model

Folkard and Åkerstedt (1992) built upon Borbély's (1982) two-process model of sleep regulation to include a sleep inertia component in order to more accurately predict alertness levels at any point on any given sleep-wake schedule. The seminal two-process model of sleep regulation dictates that sleep propensity is determined by a circadian factor (Process C) and a homeostatic sleep factor (Process S; Borbély, 1982). Process C refers to the endogenous circadian rhythm; a sinusoidal component that is inversely related to the core body temperature rhythm and is relatively unaffected by small changes in sleep timing or extent of prior wakefulness. Process S is a homeostatic component that refers to the exponential decrease in alertness with continuous wakefulness. Its reversal during sleep is dependent on delta wave activity (i.e., deep sleep), and alertness shows an exponential growth during sleep. In the two-process model, Process S and Process C hypothetically interact to influence an individual's sleepiness or propensity for sleep (Borbély, 1982).

Folkard and Åkerstedt (1992) wished to theoretically account for the sleep inertia phenomenon by proposing a third process, Process W. They described Process W as a temporary increase followed by a rapid decay of sleep propensity immediately upon awakening from a substantial period of sleep or following an enforced awakening. It was described as a transitory exponential deviation from Process S, reflecting a wake up effect. Folkard and Åkerstedt's model was mathematical in concept and empirically

based. It is an even more sophisticated model of sleep inertia and sleep processes that integrates the variables identified by Dinges (1990) into a mathematical model that establishes the continuous nature of the sleep-wake transition and can predict alertness in any sleep/wake schedule.

Alexander Borbély, author of the original two-process model of sleep regulation stated recently that "it is inevitable that the two-process model evolves to accommodate new data generated by advances of recording and analysis techniques" (Borbély, 2009, p. 1). Indeed, Achermann and Borbély (1994) had further refined the three-process model and modified Process W to account for both sleep inertia and "wake inertia", which reflects the time taken to fall asleep, or sleep-onset latency. Further to this, additional mathematical models have been proposed to account for 1) regional differences in slow-wave activity across the cortex during sleep, proposed as Process Z (see Zavada, Strijkstra, Boerema, Daan, & Beersma, 2009), and 2) changes in subjective alertness and neurobehavioural performance (or 'cognitive throughput') during sleep inertia (e.g., Jewett et al., 1999; Jewett & Kronauer, 1999). Using data from sleep inertia studies, sleep deprivation studies initiated across all circadian phases, 28-hour forced desynchrony protocols, and alertness and performance dose response curves to sleep, Jewett and colleagues constructed initial models of subjective alertness and neurobehavioural performance which were then refined using data from over 100 studies using 30- to 50-hour sleep deprivation protocols in which subjects awoke at their habitual times. Their models of subjective alertness and neurobehavioural performance are determined by the interactions between Process S, Process C and Process W, in addition to incorporating the effect of light (Process L) on the circadian component (Process C). In their model, predictions of alertness and neurobehavioural performance are based on an asymptotic dissipation of sleep inertia (Process W), and represent the levels of each that would be expected if a person were to be awoken at any particular moment during sleep.

Whilst most mathematical models of neurobehavioural performance and alertness predict interactions between homeostatic, circadian and sleep inertia components, there are often

fundamental differences in their equations as a function of different assumptions regarding the underlying physiology (Klerman & St. Hilaire, 2007). Indeed, despite the comprehensive nature of these descriptive and predictive models, the underlying physiological mechanisms of sleep inertia are largely unknown (Kräuchi et al., 2004). Nonetheless, mathematical models of performance and alertness have both scientific and practical applications, particularly in the context of predicting performance abilities under a variety of sleep-wake schedules, including rotating shift work and transmeridian travel (Jewett & Kronauer, 1999).

2.2 Refining the models: The parameters of sleep inertia

Whilst the above theories have helped scientists and sleep researchers to conceptualise and understand sleep inertia as a biological process, it is the experimental data that has really helped to refine our understanding of the sleep inertia phenomenon and delineate the factors and contingencies that predict its effects. A number of factors that influence the sleep inertia process have been identified through a myriad of experimental investigations. These include pre-awakening stage of sleep, sleep deprivation, and circadian and time-of-day effects. The time course of sleep inertia is another important factor to consider.

2.2.1 Pre-awakening stage of sleep

A number of studies have shown that the pre-awakening stage of sleep is an important determinant of sleep inertia. As described earlier, Broughton (1968) first documented physiological evidence of sleep inertia following awakening from slow wave sleep, but found no such physiological evidence following awakening from REM sleep. Through a myriad of experimental research, Broughton's findings have been verified from a behavioural and cognitive perspective. For example, Stones (1977) and Bonnet (1983) found impairments in both immediate and delayed recall of a list-learning task following awakening from stage 4 (deep) compared with stage 2 (light) sleep. Webb and Agnew (1964) investigated discrimination reaction times and overall efficiency in completing a serial response task. They found significant performance decrements when participants were awoken from stage 4 sleep during an afternoon nap, representing a 12% decline in

performance compared to pre-nap levels. Significant decrements in reaction time were also found by Feltin and Broughton (1968) following awakening from slow wave sleep compared to REM sleep. Similarly, Silva and Duffy (2008) found that older adults performed more poorly on the Digit Symbol Substitution Test, a test of divided attention and psychomotor speed, when awoken from non-REM compared to REM sleep stages during the biological night. In an experimental nap paradigm, Stampi, Mullington, Rivers, Campos & Broughton (1990) used the Memory and Search Test and the Descending Subtraction Task (DST), a complex subtraction task with a high working memory load, to investigate cognitive performance following naps of varying lengths. They found increased performance decrements on the DST following awakening from nap schedules that were long enough to initiate slow wave sleep, but not quite long enough for participants to cycle through slow wave sleep and return to light sleep prior to awakening. For example, performance was most impaired following a 50-minute nap schedule as participants were most likely to be woken out of slow wave sleep 50 minutes post-sleep onset. In the 20-minute and 80-minute nap schedules, however, there was either no opportunity for slow wave sleep to occur (20-minute nap condition), or participants had cycled through slow wave sleep and back into light sleep or into REM sleep prior to forced awakening (80-minute nap condition), and hence sleep inertia effects were substantially less. On the Memory and Search Test however, performance decrements increased linearly with increased nap time. That is, participants performed 3%, 8%, and 14% below pre-sleep levels during the 20-minute, 50-minute and 80-minute nap conditions, respectively.

Tebbs and Foulkes (1966) found no effect of pre-awakening stage of sleep on motor grip strength. In their study, strength of grip was consistently but insignificantly poorer following REM awakenings than non-REM awakenings. Similarly, Koulack and Schultz (1974) found no difference in participants' ability to perform psychomotor and vigilance tasks following nocturnal awakenings from REM and non-REM sleep stages. They did find, however, that higher eye-movement density during REM sleep was associated with poorer performance on a vigilance task following awakening from REM sleep, compared with REM awakenings characterised by lower eye-movement density. It is possible that

cognitive performance but not motor performance is affected by pre-awakening stage of sleep (e.g., Ferrara, De Genarro, & Bertini, 2000a). Further, decreased vigilance is typically associated with sleep deprivation but not sleep inertia (see section *2.3.4.2* below), so had Koulack and Schultz (1974) used tasks tapping cognitive or neuropsychological function, they may have found differences in participants performance following REM and non-REM nocturnal awakenings. Nonetheless, the general consensus regarding the effect of pre-arousal stage of sleep on sleep inertia is that awakening from REM sleep represents intermediate effects. Performance is most greatly affected following awakening from deep slow wave sleep, whilst there is little evidence of sleep inertia-related performance decrements following awakenings from light (stage 1 and 2) sleep (Bonnet 1983; Stones, 1977; Tassi & Muzet, 2000). This is consistent with the predictions of the arousal hypothesis and Dinges' (1990) pressure for sleep model. According to the arousal hypothesis, the deeper the sleep stage prior to awakening, the greater the level of cerebral deactivation and therefore the slower the cognitive processing upon awakening. Dinges' model predicts that greater sleep depth, through a variety of mediating physiological factors, ultimately intensifies sleep inertia performance decrements.

2.2.2 Prior sleep deprivation

Prior sleep deprivation has also been shown to affect the magnitude of sleep inertia. Using an experimental nap paradigm, Dinges, Orne and Orne (1985) and Dinges, Orne, Whitehouse and Orne (1987) argued that sleep deprivation increased the amount of slow wave sleep obtained during 2-hour naps, thereby enhancing the deleterious effects of sleep inertia at abrupt post-nap awakening. Dinges et al. (1985) assessed reaction time (efficiency at answering a telephone call that terminated the 2-hour nap) and working memory performance (performance on the DST), immediately following a 2-hour nap placed at either the circadian peak or trough in body temperature. This represented periods of decreased and increased sleep propensity, respectively. The 2-hour naps were preceded by either 6, 18, 30, 42 or 54-hours of sleep deprivation. Dinges et al. found that whilst reaction time performance was most closely associated with pre-awakening stage of sleep, performance on the DST showed a linear decrease as a function of the amount

of slow wave sleep obtained during the nap. The longer participants were deprived of sleep, the more slow wave sleep they obtained in the 2-hour nap period and the more impaired their performance on the DST at awakening. Specifically, participants with 6-hours of prior sleep deprivation performed 26% below pre-nap performance, whilst participants in the 18- and 30-hour sleep deprivation conditions performed 38% below pre-nap levels, and participants in the 42- and 54-hour sleep deprivation conditions exhibited the greatest decrements, performing 71% below pre-nap performance. This linear increase in cognitive performance decrements following longer periods of sleep deprivation and increased amount of slow wave sleep obtained during a 2-hour nap was irrespective of sleep stage prior to awakening. However, contrary to speculations that it is the amount of slow wave sleep accumulated rather than the pre-awakening stage of sleep that influences the severity of sleep inertia, Maloney (2001) found no increase in sleep inertia effects on cognitive performance when comparing awakenings from the first to the second nocturnal slow wave sleep period. As Maloney's participants were not sleep deprived however, it is possible that the *combination* of sleep deprivation and increased slow wave sleep causes dose-dependent changes in cognitive performance during sleep inertia.

Balkin and Badia (1988) found a similar 'dose-dependent' increase in sleep inertia following only a small amount of sleep deprivation. Using a sleep restriction/disruption procedure in which participants slept for only 5-hours per night across four nights and were awoken each hour to perform a mathematical addition task, Balkin and Badia found performance changes suggesting that sleep inertia was enhanced by sleep deprivation in a dose-dependent fashion. Specifically, mean performance on the addition task for each night declined from 18.7 on night 1 to 10.2 on night 4. The mean number of problems attempted during the testing sessions within each night declined across nights 1 to 4, whilst the mean percentage of errors increased from 10% to 14% to 21% to 23% across testing nights. Slight performance improvements, both speed and accuracy related, were seen *within* testing nights to a similar magnitude as the decrements observed *across* testing nights. That is, error rates across the three post-awakening tests within each 20-

minute testing session declined from 25% to 10% and the mean number of addition problems attempted (speed of performance) increased from 15.2 to 18.5.

These results show that accumulating sleep deprivation over a number of nights increases the magnitude of sleep inertia performance decrements. Balkin and Badia (1988) noted, however, that the degree of performance fluctuations was similar across testing nights 1 to 4 and across post-awakening tests within each testing session *within* each night. That is, errors rates tended to decline as sleep inertia dissipated, at a similar rate that error rates increased as sleep deprivation increased across testing nights. These findings led Balkin and Badia to question whether sleep inertia effects are qualitatively different from sleep deprivation effects. The similarities between sleep inertia and sleep deprivation will be discussed more fully in section *2.3.4.2* (see below).

Using a short, one-night partial sleep deprivation paradigm, Tassi et al. (1992) found that pre-awakening stage of sleep was more predictive of sleep inertia effects than amount of sleep loss. Participants were either awoken at 1am or 4am following a 1-hour nap and performed a spatial memory task. Although there was greater sleep loss in the 4am condition, performance decrements were most adverse following the earlier awakening due to a greater likelihood that awakenings would occur out of slow wave sleep. Although the amount of prior sleep deprivation in this study was minimal, it highlights that there is a complex interplay between pre-awakening stage of sleep, prior sleep deprivation, and also a potential influence of circadian factors, in determining sleep inertia effects.

2.2.3 Circadian rhythm and time-of-day effects

The above studies demonstrate that sleep inertia effects can vary following nap sleep versus nocturnal sleep due to a number of potential factors including circadian rhythmicity, and therefore the timing of sleep periods. A number of sleep inertia studies using a variety of experimental paradigms, have hinted at the effects of a circadian rhythm influence on sleep inertia (e.g., Dinges et al., 1985; Tassi et al., 1992). These studies, however, were often conducted to investigate the effects of other sleep processes

on sleep inertia and circadian effects often emerged incidentally. Therefore, as acknowledged by the authors, their results are invariably confounded by interference from sleep deprivation, varying nap lengths, and other experimental methods. For example, whilst Dinges et al. (1985) found that awakening from naps occurring at the trough of the core body temperature produced greater sleep inertia deficits than naps occurring at the peak of the core body temperature, their results were confounded by amount of accumulated sleep deprivation and length of preceding nap. Their results are suggestive of a circadian component to sleep inertia, nonetheless, as core body temperature is known to display a sinusoidal circadian rhythm almost analogous to the circadian rhythm of sleep propensity (Kräuchi et al., 2004; Johnson et al., 1992) and therefore depth of sleep according to Dinges' (1990) pressure for sleep model. Similar results with similar confounding factors have been identified in other studies (e.g., Lavie & Weler, 1989; Naitoh, 1981). Wilkinson and Stretton (1971) found that participants performed more poorly on addition and physical co-ordination tasks when aroused later in the night (i.e., 3.30am or 5.30am) compared to afternoon baseline levels and awakenings earlier in the night (i.e., 12.30am and 1.30am). This was not true for all the experimental tasks administered however, and further, measurements of circadian rhythm timing were not taken.

To redress this gap in the understanding of true circadian influences on sleep inertia, Scheer and colleagues (2008) used a forced desynchrony protocol consisting of seven 28-hour sleep/wake cycles to investigate the effects of circadian phase on sleep inertia performance. Based on the known circadian rhythm timing of sleep propensity, they hypothesised that sleep inertia-related cognitive performance decrements, as measured by a serial addition task, would be maximal during the biological 'night' compared to the biological 'day'. Their results indeed confirmed their hypothesis and showed that sleep inertia performance decrements were 3.6 times larger when participants were awoken during the circadian nadir (approximately 11pm – 3am) compared to awakenings made during the circadian peak (approximately 3pm – 7pm). The circadian influence on sleep inertia performance was found irrespective of the pre-arousal stage of sleep. Similarly, Silva and Duffy (2008) found a circadian component to performance on the Digit Symbol

Substitution Test in a sample of older adults. In a forced desynchrony protocol, cognitive performance was poorest when testing corresponded to the late evening and night under entrained conditions. Performance on the Digit Symbol Substitution Test was least affected by sleep inertia when testing corresponded to the early afternoon under entrained conditions. This circadian influence on sleep inertia performance was also independent of pre-awakening stage of sleep (REM v. non-REM).

Naitoh, Kelly and Babkoff (1993) reasoned that because there is a circadian involvement in falling asleep, there should also be a circadian component to the reverse process of waking up, and hence, they aimed to isolate the point in the circadian rhythm when sleep inertia is maximal. Using performance on Baddeley's logical reasoning task as a dependent measure, they found no obvious rhythmic fluctuations in performance data and concluded that "there appeared to be no specific circadian time when sleep inertia is either maximal or minimal" (p. 109). These results are inconsistent with Scheer et al.'s (2008) results, however, Naitoh et al.'s experimental design is confounded by 64-hours of prior sleep deprivation with 20-minute sleep periods every 6 hours. Further, it is unclear which stage of sleep participants were awoken from immediately prior to sleep inertia testing and cognitive assessment was only conducted at 2-hourly intervals. Balkin and Badia (1988) also failed to detect a circadian pattern in sleep inertia performance over four consecutive nights of testing, however, their protocol was also confounded by increasing sleep loss. These studies highlight the complex interaction between sleep processes in determining the characteristics of sleep inertia.

Whist Scheer et al.'s (2008) results are yet to be replicated, there is strong evidence to suggest a circadian rhythm component to sleep inertia. The studies that show this effect provide statistical support for Process C in the three-process model of sleep regulation, which predicts an interaction between Process C, Process S, and Process W – a sleep inertia component. This indicates that circadian effects should be controlled for, or at the very least taken into consideration, in experimental protocol assessing sleep inertia.

2.2.4 Time course of sleep inertia

Whilst both the arousal hypothesis and the three-process model of sleep regulation would predict that sleep inertia subsides in an exponential or asymptotic manner (probably as a function of how long it is measured), controversy surrounds the exact time course of sleep inertia dissipation, with estimates ranging from several minutes to several hours (Tassi & Muzet, 2000). Amongst the earliest estimates of the time course of sleep inertia, Wilkinson and Stretton (1971) found suboptimal performance for at least 15 minutes for both behavioural and cognitive tasks when participants were awoken at various times during the night. Performance was expressed as a percentage of baseline performance as measured during the afternoon. Although their experimental protocol only incorporated formal assessment of functioning 4-15 minutes post-awakening, Wilkinson and Stretton's early study nonetheless provided a valid approximation of the time course of sleep inertia. Other studies have also estimated that sleep inertia is short-lasting, ranging from 2.5 minutes (Kolff, Hofman, Kerkhof, & Coenen, 2003), 1-15 minutes (Naitoh, 1992), 1-20 minutes (Dinges, 1990), and 5-35 minutes (Åkerstedt, Torsvall, & Gillberg, 1989). Commensurate with this, Sallinen, Härmä, Åkerstedt, Rosa, and Lillqvist (1998) found sleep inertia decrements that lasted for 10-15 minutes following early morning (i.e., between 1-4am) naps of either 30- or 50-minutes duration. Bruck and Pisani (1999) observed sleep inertia decrements on a decision-making performance task for at least 30-minutes following abrupt nocturnal awakening, with performance being most impaired during the initial 3 minutes of sleep offset. In their simulation study, Seminara and Shavelson (1969) also found sleep inertia-related cognitive performance decrements to be severest during the first 3 minutes of sleep offset.

Longer estimates of sleep inertia dissipation have also been proposed. Achermann, Werth, Dijk, & Borbély (1995) found sleep inertia-related deficits on the Memory and Search Test for approximately 54 minutes following arousal from a regular nighttime sleep episode, whilst impairments of alertness (as assessed by subjective rating) persisted for a further 30 minutes. A similar time course of sleep inertia was found following an evening nap (at 6pm), a daytime nap, and a full night of nocturnal sleep (Achermann et al.). Also assessing sleep inertia following 8-hours of regular nocturnal sleep, Jewett et

al. (1999) measured subjective alertness and cognitive performance (addition task) for 4 hours following habitual wake time over 3 consecutive days. Their results showed that under normal entrainment conditions, sleep inertia performance took 2 to 4 hours to plateau. Levels of subjective alertness were quicker to recover (time constant = 0.67-hours) than cognitive performance measures (time constant = 1.17-hours). Participants in this study were awoken from REM, stage 1, or stage 2 sleep, however this had no effect on the subsequent time course of sleep inertia dissipation.

Ferrara and colleagues (2000a) also assessed cognitive functioning during sleep inertia following an undisturbed night of sleep in the laboratory occurring after an adaptation night. They found no sleep inertia effect on the DST under this condition. They did, however, find a sleep inertia effect on this cognitive performance task following a night where participants were awoken out of stage 2 sleep after 2-hours, and then 5-hours of accrued sleep. During this condition, and a condition of accumulated selective slow wave sleep deprivation conducted on a separate night (the second of two consecutive nights of slow wave sleep deprivation), cognitive performance on the DST showed a linear trend of sleep inertia over 75 minutes, yet performance had reached baseline levels at 30-45 minutes post-arousal. They found a quadratic (inverted U-shape) trend of performance during the first 75-minutes of sleep inertia during the adaptation night, the first night of slow wave sleep deprivation, and also during a recovery night. Interestingly, the baseline night was the only night not to show cognitive effects of sleep inertia on the DST. Of the five experimental nights (excluding the adaptation night) this is also theoretically the only night without concomitant sleep deprivation. However, the authors did not specify if or how they controlled for or prevented sleep deprivation prior to the adaptation night.

Indeed, sleep deprivation has been shown to prolong the effects of sleep inertia and delay return to pre-sleep performance levels (Naitoh, 1981; Haslam, 1985; Tassi & Muzet, 2000). Some studies have shown that sleep deprivation can extend sleep inertia effects for up to 4 hours following a short nap (e.g., Naitoh, 1981; Haslam, 1985). Tietzel and Lack (2001) found that increased sleep inertia effects offset the benefits of a 30-minute

nap following a night of partial sleep deprivation (4.7-hours total sleep time). They found decreased performance on cognitive tasks for up to 35 minutes following a 30-minute nap in partially sleep deprived participants.

Due to methodological differences between studies and the diversity of their dependent measures, it is difficult to determine the precise time course of sleep inertia. A few studies have failed to make continuous assessments of cognitive performance during the sleep inertia period, instead making an assessment after awakening and then repeating the assessment 2 or more hours later (e.g., Angus, Pigeau, & Heslegrave, 1992; Naitoh et al., 1993). These studies do not allow an accurate estimation of the duration of sleep inertia and have not been included in this review. After evaluating the suitable literature, a predominance of studies suggest that the acute effects of sleep inertia last from 15-30 minutes (see Table 1, p. 25), and dissipate in an exponential manner, eventually reaching an asymptote if measured for long enough. It is unclear, however, if the nature of the task affects the time course of sleep inertia and further, it depends how the dissipation of sleep inertia is methodologically defined (Tassi & Muzet, 2000). In some studies, sleep inertia performance is matched to pre-sleep performance, and in others performance is monitored until it levels off or reaches an asymptote. It is also imperative to consider circadian influences and the effects of prior sleep deprivation when evaluating the time course of sleep inertia.

2.2.5 Summary

A number of factors interact to characterise the parameters of sleep inertia. These can be summarised as follows:

1. Theoretically and experimentally, sleep inertia effects are intensified following arousal from deep slow wave sleep compared with REM or light sleep.
2. Prior sleep deprivation enhances and prolongs sleep inertia effects.
3. There appears to be a circadian influence on sleep inertia.
4. The time course of acute effects of sleep inertia on cognitive performance is approximately 15-30 minutes, with the severest impairment occurring within the first 3 minutes of sleep offset.

2.3 Sleep inertia and neurocognitive functioning

Psychophysiological research has been concerned with the effects of sleep inertia on cognition and behaviour since the early 1960s (Tassi & Muzet, 2000). As can be inferred by the variety of research presented above, sleep inertia-related performance decrements are well-documented in a number of cognitive domains. Performance on tasks of basic motor functions such as grip strength (e.g., Jeanneret & Webb, 1963; Tebbs & Foulkes, 1966), auditory and visual reaction time (e.g., Matchock & Mordkoff, 2007; Sallinen et al., 1998; Wilkinson & Stretton, 1971), simple mental arithmetic (e.g., Balkin & Badia, 1988; Hofer-Tinguely et al., 2005; Jewett et al., 1999; Wertz et al., 2006), and more complex cognitive functions including memory (Achermann et al., 1995; Bonnet, 1983; Tassi et al., 1992; Salamé et al., 1995; Stones, 1977) and logical reasoning (Hou, Huangfu, Zhang, & Miao, 2007; Salamé et al., 1995) have been shown to be consistently impaired following abrupt and natural awakening from sleep. In regards to the neurocognitive processes relevant to emergency fire escape behaviours, working memory and attentional processes are also known to be affected under conditions of sleep inertia.

2.3.1 Working memory

Working memory performance, as measured by the DST, took up to 45 minutes to recover to baseline levels following stage 2 sleep awakening from regular nocturnal sleep (Ferrara et al., 2000a). Sleep inertia effects were also evident on the DST following awakening from a night of selective slow-wave sleep deprivation and also following a nocturnal recovery sleep, taking up to 75 minutes to completely dissipate under these conditions (Ferrara et al.). It was found that performance accuracy on this task (i.e., ratio of correct responses to number of responses) and not performance speed (i.e., overall number of responses produced) was impaired at morning awakenings following nocturnal sleep under both regular, sleep deprivation, and recovery sleep conditions.

Tassi, Bonnefond, Hoeft, Eschenlauer and Muzetand (2003) also found impaired performance on the DST at morning awakenings in partially sleep deprived and non-sleep deprived individuals. They presented the DST in two forms; the regular (complex) version, and a simpler version in which subjects progressively subtracted the same digit,

rather than a digit that changed after every calculation. Interestingly, Tassi et al. found that performance speed was affected during the first 15 minutes of the sleep inertia period for all subjects (sleep deprived and non-sleep deprived) for the simple version of the DST only (with the complex version showing only mild reductions in speed of performance). The authors reasoned that the increased difficulty of the complex version of the DST had an activating effect on sleep deprived participants' level of arousal, and thereby, in keeping with the principles of the Yerkes-Dodson Law, mental slowing or reduced speed of performance was not observed due to an increased level of basal arousal compared to the simple version of the task. Performance accuracy on the DST was impaired from 0-15 minutes and 45-60 minutes after awakening for the complex/regular version of the DST, for the sleep deprived group only. The authors did not acknowledge, however, the possibility that reduced performance accuracy may have been observed for the simple version of the DST had there not been such a high potential for ceiling effects (within the realms of participants' individual capacity). Speed accuracy trade-off effects will be discussed more fully in section *2.3.3* (see below).

Sleep inertia effects have also been found on working memory tasks assessed following arousal from naps. Dinges et al. (1985) found substantially decreased working memory performance on the DST following a 2-hour nap in a sustained operations study where participants were exposed to varying levels of sleep deprivation. Performance decrements on this task were a function of the amount of accumulated sleep loss and the circadian timing of the nap. The dose-dependent response found in this study demonstrates a clear relationship between sleep inertia, the factors that influence its effects, and working memory performance. Similarly, Dinges, Orne, Evans and Orne (1981) found a 25-26% reduction in DST performance compared to pre-sleep levels following 1- and 2-hour daytime naps in non-sleep deprived subjects. Further to this, Mullington and Broughton (1994) also found working memory deficits following short daytime naps in a narcolepsy population. Using the DST and a four-choice reaction time test, sleep inertia effects were evident for approximately 20 minutes following waking from afternoon or evening naps of around 30 minutes duration. Similarly, Hou et al. (2007) assessed both verbal and spatial aspects of working memory functioning

following a 1-hour nap in sleep deprived subjects. In their study, under conditions of sleep inertia, it took 30 minutes for spatial working memory performance to reach baseline levels, and verbal working memory impairments were evident for up to 2 hours post-nap. In Frey's (2008) study of over 300 young adults, results showed that cognitive performance during sleep inertia varied depending on the task. *All* subjects, however, were impaired on the working memory task upon awakening from sleep.

2.3.2 Attention

Attentional processes appear to be particularly sensitive to the effects of sleep inertia, and indeed, sleep inertia effects have been documented for a wide variety of attentional processes including event-related potentials (e.g., Bastuji, Perrin, & Garcia-Larrea, 2003), visual discriminant attention (Matchock & Mordkoff, 2007), sustained attention, selective attention, divided attention and mental tracking (Tietzel & Lack, 2001). Tietzel and Lack investigated the recuperative benefits of short daytime naps against the performance decrements associated with sleep inertia. They increased the duration of a selective attention task, the well-known Letter Cancellation Test, in order to simultaneously assess sustained and selective attentional functions. They also used the Symbol Digit Substitution Test to capture sleep inertia effects, which recruits divided attention and mental tracking. The results showed a significant sleep inertia effect on attentional functions following a 30-minute nap period in partially sleep-deprived subjects. Similarly, Silva and Duffy (2008) also found impaired performance on the Digit Symbol Substitution Test (a modified version of the Symbol Digit Substitution Test) when sleep was disrupted both during the 'biological day' and 'biological night' in a forced desynchrony protocol. Please refer to Table 1 for a summary of the cognitive tasks assessed in a variety of sleep inertia paradigms, and the associated estimations of the time course of sleep inertia.

Table 1.

Studies of sleep inertia by cognitive domain: Task, methodology and estimates of time course.

Study	Task	Duration	Methods
Motor Strength.			
Jeanneret & Webb (1963)	Grip Strength	Not revealed	Nocturnal sleep study.
Tebbs & Foulkes (1966)	Grip strength	Not revealed	Nocturnal sleep study, disrupted sleep.
Reaction Time.			
Webb & Agnew (1964)	Serial response time	Not revealed	Disrupted sleep.
Wilkinson & Stretton (1971)	Reaction time Addition task Motor co-ordination	At least 15 minutes	Nocturnal sleep study, disrupted sleep.
Dinges et al. (1981)	Simple reaction time	5 – 35 minutes	Nap study.
Sallinen et al. (1998)	Visual reaction time	10 – 15 minutes (following 30 or 50-min naps)	Nap study in shift work paradigm (nighttime naps).
Matchock & Mordkoff (2007)	Visual attention Reaction time	At least 20 minutes	Nocturnal sleep study, disrupted sleep.
Psychomotor Performance.			
Koulack & Schultz (1974)	Psychomotor speed Vigilance task	Not revealed	Nocturnal sleep study, disrupted sleep
Bruck & Kritikos (2007)	Psychomotor speed Physical performance	Not revealed	Nocturnal sleep study, disrupted sleep.
Simple Mental Arithmetic.			
Balkin & Badia (1988)	5-min Addition test	Not revealed	Nocturnal, restricted, disrupted sleep.
Jewett et al. (1999)	2-min Addition task	2 – 4 hours to reach asymptote	Nocturnal sleep study (free of time cues).
Hofer-Tinguely et al. (2005)	Addition task Auditory reaction time	20 – 60 minutes following 2-hour nap	Nap study.
Wertz et al. (2006)	Addition task	Approx. 1-hour, severest impairments within first 3 minutes	Nocturnal sleep study, disrupted sleep v. sleep deprivation.
Scheer et al. (2008)	Serial addition test	At least 20 minutes	Disrupted sleep/forced desynchrony protocol.

Higher-level Attention & Working Memory.			
Dinges et al. (1985)	DST Reaction time	Not revealed	Nap & sleep deprivation study.
Stampi et al. (1990)	DST Memory and search test	Not revealed	Nap study.
Stampi & Davis (1991)	DST Memory and search test	Not revealed	Sleep restriction study.
Mullington & Broughton (1994)	DST (2-mins) Forced-choice reaction time	20 minutes following 30-min (short) nap, no SI effect following long nap	Daytime nap study (narcoleptic subjects).
Ferrara et al. (2000a)	DST Auditory reaction time Finger tapping	At least 75 minutes (30 – 45 minutes to reach baseline levels	Nocturnal sleep and selective SWS deprivation.
Tietzel & Lack (2001)	SDST LCT	35 minutes following 30-min nap	Restricted sleep/Nap study.
Tassi et al. (2003)	DST (complex and simple)	15 – 60 minutes	Partial sleep deprivation vs. no sleep deprivation.
Hou et al. (2007)	Verbal working memory Spatial working memory Logical thinking test	30 – 120 minutes	Nap (1-h) & sleep deprivation (30-h) study.
Frey (2008)	Working memory task Executive task	Not revealed	Nocturnal sleep study.
Silva & Duffy (2008)	Digit Symbol Substitution Test	At least 30 minutes	Disrupted sleep/forced desynchrony protocol.
Memory.			
Stones (1977)	Learning and memory	Not revealed.	Nocturnal sleep study, disrupted sleep.
Bonnet (1983)	Short and long term memory	Up to 30 minutes	Nocturnal sleep study, disrupted sleep.
Tassi et al. (1992)	Spatial Memory test	15 minutes (following 1-hour nap)	Nap study (nighttime naps).
Achermann et al. (1995)	Memory search task	Approx. 1 hour (0.9h)	Nocturnal sleep and daytime nap study.
Salamé et al. (1995)	Spatial Memory task Logical Reasoning task	24 – 27 minutes	Nap & sleep deprivation study.
Executive Functions.			
Bruck & Pisani (1999)	Decision-making task	At least 30 minutes, severest impairments within first 3 minutes	Nocturnal sleep study, disrupted sleep.

Tassi et al. (2006)	Stroop test	1 hour	Nocturnal sleep study, partial sleep deprivation v. no sleep deprivation.
Simulation.			
Langdon & Harman (1961)	Flight simulation	At least 10 minutes	Nocturnal sleep study, varying intervals of sleep.
Hartman & Langdon (1965)	Flight simulation	At least 10 minutes	Nocturnal sleep study, varying intervals of sleep.
Seminara & Shavelson (1969)	Response time: space crew performance tasks	9 – 12 minutes, severest impairment within first 3 minutes	Nocturnal sleep study v. awake (daytime) performance.

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test.

2.3.3 Speed-accuracy trade-off effect

A large majority of the literature assessing cognitive performance in sleep studies has found that sleep inertia affects the speed of cognitive processing. Hence, a speed-accuracy trade-off effect is identified whereby behavioural and cognitive performance is slowed during conditions of sleep inertia, whilst performance accuracy is more-or-less maintained. This effect of cognitive slowing, or speed-accuracy trade-off, is so consistent that most studies indeed only measure performance speed or reaction time on a variety of cognitive tasks, and do not measure, or report, performance accuracy (e.g., Achermann et al.,1995; Jewett et al., 1999; Tassi et al., 1992; Webb & Agnew, 1964). As described above, Tassi et al. (2003) found a complex interaction between performance speed, performance accuracy, and task complexity on the DST. They found a similar pattern on a Stroop Test, whereby performance speed was affected by sleep inertia initially (i.e., up to 30 minutes), but performance accuracy began to decrease in the second half-hour block post-awakening. Their interesting results raise questions about the effects of task complexity, task novelty, arousal and boredom on cognitive performance.

In contrast, Ferrara and colleagues (Ferrara et al., 2000a; Ferrara, De Gennaro, Casagrande, & Bertini, 2000b) have found that performance accuracy on the DST and *not* performance speed was sensitive to sleep inertia under varying conditions, including

waking up from regular nocturnal sleep and also under conditions of slow wave sleep deprivation. They have concluded that "during the sleep-wake transition, cognitive performance *accuracy* is more impaired than performance *speed*" (Ferrara et al., 2000b, p. 440). In these studies, however, Ferrara and colleagues' participants performed the DST at least 50 times over six consecutive nights following pre-experiment training to asymptotic levels on this task. With that many trials, it is statistically much more likely that they would find differences where differences do not exist. Further, their results are contradictory to those of Stampi and Davis (1991) who found that performance speed on the DST was so significantly impaired by sleep inertia that the decrements found on this task surpassed decrements found on their other cognitive measure, the Memory and Search Test. The Memory and Search Test has previously been shown to be sensitive to sleep inertia (e.g., Achermann et al., 1995). Mullington and Broughton (1994) also assessed sleep inertia effects using the DST in a sample with narcolepsy. Whilst they claimed to measure both speed and accuracy components on this task, only the accuracy of participants' performance was reported; it showed inconsistent sleep inertia effects across a variety of nap schedules, even demonstrating *improvement* in task accuracy following an arousal. The pattern of results was unable to be explained by nap length or circadian influences. The results of a subsequent study that compared the cognitive performance of narcolepsy subjects when sleepy and alert (Hood & Bruck, 1996) would suggest that such improvement in performance may be an artifact of the alerting effects of the nap, particularly when contrasted against narcoleptic subjects' greater baseline levels of sleepiness. Other studies in healthy populations have shown no effect of sleep inertia on task performance accuracy (e.g., Hofer-Tinguely et al., 2005; Naitoh, 1981).

Whilst a small number of studies have found that sleep inertia affects performance accuracy, these have been under a variety of experimental paradigms, and therefore the 'accuracy-speed trade-off' effect has not yet been replicated under the same procedure twice. The *speed-accuracy* trade-off effect, on the other hand, has been replicated a number of times, using a wide range of cognitive tasks as dependent measures, including addition tests (Balkin & Badia, 1988; Hofer-Tinguely et al., 2005), spatial memory tasks (Salamé et al 1995; Tassi et al., 1992), a logical reasoning task (Salamé et al 1995), the

DST (Stampi & Davis, 1991), a visual attention task (Matchock & Mordkoff, 2007), and real-world space tasks in a simulation study (Seminara & Shavelson, 1969). Indeed, under conditions of 'normal' sleep inertia, i.e., in the absence of concomitant sleep deprivation, most studies demonstrate that only performance speed is adversely affected (Tassi & Muzet, 2000). Further to this, the speed-accuracy trade-off effect, or slowed speed of performance under conditions of sleep inertia is consistent with the basic tenet of the arousal hypothesis which posits neurophysiological evidence for decreased cerebral perfusion and responsiveness and cognitive slowing during the sleep-wake transition (Hajak et al., 1994; Tassi & Muzet, 2000; see section *2.1.1* above).

2.3.4 Simulation studies: Sleep inertia and emergency behaviour

Bruck and Pisani (1999) were the first to investigate the effects of sleep inertia on decision-making; a higher-order cognitive process relevant to emergency situations and on-call shift-workers such as medical registrars and emergency workers. Using a computer-animated fire situation task, their pioneering research demonstrated that under conditions of sleep inertia, decision-making performance is reduced by 51% compared to baseline levels in the first 3 minutes post-arousal. Performance on the task remained sub-optimal (approximately 20% below baseline) for up to 30 minutes after awakening. In this study, nocturnal awakenings occurred from both REM and slow wave sleep (order counterbalanced) between the hours of midnight and 4am, when most residential fires occur (Jones, 1983). Whilst their research is important and informative, it is also important to consider the results of simulation studies as their inherent validity provides more generalisable estimations of sleep inertia effects on performance in real-world situations.

Given the ethical implications of exposing participants to emergency and other highly stressful situations, however, only a small handful of studies have investigated sleep inertia effects in 'real-life' emergency studies or in simulation paradigms. Not surprisingly, the majority of this research is quite dated. In 1969, Seminara and Shavelson assessed space-typical emergency responses in a 5-day lunar mission simulation study. With the aim of investigating the advantages of simultaneous vs.

staggered sleep schedules, participants were required to perform a number of space crew tasks in response to an alarm which sounded during sleep and daytime awake periods. They found significantly impaired performance on an alarm disable task, a monitoring task requiring a "go/no go" decision at completion, a control panel task, and a pressure suit donning task when participants were awoken during sleep and asked to immediately perform these space-crew emergency response tasks. Participants took 36% more time to perform the monitoring task and 12.6% more time to don the space suit when responding from sleep compared to wakefulness. The alarm disable task was not significantly different across conditions due to an inflated error term, however. Overall, participants took 23% longer to perform the emergency response tasks when responding to an alarm under conditions of sleep inertia. They found that performance on the control panel task was most significantly impaired at 0 – 3 minutes post-arousal, but decrements in performance were still evident at the final trial which was initiated at 9 minutes post-arousal. These results indicate that time-critical responses to a space crew emergency situation are affected by sleep inertia.

Seminara and Shavelson's (1969) study was one of a series of studies comparing psychomotor performance under conditions of sleep inertia and normal awake periods in aerospace research. The series of studies developed as a result of a crash due to pilot error when a pilot had to respond immediately following sleep whilst on nighttime alert duty. Following this accident, new policies meant that all pilots had to remain awake during alert duty. In the context of this new policy, Hartman and colleagues (e.g., Hartman & Langdon, 1965; Langdon & Hartman, 1961) also investigated the effect of different sleep schedules (e.g., simultaneous versus staggered) on pilot performance. They used a Complex Behaviour Simulator and performance on a piloting task and found that operation of these tasks was significantly degraded following abrupt arousal from sleep. Further, recovery was gradual as sleep inertia effects had not yet dissipated after 10 minutes of continuous post-arousal performance.

Also in the realm of aerospace medicine, Ribak and colleagues (1983) retrospectively studied Air Force flying accidents that were attributed to pilot error for the non-wartime

period of 1968-1980. They computed the frequency of hourly accidents for each year, each month, each day of the week, and each calendar day. Their computations revealed a diurnal pattern which was independent of the frequency of the flights, but most closely related to the sleep-wake schedules of the pilots. In particular, the calculated Hourly Accident Coefficient was highest at the point pertaining to the time of pilots' waking from nocturnal sleep. This retrospective research demonstrates the implications of sleep inertia for real-life performance of aerospace duties. More recently, an airforce mishap was also examined retrospectively to determine the factors that contributed to the accidental aerodynamic stall during a long-haul flight. Through the use of computerized fatigue modeling and other methods, it was found that multiple elements of fatigue were responsible for this mishap. These included acute and cumulative fatigue, circadian disruptions, and sleep inertia (Armentrout, Holland, O'Toole, & Ercoline, 2006).

Whilst this small selection of studies actually represents the entire catalogue of "field" studies in sleep inertia, the effects of sleep loss and sleep deprivation have been studied in the context of military performance and on-call emergency work such as that of medical registrars and interns. For example, Taffinder et al. (1998) found 20% more errors and a 14% reduction in efficiency for medical residents undertaking procedural skills tasks under conditions of sleep deprivation. Similarly, surgical physicians and medical students were found to show deteriorated performance in laparoscopic procedures during the daytime following a night of on-call duty in a surgical department with a total sleep time of less than 3 hours (Grantcharov, Bardram, Funch-Jensen, & Rosenberg, 2001). There was a two-fold increase in errors in surgical skill and dexterity, and the speed at which they could perform the simulated procedure deteriorated by 38% compared to baseline daytime performance.

Sleep deprivation has also been shown to affect speed of performance in the military field. Haslam (1982) assessed infantry soldiers' shooting performance, vigilance, physical fitness and performance on two cognitive tasks in a 9-day sleep deprivation paradigm in which soldiers were partially sleep deprived following a 90-hour period with no scheduled sleep (and very little unscheduled sleep). Results showed that performance

on the military tasks reduced by 25% due to sleep deprivation and that this was due to reduced attention, rather than decrements in skills, e.g., shooting skills. Haslam found that cognitive performance on a logical reasoning task and a decoding task showed decrements in speed (i.e., the number of responses produced), whilst accuracy on these tasks was unchanged under conditions of sleep deprivation. Performance reduction was up to 35% below average baseline levels for the logical reasoning task, whilst performance on the decoding task was approximately 50% of the average baseline value. Vigilance and cognitive task performance deteriorated to a greater extent and more quickly than physical and military tasks, with performance deteriorating after one night of sleep loss. Haslam notes, however, that when considering the results of the cognitive tasks, one should keep in mind that "there was no real spur to awakening, such as threatening or demanding situation, to provide arousing stimulus" (p. 174) compared to the shooting tasks, which although done in a simulation paradigm, are higher on military realism (and therefore arousal) than the cognitive tasks. This begs the question of the affects of arousal, stress, and adrenalin on cognitive performance during sleep inertia.

2.3.4.1 Effect of arousal and stress on cognitive performance during sleep inertia

As already described, a number of studies have acknowledged, either directly or indirectly, the role of task complexity and/or importance in determining cognitive performance during sleep inertia. These factors are mediated, it is believed, by their arousing effect on persons experiencing sleep inertia. According to the Yerkes-Dodson law, performance is optimal at moderate levels of arousal, but too much arousal or not enough arousal is associated with decreased performance. Tassi et al. (2003) used the Yerkes-Dodson principle to explain how decreases in performance speed were evident on a simple version of the DST, but not on a more complex version of the same task. According to the authors, a lower basal level of arousal, such as that found during conditions of sleep inertia, was required to successfully execute the complex task, as the complexity of the task itself contributed to participants' level of activation. The simple task, however, was not arousing enough to offset the de-arousing and cognitive slowing effects of sleep inertia.

Tassi et al.'s (2003) results and theoretical postulations are consistent with findings from field studies. As described above, Haslam (1982) found that performance on cognitive tasks was affected to a greater extent than performance on real-world military tasks during a sleep deprivation paradigm. It is likely that the real-world military tasks were more important and significant, and therefore more arousing to the infantry soldiers who participated in the study. Further to this, Seminara and Shavelson (1969) found that complex aerospace tasks (e.g., product organising) were less affected by sleep inertia than more simple ones (e.g., control panel task and monitoring task) upon arousal from sleep in an astronaut population.

The postulated effect of task complexity on arousal to explain these results seems parsimonious. Given that the effects are present in field and simulation studies, however, it is important to consider the effect of stress on performance under conditions of sleep inertia. Stress is known to impair cognitive function through the activation of the hypothalamic-pituitary-adrenal axis. In humans and other mammals, adrenal steroid secretion increases in response to stress, directly increasing the amount of circulating glucocorticoids. Glucocorticoids have important direct effects on the brain, and particularly the hippocampus in the medial aspect of the temporal lobe where there is a high density of adrenal steroid receptors (McEwen, Weiss, & Schwartz, 1968). The primary role of the hippocampus is the acquisition and long-term storage of new information, particularly in the form of declarative memories. It is not surprising then, that the main effect of stress on cognitive functioning is to impair memory storage and retrieval processes as a result of significant elevations of circulating glucocorticoids (de Quervain, Roozendaal, & McGaugh, 1998; Lupien et al., 1998; Newcomer et al., 1999; Porter & Landfield, 1998). Mild psychological stress (e.g., exam stress, exposure to a stressful video game) has been found to affect attentional processes (Skosnik, Chatterton, Swisher, & Park, 2000; Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000), although these changes are not consistently correlated to increasing cortisol levels and therefore may represent spurious effects. High levels of circulating corticosteroids is also associated with cognitive decline in older adults (Karlamangla, Singer, Chodosh,

McEwen, & Seeman, 2005), but no other specific cognitive impairments have been described in normal subjects.

Knowing this, it is possible to postulate that unless memory process are being specifically investigated, stress is not able to account for differential sleep inertia effects on complex vs. simple tasks (or 'important' vs. 'non important' tasks) in both experimental paradigms and real-world or simulation studies. If stress affects cognitive functioning so selectively, however, is it possible that there are specific tasks and performance indices mainly affected by sleep inertia? And, if studies of sleep deprivation also show the arousing effects of complex or important tasks, are the cognitions selectively affected by sleep inertia different to those affected by sleep deprivation? Are sleep inertia and sleep deprivation conceptually or functionally the same?

2.3.4.2 Is sleep inertia the same as sleep deprivation?

Whilst prior sleep deprivation is known to enhance the cognitive decrements associated with sleep inertia, the conceptualisation of sleep deprivation and sleep inertia as similar processes remains controversial. Sleep deprivation produces sleepiness, which *accumulates* with continued wakefulness, whilst sleep inertia effects *dissipate* with continued wakefulness, as disengagement from the prior sleep state is completed. Therefore, one difference between sleep loss and sleep inertia is the "direction in which alertness is progressing" (Balkin & Badia, 1988, p. 247). This suggests that sleep loss and sleep inertia are unlikely to be physiologically equivalent states and therefore there may be qualitative and quantitative differences between the two states in their manifestation of behavioural and neurocognitive deficits.

Sleep inertia and sleep deprivation both impair cognitive functioning, and both tend to produce a speed-accuracy trade-off effect in task performance (e.g., for sleep deprivation: Thorne, Genser, Sing, & Hegge, 1985; Williams & Lubin, 1967), although there is a more recent indication that decrements in speed *and* accuracy of performance are associated with partial or total sleep deprivation (Mallis, Banks, & Dinges, 2007). It seems, however, that this may be where the comparative similarities cease. Some studies

show that the magnitude of performance deficits during sleep inertia may be worse than those produced by sleep deprivation. The suggestion that cognitive performance is more impaired following nocturnal awakenings than when subjects have remained awake throughout the night has been recognised for some time (e.g., Fort & Mills, 1972; Rosa, Bonnet, & Warm, 1973). Recently, Wertz et al. (2006) found that sleep inertia effects on an addition task were significantly worse than decrements due to sleep deprivation, assessed every 2 hours across a 26-hour sleep deprivation protocol. Whilst the performance decrements associated with 24 hours of sleep deprivation have been likened to alcohol intoxication of up to 0.05 BAC (Dawson & Reid, 1997; Roehrs, Burduvali, Bonahoom, Drake, & Roth, 2003), it seems that sleep inertia effects may be significantly more severe (Frey, 2008). Not all studies, however, have consistently shown that sleep inertia effects on cognitive performance are more severe than those produced by sleep deprivation. In a repeated-measures design, Hou et al. (2007) subjected participants to 30 hours of sleep deprivation on two separate occasions. On one of these occasions, participants were allowed a 1-hour nap. Whilst a significant sleep inertia effect on cognitive performance was observed following the short nap, they found no significant difference in performance between the nap and no-nap conditions, suggesting that cognitive performance under conditions of sleep inertia and concomitant sleep deprivation is not significantly worse than during conditions of sleep deprivation alone.

Although it appears that sleepiness and sleep inertia may have different *quantitative* effects on cognitive functioning, are they similar *qualitatively*? If sleep inertia were basically sleepiness then we would expect similar decrements with sleep deprivation as with sleep inertia, yet this is not entirely the case. Sleep deprivation or the effects of sleep loss are characterised by lowered vigilance, microsleeps, and increasing fluctuations in alertness, in addition to cognitive changes such as poor short-term recall and multitasking or divergent thinking (Banks & Dinges, 2007; Mallis et al., 2007). Lapses appear to be a specific feature of sleep-loss sleepiness and not sleep inertia (Miccoli, Versace, Koterle, & Cavallero, 2008). This suggests that sleep inertia is different to simple sleepiness as caused by sleep deprivation or sleep loss, although the evidence is tentative.

It is known that performance decrements with simple sleepiness can be quite readily reversed for short-term tasks by a heightened state of arousal, especially if associated with increased motivation (Horne & Pettitt, 1985). In addition, a small selection of research has shown that continuous low frequency noise can alleviate the cognitive decrements associated with sleep inertia under conditions of partial sleep deprivation by increasing arousal and offsetting hypovigilance (Koelega & Brinkman, 1986; Tassi et al., 1992). In addition, increased task complexity can potentially eliminate sleep inertia decrements by increasing arousal (Tassi et al., 2003). Further research is required, however, to clarify the exact nature of the arousal, and the precise role of task complexity, task importance, novelty, or motivation on eliminating sleep inertia effects on cognitive functioning. These effects could simply be an artifact of the dependent variable used in each study (Frey, 2008; Tassi & Muzet, 2000).

The conceptual argument of the similarity between sleep inertia and sleep deprivation is controversial and unresolved. It is nonetheless an important issue that not only has theoretical implications for our understanding of sleep processes, but practical and operational implications. From a logistical perspective, it is important to understand the effects of sleep loss on sleepiness and performance against the adverse effects of sleep inertia on abrupt awakening from sleep due to a possible emergency, in both the on-call working population and to understand sleep-scheduling for the shift-work population.

2.3.5 Summary

Table 1 provides an overview of the behavioural and cognitive functions empirically demonstrated to show a sleep inertia effect. As is evident from this table, sleep researchers have used a variety of methods (i.e., nocturnal sleep studies, nap studies, forced desynchrony protocols, sleep restriction and deprivation studies) to show consistent sleep inertia effects for both simple and higher-order cognitive processes. While these findings are robust and have important implications for our understanding of the relationship between sleep processes and cognitive functions, a number of neuropsychological domains have been left untapped. Hence, the full extent of sleep inertia effects on neurocognitive functioning is unknown. Significant methodological

advancements are required to comprehensively capture/measure the interface between neuropsychological functioning and sleep processes during sleep inertia and other degrees of "wakefulness" (Schulz & Salzarulo, 1997). Further research is also required to determine the exact nature of the effects of stress and arousal on sleep inertia. Comparative data on the cognitive effects of sleep deprivation and sleep inertia will advance our understanding of the similarities and differences between these two physiologically disparate yet seemingly functionally similar sleep processes.

2.4 Subjective sleepiness and sleep inertia

As well as understanding sleep inertia from a neurophysiological and cognitive perspective, it is similarly important to understand sleep inertia from a subjective perspective. A number of studies have reported correlations between self-reports of subjective sleepiness and alertness and sleep inertia, demonstrating that sleep inertia also suppresses subjective experiences of alertness, and validating the objective performance measures with subjective experiences. Using a variety of measures, but most commonly the Karolinska Sleepiness Scale (KSS) and the Stanford Sleepiness Scale (SSS), sleep inertia and feelings of alertness have been significantly correlated in a number of experimental paradigms including naps studies (e.g., Hofer-Tinguely et al., 2005; Tietzel & Lack, 2001), sleep deprivation studies (e.g., Hou et al., 2007) and free-running studies (e.g., Jewett et al., 1999).

Bruck and Pisani (1999) found that subjective ratings of sleepiness on the KSS were significantly higher (i.e., higher levels of subjective sleepiness) following REM and SWS arousals than at baseline. Similarly, Kräuchi et al. (2004) found that KSS ratings were not only correlated with thermoregulatory processes predicting the onset and offset of sleep, but both the subjective measure (KSS) and the physiological measure (distal-proximal skin temperature gradient) shared the same time course when measured simultaneously under sleep inertia conditions following (1) regular nocturnal sleep and (2) an afternoon nap. Further, the KSS has been shown to be correlated with other visual analog scales of sleepiness and laboratory performance measures in a sleep deprivation paradigm (Gillberg, Kecklund, & Åkerstedt, 1994). Babkoff, Caspy and Mikulincer

(1991) found significant correlations between computerized self-report sleepiness scales (a Hebrew version of the SSS and a visual analog scale) and circadian rhythm timing. These studies indicate that subjective sleepiness ratings show gross changes in concert with sleep inertia and other sleep-related processes. However, they are not always precisely correlated with objective performance measures of sleep inertia nor do they always reflect the magnitude of the performance decrements (e.g., Brooks & Lack, 2005; Bruck & Pisani, 1999). For example, Bruck and Pisani (1999) found no significant relationship between an objective performance task assessing decision-making and subjective ratings of sleepiness and clearheadedness under conditions of sleep inertia following nocturnal awakening. Using the SSS, Dinges et al. (1987) found that ratings increased as sleep loss progressed, but that SSS scores did not reflect observed improvements in performance following a short nap. Similarly, some subjective measures of sleepiness are shown to reach the highest level of alertness long before objective performance measures confirm that sleep inertia effects are completely dissipated, or subjective and objective measures proceed in opposite directions (e.g., Achermann et al., 1995). However, this may represent an inherent limitation of subjective measures in that they are less sensitive than objective performance measures in capturing sleepiness and levels of alertness. Further, interpretation of self-report measures is often idiosyncratic which can threaten their validity (Barker, Pistrang, & Elliot, 2002). It is nonetheless well-established in the literature that subjective correlates of sleep inertia can be accurately obtained using visual analog rating scales such as the KSS and the SSS (e.g., Bruck & Pisani, 1999; Ferrara et al., 2000a; Hou et al., 2007; Jewett et al., 1999; Matchock & Mordkoff, 2007; Salinen et al., 1998; Tietzel & Lack, 2001)

2.5 Alcohol

Alcohol is a neurotoxin that acts as a central nervous system (CNS) depressant, and has effects similar to those of some tranquilising and hypnotic drugs. Moderate alcohol intake is typically defined as one to two standard doses which provides 21-42 millilitres of alcohol in a day; definitions of heavy or high alcohol intake start at four to five standard doses per day (Arciniegas & Beresford, 2001). The metabolism of alcohol

initiates chains of biochemical and physiological reactions that involve many other organ systems of the body in addition to the CNS. Its metabolism proceeds through many different routes, and therefore alcohol has many different effects on the CNS and on other organ tissues (Arciniegas & Beresford, 2001). Neurophysiological studies demonstrate that alcohol causes bilateral increases in hemispheric cerebral blood flow, but with significant regional differences. At low doses, alcohol causes increased regional cerebral blood flow (rCBF) in prefrontal regions, which then decreases at higher doses. Increased rCBF is seen in temporal regions at higher doses of alcohol, which continues to increase in a dose-response manner (Sano et al., 1993).

2.5.1 Alcohol metabolisation

The effects of a dose of alcohol are biphasic. When alcohol is consumed, blood alcohol concentration (BAC) rises quite rapidly, reaching its peak level in approximately 80 – 90 minutes; this is referred to as the ascending limb of the BAC curve (Schweizer & Vogel-Sprott, 2008). During the descending limb of the BAC curve, BAC levels gradually decline over 3 or more hours, depending on the level of consumption. A number of factors are known to influence the metabolism of alcohol including food, gender, body weight and medications amongst other things (National Institute on Alcohol Abuse and Alcoholism, 1997). At moderate doses, 50% of peak BAC is metabolised within 3.6 hours (Williams & Salamy, 1972).

2.5.2 Alcohol and the sleeping brain

A number of studies have shown that the rate of elimination of alcohol from blood is equivalent during waking and sleep (e.g., Rundell et al., 1972; Williams & Salamy, 1972), and hence, the systematic effects of alcohol on sleep are evident in the first 4 hours. Higher BACs will exert longer range influences on both the sleeping and awake brain. The initial effect of alcohol on the sleeping brain is sedative (i.e., rapid sleep onset, reduced body movements and increased slow wave sleep) and affects the rostral and caudal aspects of the brainstem and reticular activating system, inhibiting neuronal activity in these regions and effectively decreasing arousal levels (Williams & Salamy, 1972). Alcohol also has direct effects on the circadian pacemaker, causing altered

rhythmicity in both alcoholics and social drinkers (Rosenwasser, 2001). However, further research is required to characterise the complex interaction between alcohol and chronobiological processes.

Experimental studies have been at the forefront of elucidating the effects of alcohol on the sleep architecture and electroencephalography of the sleeping brain. Studies consistently show that the effects of alcohol on sleep architecture and EEG occur during the first half of the night, with rebound effects evident during the second half of the night, for moderate levels of intoxication. The following section discusses the major experimental advances in understanding the impact of alcohol on the sleeping brain.

2.6 Alcohol and sleep

There has been awareness for many centuries that alcohol consumption and withdrawal alters sleep patterns, however it was not until the late 1960s that these effects were subjected to experimental analysis (Williams & Salamy, 1972). Studies investigating the effects of both single and repeated doses of alcohol on the normal sleep cycle have generally been consistent in their findings. Mullin, Kleitman and Cooperman (1933) are heralded as the first research team to empirically investigate the effects of a single dose of alcohol on sleep phenomena. They administered 300 – 375 millilitres of a 19% alcohol beverage (the equivalent of about 1 litre of light wine) to four young adults 45 minutes prior to retiring to bed. Body movements and rectal temperature were measured throughout the night and it was found that alcohol caused a significant reduction in both these variables in the first half of the night, with an increase to baseline levels in the second half of the night. It appeared that the de-arousing and sedative effects of alcohol were only evident in the first half of the sleep period (Mullin et al., 1933).

The first investigation into the effects of alcohol on polysomnographically-measured sleep *patterns* occurred in 1963 when Gresham, Webb, and Williams investigated changes in REM sleep following a single dose of alcohol. Taking into account the known effects of CNS depressants on sleep (i.e., decreased frequency and increased amplitude of EEG waves), they hypothesised that alcohol would increase the depth of sleep in their

seven young adult volunteers, thereby diminishing light and REM sleep. The study was conducted over five nights, on two of which subjects received either 1 gram of alcohol per kilogram of body weight, or 0.005 g/kg of caffeine. Whilst caffeine had no effect on REM sleep architecture, the single dose of alcohol significantly reduced the amount of REM sleep obtained. Although the data appeared to support Gresham and colleagues' experimental hypothesis, the theoretical underpinnings of their predictions were wrong, as subsequent studies showed that alcohol affects the duration of sleep stages rather than their periodicity or the depth of sleep (e.g., Knowles, Laverty, & Kuechler, 1968; Williams & Salamy, 1972; Yules, Lippman, & Freedman, 1967).

Yules and colleagues conducted a series of experiments to delineate the effects of alcohol on sleep when ingested at different time points prior to retiring to bed, in order to assess the role of BAC levels. When subjects ingested alcohol immediately prior to going to bed on five consecutive nights, they found that REM sleep time was significantly decreased on the first alcohol night, then returned to baseline levels on the subsequent three alcohol nights, before exceeding baseline levels on the fifth alcohol night and for the following two recovery nights. By the fourth recovery night, REM sleep time had again returned to baseline levels (Yules, Freedman, & Chandler, 1966). In a second experiment where alcohol was administered 4 hours before subjects retired to bed, and was therefore 50% metabolised prior to sleep onset, REM sleep was found to decrease in duration for the first *two* alcohol nights, and returned to baseline levels on the third night of alcohol administration (Yules et al., 1967).

In this second experiment, half of Yules and colleagues' subject pool underwent five nights of alcohol administration 4 hours prior to a full 7-hour nocturnal sleep, and the other half of the subject pool underwent three alcohol nights. Further analyses revealed that during the fourth and fifth nights of alcohol administration, there was no further reduction in length of REM sleep periods below baseline levels. A REM-rebound effect was established in this study as the authors noted that when alcohol administration ceased, the amount of REM sleep obtained increased, particularly on the first recovery

night, and then progressively decreased to baseline levels over the subsequent four recovery nights.

The results of these studies indicate that the magnitude of the effect of alcohol on REM sleep is a direct function of BAC but is confounded by both adaptation and compensation effects. When Yules et al.'s subjects consumed alcohol immediately prior to retiring to bed, alcohol effects on REM sleep were evident on the first alcohol night only, with adaptation effects evident immediately thereafter (i.e., reductions in REM sleep were not detected on the following three alcohol nights). When the same amount of alcohol was imbibed 4 hours prior to bed, and therefore subjects' BAC level was significantly reduced at sleep onset, it took *two* nights for adaptation effects to occur. Consistent with the findings of Mullin et al. (1933) alcohol administration effects on REM sleep occurred during the first half of the nocturnal sleep period, whilst REM-rebound effects were evident during the second half of the sleep period on both alcohol nights and recovery nights. REM compensation effects occurred beyond the period of alcohol intoxication indicating that they cannot be a direct result of alcohol on the CNS. Instead, the nature of these changes stipulates that alcohol produces a self-sustaining dysregulation of REM sleep processes.

It was specifically the length of the REM sleep periods that was affected by alcohol in these studies as there was no systematic change in the number of REM periods or the latency of the onset of the first REM episode between alcohol and baseline or recovery nights. Interestingly, Yules and colleagues (1966; 1967) found that when alcohol-related reductions in REM sleep occurred, there was an inverse alteration in the length of stage 2 sleep, but no change in stages 3 or 4.

With the effect of alcohol on REM sleep well-established, research began to look more closely at the effect of repeated dosages of alcohol on sleep. In order to systematically evaluate the effect of single versus repeated doses of alcohol on physiological sleep architecture, Rundell, Lester, Griffiths, and Williams (1972) conducted two separate experiments with young male subjects. In the first experiment, subjects were given a

single dose (0.9 grams of 95% alcohol per kilogram of body weight) of alcohol, divided into three drinks consumed over an hour, 30 minutes prior to retiring to bed. BACs in these subjects ranged from 50 to 90 mg percent, with a mean of 75 mg percent. The same protocol was followed in the repeated-dose experiment where subjects were exposed to the same level of alcohol for three consecutive nights.

In the single-dose study, sleep onset latency and latency to slow wave sleep were significantly reduced compared to baseline levels. Sleep onset was signified by the first stage 2 sleep spindle, which occurred an average of 10.7 minutes after going to bed following a single dose of alcohol; a significant reduction from a baseline average of 15.7 minutes. Latency to slow wave sleep (from stage 2 sleep) was an average of 23.7 minutes during baseline nights, and an average of 15.4 minutes during the alcohol night. These changes in sleep architecture represent the sedative effects of alcohol, a known CNS depressant. Further to this, however, the length of the first REM sleep episode was significantly reduced from 18.6 minutes during baseline, to 12.1 minutes during the alcohol night. In the second half of the sleep period there was a significant "rebound" increase in REM sleep and a corresponding reduction in stage 2 sleep. Alcohol and time of night interacted significantly to shift the distribution of REM sleep to the last 4 hours of the night. There was a significant loss of high frequency beta rhythms in the EEG and an increase in the amount and synchronicity of alpha rhythms, consistent with previous findings that alcohol has direct effects on the brain (Rundell et al., 1972). There was no significant difference in the distribution of the percent stages of sleep between the baseline and recovery nights in the single-dose study, however, sleep onset latency and latency to REM sleep (from stage 2 sleep) were both significantly reduced in the recovery night compared to baseline conditions.

In the three-night repeated-dose study, a reduction in REM time was observed in the first half of the night on the first alcohol night, consistent with the findings of the single-dose study. There was no consistent reduction of REM sleep in the second or third alcohol nights, commensurate with the adaptation effects observed in Yules et al.'s (1967) study. The sedative effects of alcohol observed in the single-dose study were apparent as trends

in the repeated-dose study, with significant results appearing only when the three alcohol nights were combined prior to analysis. Changes in EEG frequencies in the repeated-dose study also mirrored those found in the single-dose study but did not reach significance. These results are also indicative of adaptation effects. Rundell et al.'s (1972) results indicate that with a single dose of alcohol, both alcohol effects and rebound effects are contained within the one night, and alcohol effects do not persist beyond the night of ingestion (although compensation effects are observed on recovery nights). When alcohol is administered repeatedly, however, adaptation effects are observed and changes in sleep architecture and EEG content are minimised.

The most recent studies also consistently demonstrate the same effect of single (e.g., Van Reen, Jenni, & Carskadon, 2006) and repeated (Feige et al., 2006) doses of alcohol on sleep polysomnography. In a sample of healthy young women, Van Reen et al. (2006) found the same REM sleep and sedative effects demonstrated in previous studies when they administered a single, moderate dose of alcohol to their subjects. Compared to a placebo condition, they found a reduction in REM sleep time, an increase in stage 4 sleep in the first 2-hour interval, and increased EEG power in the alpha range during non-REM sleep. Feige and colleagues (2006) used a within-subjects crossover design to investigate sleep changes associated with repeated-doses of alcohol of different amounts. Healthy subjects received moderate doses of alcohol (0.03 BAC) representing "normal social drinking" and higher doses of alcohol (0.1 BAC) representing "alcohol abuse", with a 1-week washout period. Interestingly, no changes in sleep polysomnography or subjective parameters of sleep were found when moderate levels of alcohol were administered at bedtime for three consecutive nights. With higher doses of alcohol, however, both sedative and REM sleep effects of alcohol were observed. Subjects had reduced sleep onset latency, a reduced number of wake periods, decreased stage 1 and REM sleep, and increased slow wave sleep in the first half of the night, with rebound effects evident in the second half of the night (increased stage 1 and REM sleep). Feige and colleagues found no evidence of rebound or withdrawal effects in the two recovery nights following three consecutive nights of 0.1 BAC.

The sedative effects of alcohol (i.e., increased slow wave sleep at the expense of stage 2 sleep) are also observed in short naps of 1-hour duration (Van, O'Boyle, & Hume, 1995), and have significant implications for arousal thresholds. Using the robust methodology of a repeated-measures design, Ball and Bruck (2004) demonstrated that moderate (0.05 BAC) and higher (0.08 BAC) levels of alcohol intoxication significantly impaired healthy young adults' ability to awaken to three different types of alarm signal, when compared to their awakening threshold on a sober night.

2.6.1 Alcohol and arousal thresholds

Persons are likely to be significantly more difficult to arouse from sleep if arousal is attempted during the ascending limb of the BAC curve or the first half of the sleep period, due to the sedative effects of alcohol. Indeed, Ball and Bruck (2004) found significantly increased awakening thresholds for participants who imbibed moderate (0.05 BAC) and higher (0.08 BAC) levels of alcohol immediately prior to bed and were awoken 90 seconds into stage 4 sleep. Using a modified method of discrete limits to awaken participants, alarms were initially commenced at 35 dBA (the sound intensity of a whisper) and then increased in 5 dBA increments after 30 seconds up to a maximum level of 95 dBA (equivalent to loud industrial noise). During 36% of trials at 0.05 BAC, participants failed to wake to the alarm prior to the 95 dBA presentation, or failed to wake at all. This was increased to 42% of trials in participants who were intoxicated to the 0.08 BAC level.

Further research shows that visual and tactile alarms have even greater arousal thresholds than auditory alarms in attempting to wake alcohol-affected individuals (Bruck, Thomas, & Ball, 2007). In a within-subjects repeated-measures design, young adults intoxicated to the level of 0.05 BAC, were awoken from stage 4 sleep with either an auditory (alarm signal), tactile (bed or pillow shaker) or visual (strobe light) stimulus. Results showed that moderate levels of alcohol intoxication greatly impaired one's ability to awaken to an alarm, particularly for arousal by tactile or visual stimulus. Only 24% of participants awoke to the strobe light when it was presented at or below benchmark intensity (i.e. commensurate with US Fire Standards), and 32% did not wake to this stimulus at all. An

average of 61% of individuals woke to the two tactile stimuli at or below benchmark levels (i.e., intensity as purchased), whilst approximately 28% slept through all intensity level presentations of this stimulus. The results for the auditory alarm were significantly better. Averaging the results for four different alarm types, 85% of participants awoke to the auditory alarms at or below benchmark intensity (75 dBA), 12% woke to the auditory alarms when presented above benchmark levels, and only 2.8% of participants did not wake to the auditory alarms at all. Nonetheless, moderate levels of alcohol intoxication still significantly increased participants' arousal thresholds when awoken from stage 4 sleep. The results of Ball and Bruck (2004) and Bruck et al. (2007) combined would indicate that alcohol may have a dose-dependent effect on arousal thresholds, with higher levels of alcohol intoxication causing greater increases in arousal thresholds than moderate levels of alcohol intoxication, and with tactile and visual alarms being less successful than auditory signals in waking alcohol-impaired young adults.

2.7 Alcohol and neurocognitive functioning

It has long been recognised in the research literature and the wider community that alcohol consumption is associated with changes in cognitive functioning, most notably psychomotor speed, memory, attention, and judgment (Allen, Frantom, Forrest, & Strauss, 2006). There is experimental evidence to support that alcohol impairs a number of more specific cognitive processes that are subcomponents of the aforementioned cognitive domains, including working memory (e.g., Finn, Justus, Mazas, & Steinmetz, 1999; Petros, 1985), event-related potentials (e.g., Lukas, Mendelson, Kouri, Bolduc, & Amass, 1990; Wall & Ehlers, 1995), attentional shifting (Jääskeläinnen, Schröger, & Näätänen, 1999), selective attention (e.g., Abroms & Fillmore, 2004; Fillmore, Dixon, & Schweizer, 2000), divided attention (e.g., Lex, Rhoades, Teoh, & Mendelson, 1994), verbal memory (e.g., Jones, 1973; Schweizer et al., 2006), visual memory (e.g., Schweizer et al., 2006) and learning (Pihl, Paylan, Gentes-Hawn, & Hoaken, 2003). There are two prominent theoretical orientations regarding the precise mechanism through which alcohol affects these cognitive processes: (1) alcohol interferes with the focus of attention (e.g., Steele & Josephs, 1990) and (2) alcohol restricts response inhibition (e.g., Fillmore & Vogel-Sprott, 1999). According to the attention-allocation

model, alcohol intoxication restricts attentional resources so that only the most salient cues in the environment are attended to at the expense of fully processing all the available information. Experimentally, this is observed as poor performance on tasks of divided attention, whilst performance is essentially preserved on tasks that participants perceive to be the most important in a multi-task paradigm (Bartholow et al., 2003). Behaviourally, the attention-allocation model has been used to explicate alcohol-related social behaviours such as aggression (e.g., Graham et al., 1998) and sexual risk-taking among adolescents (e.g., Cooper & Orcutt, 1997). The response inhibition model is based on a theory of cognitive control that deems behavioural activation and behavioural inhibition to be cognitively independent. According to the model, alcohol selectively impairs behavioural inhibition and this is demonstrated as poor performance on go/no-go tasks where participants are required to inhibit a prepotent or primed response (e.g., Fillmore & Vogel-Sprott, 1999).

Bartholow et al. (2003) found support for the response inhibition model in a group of 45 young healthy adults when performing a modified flanker task under conditions of either placebo levels (0.04 g/kg), moderate levels (0.40 g/kg) or higher levels (0.80 g/kg) of alcohol intoxication. In the experimental task the target stimulus was flanked by response-compatible or response-incompatible letters. ERP data and measures of response speed and performance accuracy revealed that alcohol affected participants' accuracy but not their speed of performance. This lead Bartholow et al. to conclude that response selection processes and not attentional processes were affected in their alcohol-impaired subjects.

2.7.1 'Accuracy-speed trade-off effects'

It would appear from Bartholow et al.'s (2003) study that alcohol selectively affects the accuracy of task performance, and not the speed. Recent studies indicate, however, that performance speed and accuracy interact in a complex manner with the biphasic segments of the BAC curve. Further complicating the interaction is the fact that, similar to the effects of alcohol on sleep architecture, recovery and adaptation effects also apply to the effect of alcohol on cognitive functioning. In their recent meta-analysis, Schweizer

and Vogel-Sprott (2008) identified that on tasks of inhibition and information processing, performance accuracy was affected on both the ascending and descending limbs of the BAC curve, whilst impairments in speed of performance were only evident on the ascending limb. Similar trends are evident on tasks of selective attention and learning. This pattern of results indicates that whilst speed of performance is affected by alcohol, it shows acute tolerance effects. That is, speed of performance impairment is substantially less on the descending limb of the BAC curve, when compared to the equivalent BAC level on the ascending limb.

Further to this, a number of studies have assessed a variety of neurocognitive functions on both the ascending and descending limbs of the BAC curve, but not at equivalent BAC levels. In most studies, performance was tested at a declining BAC level that was lower than the rising BAC test. Given that lower BAC levels are expected to produce weaker effects, a reduction in the impairment seen during the ascending limb is expected when performance in assessed on the descending limb; such an observation would be indicative of recovery effects (Schweizer & Vogel-Sprott, 2008). Indeed, such effects were observed for speed of performance but not performance accuracy on tasks of inhibition, working memory, and learning. It appears that performance accuracy is affected by alcohol bi-phasically whilst speed of performance is affected initially, but consistently shows acute tolerance and recovery effects (Schweizer & Vogel-Sprott, 2008). These appear to be robust findings as 94% (17/18) of tasks included in the review that assessed errors or task accuracy failed to show acute tolerance or recovery, whilst 100% (11/11) of tasks that assessed reaction time or speed of performance demonstrated acute tolerance and recovery during declining BACs.

2.8 Interactions between alcohol and sleep processes

Due to the high risk posed to safety when sleepiness and alcohol are combined, most studies investigating the interaction between alcohol and sleep processes have primarily assessed effects on driving performance. A variety of carefully executed and methodologically robust studies clearly demonstrate that the combination of sleep restriction and acute doses of alcohol impair driving performance to a greater degree than

sleep restriction (Banks, Catcheside, Lack, Grunstein & McEvoy, 2004; Vakulin et al., 2007) or alcohol intoxication (Howard et al., 2007) alone. This occurs during both daytime and nighttime assessments indicating that the detrimental effects of alcohol and sleep restriction are evident at different points on the circadian phase. Vakulin et al. (2007) assessed driving simulator performance in a group of healthy young men in a repeated measures study under conditions of:

- Normal sleep without alcohol
- Sleep restriction alone (4 hours)
- Sleep restriction in combination with 0.025 g/dL of alcohol and
- Sleep restriction in combination with 0.035 g/dL of alcohol.

Participants undertook a 70-minute afternoon session of driving simulation commencing at 2pm and were assessed for steering deviation, braking reaction time, and number of collisions. The results indicated that compared to normal sleep or sleep restriction alone, the combination of sleep restriction and the higher dose of alcohol significantly increased steering deviation, with concomitant increases in alpha/theta EEG activity throughout the simulation and significant increases in self-reported sleepiness and negative driving performance ratings. Whilst effectively controlling for individual differences in driving ability and approach to self-report measures by using a within-subjects design, the study shows that combining low doses of alcohol and sleep restriction impairs both objective and subjective performance indicators on a driving simulation task, more so than sleep restriction alone.

In a similar study, Banks and colleagues (2004) assessed driving simulator performance during the nighttime. Healthy young adults were assessed on driving simulator performance and their ability to predict crash risk at 1am on two occasions with a 1-week washout period; once under conditions of sleep restriction (5 hours in bed) and once under conditions of combined sleep restriction and alcohol intoxication (mean BAC = 0.035 g/dL, *SD* = 0.015 g/dL). EEG revealed increased microsleeps during the combined condition when compared to sleep restriction alone. In addition to this, the combination of sleep restriction and legal levels of alcohol intoxication significantly reduced participants' performance on the driving simulator and eliminated their ability to

accurately predict crash risk. Banks and colleagues (2005) have since shown, in a within-subjects experimental design study, that the Maintenance of Wakefulness Test (MWT; conducted prior to the driving simulation) can accurately predict driving performance and EEG-determined microsleeps in healthy persons who are both sleep deprived and alcohol impaired. In their study, sleep latency during a 40-minute MWT was inversely correlated with steering deviation, braking reaction time, number of crashes and number of microsleeps. The MWT also accurately predicted braking reaction time on the driving simulation task in a group of partially sleep-deprived healthy persons. These findings have important implications for predicting and preventing perilous driving practices that threaten community safety.

Howard and colleagues (2007) found similar decrements in the driving simulation performance of a group of volunteer professional drivers following an acute dose of alcohol in combination with mild sleep deprivation. Participants in this study were assessed on a driving simulation task and a psychomotor vigilance task under four different conditions, including

- alcohol intoxication (measured at 0.03 BAC and 0.05 BAC) in a non-sleep deprived state (i.e., 12-15 hours awake),
- alcohol intoxication (measured at 0.03 BAC and 0.026 BAC) in a sleep deprived state (18-21 hours awake), and
- both the non-sleep deprived and sleep deprived conditions without alcohol.

Professional drivers had significantly reduced reaction time and significantly more lapses on the vigilance task, and significantly greater variation in lane position and speed on the driving simulation task when performing under conditions of extended wakefulness (18-21 hours awake) and low-dose alcohol (0.03 BAC) compared to a condition of alcohol intoxication (0.05 BAC) only. Therefore, the combination of mild sleep deprivation and low-dose alcohol poses greater risks to driver safety than alcohol intoxication to a level known to appreciably increase the risk of having a crash (Howard et al., 2007).

The detrimental effects of combined alcohol and prolonged wakefulness were also observed in a group of young healthy males in a 30-minute driving simulation task

(Arnedt, Wilde, Munt, & Maclean, 2000). Subjects provided self-report ratings of sleepiness and made simultaneous and retrospective ratings of their impairment regarding driving simulator operation. A synergistic interaction was observed between 20 hours of prolonged wakefulness and alcohol impairment to 0.08 BAC, whereby ratings of subjective sleepiness were significantly greater under this condition than what would be expected from the additive effects of the conditions of alcohol impairment and prolonged wakefulness alone. Driving performance was also worse under this condition, but the decrements did not reach significance. Arnedt et al. (2000) found a modest association between subjective and objective levels of impairment during the combined condition, suggesting that subjects had only a moderate appreciation of the magnitude of their performance decrements. Subjects performed the driving simulator task at two occasions under conditions of alcohol intoxication; once at 30 minutes post-ingestion of alcohol and again at 90 minutes post-ingestion, timed to correspond with the ascending and descending limbs of the BAC curve, respectively. BAC levels were lower at the second session which corresponds to the descending limb of the BAC curve, however, subjects consistently performed worse during this session.

Although these results are discrepant to a myriad of studies that suggest performance decrements are greater during the ascending limb of the BAC curve (e.g., Gengo, Gabos, & Straley, 1990; Hurst & Bagley, 1972; Jones, 1973; Nicholson et al., 1992; Young, 1970), Arnedt et al.'s (2000) subjects were assessed at 4am on the driving simulation task whilst the studies mentioned above were conducted during the daytime with no concomitant sleep deprivation. Therefore, it is likely that the prolonged decrements found at 90 minutes post-ingestion are due to a combination of higher levels of sleepiness at the second session and circadian influence on performance. However, Arnedt et al.'s study is lacking comparable data that would allow them to determine the isolated effects of prolonged wakefulness in their subject sample, as subjects were not tested without alcohol on two comparable occasions. Driving simulation performance at the second session is therefore confounded with time awake, BAC level and the effects of having to perform the task again.

Not all studies assessing driving performance show consistent effects of combined alcohol intoxication and sleep deprivation. Huntley and Centybear (1974) found that at moderately high doses of alcohol (0.09 BAC), similar to the intoxication level of Arnedt et al.'s subjects, sleep deprivation (29 hours) and alcohol produced an antagonistic reaction whereby sleep deprivation *reduced* the influence of alcohol on course steering rate on a driving performance task. Wilkinson & Colquhoun (1968) found that at moderate blood alcohol concentration levels (i.e., 0.03 BAC or greater), sleep deprivation enhanced alcohol-related deficits on a continuous performance task, whereas at low blood alcohol concentration levels (i.e., less than 0.03 BAC), sleep deprivation tempered the effects of alcohol-related impairments in reaction time; an antagonistic interaction. It appears, however, that these latter studies did not consider the role of circadian phase or the biphasic effects of alcohol metabolism in their experimental protocol.

In addition to driving performance, decrements associated with the interaction between alcohol and sleep processes has been found for a small number of simpler cognitive processes including event-related potentials (Krull, Smith, Sinha, & Parsons, 1993; Peeke, et al., 1980) and reaction time and performance accuracy on a matching and categorisation task requiring a choice of four response options (Peeke et al., 1980). Peeke et al. (1980) measured a variety of physiological and cognitive variables 40 minutes after alcohol consumption commenced, at two levels of intoxication (0.90 mL/kg and 0.45 mL/kg), under conditions of 0 and 26 hours sleep deprivation in a within-subjects partial cross-over design. Mild performance impairments were evident during conditions of sleep deprivation alone and alcohol impairment alone, as characterised by reduced subjective alertness and increased latency of cortical evoked potential components. Increases in state anxiety were observed in the sleep deprivation alone condition, whilst alcohol produced increases in heart rate. When alcohol and sleep deprivation were combined, measures of heart rate, self-reported alertness, state anxiety, latency of early evoked potential components and reaction time on the categorisation task exhibited an antagonistic interaction. Synergistic effects were evident on late evoked potential components and performance accuracy on the categorisation task. It is interesting to note the speed-accuracy trade-off effect on the cognitive task, whereby

speed of performance increased antagonistically and accuracy of performance decreased synergistically when alcohol and sleep deprivation were combined. Although it is not reported, it can be deduced from the experimental protocol that Peeke et al's subjects were on the ascending limb of the BAC curve when these measurements were taken. Their results are in agreement with Bartholow et al. (2003) who found that alcohol affected participants' accuracy on a modified flanker task, but not their speed of performance.

A review of the limited literature assessing alcohol and sleep processes indicates that the combined interaction produces effects on tasks assessing complex cognitive processes greater than those produced by either factor alone, sometimes to the extent of generating a synergistic interaction. This is not true for all tasks or all studies, however, and may be dependent on the level of intoxication, the type of task, circadian phase, and the position on the BAC curve of alcohol metabolism at which cognitive performance is assessed.

2.9 Rationale

The interaction between alcohol impairment and sleep inertia has not been empirically investigated previously. However, research demonstrates that alcohol impairment and sleep process do interact, although the precise nature of this interaction may be hard to predict. Both alcohol and sleep inertia are ubiquitous factors that have important implications for human performance in an emergency waking context, including escape from a fire emergency. Indeed, research shows that alcohol and being asleep in a residential home are two important risk factors for death in a fire.

2.10 Aims and hypotheses

The current study aims to investigate the interaction of sleep inertia and moderate alcohol impairment on neurocognitive processes relevant to a fire emergency situation. In doing so, neurocognitive functioning and self-reports of subjective sleepiness and clearheadedness will be assessed under conditions of sleep inertia alone, alcohol impairment alone and combined sleep inertia and alcohol impairment in a within-subjects repeated-measures design, as per the protocol summarised below in Table 2. Note that,

while counterbalancing of the alcohol and sober nights would have been the most desirable design, this study was conducted within the framework of a larger study on arousal thresholds to different emergency notification signals (Bruck et al., 2007) and counterbalancing was not possible. Implications of this will be discussed in section *5.7*.

Table 2.

Summary of cognitive testing conditions.

	Night 1 & 2: ALCOHOL	**Night 3: SOBER**
Time 1 (2 hours prior to participant's bedtime)	Baseline sober	Baseline sober
Time 2 (immediately prior to lights out)	Baseline 0.05 BAC ***Alcohol Only***	
Time 3 (immediately after final awakening)	Sleep inertia 1 ***Combined Alcohol & Sleep Inertia (0-10 minutes)***	Sleep inertia 1 ***Sleep Inertia Only (0-10 minutes)***
Time 4 (10 minutes post awakening, immediately following Time 3)	Sleep inertia 2 ***Combined Alcohol & Sleep Inertia (10-20 minutes)***	Sleep inertia 2 ***Sleep Inertia Only (10-20 minutes)***

The study will consider four hypotheses, with the first having two parts. The three cognitive measures referred to below are the Descending Subtraction Task (DST), Symbol Digit Substitution Task (SDST) and Letter Cancellation Test (LCT). Subjective sleepiness and clearheadness will each be assessed by subjective ratings on a 9-point (Karolinska Sleepiness Scale; KSS) and 5-point (Clearheadedness Rating Scale; CH) visual analog scale, respectively. Justifications of the inclusion of the dependent measures will be discussed in sections *3.3.1* (conceptual) and *3.2* (operationalisations). 'Time' and 'night' (e.g., sober or alcohol) information relate to Table 2.

2.10.1 Hypothesis 1a

Conditions of sleep inertia will produce cognitive performance decrements, increased subjective sleepiness and decreased subjective clearheadedness, compared to 'baseline' conditions.

Operationalisation.

Participants will perform significantly poorer on the cognitive measures (DST, SDST, LCT) and report significantly higher levels of subjective sleepiness and lower levels of subjective clearheadedness on the rating scales during the sleep inertia condition (sober, time 3) relative to the baseline condition (sober, time 1).

Rationale.

A vast body of literature (e.g., Balkin & Badia, 1988; Dinges et al., 1985; Jewett et al., 1999; Wertz et al., 2006) demonstrates consistent sleep inertia effects across a variety of neurocognitive functions, including those required in emergency situations such as attentional processes (e.g., Ferrara et al., 2000a; Tietzel & Lack, 2001), working memory (e.g., Dinges et al., 1985; Tassi et al., 2003), and psychomotor speed (e.g., Bruck & Kritikos, 2007; Koulack & Shultz, 1974). This experimental evidence is consistent with the arousal hypothesis and other theoretical models of sleep inertia.

2.10.2 Hypothesis 1b

The speed, but not the accuracy, of cognitive performance will be affected under conditions of sleep inertia, relative to 'baseline' conditions.

Operationalisation.

Participants will produce significantly less responses on the cognitive measures during the sleep inertia condition (sober, time 3 & 4) relative to the baseline condition (sober, time 1). Hence, the decrements in cognitive performance during the sleep inertia condition will be characterised by a reduced efficiency in performance (i.e., reduced number of total responses for the duration of each task), whilst performance accuracy will be unaffected.

Rationale.

The basic tenet of the arousal hypothesis (Malmo, 1959) predicts that the performance decrements observed during sleep inertia are produced by a depression of the arousal system, causing in turn, an overall reduction in neurocognitive processing. This general

slowing of cognitive processes is largely independent of the type of task, and in the absence of concomitant sleep deprivation, is also unrelated to the level of accuracy of cognitive performance (see Tassi & Muzet, 2000). Since the mid-1960s when sleep inertia was first systematically investigated, a number of empirical studies have confirmed that conditions of sleep inertia selectively affect the speed and not the accuracy at which cognitive tasks can be performed (e.g., Jewett et al., 1999; Seminara & Shavelson, 1969; Webb & Agnew, 1964).

2.10.3 Hypothesis 2

Moderate alcohol consumption will produce cognitive performance decrements compared to 'sober' conditions.

Operationalisation.

Participants will perform significantly poorer on the cognitive measures (DST, SDST, LCT) during conditions of 0.05 BAC (alcohol, time 2) relative to the 'sober' baseline condition (alcohol, time 1).

Rationale.

It has long been recognised in the research literature and the wider community that alcohol consumption is associated with changes in cognitive processing, most notably psychomotor speed, memory, attention, and judgment (Allen et al., 2006). Experimental data supports changes in information processing, selective attention, and working memory abilities, amongst others, during both the ascending and descending limbs of the BAC curve (Schweizer & Vogel-Sprott, 2008). This experimental evidence is consistent with the known physiological effects of alcohol, a central nervous system depressant, on the brain, particularly prefrontal regions (Sano et al., 1993) that are involved in higher-level cognitions including working memory and executing attentional control (Hannay, Howieson, Loring, Fischer, & Lezak, 2004; Sano et al., 1993).

2.10.4 Hypothesis 3

Conditions of combined sleep inertia and alcohol impairment will produce cognitive performance decrements, increased subjective sleepiness and decreased subjective clearheadedness, compared to conditions of sleep inertia alone or alcohol impairment alone.

Operationalisation.

Participants will perform significantly poorer on the cognitive measures (DST, SDST, LCT) and report significantly higher levels of subjective sleepiness and lower levels of subjective clearheadedness on the rating scales (KSS, CH) during the combined sleep inertia and alcohol condition (alcohol, time 3 & 4) relative to both the sleep inertia alone (sober, time 3 & 4) and alcohol alone (alcohol, time 2) conditions.

Rationale.

Given our knowledge of the detrimental effects of both sleep inertia and alcohol on cognitive performance, it is predicted that when these two factors are combined, even further decrements in cognitive functioning will result. Although the current study is the first to investigate the combined effects of sleep inertia and alcohol consumption on neurocognitive processing, early research has shown that alcohol and sleep processes, specifically sleep deprivation, do interact. Wilkinson & Colquhoun (1968) found that at moderate blood alcohol concentration levels (i.e., BAC 0.03 or greater), sleep deprivation enhanced neurocognitive deficits, whereas at low blood alcohol concentration levels, sleep deprivation tempered the effects of alcohol-related impairments in reaction time; an antagonistic interaction. Peeke and colleagues (1980) found a similarly complex interaction between 26 hours of sleep deprivation and moderate alcohol intake. They reported a synergistic increase in task errors and P_{300} evoked potential latency, whilst decrements in alertness and reaction time found during conditions of sleep deprivation and alcohol consumption alone were absent during the combined condition; another antagonistic result. A number of studies report greater performance decrements on driving simulation tasks during conditions of combined alcohol impairment and partial sleep deprivation, than under conditions of partial sleep loss (e.g., Banks et al., 2004;

Vakulin et al., 2007) or alcohol intoxication alone (e.g., Howard et al., 2007). Although sleep deprivation and sleep inertia are not necessarily believed to be theoretically or functionally the same, previous research indicates that alcohol and sleep processes do indeed interact, although the precise nature of this interaction may be hard to predict.

2.10.5 Hypothesis 4

Subjective feelings of sleepiness and clearheadedness will correlate with each other.

Operationalisation.

Participants will self-report lower levels of clearheadedness (i.e., higher scores on the CH) when they report higher levels of sleepiness (i.e., lower scores on the KSS). Self-reports of subjective sleepiness and clearheadedness will be significantly correlated during all conditions.

Rationale.

A number of studies demonstrate that sleep inertia suppresses subjective feelings of alertness (e.g., Bruck & Pisani, 1999; Jewett et al., 1999; Hofer-Tinguely et al., 2005; Hou et al., 2007; Tietzel & Lack, 2001) and that self-report measures of alertness during sleep inertia are generally consistent with physiological parameters of sleep and sleep inertia such as thermoregulation and circadian timing (e.g., Babkoff et al., 1991; Kräuchi et al., 2004). Further, various subjective self-report measures are often correlated when administered under conditions of sleep inertia (e.g., Bruck & Pisani, 1999; Gillberg et al., 1994). Given that increased subjective sleepiness and decreased subjective clearheadedness (grogginess) are both established features of sleep inertia and may relate mutually under other circumstances, it is expected that ratings on these measures will change in unison under conditions of sleep inertia, alcohol impairment, and combined sleep inertia and alcohol impairment, reflecting changes in levels of subjective sleepiness and clearheadedness.

CHAPTER THREE: METHOD

3.1 Participants

Twenty-four young adults aged between 18 and 26 years (M = 20.7, SD = 2.4) participated in the current study. The intent was to have males and females equally represented, and the final cohort of participants comprised 11 males and 13 females. Participation was on a voluntary basis contingent upon meeting selection criteria for the study. Selection criteria required that participants must not be taking any medication that affected their sleep, usually not have significant difficulty falling asleep, not have a sleep disorder, and be aged between 18 and 26 years. A self-reported absence of physical or neurological conditions that affect the ability to perceive or respond to visual, tactile, or auditory stimuli, and a self-reported regular consumption of alcohol (i.e., consumed alcohol at least one night per week; criterion designed according to the National Drug Strategy, 1998; Adhikari & Summerill, 2000) were also required to meet eligibility criteria for the study. In addition to this, participants were screened to ensure they met a hearing threshold criterion, defined as a threshold below 20 dBA across a range of frequencies. Of the 35 people recruited overall, six potential participants were excluded on this latter criteria. Attrition was 17% due to participant 'drop out' (i.e., five of the 29 eligible participants discontinued their involvement prior to the completion of the study, three after completing two study nights and two after completing a single study night). Participants were remunerated $80 per night for their time, and received a completion bonus of $75 for participating in three study nights.

3.2 Apparatus

The following tasks were specifically chosen to capture the cognitive functions believed to underpin the performance of an effective evacuation during an emergency fire situation (see section *3.3.1*).

3.2.1 The Descending Subtraction Task (DST)

The Descending Subtraction Task (DST) is a demanding verbal arithmetic task that recruits working memory functions (Tassi et al., 2003). Tasks of working memory

function tap the ability to simultaneously retain information in memory (e.g., multi-digit numbers) and manipulate or perform some operation on it (e.g., subtraction). In the DST, participants are verbally given a three-digit number, such as 702, which they are required to repeat aloud as their initial response. Participants are then required to mentally subtract the number 9 from this starting number (702), and verbally produce the answer (693). This answer becomes the new number from which 8 must be subtracted. The subtrahend, the number that is subtracted, progressively decreases by 1 with each calculation, until the subtrahend reaches the value of 2. At this point, the participant returns to subtracting the number 9 and continues the descending subtraction sequence. If participants fail to respond within 20 seconds, or indicate that they have become lost in the sequence, they are prompted to take a guess and continue. In keeping with the intended administration of the test, participants were instructed to "work as fast as possible and keep a steady pace, but try to remain as accurate as possible" (Dinges et al., 1985, p. 42; see Appendix A for administration instructions and scoring template). Participants did not receive feedback on their performance during the task, however self-correcting was allowed. The DST, performed continuously for 3 minutes, yields measures of both speed and accuracy. Calculating the number of responses provided a measure of speed, whilst responses were assessed for errors to determine a percentage accuracy score (i.e., number of correct responses divided by total number of responses). Twelve even 3-digit numbers were generated to cover each condition over three nights and during the practice trial, and these were randomly counterbalanced across participants. Therefore, each participant received a different starting number for each condition of the study.

The DST, administered as described by Dinges et al. (1985), considerably taxes working memory functioning. In addition to performing the simple arithmetic calculations, participants need to "hold on-line" both the number to subtract *from*, and the subtrahend, both of which change after each response. This places a high cognitive load on the phonological loop and the central executive in the working memory store. The task was non-intrusive and could be performed with participants lying in bed. It is these properties that made the DST optimal for capturing the acute effects of sleep inertia on cognitive

functioning, as assessment could begin within seconds of sleep offset. The DST has been previously shown to be sensitive to conditions of sleep inertia (e.g., Dinges et al., 1985; Ferrara et al., 2000a; Ferrara et al., 2000b; Tassi et al., 2003) and is one of the most widely used tools to examine neurocogntive functioning upon awakening (Ferrara et al., 2000a).

3.2.2 The Symbol Digit Substitution Task (SDST)

Originally published by Aaron Smith in 1973 as a screening tool for the detection of cerebral dysfunction (Strauss, Sherman, & Spreen, 2006), the Symbol Digit Modalities Test, also known as the Symbol Digit Substitution Task (SDST) in alternative form, is now commonly used as an assessment of divided attention (e.g., Ponsford & Kinsella, 1992; Strauss et al., 2006). This task also recruits visual scanning, tracking, and psychomotor speed functions and requires a grapho-motor response (Strauss et al., 2006). In this pencil-and-paper task, participants are presented with a coding key which consists of a number paired with an arbitrary abstract symbol; the numbers 1 to 9 correspond to different symbols. On this same response page, several rows of the abstract symbols are presented at random, with empty boxes below in which participants are required to fill-in the corresponding digit. Participants are required to work quickly and accurately to fill-in as many boxes *in order* as possible within a 2 minute time period. Scores are calculated by summing the number of *correctly completed* items, and points are not deducted for errors on this task. Hence, measures of speed and accuracy are combined in this task as the number of correct responses produced in the allocated time is calculated. Particularly speedy and accurate performance also recruits working memory functions as the participant is required to have held some symbol-digit pairs in accessible consciousness (Strauss et al., 2006). Ten parallel forms of the SDST, developed by Teitzel and Lack (2001; see Appendix C for an example) were utilised in the current study and randomly counterbalanced across conditions for each participant. Each form has its own set of unique symbols that correspond to the numbers 1 to 9 and are, according to pre-testing conducted by Teitzel and Lack (2001), of equal difficulty.

The original Symbol Digit Modalities Test has a test-retest reliability of .80 in normal populations (with an average test-retest interval of 29 days; Strauss et al., 2006) and correlates highly with other tests of sustained attention (e.g., the Test of Everyday Attention) and divided attention and tracking (e.g., the Digit-Symbol/Coding subtest of the Wechsler Adult Intelligence Scale; Strauss et al., 2006; Hinton-Bayre & Geffen, 2005). Alternate forms, such as those developed by Hinton-Bayre, Geffen, and McFarland (1997), are reported to have an internal consistency of $r > 0.87$, and adequate test-retest reliability, however, practice effects have been found on retesting using parallel alternate forms (Hinton-Bayre & Geffen, 2005; Hinton-Bayre et al., 1997), unless the test-retest interval is yearly or longer (Uchiyama et al., 1994). Hence, two baseline assessments are recommended prior to formal testing to alleviate practice effects (Hinton-Bayre, Geffen, Geffen, McFarland, & Friis, 1999). The SDST used in the current study has been previously shown to be sensitive to conditions of sleep inertia (e.g., Tietzel & Lack, 2001).

3.2.3 The Letter Cancellation Test (LCT)

The Letter Cancellation Test (LCT) is a pencil-and-paper task that assesses selective attention by requiring participants to selectively attend to two predetermined stimuli and ignore competing stimuli. When administered for an extended period of time, it also assesses sustained attention. Similar to the SDST, the LCT also recruits visual scanning and motor speed skills. Participants were presented with a large 32 x 45 matrix of random capital letters and asked to search for two particular stimuli, e.g., 'A' and 'N' (see Appendix D). Participants were required to mark these stimuli with a highlighter working through the matrix line-by-line from left to right without skipping any rows. The dependent measure was the number of correct identifications in a 4 minute time period; this calculation combines measures of speed and accuracy. There are a number of variants of the LCT (Lezak, Howieson, & Loring, 2004). Ten parallel forms of the LCT, constructed by Tietzel and Lack (2001), were utilised during the current study and randomly counterbalanced across conditions for each participant. Whilst the same test matrix is used each time, the target letters change in each alternative form of the task.

There are 160 targets in each parallel form, representing approximately 11% of the matrix.

Data are not available on the psychometric properties of the LCT, although other similar tests of visual attention (e.g., Bells Cancellation Test) are known to be psychometrically sound (Strauss et al., 2006). The LCT has been previously shown to be sensitive to performance changes associated with sleep processes (e.g., Foret, 1992; Fort & Mills, 1972; Tietzel & Lack, 2001). Under conditions of sustained attention (i.e., task duration of 4 minutes), Tietzel and Lack (2001) found the LCT to be sensitive to the effects of sleep inertia.

3.2.4 The Karolinska Sleepiness Scale (KSS)

The Karolinska Sleepiness Scale (KSS), constructed by Åkerstedt and Gillberg (1990), is a 9-point Likert-type self-report scale used to subjectively assess sleepiness. The anchor points of the scale are (1) 'extremely sleepy, fighting sleep' and (9) 'extremely alert', with seven gradations of sleepiness between and verbal descriptions of every second point. The median point of the scale is (5) 'neither alert nor sleepy' (see Appendix E). Participants were asked to rate how they felt (level of sleepiness) right at that moment. Lower scores on the KSS indicate greater levels of sleepiness. The KSS has been used extensively in previous sleep inertia research to provide a subjective measure of sleepiness (e.g., Bruck & Pisani, 1999; Kräuchi et al., 2004; Sallinen et al., 1998), and correlates highly with EEG (Åkerstedt & Gillberg, 1990; Kaida et al., 2006), EOG (Åkerstedt & Gillberg, 1990) and behavioural indicators of sleepiness (Gillberg et al., 1994; Kaida et al., 2006). It also correlates highly (r = 0.73-0.86) with subjective sleepiness measured on a different visual analog scale (Gillberg et al., 1994).

3.2.5 The Clearheadedness Rating Scale (CH)

The Clearheadedness Rating Scale (CH), developed by Bruck and Pisani (1999), is a 5-point Likert-type scale used to subjectively assess clearheadedness, or the grogginess associated with sleep inertia. Participants rated their current level of clearheadedness from (1) 'extremely' clearheaded to (5) 'not at all' clearheaded. The median point of the

scale is (3) 'moderately' clearheaded (see Appendix F). Participants were required to rate how clearheaded they felt right at that moment. Higher scores on the CH correspond to *lower* levels of clearheadedness (i.e., greater levels of grogginess). Although it has not been systematically validated, there is evidence that the CH provides a valid and sensitive assessment of clearheadedness during an experimental sleep inertia protocol (Bruck & Pisani, 1999).

3.3 Design

The study used a within-subjects repeated measures experimental design to compare the effects of sleep inertia and alcohol-impairment on neurocognitive function. Specifically, working memory, divided attention, and selective and sustained attention, in addition to mental tracking, visual scanning and psychomotor speed functions operationalised into three neuropsychological tasks, were examined as dependent measures. These tasks were specifically chosen to capture the cognitive functions that underpin the performance of an effective evacuation during an emergency fire situation, as explained below.

3.3.1 Effective fire evacuation behaviours

Mental tracking, visual scanning, working memory, sustained attention and higher-order attentional processes such as selective attention and divided attention are deemed to be the most important cognitions recruited during an emergency evacuation. Mental tracking is required in an emergency situation to execute an escape strategy in a synthesised and sequential manner, as are the lower-order functions of visual scanning and motor speed. Working memory is the active, on-line system that holds information in current consciousness and manipulates it, or updates and re-evaluates it as new information comes to hand. It is therefore imperative for effective evacuation in a rapidly changing environment that can occur during a fire emergency. Persons in fire emergency situations may recruit working memory functions for several reasons, for example, to hold an image in their mind of the spatial layout of their residence in order to perform a mental checklist of the rooms visited and successfully evacuated.

Sustained attention is the ability to continuously focus on an aspect of experience, and is aligned with a readiness to respond to small and random changes in one's environment. Selective attention is the ability to focus on a particular stimuli or set of stimuli whilst effectively ignoring irrelevant stimuli. Both sustained attention and selective attention are vital functions in fire emergency situations in which one must maintain focus on an escape strategy or route whilst detecting small changes in the environment (e.g., more or less smoke coming from one room) and assess the effect of these changes on their evacuation plan (working memory). Persons must be able to select out the important information from their environment in order to make a safe and timely escape; the ability to ignore irrelevant stimuli will assist in a timely evacuation and maximise one's ability to prioritise and collect the most essential items before evacuation. Divided attention involves concentrating on more than one activity or stimulus at the same time, and is crucial to the successful evacuation from fire emergency situations in which persons are required to gather persons or belongings together whilst simultaneously monitoring the progress of the fire and/or other objects that may impede their escape goal.

3.4 Procedure

Data for the current study were obtained as part of a larger study investigating the waking effectiveness of alarms (auditory, visual and tactile) for the alcohol impaired (Bruck et al., 2007). Procedures for the current study were built in to the protocol of the larger study, which imposed some limitations on the methodological design. The three most important of these were (1) unable to counterbalance alcohol and sober testing nights in the current study as two consecutive alcohol testing nights were required to follow the protocol for the larger study, which involved alcohol nights only, (2) participants were potentially awoken by various stimuli three times per night in keeping with protocol for the larger study, and sleep inertia testing was undertaken after the third and final awakening, and (3) the intensities of the waking stimuli were gradually increased, with the result that not all awakenings may have been immediately from stage 4 sleep. Implications of these will be addressed in section *5.7*. The research was approved by the Victoria University Human Experimentation Ethics Committee.

3.4.1 Recruitment

Participants for the current study were recruited from the student body at Victoria University and their personal contacts. In addition to promotional talks at lectures and word of mouth advertising, flyers were distributed at the Footscray Park and St Albans campuses of Victoria University advertising for volunteers for the study (see Appendix G). Participants were recruited for three study nights, the first two involving alcohol consumption. The Participant Information Sheet outlined the requirements of the study and can be found in Appendix H. Informed consent was obtained from all participants prior to commencing screening.

3.4.2 Screening and practice trial

During screening, participants underwent a free hearing test to determine that their hearing (bilaterally) was average or better for their age, to ensure they met eligibility criteria. Tones at 500, 1000, 2000 and 4000 Hz were tested for each participant using an audiometer (Endomed SA 201/2 #13355) with specialised headphones which allowed field testing in quiet environments and eliminated the need for a sound chamber. Potential participants whose average hearing threshold was deemed to be above 20 dBA, were referred to H.E.A.R Hearing Service Victoria and were not invited to participate further. Those who passed the hearing test were assigned to a sleep technician (participants and sleep technicians were gender-matched where possible) who contacted them to organise mutually convenient dates for the testing phase. During this initial contact, sleep technicians also communicated with participants the importance of avoiding alcohol on the day of testing and ensuring sufficient sleep the night prior to testing to avoid sleep deprivation (which enhances sleep inertia effects, see section *2.2.2*). This was checked on each night of testing using the Prior Sleep and Alcohol Consumption Screening Questionnaire which was designed for the purposes of this study (see Appendix I). The Prior Sleep and Alcohol Consumption Screening Questionnaire was qualitatively analysed (initially by the sleep technician) to check participants' level of compliance with the pre-testing requirements of the study.

During the 30-minute initial screening session, conducted at a place of convenience, participants completed the DST, SDST, and LCT tasks once. This procedure guaranteed that all participants were experienced with task requirements prior to commencing the study, ensuring uniformity in regards to task familiarity. Nine trials of the DST (Dinges et al., 1985) and two baseline trials of the SDST (Hinton-Bayre et al., 1999) are recommended to eliminate and/or temper practice effects.

3.4.3 Experimental protocol

3.4.3.1 Testing environment and apparatus

During the testing phase, participants were typically assessed in their own homes to promote ecological validity. This involved participants sleeping alone in their bedroom with the door closed and with the sleep technician and recording equipment set up in the hallway or an adjacent room. Five participants, at their own request, were tested at the Victoria University Sleep Laboratory, located at the St Albans campus. Participants slept in one of two separate bedrooms with the door closed, and sleep technicians operated from the adjacent experimental room. Once a testing venue was chosen, it remained the same for all three testing nights.

Sleep technicians met participants at the agreed testing venue 2 hours prior to their usual bedtime, to account for natural variations in circadian rhythm timing (see section *2.2.3*). This meant that all preparatory equipment could be set up and all baseline tasks could be complete in time for the participant to retire to bed and fall asleep at the time that is typical or routine for them. Upon arrival, all electronic equipment was set up including polysomnographic recording equipment, and auditory, tactile and visual stimuli (note that visual stimuli were never presented as the third and final awakening stimulus so data pertaining to this were not used in the current study). Polysomnographic recordings were conducted using the Compumedics Siesta wireless data acquisition system or Compumedics Series E data acquisition system. The sleep equipment (EEG electrodes etc., see below) transmitted EEG data, either via radio waves or a cable, to a laptop monitored by a sleep technician. Auditory alarm sounds (four types in total) were operated from the laptop and emitted via speakers placed 1 metre from the centre of the

participant's pillow, directly facing the pillow. One Kevlar Car speaker, 40 Watts RMS (Response Precision Brand) and one Hylex PA Amplifier PA-50W were used and were attached to the laptop via a 10 metre extension cord. Auditory alarm signals were presented initially at 55 dBA (sound intensity of a normal to loud conversation) increasing in 10 dBA increments until 95 dBA (equivalent to industrial noise) or the participant woke up, whichever occurred first. Three of the alarm signals were created on the computer and one was a recording of the current Australian Standard smoke alarm signal. Analysis of the effects of different alarm signals on waking from sleep is beyond the scope of this thesis (please consult Bruck et al. (2007) for further information). Sound levels were calibrated (at the pillow) in each bedroom at either the participant's house or the sleep laboratory each night prior to testing using a Lutron SL-4001 Sound Level Meter.

Two types of tactile stimuli were used; a bed shaker and a pillow shaker. The pillow shaker was adapted from the Bellman and Smyfon AB of Sweden "Visit" bed shaker (recommended for placement under the pillow). To prevent the device from shaking loose under the pillow, it was placed inside a small linen bag and attached to the underside of the centre of the sleeper's top pillow with a safety pin. This was consistent with the recommended placement discussed within the local deaf community. The bed shaker was adapted from the Vibralarm VSS12 device and was placed under the mattress at a point deemed to be directly under the sleeper's navel.

Using a modified method of discrete limits, each signal was presented for a 'discrete' 30 second period followed by a 30 second period of signal offset. If the participant did not wake, the signal was presented again for 30 seconds at an increased intensity. This onset-offset pattern was repeated until the highest intensity level was reached (all awakening stimuli had five levels of intensity, for the tactile stimuli this was achieved by controlling input voltage to predetermined documented levels). If the participant still did not wake, the highest intensity level of the stimulus was presented continuously for 3.5 minutes following the normal 30 second offset. The alternating onset-offset pattern of presentation meant that each time a signal was presented, regardless of intensity, it

commenced from nil intensity, simulating the sudden onset of an emergency signal. It is stated in the research literature that if a person is going to respond to a smoke alarm, they will typically do so within the first 30 seconds of its onset (Bruck & Horasan, 1995), hence, the 30 second time period was deemed sufficient to allow most participants to respond to the signals at any given intensity.

Auditory (alarm signals operated via laptop and speaker system), tactile (bed or pillow shaker) and visual (strobe light, not used in the current study) stimuli equipment were set up every testing night so that participants were unaware of the arousal method to be used. Participants were informed that *any* arousal stimulus could be used to wake them, i.e., they may wake to something they see, hear or feel, thereby reducing expectation effects. Participants were potentially awoken three times each night from stage 4 sleep (see below), using different stimuli each time. The final (third) awakening signal (relevant to the current study) was always either auditory or tactile, however. Final awakening via auditory or tactile stimulus was counterbalanced across participants between the two alcohol nights. For the final awakening on the 'sober' night, all participants were awoken with an auditory stimulus.

A response button was placed beside the participant's bed which they were instructed to press three times immediately upon wakening. When the button was pressed, it illuminated a small blue light located near the sleep technician and the polysomnographic recording equipment. The behavioural response button and the small blue light were connected via a 10 metre extension cord. Behavioural response times and signal intensity required for waking were recorded via a specialised computer program run from the laptop.

The study consisted of two nights of testing involving alcohol administration (night 1 and night 2) and one night of testing without (night 3), known as the 'sober' night (always the final night).

3.4.3.2 'Alcohol' nights (night 1 & night 2)

Testing for nights 1 and 2 was usually conducted 2 weeks apart (M = 17.14 days), and always with a minimum of three intervening nights to allow for adequate sleep recovery. Prior to setting up the equipment, participants were first instructed to complete the Prior Sleep and Alcohol Consumption Screening Questionnaire (see Appendix I). This was primarily used as a screening measure to check participants' level of compliance with the pre-testing requirements of the study. If participants reported that their sleep was "much worse than usual", or reported that they had consumed alcohol since 4pm that day, the testing was aborted and rescheduled for another night. This was designed to ensure that participants were not sleep deprived at the time of testing and that there were no 'hangover' or confounding effects of prior alcohol use on either alcohol or sober testing nights. Although this procedure relied on participant's honesty, their motivation to be co-operative subjects was found to be high and they were always given the option of rescheduling testing for a different night, via a prior phone call to their sleep technician.

Once the equipment was set up and the sound levels were calibrated, electroencephalogram (EEG) electrodes for polysomnographic recording were fitted. Ten electrodes were attached according to the standard configuration described by Rechtschaffen & Kales (1968). Electroencephalogram (EEG) electrodes were attached at C3, C4, A1 and A2. Electro-oculogram (EOG) electrodes were placed at approximately 1cm above the outer canthus of the eye on one side, and at approximately 1cm below the outer canthus of the other eye, and electromyogram (EMG) electrodes were placed beneath the chin. Additionally, a reference electrode was affixed to the middle of the forehead, and a ground electrode was placed at the collarbone. Please see Appendix J for a diagrammatic representation of electrode placement. Prior to electrode placement, the skin was cleaned firstly with an alcohol swab, and then with Nuprep abrasive cream. Gold cup electrodes with Grass Electrode Cream were used for the scalp electrodes (C3 and C4), and mini-dot snap-on electrodes were used for all others.

Baseline cognitive testing commenced 2 hours prior to participants' usual bedtime, again to account for individual variations in circadian rhythm timing. Sleep technicians

administered one block of cognitive testing, known as the 'baseline sober condition' (time 1). Each block of cognitive testing was 10 minutes in duration and involved completing one trial of the DST (3 minutes), the SDST (2 minutes), the LCT (4 minutes) and the KSS and CH, always in that order. Cognitive tasks were not counterbalanced so that performance on the individual tasks could be directly comparable across conditions and the time course of sleep inertia effects could be assessed by comparing blocks of cognitive testing.

Alcohol administration commenced following the baseline sober condition of cognitive testing. Alcohol was administered in measured standard doses as specified by the Australian Transport Safety Bureau. The operational definition of one standard dose used for the current study was one 30ml dosage of vodka (Vodka Smirnoff, 37.5% alcohol volume), mixed with equal parts of orange juice (60ml total). Multiple doses were often administered as one drink before a participant was breathalysed (for example, a single 180ml drink consisting of three doses). Participants consumed as many beverages prepared in this manner, at their own pace, as was required for them to reach a blood alcohol concentration (BAC) of 0.05%, determined by breath-test analysis. An initial estimate of the number of beverages required to reach the desired BAC level on any given night was determined by the sleep technician in consultation with the participant. A number of factors known to affect the absorption of alcohol were considered, including the participant's previous experience with alcohol, their sex, their weight, time since their last meal, etc. Sleep technicians were instructed to make conservative estimates to minimise the possibility of overshooting the desired level. The first breath test analysis occurred 10 minutes after the first alcoholic drink was consumed (see Appendix K for BAC testing procedural details. This appendix is taken from Bruck et al., 2007). Several Lion Alcometre S-D2 breathalysers were loaned to the study by the Victoria Police to measure BAC levels. For further details of these units please see Appendix K.

Once the required alcohol level was reached (0.05 BAC + 0.01) participants were settled in bed and informed of the procedure to follow upon becoming aware of a signal. They were instructed to press the behavioural response button located at their bedside three

times to indicate their arousal immediately after detecting a signal. At this point they were reminded that the signal may be something they could see, hear, or feel. Participants then completed a second 10-minute block of cognitive testing, known as the 'baseline 0.05 BAC condition' (time 2) sitting up in bed, using a stable table to complete pencil-and-paper tasks. Following this, a BAC reading was taken and this measurement was used as a record of BAC prior to sleep. Lights were then extinguished and participants were left to fall asleep.

Sleep technicians monitored the participant's EEG output until stage 4 sleep was confirmed for a minimum of three consecutive 30-second epochs as determined by the criteria specified by Rechtschaffen & Kales (1968). Once stage 4 sleep was confirmed, the sleep technician activated the signal delivery system to present the required arousal signal (auditory, tactile or visual) at the lowest experimental level. All signals were presented during stage 4 sleep (see section *2.2.1*) but continued to be presented using the modified method of discrete limits even if a sleep stage changed. When a participant responded by pressing the behavioural response button, the sleep technician alerted the signal delivery program to record the exact time, and the stimulus was terminated. Participants were then allowed to return to sleep. Participants were potentially awoken twice more according to this procedure (due to investigations related to the research protocol with which this study was combined), again using either auditory, tactile or visual stimuli. Upon the third awakening (only auditory or tactile stimuli were used for the third and final awakening of each night), the sleep technician entered the bedroom and immediately instructed the participant to commence the DST whilst still lying in bed with the lights out, with some illumination available from the adjacent room or hallway (the DST instructions were modified for this condition so that the task could begin as soon after sleep offset as possible, see Appendix B). This was the commencement of the third 10-minute block of cognitive testing, known as the 'sleep inertia 1 condition' (time 3). Following the 3 minutes duration of the DST, the lights were illuminated and participants were breathalysed to determine the exact BAC level during performance on the tasks during the sleep inertia conditions (see Appendix L for raw BAC data). They then completed the remainder of the block of cognitive testing, followed immediately by

a second 10-minute block of cognitive testing, known as the 'sleep inertia 2 condition' (time 4). Participants were then breath-tested again to achieve a final BAC reading (see Appendix L), instructed to return to sleep, and were left to sleep uninterrupted. See Table 2.1 for a summary of the cognitive testing conditions, which is reproduced here from Chapter 2 for reference.

3.4.3.3 'Sober' night (night 3)

The 'sober' testing night (night 3) was conducted an average of 117.33 days (*SD* = 50.92) following night 2, and was always the final night (as dictated by the protocol of the larger study with which this study was combined). Sober testing nights were conducted in the same manner as the 'alcohol' nights, however without alcohol administration and therefore without the baseline 0.05 BAC condition (time 2). This meant that there were only three 10-minute blocks of cognitive testing on the sober night compared to four 10-minute blocks of cognitive testing on nights involving alcohol administration (see Table 2.1). On sober testing nights, a "dummy run" breath-test was undertaken following administration of the DST in the 'sleep inertia 1 condition' for purposes of experimental control and consistency between the sober and alcohol nights. On all testing nights, participants were assessed under conditions of sleep inertia an average of 99.73 minutes (*SD* = 62.11 minutes) after their usual bedtime.

Table 2.1.

Summary of cognitive testing conditions.

	Night 1 & 2: ALCOHOL	**Night 3: SOBER**
Time 1 (2 hours prior to participant's bedtime)	Baseline sober	Baseline sober
Time 2 (immediately prior to lights out)	Baseline 0.05 BAC ***Alcohol Only***	
Time 3 (immediately after final awakening)	Sleep inertia 1 ***Combined Alcohol & Sleep Inertia (0-10 minutes)***	Sleep inertia 1 ***Sleep Inertia Only (0-10 minutes)***
Time 4 (10 minutes post awakening, immediately following Time 3)	Sleep inertia 2 ***Combined Alcohol & Sleep Inertia (10-20 minutes)***	Sleep inertia 2 ***Sleep Inertia Only (10-20 minutes)***

3.5 Data analysis

All hypotheses were tested using a repeated-measures multivariate analysis of variance (MANOVA) with condition as the independent variable and performance on the three cognitive tasks and two subjective rating scales as dependent variables. Alpha was set at .05.

Repeated-measures statistics are a particularly powerful statistical method as they eliminate variance due to individual differences, inherent in independent-samples designs. They are robust to minor violations to their underlying assumptions, and smaller sample sizes are required to uncover real differences where they exist (Gravetter, 2006). The disadvantages of repeated-measures designs include the potential for confounding practice effects and the fact that any missing data necessitates the exclusion of the entire case for relevant analyses.

In addition, a number of preliminary analyses were conducted. Due to the fact that the procedures for the current study were built into a larger study investigating the waking effectiveness of alarms (auditory, visual and tactile) for the alcohol impaired, preliminary analyses were conducted to determine the effect, if any, of different arousal stimuli on performance during sleep inertia. Auditory and tactile arousal stimuli were used as the final awakenings immediately prior to sleep inertia testing on nights 1 and 2 (alcohol nights), and therefore these data were investigated. Following this, evidence of adaptation effects and practice effects were examined.

CHAPTER FOUR: RESULTS

4.1 Data screening

The data were screened in accordance with criteria recommended by Tabachnick and Fidell (2001).

Sample size.

With 24 cases and less than 1% of data missing from the total data points on the dependent measures, there are more cases than dependent variables in every cell, ensuring sufficient power.

Normality of sampling distribution.

Based on visual inspection of histograms, evaluation of skewness and kurtosis values, and Kolmogorov-Smirnov statistic values of $p > .05$, three measures displayed a non-normal distribution, namely the Descending Subtraction Task (DST), the Karolinska Sleepiness Scale (KSS) and the Clearheadedness Rating Scale (CH). Specifically, the number of responses on the DST demonstrated moderate positive skewness whilst percentage accuracy on the DST demonstrated moderate negative skewness. Responses on the subjective rating scales were either positively or negatively skewed in the direction expected for each condition (e.g., responses on the CH were negatively skewed during the baseline condition). The application of square root and logarithm transformations sufficiently corrected the normality of these data, but did not change the outcome of hypothesis testing. Therefore, for ease of interpretation, the untransformed data were used and reported in subsequent analyses.

For some analyses the sample size was sufficient to produce 20 degrees of freedom for error in the univariate case ensuring the robustness of the test (in combination with equal sample sizes across measures and use of two-tailed tests) in regards to multivariate normality. In analyses where this was not the case, the univariate *F* was deemed to be robust to potential modest violations of multivariate normality as the sample size was above 20 and the violations were not due to the presence of outliers (see below).

Outliers.

Several univariate outliers were detected through visual inspection of box plots. In accordance with Tabachnick and Fidell's (2001) criteria, these data points were given a raw score one unit above or below the next most extreme case, depending on the direction of the outlying value. This procedure was successful in abating the influence of outlying cases.

Mahalanobis distance (χ^2 = 22.46; Tabachnick & Fidell, 2001) was used to test for the presence of multivariate outliers. With the application of a criterion of $p < .001$, no multivariate outliers were detected in the present sample.

Homogeneity of the variance-covariance matrices.

Theoretically, due to the repeated measures design, sample sizes are equal across all conditions ensuring the robustness of significance tests and satisfying the assumption of the homogeneity of the variance/co-variance matrix. Statistically, homogeneity of variance is assessed using Mauchly's test of Sphericity, provided routinely in SPSS MANOVA output. When Mauchly's test of Sphericity was significant ($p < .05$), violating the underlying assumption of homogeneity of variance, the Huynh-Feldt values and not the Sphericity Assumed values were analysed and reported.

Linearity.

The data did not violate the assumptions of linearity according to inspection of bivariate scatterplots; no curvilinearity was detected.

Multicollinearity and singularity.

An absence of multicollinearity was demonstrated through correlation of the dependent variables, using point biserial correlations and Pearson's product-moment correlations (all less than .70; Tabachnick & Fidell, 2001).

4.2 Preliminary analyses

4.2.1 Auditory versus tactile awakening signal

To investigate the effect of different arousal signals on cognitive functioning and subjective sleepiness and clearheadedness during sleep inertia, performance measures and self-report ratings were compared following awakening via auditory alarm signal (auditory condition) versus awakening via bed or pillow shaker (tactile condition). Nineteen of the total 24 participants were awoken by both an auditory and tactile stimulus on separate nights (across nights 1 and 2 with the order counterbalanced across participants) immediately prior to sleep inertia testing (time 3). The data for these participants were included in the analyses, and the descriptive and inferential statistics are presented in Table 3. The remaining five participants were awoken with only one type of stimulus (typically auditory) across the testing nights.

Table 3.

Means, standard deviations and within-subjects MANOVA univariate results for cognitive performance and subjective ratings following awakening with an auditory vs. tactile stimulus (n = 19).

	Auditory		Tactile			
	M	*SD*	*M*	*SD*	*F*(1,18)	*p*
Cognitive functioning						
DST accuracy	71.71	14.63	70.81	18.46	0.06	0.82
DST speed	28.11	14.45	27.00	10.93	0.52	0.48
SDST # correct	70.95	15.22	72.53	15.24	0.28	0.60
LCT # correct	85.58	19.54	86.47	24.73	0.05	0.83
Subjective ratings						
KSS	8.37	0.68	7.95	1.01	2.94	0.10
CH	2.11	1.15	2.37	0.96	2.03	0.17

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.

A single factor repeated-measures MANOVA was performed on six dependent variables: working memory accuracy (DST accuracy), working memory speed (DST number of responses produced), divided attention/mental tracking/psychomotor speed (Symbol Digit

Substitution Task (SDST) number of correct responses), selective/sustained attention/visual scanning (Letter Cancellation Test (LCT) number of correct responses), subjective sleepiness (KSS score), and subjective clearheadedness (CH score) measured during the first 10 minutes of sleep inertia (time 3). The independent variable was type of arousal stimulus (auditory and tactile). There was no significant effect for sensory mode, *Wilks' Lambda* = 0.68, $F(6,13) = 1.04$, $p = .443$. Univariate results are shown in Table 3, and actual *p* values are reported to show that the results are far from approaching statistical significance.

As expected, these results indicate that arousal from sleep by different sensory stimuli (i.e., auditory or tactile) did not affect the nature of cognitive performance or feelings of sleepiness and clearheadedness during sleep inertia. Given the lack of difference between the two arousal methods, it was theoretically acceptable that the sleep inertia performance and subjective ratings data from both auditory and tactile stimuli awakenings could be pooled together within their respective nights (i.e., night 1 or night 2). Further, data from each of nights 1 and 2 are now both directly comparable, as 'alcohol' nights, with data from night 3, the 'sober' night. First, however, evidence of adaptation effects on performance tasks and subjective ratings must be investigated, as repeated-measures designs are particularly vulnerable to confounding practice effects.

4.2.2 Investigation of practice effects and adaptation effects

A series of single factor repeated-measures MANOVAs was conducted to examine differences on the six dependent measures between each condition, or time point, of nights 1 and 2. Four separate repeated-measures MANOVAs were conducted, one for each time point with night 1 versus night 2 as the independent variable; baseline sober (time 1), baseline 0.05 blood alcohol concentration (BAC; time 2), sleep inertia 1 (0-10 minutes post-awakening; time 3) and sleep inertia 2 (10-20 minutes post-awakening; time 4). The risk for alpha inflation due to multiple analyses is acknowledged, however these analyses were planned from the outset as part of the design of the study. Table 4 presents the inferential statistics of the univariate analyses. Partial eta squared was calculated to

determine the strength of the effects, and is also presented in Table 4. Partial eta squared values range from 0 to 1, with larger values representing larger effects (Cohen, 1988).

Table 4.

Comparison of cognitive performance and subjective ratings across each time point with night 1 versus night 2 as the independent variable; univariate p *values and effect sizes (n = 24).*

	Baseline sober	*Baseline .05 BAC*	*Sleep inertia 1*	*Sleep inertia 2*	*Partial η^2 (baseline sober)*
Cognitive functioning					
DST accuracy	.022*	ns	ns	ns	0.22
DST speed	.047*	.019*◊	ns	ns	0.17
SDST # correct	ns	ns	ns	ns	-
LCT # correct	ns	ns	ns	ns	-
Subjective ratings					
KSS	ns	ns	ns	ns	-
CH	.029*	ns	ns	ns	0.20

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale. ns = not significant.
* = $p < .05$.
◊ = overall MANOVA was not significant for this time point.

There was an overall significant difference between the baseline sober condition (time 1) of night 1 and the baseline sober condition (time 1) of night 2, *Wilks' Lambda* = 0.35, $F(6,17) = 5.35$, $p = .003$, *partial* $\eta^2 = 0.65$. Univariate tests revealed this difference was predominantly due to significantly better scores on the DST on night 2 compared to night 1. Specifically, participants produced more responses on the DST (working memory speed; $F(1,22) = 4.41$, $p = .047$) and were more accurate in their responses (working memory accuracy; $F(1,22) = 6.03$, $p = .022$) on night 2. The performance task results are consistent with the known practice effects of the DST, in which there is a "substantial practice effect over the first nine trials" (Dinges et al., 1985). Participants also self-reported feeling more clearheaded during the baseline sober condition of night 2 compared to night 1, $F(1,22) = 5.46$, $p = .029$. A single factor repeated-measures

MANOVA revealed no overall significant difference, however, between the baseline 0.05 BAC condition (time 2) of night 1 and the baseline 0.05 BAC condition (time 2) of night 2, Wilks' Lambda = 0.62, $F(6,18) = 1.85$, $p = .145$, when three trials (including screening) would have been completed. These results indicate that practice effects on the DST began to dissipate by the third trial. Importantly, there were no significant differences on the six dependent measures between the sleep inertia conditions of night 1 and the sleep inertia conditions of night 2 for either time 3 or time 4, *Wilks' Lambda* = 0.59, $F(6,18) = 2.07$, $p = .108$ (sleep inertia 1; time 3) and *Wilks' Lambda* = 0.68, $F(6,18) = 1.39$, $p = .270$ (sleep inertia 2; time 4), respectively.

To confirm that practice effects were indeed completely diminished after the first baseline trials, a further comparison was made between performance on the dependent measures during the baseline sober condition (time 1) of night 2 and the baseline sober condition (time 1) of night 3, the final testing night. A single factor repeated-measures MANOVA revealed no significant difference between the conditions, *Wilks' Lambda* = 0.65, $F(6,17) = 1.52$, $p = .231$, indicating that participants maintained their improvement on the DST gained over the first 2-3 trials, and that practice effects were no longer operating.

A statistically significant difference between the baseline sober condition (time 1) of night 1 and the baseline sober condition (time 1) of night 3 also confirms the presence of practice effects for the DST, *Wilks' Lambda* = 0.50, $F(6,18) = 3.05$, $p = .031$. Participants produced significantly more responses on the DST during the baseline sober condition (time 1) of night 3 compared to night 1, $F(1,23) = 4.83$, $p = .038$. They also reported significantly higher levels of clearheadedness during the baseline sober condition (time 1) of night 3 compared to night 1, $F(1,23) = 5.66$, $p = .026$.

In sum, participants' performance on the DST was significantly worse at baseline (time 1) on night 1, compared to both night 2 and night 3 baseline (time 1) conditions. Practice effects had completely dissipated by the baseline 0.05 BAC condition of night 1 (time 2) following 2-3 trials, as there were no longer statistically detectable differences in

performance during this condition of night 1 and night 2. The plateau in performance persisted at night 3, an average of 117.33 (*SD* = 50.92) days following night 2, as there was no significant difference in performance between night 2 baseline (time 1) and night 3 baseline (time 1) conditions. This indicates that practice effects were no longer operating. It also indicates that on night 3 participants retained some familiarisation with the task from night 2, even though night 2 was, on average, 117 days earlier. See Figure 1 for a graphical representation of practice effects on the number of responses produced on the DST.

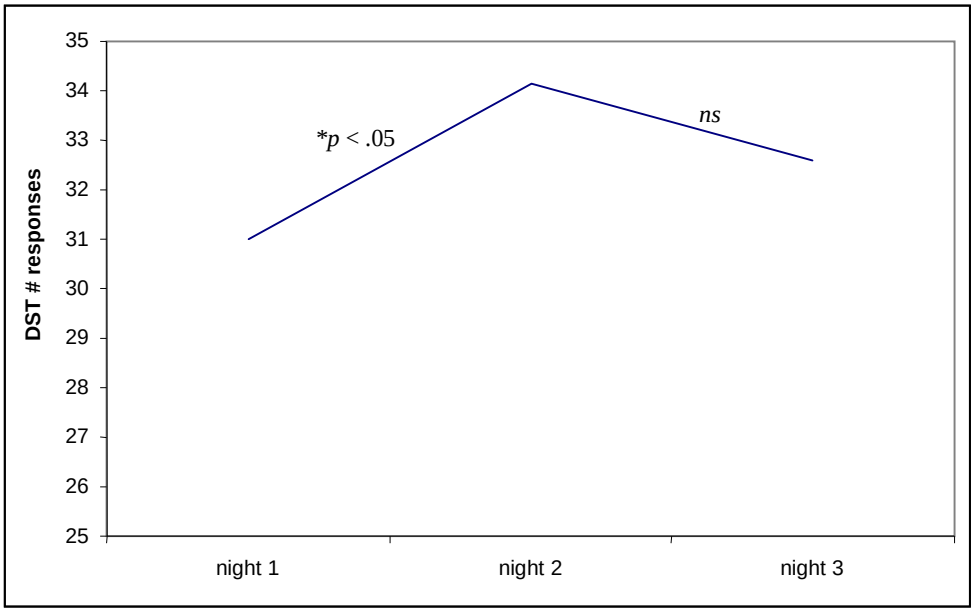


Figure 1. Mean Descending Subtraction Task number of responses for the baseline sober condition of each testing night.

Due to the presence of practice effects on the DST during the first night of testing, it is henceforth considered to be an 'adaptation night' where participants were able to familiarise themselves with the cognitive tasks and rating scales in order to counteract practice effects. This also allowed participants to become acquainted with the experimental protocol and EEG equipment to avoid the 'first night effect' which is known to distort sleep architecture and increase the amount of alpha-wave activity detected on EEG, having a wake-promoting effect and increasing sleep-onset latency (Tamaki, Nittono, Hayashi, & Hori, 2005). The 'first night effect' is a commonly

encountered threat to the validity of sleep research that involves participants spending a night in an unfamiliar setting; typically the sleep laboratory. Avoiding the 'first night effect' is particularly pertinent to the current study as possible increased sleep-onset latencies during night 1 would ultimately affect the BAC level at sleep inertia testing.

Hereafter night 2 performance data will be used as the 'alcohol' night, and night 3 performance data will be used as the 'sober' night. Night 1 performance data will not be used in the analyses.

4.3 Sleep inertia effect

A single factor repeated-measures MANOVA was conducted to determine the effect of sleep inertia (in isolation) on participants' performance on the cognitive tasks and subjective rating scales. Using time (three occasions, times 1, 3 & 4) as the independent variable and performance on the three tasks and two scales on the 'sober' night (night 3) as the dependent measures, a significant overall sleep inertia effect was found, *Wilks' Lambda* = 0.09, $F(12,12) = 10.73$, $p < .001$, *partial* $\eta^2 = 0.92$.

Univariate analyses revealed that performance on each of the cognitive measures, with the exception of percentage accuracy on the DST, was significantly better during the baseline sober condition (time 1) than during the first 10 minutes of sleep inertia (time 3). Similarly, participants reported significantly lower levels of sleepiness and significantly higher levels of clearheadedness during the baseline sober condition than during the first 10 minutes of sleep inertia. See Table 5 for descriptive and inferential statistics.

Table 5.

Means, standard deviations and within-subjects univariate MANOVA results for cognitive performance and subjective ratings during the baseline sober (time 1) and sleep inertia 1(time 3) conditions of night 3, the 'sober' night.

	Baseline sober		Sleep inertia 1				
	M	*SD*	*M*	*SD*	*F*(2,46)	*p*	*Partial* η^2
Cognitive functioning							
DST accuracy	81.20	11.57	77.21	17.08	3.19	.063†	0.12
DST speed	32.58	10.68	25.88	10.30	25.59	.000***	0.53
SDST # correct	88.50	15.39	69.21	12.50	37.81	.000***†	0.62
LCT # correct	104.08	24.45	84.25	27.58	18.34	.000***	0.44
Subjective ratings							
KSS	6.88	1.19	7.83	1.05	5.00	.011*	0.18
CH	3.15	0.85	2.50	0.89	8.73	.001**	0.28

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.
* = $p < .05$. ** = $p < .01$. *** = $p < .001$.
† Huyhn-Feldt statistic used as Mauchly's test of Sphericity was $p < .05$.

Participants performed significantly poorer on the cognitive measures, felt sleepier, and felt less clearheaded during the sleep inertia condition compared to the baseline condition, representing an overall sleep inertia effect for cognitive performance and subjective ratings. In addition, when considering performance on the DST alone, it appears that a speed accuracy trade-off effect is operating whereby the accuracy of participants' responses is maintained, but the speed of their performance is adversely affected by the presence of sleep inertia.

Simple contrasts revealed a recovery effect for two of the dependent measures when performance during 0-10 minutes of sleep inertia (time 3) was compared with performance during 10-20 minutes of sleep inertia (time 4). A recovery effect is defined as a statistically significant improvement during the 20 minute time course of sleep inertia, i.e., a statistically significant improvement in performance on the cognitive tasks

or subjective ratings from the first (time 3) to the second sleep inertia condition (time 4). Overall performance on the SDST ($F(1,23) = 41.92$, $p < .001$, *partial* $\eta^2 = 0.65$) and speed of performance on the DST ($F(1,23) = 26.03$, $p < .001$, *partial* $\eta^2 = 0.53$) demonstrated a recovery effect from 0-10 minutes of sleep inertia (time 3) to 10-20 minutes of sleep inertia (time 4). No recovery effect was observed for the LCT or the subjective rating scales ($p > .05$). The descriptive and inferential statistics are presented in Table 6.

Table 6.

Means, standard deviations and within-subjects univariate MANOVA results for cognitive performance and subjective ratings during the sleep inertia 1 (time 3) and sleep inertia 2 (time 4) conditions of night 3, the 'sober' night.

	Recovery effect (time 3 v. time 4)						
	Sleep inertia 1		*Sleep inertia 2*				
	M	*SD*	*M*	*SD*	*F*(2,46)	*p*	Partial η^2
Cognitive functioning							
DST accuracy	77.21	17.08	84.54	14.47	9.36	.006**†◊	0.29
DST speed	25.88	10.30	30.63	11.73	26.03	.000***	0.53
SDST # correct	69.21	12.50	80.83	14.40	41.92	.000***†	0.65
LCT # correct	84.25	27.58	91.29	20.88	3.89	.061	0.15
Subjective ratings							
KSS	7.83	1.05	7.48	0.83	2.37	.137	0.09
CH	2.50	0.89	2.75	0.90	4.06	.056	0.15

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.
* = $p < .05$. ** = $p < .01$. *** = $p < .001$.
† Huyhn-Feldt statistic used as Mauchly's test of Sphericity was $p < .05$.
◊ = overall MANOVA was not significant for this variable.

Recovery to baseline levels occurred when performance at 10-20 minutes of sleep inertia (time 4) had improved to the extent that it was now (statistically) indistinguishable from baseline performance (time 1). Only performance speed on the DST ($F(1,23) = 4.10$, $p = .055$) and level of subjective sleepiness on the KSS ($F(1,23) = 3.77$, $p = .065$) recovered to baseline levels (i.e., $p > .05$ when comparing performance at sleep inertia 2 (time 4) to

baseline performance (time 1). The descriptive and inferential statistics are presented in Table 7.

Table 7.

Means, standard deviations and within-subjects univariate MANOVA results for cognitive performance and subjective ratings during the sleep inertia 2 (time 4) and baseline sober (time 1) conditions of night 3, the 'sober' night. Note that non-significant results indicate a recovery to baseline effect.

	Recovery to baseline (time 4 v. time 1)$^{\Delta}$						
	Sleep inertia 2		*Baseline sober*				
	M	*SD*	*M*	*SD*	*F*(2,46)	*p*	Partial η^2
Cognitive functioning							
DST accuracy	84.54	14.47	81.20	11.57	1.74	.201$^{\dagger\lozenge}$	0.07
DST speed	30.63	11.73	32.58	10.68	4.10	.055	0.15
SDST # correct	80.83	14.40	88.50	15.39	14.50	.001**†	0.38
LCT # correct	91.29	20.88	104.08	24.45	22.36	.000***	0.49
Subjective ratings							
KSS	7.48	0.83	6.88	1.19	3.77	.065	0.14
CH	2.75	0.90	3.15	0.85	5.25	.032*	0.19

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.
* = $p < .05$. ** = $p < .01$. *** = $p < .001$.
† Huyhn-Feldt statistic used as Mauchly's test of Sphericity was $p < .05$.
◊ = overall MANOVA was not significant for this variable.

4.4 Alcohol intake effect

A single factor repeated-measures MANOVA was conducted to determine the effects of alcohol (in isolation) on participant's performance on the cognitive tasks and subjective measures. Using time as the independent variable (two occasions; time 1 versus time 2) and performance on the three tasks and two scales on the 'alcohol' night (night 2) as the dependent measures, a significant overall effect for alcohol was found, *Wilks' Lambda* = 0.20, $F(6,17) = 11.60$, $p < .001$, *partial* $\eta^2 = 0.80$.

Univariate analyses revealed that performance on each of the cognitive measures, with the exception of number of responses on the DST, was significantly better during the baseline sober condition (time 1) than during the baseline 0.05 BAC condition (time 2). Similarly, participants reported significantly lower levels of sleepiness and significantly higher levels of clearheadedness during the baseline sober condition (time 1) than during the baseline 0.05 BAC condition (time 2). See Table 8 for descriptive and inferential statistics.

Table 8.

Means, standard deviations and within-subjects univariate MANOVA results for cognitive performance and subjective ratings during the baseline sober (time 1) and baseline 0.05 BAC (time 2) conditions of night 2, the 'alcohol' night.

	Baseline sober		Baseline 0.05 BAC				
	M	*SD*	*M*	*SD*	*F*(1,22)	*p*	Partial η^2
Cognitive functioning							
DST accuracy	86.09	9.43	79.43	13.48	19.00	.000***	0.46
DST # responses	32.91	11.83	32.22	12.15	0.68	.420	0.03
SDST # correct	87.48	12.24	80.61	15.98	7.34	.013*	0.25
LCT # correct	107.39	20.35	98.48	20.00	15.00	.001**	0.41
Subjective ratings							
KSS	5.96	1.52	7.87	0.69	40.41	.000***	0.65
CH	3.26	0.69	2.30	0.89	30.95	.000***	0.59

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.
* = $p < .05$. ** = $p < .01$. *** = $p < .001$.

Decrements in cognitive performance, increased subjective sleepiness and decreased subjective clearhdeadedness were observed following alcohol administration to 0.05 BAC. A speed-accuracy trade-off was again observed for the DST, however, in the reverse direction compared to conditions of sleep inertia; participants were able to maintain baseline levels of speed, but at the cost of reduced accuracy of performance, i.e., they produced the same number of responses overall, but their responses were less accurate. This is referred to as an 'accuracy-speed trade-off' effect in the current study.

4.5 Preliminary summary

The above analyses demonstrate that, separately, sleep inertia and alcohol affected participants' cognitive functioning and levels of subjective sleepiness and clearheadedness. A speed-accuracy trade-off effect was found for working memory performance as measured by the DST. Sleep inertia detrimentally affected performance speed but not accuracy, and alcohol caused impairments in performance accuracy whilst performance speed was unaffected. Elements of speed and accuracy were not assessable on the SDST and LCT tasks due to a lack of variation in scores (e.g., errors on the SDST and errors of commission on the LCT were 0 or 1), possibly due to the relative simplicity of these tasks when compared with the complexity of the DST.

4.6 Alcohol and sleep inertia effects

4.6.1 Within 10 minutes post-awakening

At 3 minutes post-awakening (breathalyser test taken immediately following administration of the DST), BAC levels varied between 0.02 and 0.05 (M = 0.037, SD = 0.01), with 50% of participants at or above 0.04 BAC (see Appendix L for raw data). Conditions of alcohol only (night 2, time 2) were compared with conditions of sleep inertia only at 10 minutes post-awakening (night 3, time 3) and conditions of combined alcohol impairment and sleep inertia (night 2, time 3), also at 10 minutes post-awakening. Using time (three conditions) as the independent variable and performance on the three performance tasks and two self-report scales as the dependent measures, a repeated-measures MANOVA revealed a significant effect for cognitive performance and subjective ratings (*Wilks' Lambda* = 0.20, $F(12,12) = 4.00$, $p = .012$, *partial* $\eta^2 = 0.80$) at 0-10 minutes post-awakening.

The MANOVA univariate analyses and Simple contrasts revealed that conditions of combined alcohol impairment and sleep inertia (night 2, time 3) produced task performance that was significantly worse than alcohol impairment alone (night 2, time 2), but not significantly worse than that produced by sleep inertia alone (night 3, time 3; see Figure 2). This was the case for all tasks with the exception of performance accuracy on the DST. Further, conditions of sleep inertia alone produced greater impairments in task

performance (with the exception of performance accuracy on the DST) than during conditions of alcohol impairment alone. Ratings of subjective sleepiness and clearheadedness were not significantly different between conditions. Descriptive and inferential statistics are presented in Table 9 below.

Table 9.

Means, standard deviations and within-subjects univariate MANOVA results for cognitive performance and subjective ratings during the alcohol only, sleep inertia only and combined sleep inert and alcohol conditions at 0-10 minutes post-awakening.

	Alcohol only		Sleep inertia only		Combined			
	M	*SD*	*M*	*SD*	*M*	*SD*	*F(2,46)*	*p*
Cognitive functioning								
DST accuracy	79.17	13.25	77.21	17.08	75.27	16.36	0.94	.397
DST speed	31.96	11.95	25.87	10.30	26.92	11.00	17.09	.000***
SDST # correct	80.29	15.70	69.21	12.50	71.17	15.00	9.37	.000***
LCT # correct	98.46	19.56	84.25	27.58	85.92	20.92	10.35	.000***
Subjective ratings								
KSS	7.83	0.70	7.83	1.05	8.25	0.74	2.75	.087†
CH	2.42	1.02	2.50	0.89	2.12	0.99	2.45	.097

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.
* = $p < .05$. ** = $p < .01$. *** = $p < .001$.
† Huyhn-Feldt statistic used as Mauchly's test of Sphericity was $p < .05$.

These results indicate that, at 0-10 minutes post-awakening, sleep inertia effects 'override' alcohol effects as the combination of sleep inertia and alcohol impairment produces greater performance decrements than alcohol impairment alone, but not sleep inertia alone. Similarly, performance decrements are greater during the sleep inertia alone condition than during the alcohol alone condition. This is true for all cognitive tasks, including performance speed on the DST. Performance accuracy on the DST, however, is not significantly different between these conditions, as represented in Figure 2. Figure 2 provides a graphical summary of the key performance results during the first 10 minutes of sleep inertia.

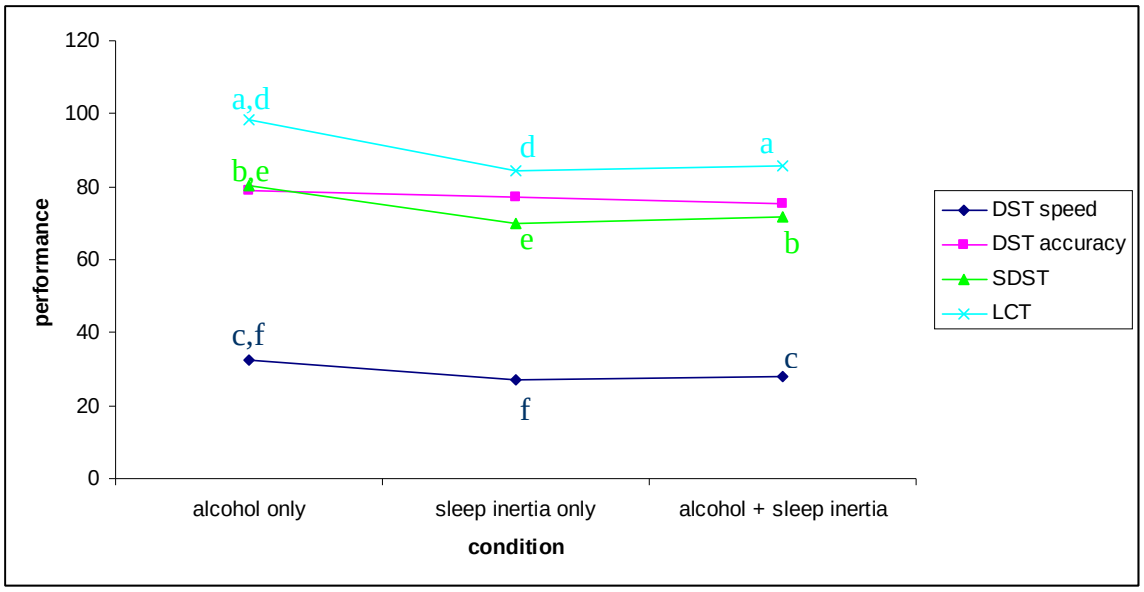


Figure 2. Mean performance scores for the DST, SDST and LCT at 0-10 minutes post-awakening.

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test.

Note. Differences between points indicated by the same letter are significant ($p < .05$).

4.6.2 Within 20 minutes post-awakening

At 20 minutes post-awakening, BAC levels varied between 0.01 and 0.05 ($M = 0.034$, $SD = 0.01$), with 50% of participants at or above 0.04 BAC (see Appendix L for raw data). Conditions of alcohol only (night 2, time 2) were compared with conditions of sleep inertia within 20 minutes post-awakening (night 3, time 4) and conditions of combined alcohol impairment and sleep inertia (night 2, time 4), also within 20 minutes post-awakening. Using time (three conditions) as the independent variable and performance on the three performance tasks and two self-report scales as the dependent measures, a repeated-measures MANOVA revealed a significant effect for cognitive performance and subjective ratings (*Wilks' Lambda* = 0.21, $F(12,12) = 3.82$, $p = .014$, *partial* $\eta^2 = 0.79$) at 10-20 minutes post-awakening.

The MANOVA univariate analyses and Simple contrasts revealed that conditions of combined alcohol impairment and sleep inertia (night 2, time 4) no longer produced task performance that was significantly worse than alcohol impairment alone (night 2, time 2), with the exception of performance on the SDST (see Figure 3). In addition, conditions of combined alcohol impairment and sleep inertia now produced performance decrements

greater than those produced by sleep inertia alone (night 3, time 4) for performance accuracy on the DST, performance on the SDST, and ratings of subjective sleepiness on the KSS (see Figure 4). Further, performance decrements were greater during the alcohol alone condition than during the sleep inertia alone condition for performance accuracy on the DST and performance on the LCT. For the LCT, performance decrements were also greater during the alcohol alone condition than during the combined condition. Speed of performance on the DST, and ratings of subjective clearheadedness on the CH were not significantly different between conditions. Descriptive and inferential statistics are presented in Table 10 below.

Table 10.

Means, standard deviations and within-subjects univariate MANOVA results for cognitive performance and subjective ratings during the alcohol only, sleep inertia only and combined sleep inert and alcohol conditions at 10-20 minutes post-awakening.

	Alcohol only		Sleep inertia only		Combined			
	M	*SD*	*M*	*SD*	*M*	*SD*	*F(2,46)*	*p*
Cognitive functioning								
DST accuracy	79.17	13.25	84.54	11.47	77.73	15.46	4.89	.012*
DST speed	31.96	11.95	30.63	11.73	30.62	12.54	1.05	.345†
SDST # correct	80.29	15.70	80.83	14.40	73.71	15.71	5.44	.008**
LCT # correct	98.46	19.56	91.29	20.88	89.42	25.55	3.26	.062†
Subjective ratings								
KSS	7.83	0.70	7.48	0.83	7.96	1.00	3.22	.049*
CH	2.42	1.02	2.75	0.90	2.42	1.02	1.74	.118

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale.
* = $p < .05$. ** = $p < .01$. *** = $p < .001$.
† Huyhn-Feldt statistic used as Mauchly's test of Sphericity was $p < .05$.

These results indicate that at 10-20 minutes post-awakening, sleep inertia effects have essentially dissipated. Alcohol effects are now 'unmasked' on two of the dependent measures that were not showing significant differences at 0-10 minutes post-awakening,

namely performance accuracy on the DST and ratings of subjective sleepiness on the KSS (i.e., scores under conditions of combined alcohol impairment and sleep inertia are now significantly *worse* (performance task) or *increased* (sleepiness subjective rating) than under conditions of sleep inertia only). Further, alcohol effects are also 'unmasked' on LCT performance, to the extent that scores under conditions of alcohol impairment are now significantly *worse* than under conditions of combined alcohol impairment and sleep inertia and under conditions of sleep inertia only. A combined interaction appears to be operating on performance on the SDST, as performance decrements during the combined alcohol impairment and sleep inertia condition are now greater than those found during the alcohol alone and sleep inertia alone conditions, which are not significantly different from each other.

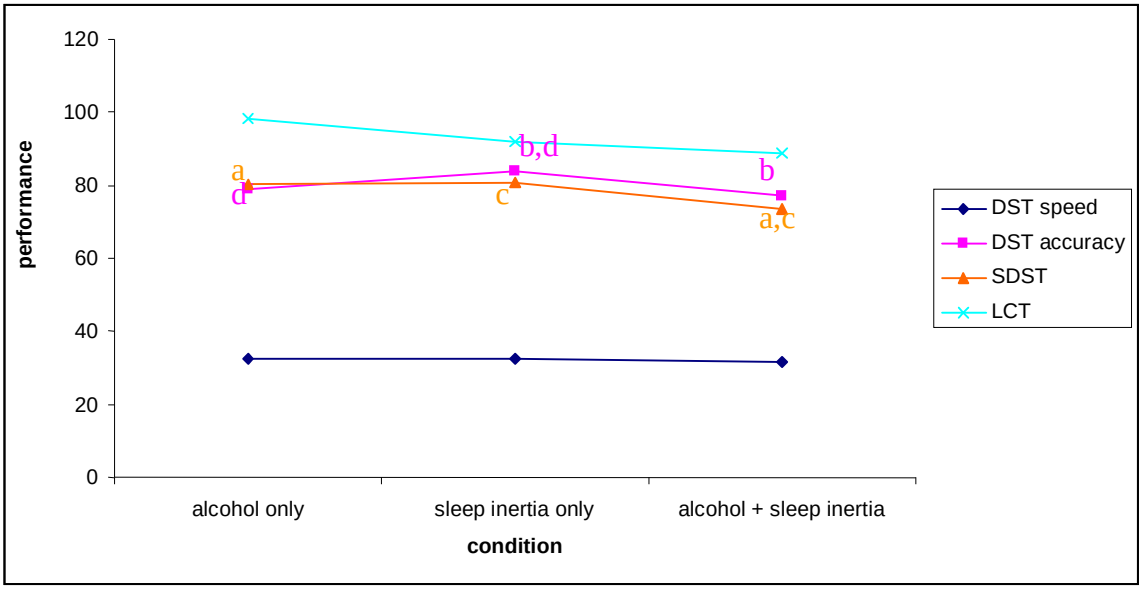


Figure 3. Mean performance scores for the DST, SDST and LCT for conditions within 20 minutes post-awakening.

Note. DST = Descending Subtraction Task. SDST = Symbol Digit Substitution Task. LCT = Letter Cancellation Test.
Note. Differences between points indicated by the same letter are significant ($p < .05$).

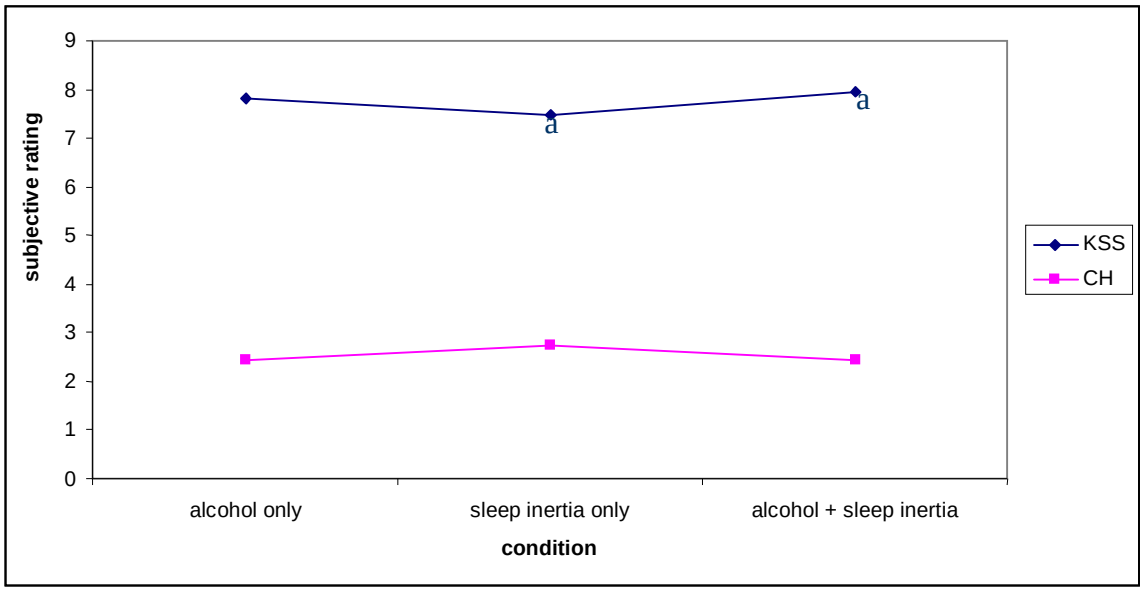


Figure 4. Mean subjective rating scores for the KSS and CH for conditions within 20 minutes post-awakening.

Note. KSS = Karolinska Sleepiness Scale. CH = Clearheadedness Rating Scale. Higher scores on the KSS = less sleepiness (more alert), higher scores on the CH = less clearheaded (more 'grogginess').

4.6.3 Summary

Mental tracking, visual scanning, psychomotor speed, attentional functions and speed of working memory performance are significantly worse under conditions of the first 10 minutes of sleep inertia than conditions of alcohol impairment. Further, cognitive performance is significantly worse under conditions of combined alcohol impairment and sleep inertia than conditions of alcohol impairment alone. There are, however, no significant differences in performance between conditions of combined alcohol impairment and sleep inertia and conditions of sleep inertia only. Within 20 minutes post-awakening, sleep inertia effects have dissipated to unmask pre-existing alcohol effects on working memory performance accuracy, selective/sustained attention, and levels of subjective sleepiness. A combined effect is found for divided attention/mental tracking/psychomotor speed (SDST) performance only within 10-20 minutes post-awakening, although this is possibly a spurious result. Moderate alcohol impairment and sleep inertia do not demonstrate a combined effect on other cognitive measures or self-report ratings. Table 11 below summarises the results.

Table 11.

Summary of key performance and subjective ratings differences at 0-10 and 10-20 minutes post-awakening.

0-10 minutes post-awakening	**10-20 minutes post-awakening**
SI < A (* DST speed, SDST, LCT)	A < SI (* DST accuracy, LCT)
SI + A < A (* DST speed, SDST, LCT)	SI + A < A (* SDST only)
SI + A = SI (* DST speed, SDST, LCT)	SI + A < SI (* SDST, DST accuracy & KSS)
	A < SI + A (* LCT only)

Note. SI = sleep inertia. A = alcohol. SI + A = combined effects.
* $p < .05$.
Note. Lower performance and subjective rating scores indicate more impairment in this table.

4.7 Correlations between subjective ratings

There were medium to large negative correlations between scores on the KSS and CH at all (sober and alcohol impaired) time points across nights 2 and 3, according to Pearson's *r* correlation interpretation parameters provided by Cohen (1988). These negative correlations indicate that as participants were reporting increased levels of sleepiness they were reporting a corresponding decrease in levels of clearheadedness. KSS and CH ratings were *significantly* correlated at night 2 baseline sober (time 1; $r = -.639$, $p = .001$), night 3 (sober) sleep inertia 1 (time 3; $r = -.468$, $p = .021$) and night 3 (sober) sleep inertia 2 (time 4; $r = -.476$, $p = .019$). This indicates that self-reports of sleepiness and clearheadedness were significantly correlated at all experimental conditions that did not involve alcohol.

KSS and CH ratings were moderately but not significantly correlated during conditions involving alcohol, indicating that alcohol affected sleepiness and clearheadedness similarly but not in perfect unison. Post-hoc analyses revealed no significant correlations between subjective self-report measures and objective cognitive performance measures at any of the assessment time points in the current study.

CHAPTER FIVE: DISCUSSION

5.1 Overview

Sleep inertia is a psychophysiological phenomenon that significantly impairs one's neurocognitive integrity and the ability to execute behaviours efficiently and effectively. It may have significant implications for human performance in an emergency waking context, including escape from a fire emergency. Alcohol is a central nervous system depressant that also impairs cognitive and behavioural functioning. Research shows that being alcohol-affected and being asleep in a residential home are two important risk factors for death in a fire (Barillo & Goode, 1996; Karter, 1986; Runyan et al., 1992). The current study aimed to contribute to the understanding of fire death vulnerability afforded by alcohol intoxication. Specifically, it investigated people's ability to perform effective fire escape behaviours during conditions of acute sleep inertia whilst under the influence of moderate levels of alcohol.

The study was the first to investigate the interaction of sleep inertia and alcohol impairment on neurocognitive functioning. Previous research indicates that alcohol and sleep processes do interact, sometimes synergistically, to produce performance decrements greater than the sum of either factor alone (e.g., Peeke et al., 1980). The results from prior studies are inconsistent, however, and the precise nature of this interaction is hard to predict, owing to a number of variables including the biphasic effects of alcohol metabolisation, level of intoxication, and the nature of the behaviour or cognitive process under investigation. Overall, the present study found that sleep inertia and moderate levels of alcohol intoxication do not interact to produce a combined effect on a variety of simple and complex neurocognitive functions relevant to performing effective fire emergency escape behaviours. Indeed a rather complex interaction was observed secondary to speed-accuracy trade-off effects, task complexity, and the time course dissipation of sleep inertia on different neurocognitive processes. The following discussion addresses the results of the current study with specific reference to previous research and the hypotheses investigated. Directions for future research will be explored in light of identified limitations of the current study.

5.2 Sleep inertia

The present study was successful in replicating the sleep inertia effect on neurocognitive functioning using a variety of tasks to tap working memory functioning and attentional processes, with secondary assessment of psychomotor performance, mental tracking and visual scanning. It was hypothesised that conditions of sleep inertia would produce cognitive performance decrements, increased subjective sleepiness and decreased subjective clearheadedness, compared to 'baseline' conditions. Consistent with the predictions of Hypothesis 1a, there was significant impairment on measures of speed of working memory performance (Descending Subtraction Task; DST), divided attention (Symbol Digit Substitution Task; SDST) and selective/sustained attention (Letter Cancellation Test; LCT) during sleep inertia. Accuracy of working memory performance was the only measure that failed to show impairment under conditions of sleep inertia (see section *5.3* below for discussion). Participants also felt significantly sleepier (Karolinska Sleepiness Scale; KSS) and significantly less clearheaded (Clearheadedness Rating Scale; CH) during conditions of sleep inertia compared to baseline, demonstrating that both objective and subjective experiences of sleep inertia were captured in the current study. These results are commensurate with previous research which has demonstrated that sleep inertia affects a variety of neurocognitive functions, including working memory, attentional processes and psychomotor performance (as shown in Table 1), and indeed these effects have been shown previously using the same measures employed in the current study (e.g., Dinges et al., 1985; Tassi et al., 2003; Tietzel & Lack, 2001). Subjective experiences of sleep inertia have also been demonstrated in previous research using both the KSS and the CH (e.g., Bruck & Pisani, 1999; Kräuchi et al., 2004).

In the current study, participants were assessed under conditions of sleep inertia immediately following awakening from signals that began to be presented during stage 4 sleep; the deepest stage of slow wave sleep that is associated with a more synchronised EEG and therefore a greater level of cognitive slowing upon awakening according to the arousal hypothesis (see section *2.1.1*). Participants were assessed under conditions of sleep inertia approximately 1.6 hours after their usual bedtime, so that they were

generally at the downward point on their circadian rhythm, approaching the nadir. Research indicates that awakenings at the trough of the circadian rhythm or during the 'biological night' are associated with greater cognitive deficits than the peak of the circadian rhythm or the 'biological day'. Therefore the significant results and the moderate to large effect sizes obtained in the study are also commensurate with the current literature which indicates that both pre-awakening stage of sleep and circadian phase contribute to and enhance the effects of sleep inertia (see sections *2.2.1* and *2.2.3*, respectively for details of the supporting literature).

Sleep inertia effects were evident regardless of the arousal method. That is, there was no difference in objective or subjective sleep inertia effects when participants were awoken with an auditory or tactile stimulus. These results are also consistent with the current literature. Whilst a small selection of literature has shown that continuous low frequency noise can alleviate the cognitive decrements associated with sleep inertia under conditions of partial sleep deprivation (e.g., Tassi et al., 1992), increase arousal and offset hypovigilance (Koelega & Brinkman, 1986), there is no experimental evidence or theoretical postulations to predict that arousal by short-duration auditory stimuli would have a differential arousing effect compared to an awakening stimulus presented in a different sensory mode, e.g., visual or tactile. This result suggests that regardless of how persons are awoken to an emergency situation (auditory – e.g., sound of fire alarm or actual fire itself, or tactile – e.g., being shaken awake by another person), there is no difference in the resultant sleep inertia effects. However, as the two stimuli conditions, auditory and tactile, were only tested under conditions of alcohol impairment, some caution is needed in generalising this to sober conditions.

Controversy surrounds the precise time course of sleep inertia dissipation with estimates ranging from minutes to hours (Tassi & Muzet, 2000). In the current study, different measures showed a different time course of sleep inertia, with some measures demonstrating a complete dissipation of sleep inertia effects within the 20 minute assessment period, whilst other measures demonstrated a trend towards recovery but did not improve significantly. Speed of working memory (DST), divided attention (SDST)

and subjective feelings of sleepiness (KSS) demonstrated some recovery of performance throughout the 20 minute assessment of sleep inertia. Speed of working memory performance and subjective feelings of sleepiness recovered to baseline levels, whilst divided attention performance demonstrated some improvement, but performance still remained sub-optimal within 20 minutes post-arousal. These results are consistent with the extant literature which demonstrates that sleep inertia effects generally last 15-30 minutes (see section *2.2.4*), with the severest impairments occurring in the initial minutes of sleep offset, but that in addition to this, the duration of sleep inertia may be different for different types of cognitive processes, and indeed may be a task-dependent effect (e.g., Jewett et al., 1999; Merica & Fortune, 2004; Tassi & Muzet, 2000). Further, the time course dissipation of subjective experiences of sleep inertia does not generally match that found for cognitive correlates. For example, Jewett and colleagues (1999) found that the time constant for dissipation of sleep inertia on cognitive tasks was much larger than that of subjective alertness, partially consistent with the results of the current study. They found that self-reports of sleepiness took 0.67 hours to dissipate, whilst cognitive throughput was impaired for up to 1.17 hours post-awakening. In comparing their results to the dissipation of cognitive measures in other studies of sleep inertia, Jewett et al. (1999) concluded that "some neurobehavioural functions may be more sensitive to sleep inertia than others" (p. 6). This reasoning could account for the current findings in which levels of subjective sleepiness recovered to baseline levels whilst the cognitive tasks were variable in their level of recovery within the 20 minute sleep inertia period.

Interestingly, the most complex task in the assessment battery (the DST) was the only cognitive task to show complete recovery to baseline levels within the 20 minute sleep inertia period. This indicates that there was a rapid dissipation of sleep inertia effects for this complex task within the first 10 minutes post-arousal. The argument for the arousing effects of complex tasks would predict that the greater the physiological sleepiness, the more dependent one is on the environment for stimulation to maintain wakefulness. Therefore, an interesting environment or complex task can mask physiological sleepiness, whilst a boring task or environment can enhance it (Babkoff et al., 1991). Given that the

DST was the most complex task in the experimental protocol, and was always administered in the first 3 minutes of sleep offset, i.e., the time of greatest physiological sleepiness, it is possible that the complexity of this task was stimulating enough to cause a sharper-than-usual increase in cerebral metabolism, and therefore demonstrate the most rapid recovery of sleep inertia effects. It is difficult however, to extrapolate findings regarding physiological sleepiness to those pertaining to sleep inertia given the controversy surrounding the similarities and differences between these two sleep-related processes. The current finding suggests that, like sleep deprivation (e.g. Horne & Pettitt, 1985), sleep inertia effects after the first 10 minutes or so are also sensitive to increases in task-related arousal, complexity, or motivation, and that the sleepiness caused by sleep deprivation and ongoing sleep inertia may respond in the same way to complex, arousing, or motivating tasks. Other data in the current study and the wider literature, however, supports the conceptualisation of sleep deprivation and sleep inertia as different processes (see section *5.5.3* below).

5.3 Speed-accuracy trade-off effects

A speed-accuracy trade-off effect was observed for working memory functioning (DST), whereby sleep inertia affected speed of working memory performance, but accuracy was unimpaired. These findings accede to widespread conjecture regarding the particular sensitivity of frontal brain regions to cognitive slowing upon arousal from sleep. According to the arousal hypothesis (see section *2.1.1*), sleep inertia causes a general cognitive slowing which essentially reduces the efficiency of one's cognitive and behavioural performance, but performance is no more erroneous than what is typical for the individual. Neurophysiological research demonstrates that frontal lobe functions, or cognitions that are anatomically represented in the anterior regions of the brain, such as working memory, are the slowest to recover during conditions of sleep inertia. Cerebral blood flow can take up to 15 minutes to re-perfuse to wake time levels in anterior brain regions according to PET studies (Balkin et al., 2002).

The speed-accuracy trade-off effect observed on the DST is consistent with other studies that have also shown such effects under conditions of sleep inertia on both the DST (e.g.,

Stampi & Davis, 1991) and other addition tasks (e.g., Balkin & Badia, 1988; Hofer-Tinguely et al., 2005; Jewett et al., 1999). The speed-accuracy trade-off effect observed in the current study is also consistent with a myriad of previous research that demonstrates that speed of cognition, and not accuracy, is affected on a wide variety of performance tasks under conditions of sleep inertia (e.g., Matchock & Mordkoff, 2007; Salamé et al., 1995; Seminara & Shavelson, 1969; Tassi et al., 1992; see section *2.3.3* for details of the supporting literature).

The magnitude of the speed decrement found on this task does not support Tassi et al.'s (1993) findings that more complex tasks, such as the regular (*cf.* simple) version of the DST, are less affected by sleep inertia due to their arousing effects. However, whilst the other tasks used in the current study are relatively simple when compared to the complexity of the DST, it is not possible to assess the role of task complexity with the current data. That is, performance across tasks is not directly comparable in the current study as (1) they assess different neurocognitive functions which could be differentially affected by sleep inertia and (2) the DST was always administered immediately after sleep offset in the most acute stage of the sleep inertia process, which represents a confound when trying to assess the role of task complexity in sleep inertia (which was not an aim of the current study). It is possible, however, that the strength of the sleep inertia effect may have been even larger had a simple task been administered in the first few minutes of awakening. That is, consistent with Tassi et al.'s (1992) theoretical reasoning regarding the Yerkes-Dodson principle of optimal arousal and task complexity, it is possible that the complexity of the DST *minimised* but did not completely eradicate sleep inertia effects. The more intense stimulation of the DST (*cf.* SDST or LCT) possibly led to a sharper increase in cerebral metabolism which ultimately tempered sleep inertia effects, particularly as it occurred so soon after wake time.

The speed decrements associated with sleep inertia are often particularly observable on self-paced tasks (Dinges, 1990). Although *all* tasks used in the current study were self-paced, the nature of the SDST and LCT were that they were so simple, it is argued, that they did not permit a separate analysis of speed and accuracy components, and therefore

speed-accuracy trade-off effects were not discernible. Hypothesis 1b, which predicted that speed of performance would be affected for all cognitive measures under conditions of sleep inertia, was therefore only partially investigated/supported.

These results suggest that even in the absence of alcohol intoxication, people may have difficulty responding to an emergency fire situation in a rapid and efficient manner immediately upon awakening due to the decreased arousal, feelings of grogginess and slowed neurocognitive functioning associated with sleep inertia.

5.4 Alcohol

Previous research has demonstrated that alcohol intoxication causes transient measurable cognitive deficits, particularly in psychomotor speed, memory, attention, and judgment (Allen et al., 2006). In the current study it was hypothesised that healthy young adults would demonstrate cognitive deficits on measures of working memory and attentional processes with a psychomotor component when moderately intoxicated. In support of the predictions of Hypothesis 2, performance on tasks of working memory (DST) accuracy, divided attention (SDST) and selective and sustained attention (LCT) was significantly impaired with intoxication, when compared to baseline performance. Speed of working memory performance was the only measure that failed to show impairment under conditions of moderate alcohol intoxication. The finding of alcohol-related attentional deficits is commensurate with other studies investigating cognitive changes during intoxication (e.g., Abroms & Fillmore, 2004; Fillmore et al., 2000; Jääskeläinnen et al., 1999; Lex et al., 1994; see section *2.7* for details of the supporting literature), and in particular, decrements in divided attention performance are consistent with the attention-allocation model of alcohol-related cognitive processing deficits (Steele & Josephs, 1990). In addition, alcohol also significantly increased subjective feelings of sleepiness (KSS) and decreased subjective feelings of clearheadedness (CH), indicating that the measures used in the current study captured both objective and subjective aspects of alcohol impairment.

The 'accuracy-speed trade-off' evident on the working memory task is commensurate with the known effects of alcohol on cognitive performance during the descending limb of the blood alcohol concentration (BAC) curve. On average, the baseline 0.05 BAC condition (time 2) occurred 108.81 minutes (*SD* = 25.81 minutes) following the baseline sober condition (time 1 – immediately after which they began to consume alcohol), and therefore participants were likely to be entering the descending limb of the BAC curve (see Schweizer & Vogel-Sprott, 2008). The current literature indicates that for cognitive processes including working memory (Grattan-Miscio & Vogel-Sprott, 2005), selective attention (Fillmore et al., 2000), inhibition (Fillmore, Marczinski, & Bowman, 2005), information processing (Schweizer, Jolicœur, Vogel-Sprott, & Dixon, 2004), and new learning (Pihl et al., 2003) an 'accuracy-speed trade-off' is evident on the descending limb of the BAC curve, in which performance accuracy is compromised but performance speed is unaffected. This occurs despite impaired performance on both elements during the ascending limb and it is thought that the speed of cognitive operations exhibits "acute tolerance or recovery from impairment" (Schweizer & Vogel-Sprott, 2008, p. 246). Unfortunately the other cognitive tasks used in the current study (SDST and LCT) could not be analysed in terms of speed or accuracy components. It is believed that the relative simplicity of these tasks compared to the DST limited their sensitivity to separate assessments of speed and accuracy.

As the literature suggests, alcohol affects the accuracy with which one can perform (e.g., Bartholow et al., 2003; Peeke et al., 1980). The current study demonstrated that this is true for moderate levels of intoxication, at the legally prescribed level for driving in many western industrialised countries (0.05 BAC). Decreased accuracy of performance can partially explain why alcohol is a significant risk factor for fire fatality; as people are more likely to make errors or mistakes when under the influence of alcohol, e.g., forget to turn off the oven or mistakenly put a flammable object such as a towel on a heater or near an open flame. They may also mistakenly fail to extinguish a lit cigarette prior to falling asleep, which is a very important cause of death in fire (Leistikow, Martin, & Milano, 2000). The response-inhibition model of alcohol effects on cognitive processing would predict that alcohol-affected persons are more likely to carry-out an erroneous action that

increases their vulnerability to fire danger as their abilities for disinhibiting inappropriate actions and their self-monitoring of errors is reduced. According to the attention-allocation model of alcohol effects on cognitive processing, alcohol would impair one's ability to effectively escape from a fire emergency situation as their ability to manage multiple demanding stimuli in their immediate environment is decreased.

5.5 Sleep inertia and alcohol impairment combined

Given the known effects of cognitive slowing under conditions of sleep inertia and effect of alcohol in impairing accuracy of performance at all stages of the BAC curve (in addition to impairing speed during the ascending curve), a combination of reduced speed *and* accuracy would be expected in the presence of both sleep inertia and moderate levels of alcohol intoxication. In the current study it was hypothesised that this interaction would cause greater performance impairments and greater feelings of sleepiness and grogginess than either sleep inertia or alcohol impairment alone. Overall, however, there was no additive or synergistic interaction observed between sleep inertia and alcohol impairment, but a rather complex interaction influenced by speed-accuracy trade-off effects, task complexity, and the time course dissipation of sleep inertia and alcohol.

5.5.1 Sleep inertia and alcohol impairment combined: Within 10 minutes post-awakening

Within the first 10 minutes of sleep inertia, sleep inertia effects clearly 'overrode' alcohol effects. That is, sleep inertia alone and the combination of sleep inertia and alcohol impairment produced greater performance decrements than alcohol impairment alone. Further, decrements in working memory speed (DST) and attentional functions (SDST and LCT) were not significantly different between conditions of sleep inertia alone and combined sleep inertia and alcohol impairment.

This pattern of performance was observed for all cognitive measures, with the exception of performance accuracy on the DST, and subjective ratings (KSS and CH), which did not change significantly between conditions. Considering that sleep inertia was shown earlier to have selectively affected the speed of working memory functioning and alcohol,

as discussed above, selectively affected working memory accuracy, it is evident from the results obtained that indeed sleep inertia effects exerted a much stronger influence on cognitive performance, as working memory speed was most significantly impaired under conditions of sleep inertia only (*cf*. the alcohol alone condition), whilst working memory accuracy was unchanged across conditions.

The finding of comparable performance decrements between the sleep inertia alone and combined sleep inertia and alcohol conditions is inconsistent with previous studies that have shown that sleep processes such as sleep restriction, when combined with alcohol, produce performance decrements greater than conditions of sleep restriction alone on driving simulator performance (e.g., Banks et al., 2004; Vakulin et al., 2007). However, the concept of sleep inertia and sleep restriction/deprivation as physiologically and cognitively equivalent states is controversial with evidence to suggest that these two sleep process differentially impair cognitive functioning (see section *2.3.4.2*). Thus, the comparison of the current results to previous studies investigating the interaction of alcohol and other sleep processes is not directly valid. The current study is pioneering in its investigation of the interaction between sleep inertia and alcohol impairment, and therefore it is generally not possible to validate these results against previous findings.

These results clearly indicate that within 10 minutes post-awakening sleep inertia effects on cognitive functioning are significant. This suggests that sleep inertia may pose a greater risk to effective fire emergency escape than moderate level alcohol impairment. These are potentially important findings, considering the initial minutes post-awakening are the most crucial for safe evacuation in an emergency fire situation. These results would suggest that the role of moderate alcohol intake in significantly increasing risk of death from a fire, is most likely either; (1) in impairing one's ability to waken to their fire alarm signal, consistent with Ball and Bruck (2004), or (2) a result of high fire risk behaviours, including behaviours where the victim is intimate with the fire, or (3) a combination of both. Sleep inertia may significantly hamper safe evacuation attempts in the event that one does awaken to their fire alarm, and this is the case whether moderate alcohol intake is involved or not.

5.5.2 Sleep inertia and alcohol impairment combined: Within 20 minutes post-awakening

Within 20 minutes post-awakening, sleep inertia effects began to dissipate on most of the cognitive and subjective measures. Interestingly, this dissipation in sleep inertia effects meant that alcohol effects were essentially 'unmasked' on two measures that previously showed strong alcohol effects in the current study but no evidence of sleep inertia effects (or change) at 0-10 minutes post-awakening. That is, for working memory accuracy (DST) and subjective sleepiness (KSS), performance was now significantly worse during the combined condition than during the sleep inertia alone condition. The selective/sustained attention task (LCT) also demonstrated an 'unmasking' of alcohol effects whereby performance under conditions of alcohol only was now significantly worse than during conditions of sleep inertia only or the combined condition. The measure of divided attention (SDST) demonstrated a complex and unpredictable pattern at 10-20 minutes post-awakening whereby performance under conditions of sleep inertia and alcohol intoxication combined became significantly worse than either condition alone. This is believed to be a spurious effect as performance decrements on the measure of divided attention dissipated much more rapidly during the sleep inertia only condition than during the combined condition, and indeed had even shown significant recovery effects between 0-10 and 10-20 minutes post-awakening. Further to this, a true combined or additive effect of sleep inertia and alcohol for this measure at 10-20 minutes post-awakening is very unlikely as no other measure had displayed such an effect, and a combined or additive effect was not evident on the measure of divided attention within the first 10 minutes of sleep inertia. It is possible that the spurious effect of greater performance decrements during the combined sleep inertia and alcohol condition for the measure of divided attention (SDST) is a result of a more rapid recovery of divided attention performance during the sleep inertia alone condition, in addition to the known longer-lasting effects of alcohol compared to sleep inertia.

Indeed overall, the general pattern of results observed at 10-20 minutes post-awakening is consistent with the known longer-lasting effects of alcohol compared to sleep inertia. Research shows that sleep inertia effects typically dissipate within 15-30 minutes after

sleep-offset, whilst the descending limb of the BAC curve of alcohol metabolisation gradually dissipates over 3 or more hours, depending on the level of consumption (see section *2.5.1*).

These results indicate that after sleep inertia effects have dissipated, alcohol will continue to cause feelings of sleepiness and affect some aspects of cognitive functioning. If the effects are sufficiently severe (which will depend on level of alcohol intake), performance decrements that would present a risk for those attempting to escape from fire would be anticipated. Research shows that fire victims typically have high to very high BAC levels at the time of death, usually exceeding a BAC of 0.1 (e.g., Berl & Halpin, 1978; Brennan, 1998; Marshall et al., 1998; Paetta & Cole, 1990; see section *1.1*).

5.5.3 The interaction of sleep inertia and alcohol impairment

The current study was the first to investigate the interaction of sleep inertia and alcohol impairment on neurocognitive processes. The results indicate that, contrary to the predictions of Hypothesis 3, the combined effects of sleep inertia and moderate levels of alcohol impairment do not cause neurocognitive performance decrements greater than those caused by either factor alone, but rather that sleep inertia effects 'override' alcohol effects. A combined effect was predicted as previous studies have shown a combined interaction between alcohol and other sleep processes, namely sleep deprivation and sleep restriction, however the results have not always been consistent, with some antagonistic interactions reported. Inconsistencies in the literature mean that the true effects of the interaction between alcohol and sleep processes arising from sleep deprivation remains obscure. The extent to which such inconsistencies are relevant to the current research depends on whether or not sleep inertia and sleep deprivation are the same psychophysiological state (see section *2.3.4.2*). It is often argued that they are theoretically and physiologically different, and whilst both temporarily impair neurocognitive functioning, the nature of the impairment is not identical. Sleep deprivation tends to specifically produce attentional lapses, microsleeps and fluctuations in attention, whilst sleep inertia can cause global impairments in both simple and

complex cognitions (see section *2.3* and Table 1). Given the differences between sleep inertia and sleep deprivation, it is theoretically acceptable that these two processes do not interact with alcohol intoxication in the same manner and that indeed, sleep inertia and alcohol do not interact in combination on neurocognitive functioning to produce impairments greater than that caused by each factor alone. However, the current study is original in its investigation of the interaction between sleep inertia and moderate levels of alcohol intoxication, and a replication of these findings would serve to confirm this conclusion.

The pioneering nature of the current study also warrants an analysis of alternative explanations for the obtained results. Firstly, participants were typically awoken from stage 4 sleep, whilst nearing the trough of their circadian rhythm. As discussed earlier, awakening from slow wave sleep and awakenings during the 'biological night' are two factors that serve to enhance the effect of sleep inertia on neurocognitive functioning. Alcohol, on the other hand, was administered in a moderate dose and BAC levels were highly variable following sleep inertia testing (M = 0.034, SD = 0.01; see Appendix L for raw data). It is possible that moderate levels of alcohol are insufficient to produce an interaction with sleep inertia, and/or that sleep inertia effects were so severe in the current study as to 'override' both alcohol effects and any interaction between sleep inertia and alcohol impairment. This latter scenario could possibly be an artifact of a methodological shortcoming in the current study. That is, due to the constraints imposed by being part of a larger research protocol, alcohol and sober testing nights were unable to be counterbalanced and therefore the sober testing night always occurred as the final testing night in the three-night testing protocol. It is possible that participants had decreased motivation for the study by this point, which caused poorer performance during the sober sleep inertia only assessment.

Secondly, it is possible that the inclusion of both males and females in the subject pool changed or masked true alcohol effects in the current study. Gender differences in moderate drinking effects are well-established and some studies have shown that women are more sensitive to the effects of alcohol on cognitive functioning, particularly on tasks

involving divided attention functions (Mumenthaler, Taylor, O'Hara, & Yesavage, 1999). Inconsistencies between genders in the neurocognitive effects of alcohol may have weakened the uniformity and reliability of results and hence reduced statistical power. Most studies that investigate the effects of alcohol on sleep use single-gender subject pools (e.g., Arnedt et al., 2000; Peeke et al., 1980; Rundell et al., 1972; Vakulin et al., 2007; Van et al., 1995; Van Reen et al., 2006; Yules et al., 1967)

Thirdly, given the wide social awareness of the effects of alcohol on cognitive functioning, it is possible that participants, either intentionally or unintentionally engaged in compensatory mechanisms when under the influence of alcohol. This effect, although uncommon, has been reported in the research literature previously, but tends to be applied more to the absorption phase of alcohol, or the ascending limb of the BAC curve (e.g., Arnedt et al., 2000). When participants are aware of their intoxication, and aware of the effects of alcohol on cognitive processes, they may concentrate particularly hard on performing well, recruiting more cognitive resources than they typically would under baseline conditions. This effect serves to reduce the cognitive decrements associated with alcohol and therefore the current results may be an under-representation. Alternatively, a confirmatory bias effect is also possible, during which participants perform in a manner that confirms the research hypotheses, or what the participant believes the researcher expects. This incidental motivation is a more common threat to internal validity that typically serves to mimic or enhance the effects of the variable under investigation, and could therefore represent an over-estimation of the results of the current study. Other possible explanations for the observed results will be discussed in light of the limitations of the methodological design (see section *5.7* below).

5.6 Subjective ratings

Subjective correlates of sleep inertia are widely reported in the literature under a variety of experimental paradigms, including correlations between levels of self-reported sleepiness and physiological measures of sleep intensity (see section *2.4*). Research also shows that subjective ratings of sleep deprivation-related sleepiness are also correlated with each other (e.g., Babkoff et al., 1991; Gillberg et al., 1994). In the current study it

was hypothesised that subjective feelings of sleepiness (KSS) and clearheadedness (CH) would be significantly correlated under all conditions. With decreased alertness, disorientation and 'grogginess' being the hallmark features of sleep inertia, it was expected that self-reports of sleepiness and clearheadedness would capture these subjective experiences of sleep inertia and therefore change in parallel.

When comparing ratings of subjective sleepiness and subjective clearheadedness, results indicated that these measures were moderately to largely correlated at all time points in the current study. These correlations were always negative in direction, indicating that as levels of subjective sleepiness were increasing, levels of subjective clearheadedness were decreasing; i.e., feelings of sleepiness and grogginess corresponded. Although these measures were moderately to largely correlated at all time points, these correlations only reached significance during conditions that did *not* involve alcohol intoxication, providing partial support for Hypothesis 4. This unexpected finding may actually serve to demonstrate the differential validity of the CH. The results provide preliminary support for the idea that sleepiness and clearheadedness are different constructs and therefore manifest themselves differently under different conditions, with moderate alcohol ingestion being one of the conditions that reveal different effects. The notion that alcohol ingestion affects clearheadedness, independent of sleepiness, certainly has face validity.

Another possible explanation for the observed pattern of results is that alcohol may impair one's insight into their subjective experiences, or impair an accurate reporting of subjective experiences. This is not unreasonable to assume, given that one of the most common cognitive effects of alcohol is to impair one's judgment (Allen et al., 2006). Alternatively, alcohol is known to have idiosyncratic effects on individuals and its effects can often vary according to an individual's physiological or mood state. For example, a fatigued or sleepy person may choose to drink alcohol for its 'pick me up' effects, whilst a tense or anxious person may require a drink to relax and calm down (Peeke et al., 1980; Sher 1985). These can be both state- and trait-dependent effects, and although contingencies were put in place to control for the effects of prior sleep deprivation in the

current study (which can affect fatigue, sleepiness, physiology, and mood among other things), the subjective effects of alcohol are difficult to control due to the varied effects it can have on individuals as a function of their physiological or mood state (de Wit, Uhlenhuth, Pierri, & Johanson, 2006). This could also account for the lack of significant correlations between subjective self-reports during conditions involving alcohol intoxication.

Nonetheless, the significant correlations achieved in the study are consistent with the extant literature which demonstrates both the KSS and CH to be valid measures of sleepiness and sleep inertia, in the absence of alcohol impairment. The KSS is a particularly popular self-report tool that has been used in a wide variety of sleep study paradigms to accurately depict levels of subjective sleepiness (e.g., Bruck & Pisani, 1999; Kräuchi et al., 2004; Salinen et al., 1998). The current study is only the second study to use the CH in an experimental sleep inertia paradigm, but the current results are commensurate with those of Bruck and Pisani (1999) who found significant decrements in feelings of clearheadedness from baseline to sleep inertia, and significant correlations between the KSS and CH under conditions of baseline and sleep inertia following REM or slow wave sleep arousals in sober young adults.

Post-hoc analyses revealed no significant correlations between subjective self-ratings and objective cognitive performance measures in the current study. This adds to the general consensus in the current literature that subjective experiences of sleep inertia do not closely reflect changes in objective measures of neurocognitive performance (e.g., Achermann et al., 1995; Brooks & Lack, 2001; Bruck & Pisani, 1999). It is possible that the limited sensitivity of rating scales in capturing change (*cf*. the high sensitivity of cognitive measures) and the reduced/variable validity of self-report measures (*cf*. highly valid objective measures) means that subjective experiences and objective parameters of sleep inertia often do not correlate. It has also been suggested that the consistent dissociation between objective and subjective measures of sleep inertia found in the literature may reflect the fact that "the intensity of sleep inertia seems to make it difficult to estimate how sleepy one feels" (Dinges, 1990, p. 165), and therefore subjective self-

reports of alertness during conditions of sleep inertia often do not accurately reflect the magnitude or time course of sleep inertia captured with the more sensitive neurocognitive performance measures.

5.7 Strengths and limitations of the current study

A strength of the current study was the utilisation of a within-subjects repeated-measures design. Repeated-measures statistics are a particularly robust and powerful statistical method as they eliminate variance due to individual differences. This is especially important in the context of sleep research as individuals vary widely on important sleep parameters including sleep architecture (Buckelmüller, Landolt, Stassen, & Achermann, 2006), morningness-eveningness and circadian timing (Horne & Ostberg, 1977), in addition to their sensitivity to the effects of alcohol (de Wit et al., 2006; Sher, 1985). Due to the fact that the current study was undertaken in the context of a larger research protocol assessing the comparative waking effectiveness of different alarms (auditory, visual and tactile) for the alcohol impaired (and thus testing for that study did not include sober nights), sober and alcohol testing nights were unable to be counterbalanced. All participants undertook two alcohol nights (typically one was with auditory awakening and the other with tactile awakening, order counterbalanced) prior to the sober testing night, which represents a systematic confound and introduces the potential for practice effects (on performance tasks) or adaptation effects (on sleep architecture as per EEG). However, given that no differences were found between auditory and tactile awakenings on outcome measures between the first and second alcohol testing nights, the first alcohol night was discounted in the data analyses and regarded as an adaptation night. This therefore minimised the potential for adaptation or 'first night' effects interfering with true alcohol and sleep inertia effects on the second alcohol night or the sober testing night. It also successfully eliminated practice effects as preliminary analyses confirmed that practice effects, which were only evident on the DST, were confined to the first alcohol night anyway (see section *4.2.2*).

Although the use of the first alcohol night as an 'adaptation night' served to counter practice effects and adaptation effects, there was an average of 117 days between the

second alcohol night and the sober testing night (*cf.* a mean of 21.7 days between the first and second alcohol nights). It is difficult to determine if any adaptation may have dissipated during this unexpectedly long delay. However, there is some evidence to suggest that adaptation to sleep testing may last quite a long time in young adults. Lorenzo and Barbanoj (2002) used the same experimental paradigm to assess the sleep architecture of healthy young adults on three occasions, for four consecutive nights at each occasion, and with a *minimum* of a 1-month interval between testing occasions. They found that the first night effect was only evident on 'the very first night', and was limited to REM sleep-related variables only. Although the current study was not primarily investigating EEG-related variables or sleep architecture per se, the results of Lorenzo and Barbanoj's study demonstrates that adaptation effects on sleep architecture (especially REM-related) in healthy young subjects last *at least* 1 month. Further, measures pertaining to slow wave sleep are less sensitive to adaptation than REM sleep-related variables and this is relevant to the current study as all arousal stimuli began to be presented when the participant was in slow wave sleep. Practice effects on cognitive tasks appear to be particularly long-lasting. In clinical populations, practice effects on neuropsychological tasks are typically evident for approximately 2 years (Lezak et al., 2004) and can last up to several years in research studies (e.g., Rabbitt, Diggle, Smith, Holland, & McInnes, 2001).

It could be argued that the use of the modified method of discrete limits (see section *3.4.3.2*) to arouse participants from sleep represents a methodological flaw. The method of limits, whereby the signal is gradually increased from a low intensity, introduces the possibility that some participants will undergo a gradual awakening from stage 4 sleep through stage 3 and then stage 2 sleep prior to awakening and responding to the arousal signal, whereas other participants may experience a very abrupt awakening straight out of stage 4 sleep. An early study has shown that there is considerable reliability across individuals in their arousal thresholds across different nights (Bonnet, Johnson, & Webb, 1978). Thus light sleepers, for example, tend to be consistently so. The repeated measures design is therefore likely to minimise differences in the sleep stage at final awakening across different nights within the same individual.

During the first sleep inertia condition (time 3) of each testing night, the DST was administered in low-level lighting (this was typically light filtering in from a light source in an adjacent room or hallway), whilst under all other conditions, including the second sleep inertia condition (time 4) the DST was administered with the lights fully illuminated in the testing room (see section *3.4.3.2*). This was done so that assessment of cognitive functioning could occur as soon as possible after sleep-offset, without having to allow time for participants to readjust to lighting levels in their environment and also to avoid bright-light stress. Although this small difference represents a potential confound in the methodological design, arguably a rapid change in lighting before or during the task would also represent a methodological confound. Further, some studies have shown that sleep inertia effects on cognitive performance and subjective alertness are not sensitive to behavioural and environmental factors such as showering or being exposed to ambient light (Jewett et al., 1999). This therefore supports the direct comparison of DST performance at the first (time 3) and second (time 4) sleep inertia conditions, despite the small environmental difference between these conditions afforded by the chosen methodology. If the experimental protocol had dictated that the DST task was interrupted part-way through by illuminating lights, participants would have undoubtedly paused and lost track of their responses whilst they adjusted to the environment change (and therefore their performance would have been ultimately underestimated). By allowing participants to undertake the DST in very low-level lighting immediately following arousal from deep sleep, the most acute effects of sleep inertia on cognitive functioning were able to be captured without interruption of environmental change. Further, participants do not require light to undertake the DST task as they are not permitted to use their fingers or any other counting aids to assist them with their calculations for this task. The short time between the administration of the DST and the SDST was used to illuminate lights fully, undertake a breathalyser test and allow participants to readjust to the bright light, which they required to undertake the paper-and-pencil SDST. The fact that sleep inertia effects are not responsive to immediate environmental change such as exposure to ambient light (Jewett et al., 1999) means that avoiding intra-task interruption and avoiding delaying the assessment of sleep inertia to ensure that all tasks were undertaken in lit conditions was a better choice.

The study employed the DST as a measure of working memory performance. The DST is a popular assessment tool that has been used to capture cognitive performance in a wide variety of sleep study protocols (see section *2.3.1* for details of the supporting literature). It recruits working memory functions by requiring participants to perform calculations in their mind and produce a verbal response. Given that the aim of the current study was to assess the interaction of sleep inertia and alcohol on neurocognitive processes required for effective escape in an emergency fire situation, a *spatial* rather than verbal working memory task may have been more ecologically valid and increased the generalisability of the findings. However, the pioneering nature of the study warranted the use of a well-established and reliable tool to capture cognitive processes as soon as possible after sleep-offset, which the DST provides. Whilst verbal working memory tasks assess the phonological loop component of the working memory system and spatial working memory tasks assess the visuo-spatial sketch pad, both these components, and the entire working memory system are represented in the same anatomical location of the brain – the frontal lobes, specifically the dorsal and lateral prefrontal cortex (Baddeley, 2002; Nolte, 2002). Therefore, according to the theoretical postulations of the arousal hypothesis, the outcome of verbal versus spatial working memory assessment is unlikely to have been different.

The issue of ecological validity also extends to the role of arousal and stress, its effect on sleep inertia, and the generalisability of the current results to real-world emergency situations. Previous studies have shown that for sleep deprived individuals, physiological sleepiness can be overcome in the context of high arousal environments or increased motivation for performance such as a monetary reward (e.g., Horne & Pettit, 1985). This has also been demonstrated for short-term attentional and complex tasks for pathologically sleepy individuals, such as those with narcolepsy (Hood & Bruck, 1996), and in simulation paradigms where more important, complex or real-world tasks are less affected by sleep inertia than relatively meaningless or simple ones (e.g., Haslam, 1982; Seminara & Shavelson, 1969). A subset of the current data also indicates that complex tasks, such as the DST, can instigate rapid recovery of sleep inertia effects, possibly mediated by a sharper-than-usual increase in cerebral metabolism, as per the arousal

hypothesis. It is difficult however, to generalise these 'laboratory' results to a real-world emergency context where a person faced with a fire emergency situation would be *extremely* aroused and stressed, and very highly motivated to escape. Thus, we can be less sure that emergency arousal would affect sleep inertia and cognitive functioning in the same way as arousing/complex tasks or motivating circumstances. Indeed, an individual in a fire emergency situation is likely to be in a state of sympathetic nervous system activation, during which blood perfuses to the body's systemic organs to increase heart rate, breathing and muscle activity (Kalat, 2001). The interaction between sleep inertia and the effects of 'survival mode' or increased sympathetic activation on neurocognitive processes is unknown. Although inducing sympathetic activation responses in participants may present an ethical obstacle, a simulation paradigm is the most suitable in which to investigate the differential effects of heightened emergency arousal and stress on neurocognitive functioning and sleep inertia, and hence increase the ecological validity of the findings. Whilst the level of stress induced in such a paradigm may not parallel that experienced during a real fire emergency, it may create stressful conditions that are inherently closer to real-world emergency stress than that afforded by current laboratory models and pencil-and-paper tasks.

5.8 Conclusions and future directions

The study was the first to examine the interaction between sleep inertia and moderate levels of alcohol intoxication on neurocognitive functions relevant to performing effective fire emergency escape behaviours. Overall, the results suggest that the combination of alcohol impairment and sleep inertia do not interact in an additive or synergistic manner to produce further decrements in performance than those caused by the effects of alcohol or sleep inertia alone. Rather, sleep inertia effects tend to 'override' alcohol effects so that performance decrements during the first 10 minutes of sleep inertia are equivalent to those produced by the combination of sleep inertia and alcohol impairment, and greater than those produced by alcohol alone. This suggests that sleep inertia poses a greater risk to effective fire emergency escape than moderate level alcohol impairment. This pioneering study has demonstrated that when awoken abruptly in an

emergency situation, prior alcohol consumption to 0.05 BAC will not further impede cognitive functioning that is already compromised by a state of sleep inertia.

The current study also demonstrated that sleep inertia selectively affects the speed of neurocognitive functioning, whilst alcohol selectively affects the level of accuracy at which one can perform. In the context of fire emergency behaviours, this indicates that conditions of sleep inertia will impede effective fire emergency behaviours by slowing down the speed of cognitive operations that underpin safe evacuation behaviours. Alcohol, on the other hand, is likely to increase the chance of engaging in high fire risk behaviours, including those that involve becoming intimate with fire, as well as increasing the likelihood of cognitive or behavioural errors during an evacuation procedure, once the slowing effects of sleep inertia have dissipated.

A replication of the current findings in the context of a fire simulation paradigm would enhance the applicability of the outcomes. The arousal hypothesis was applied to the current findings in the explication of arousal, task complexity and speed-accuracy trade-off effects. Future research would benefit from the application of the arousal hypothesis to simulation studies that investigate the combined effects of sleep inertia and high level alcohol intoxication on neurocognitive functioning in a fire emergency context. By linking a simulation paradigm with this theoretical framework, an enhanced understanding of the physiological underpinnings of sleep inertia on neurocognitive processes in the context of high-level stress would be achieved. For example, the role of a stressful environment or behavioural performance task in achieving a sharper-than-usual increase in cerebral metabolism during particularly low-level arousal periods (and possibly causing a rapid recovery of sleep inertia effects) could be investigated and would increase the generalisability of the findings to a real-world emergency context. In addition, such a design could account for factors pertinent to a simulated or real emergency that the current laboratory study was unable to replicate, for example, rapid sequencing of physical and cognitive processes, in addition to high-level or 'survival mode' emergency stress. The present study, however, represents an important first step

towards understanding the cognitive (and behavioural) decrements involved in fire escape, when both sleep inertia and alcohol are implicated.

REFERENCES

Abroms, B. D., & Fillmore, M. T. (2004). Alcohol-induced impairment of inhibitory mechanisms involved in visual search. *Experimental and Clinical Pharmacology, 12*, 243-250.

Achermann, P., & Borbély, A. A. (1994). Simulation of daytime vigilance by the additive interaction of a homeostatic and a circadian process. *Biological Cybernetics, 71,* 115-121.

Achermann, P., Werth, E., Dijk, D-J., & Borbély, A. A. (1995). Time course of sleep inertia after nighttime and daytime sleep episodes. *Archives Italiennes de Biologie, 134,* 109-119.

Adhikari, P,. & Summerill, A. (2000). 1998 National Drug Strategy household survey: Detailed findings. Drug statistics series no. 6, AIHW cat. no. PHE-27. Canberra: Department of Health and Ageing.

Åkerstedt, T., & Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience, 52,* 29-37.

Åkerstedt, T., Torsvall, L., & Gillberg, M. (1989). Shiftwork and napping. In D. F. Dinges and R. J. Broughton (Eds.). *Sleep and alertness: Chronobiological, behavioral, and medical aspects of napping*, pp. 205-220. New York: Raven Press.

Allen, D. M., Frantom, L. V., Forrest, T. J., & Strauss, G. P. (2006). Neuropsychology of substance use disorders. In P. J. Snyder, P. D. Nussbaum and D. L. Robins (Eds.). *Clinical Neuropsychology: A Pocket Handbook for Assessment* (pp. 649-673). Washington DC: American Psychological Association.

Angus, R. G., Pigeau, R. A., & Heslegrave, R. J. (1992). Sustained operation studies: From the field to the laboratory. In C. Stampi (Ed.), *Why we nap* (pp. 217-241). Boston: Birkhäuser.

Arciniegas, D. B., & Beresford, T. P. (2001). *Neuropsychiatry: An introductory approach*. Cambridge: Cambridge University Press.

Armentrout, J. J., Holland, D. A., O'Toole, K. J., & Ercoline, W. R. (2006). Fatigue and related human factors in the near crash of a large military aircraft. *Aviation, Space, and Environmental Medicine*, *77*, 963-970.

Arnedt, J. T., Wilde, G. J. S., Munt, P. W., & Maclean, A. W. (2000). Simulated driving performance following prolonged wakefulness and alcohol consumption: Separate and combined contributions to impairment. *Journal of Sleep Research*, *9*, 233-241.

Australasian Fire Authorities Council (2005). Accidental fire fatalities in residential structures: Who's at risk?

Babkoff, H., Caspy, T., & Mikulincer, M. (1991). Subjective sleepiness ratings: The effects of sleep deprivation, circadian rhythmicity and cognitive performance. *Sleep*, *14*, 534-539.

Baddeley, A. D. (2002). Is working memory still working? *European Psychologist*, *7*, 85-97.

Balkin, T. J., & Badia, P. (1988). Relationship between sleep inertia and sleepiness: Cumulative effects of four nights of sleep disruption/restriction on performance following abrupt nocturnal awakenings. *Biological Psychology*, *27*, 245-258.

Balkin, T. J., Braun, A. R., Wesensten, N. J., Jeffries, K., Varga, M., Baldwin, P., Belenky, G., & Herscovitch, P. (2002). The process of awakening: A PET study of regional brain activity patterns mediating the re-establishment of alertness and consciousness. *Brain*, *125*, 2308-2319.

Balkin, T. J., Wesensten, N. J., Braun, A. R., Varga, M., Baldwin, P., Carson, R. E., Herscovitch, P., & Belenky, G. (1998). Shaking out the cobwebs: Changes in regional cerebral blood flow (rCBF) across the first 20 minutes of wakefulness. *Sleep*, *21*, 271.

Ball, M. & Bruck, D. (2004). The effect of alcohol upon response to fire alarm signals in sleeping young adults. In: Shields J. (Ed.). *Proceedings of the 3rd International Symposium on Human Behaviour in Fire* (pp. 291-302). Belfast, Interscience Communications.

Banks, S., Catcheside, P., Lack, L., Grunstein, R. R., & McEvoy, R. D. (2004). Low levels of alcohol impair driving simulator performance and reduce perception of crash risk I partially sleep deprived subjects. *Sleep*, *27*, 1063-1067.

Banks, S., Catcheside, P., Lack, L., Grunstein, R. R., & McEvoy, R. D. (2005). The Maintenance of Wakefulness Test and driving simulator performance. *Sleep*, *28*, 1381-1385.

Banks, S., & Dinges, D. F. (2007). Behavioral and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine*, *3*, 519-528.

Barillo, D. J., & Goode, R. (1996). Fire fatality study: Demographics of fire victims. *Burns, 22*, 85-88.

Barker, C., Pistrang, N., & Elliot, R. (2002). *Research methods in clinical psychology (2nd Edn)*. Chichester, West Sussex: John Wiley & Sons.

Bartholow, B. D., Pearson, M., Sher, K. J., Wieman, L. C., Fabiani, M., & Gratton, G. (2003). Effects of alcohol consumption and alcohol susceptibility on cognition: A psychophysiological examination. *Biological Psychiatry*, *64*, 167-190.

Bastuji, H., Perrin, F., & Garcia-Larrea, L. (2003). Event-related potentials during forced awakening: A tool for the study of acute sleep inertia. *Journal of Sleep Research*, *12*, 189-206.

Berl, W. G. & Halpin, B. M. (1978). Human fatalities from unwanted fires. *The Johns Hopkins University, Applied Physics Laboratory, National Bureau of Standards Report NBS-GCR-79-168.*

Bonnet, M. H. (1983). Memory for events occurring during arousal from sleep. *Psychophysiology*, *20*, 81-87.

Bonnet, M. H. (1985). Effects of sleep disruption on sleep, performance and mood. *Sleep*, *8*, 11-19.

Bonnet, M. H., Johnson, L. C., & Webb, W. B. (1978). The reliability of arousal threshold during sleep. *Psychophysiology*, *14*, 412-416.

Borbély, A. A. (1982). A two-process model of sleep regulation. *Human Neurobiology*, *1*, 195-204.

Borbély, A. A. (2009). Refining sleep homeostasis in the two-process model. *Journal of Sleep Research*, *18*, 1-2.

Brennan, P. (1998). Victims and survivors in fatal residential building fires. In J. Shields (Ed.), *Human behaviour in fire: Proceedings of the first international symposium* (pp. 157-166). Fire SERT Centre, University of Ulster.

Brooks, A., & Lack, L. C. (2005). Naps. In C. A. Kushida (Ed.), *Sleep deprivation: Clinical issues, pharmacology, and sleep loss effects* (pp. 457-474). New York: Marcel Dekker Inc.

Broughton, R. J. (1968). Sleep disorders: Disorders of arousal? *Science, 159,* 1070-1078.

Bruck, D. (2001). The who, what, where and why of waking to fire alarms: A review. *Fire Safety Journal, 36,* 623-639.

Bruck, D., Thomas, I., & Ball, M. (2007). Waking effectiveness of alarms (auditory, visual and tactile) for the alcohol impaired. *Report for the Fire Protection Research Foundation.*

Bruck, D. & Horasan, M. (1995). Non-arousal and non-action of normal sleepers in response to a smoke detector alarm. *Fire Safety Journal, 25,* 125-139.

Bruck, D., & Kritikos, A. (2007). The effect of sleep inertia on cognitive and physical functioning in older adults. *Sleep and Biological Rhythms, 5,* A4.

Bruck, D., & Pisani, D. L. (1999). The effects of sleep inertia on decision-making performance. *Journal of Sleep Research, 8,* 95-103.

Buckelmüller, J., Landolt, H-P., Stassen, H. H., & Achermann, P. (2006). Trait-like individual differences in the human sleep electroencephalogram. *Neuroscience, 138,* 351-356.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences.* Hillsdale, NJ: Lawrence Erlbaum Associates.

Cooper, M. L., & Orcutt, H. K. (1997). Drinking and sexual experience on first dates among adolescents. *Journal of Abnormal Psychology, 106*, 191-202.

Dawson, D., & Reid, K. (1997). Fatigue, alcohol and performance impairment. *Nature, 388*, 235.

de Quervain, D. J-F., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394*, 787-790.

de Wit, H., Uhlenhuth, E. H., Pierri, J., & Johanson, C. E. (2006). Individual differences in behavioral and subjective responses to alcohol. *Alcoholism: Clinical and Experimental Research, 11*, 52-59.

Dinges, D. F. (1990). Are you awake? Cognitive performance and reverie during the hypnopompic state. In R. Bootzin, J. Kihistrom and D. Schacter (Eds.), *Sleep and cognition* (pp. 159-175). Washington DC: American Psychological Society.

Dinges, D. F., Orne, E. C., Evans, F. J., Orne, M. T. (1981). Performance after naps in sleep-conducive and alerting environments. In L. C. Johnson, W. P. Colquhoun, D. I. Tepas, and M. J. Colligan (Eds.), *Biological rhythms, sleep and shift work* (pp. 539-552). New York: SP Medical and Scientific Books.

Dinges, D. F., Orne, M. T., & Orne, E. C. (1985). Assessing performance upon abrupt awakening from naps during quasi-continuous operations. *Behavior Research Methods, Instruments, and Computers, 17*, 37-45.

Dinges, D. F., Orne, M. T., Whitehouse, W. G., & Orne, E. C. (1987). Temporal placement of a nap for alertness: Contributions of circadian phase and prior wakefulness. *Sleep, 10*, 313-329.

Evans, F. J., & Orne, M. T. (1975). *Recovery from fatigue*. Frederick, MD: U.S Army Medical Research and Development Command.

Feige, B., Gann, H., Brueck, R., Hornyak, M., Litsch, S., Hohagen, F., & Riemann, D. (2006). Effects of alcohol on polysomnographically recorded sleep in healthy subjects. *Alcoholism: Clinical and Experimental Research*, *30*, 1527-1537.

Feltin, M., & Broughton, R. J. (1968). Differential effects of arousal from slow wave sleep and REM sleep. *Psychophysiology*, *5*, 231.

Ferrara, M., Curcio, G., Fratello, F., Moroni, F., Marzano, C., Pellicciari, M. C., & Gennaro, L. D. (2006). The electroencephalographic substratum of the awakening. *Behavioural Brain Research*, *167*, 237-244.

Ferrara, M., De Gennaro, L., & Bertini, M. (2000a). Time-course of sleep inertia upon awakening from nighttime sleep with different sleep homeostasis conditions. *Aviation, Space, and Environmental Medicine*, *71*, 225-229.

Ferrara, M., De Gennaro, L., Casagrande, M., & Bertini, M. (2000b). Selective slow-wave sleep deprivation and time-of-night effects on cognitive performance upon awakening. *Psychophysiology*, *37*, 440-446.

Fillmore, M. T., Dixon, M. J., & Schweizer, T. A. (2000). Differential effects of alcohol on responses to negatively and positively primed stimuli. *Journal of Studies on Alcohol*, *61*, 872-880.

Fillmore, M. T., Marczinski, C. A., & Bowman, A. M. (2005). Acute tolerance to alcohol effects on inhibitory and activational mechanisms of behavioral control. *Journal of Studies on Alcohol*, *66*, 663-672.

Fillmore, M. T., & Vogel-Sprott, M. (1999). An alcohol model of inhibitory control and its treatment in humans. *Experimental and Clinical Psychopharmacology, 7*, 49-55.

Finn, P. R., Justus, A., Mazas, C., & Steinmetz, J. E. (1999). Working memory, executive processes and the effects of alcohol on go/no-go learning: Testing a model of behavioral dysregulation and impulsivity. *Psychopharmacology, 146*, 465-472.

Folkard, S., & Åkerstedt, T. (1992). A three-process model of the regulation of alertness-sleepiness. In R. J. Broughton and R. D. Ogilvie (Eds.), *Sleep, arousal, and performance* (pp. 11-26). Boston: Birkhäuser.

Foret, J. (1992). Sleep-schedule shift: Influence on alertness and performance. In R. J. Broughton and R. D. Ogilvie (Eds.), *Sleep, arousal and performance* (pp. 164-175). Boston: Birkhauser.

Fort, A., & Mills, J. N. (1972). Influence of sleep, lack of sleep and circadian rhythm on short psychometric tests. In W. P. Colquhoun (Ed.), *Aspects of human efficiency* (pp. 115-127). New York: Plenum Press.

Frey, D. J. (2008). The influence of sleep inertia on human performance: Individual differences, aging, hypnotics, cognition and balance. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, *68*, 4295.

Gengo, F. M., Gabos, C., & Straley, C. (1990). The pharmacodynamics of ethanol: Effects on performance and judgment. *Journal of Clinical Pharmacology, 30*, 748-754.

Gillberg, M., Kecklund, G., & Åkerstedt, T. (1994). Relations between performance and subjective ratings of sleepiness during a night awake. *Sleep, 17*, 236-241.

Graham, K., Leonard, K. E., Room, R., Wild, T. C., Pihl, R. O., Bois, C., & Single, E. (1998). Current directions in research on understanding and preventing intoxicated aggression. *Addiction, 93,* 659-676.

Grantcharov, T. P., Bardram, L., Funch-Jensen, P., & Rosenberg, J. (2001). Laparoscopic performance after one night on call in a surgical department: Prospective study. *British Medical Journal, 323,* 1222-1223.

Gravetter, F. J. (2006). *Research methods for the behavioural sciences*. Belmont, CA: Thomson/Wadsworth.

Gresham, S. C., Webb, W. B., & Williams, R. L. (1963). Alcohol and caffeine: Effect on inferred visual dreaming. *Science, 140,* 1266.

Hajak, G., Klingerlhofer, J., Schulz-Varszegi, M., Matzander, G., Sander, D., Conrad, B., & Rüther, E. (1994). Relationship between cerebral blood flow velocities and cerebral electrical activity in sleep. *Sleep, 17,* 11-19.

Hannay, H. J., Howieson, D. B., Loring, D. W., Fischer, J. S., & Lezak, M. D. (2004). Neuropathology for neuropsychologists. In M. D. Lezak, D. B. Howieson and D. W. Loring (Eds), *Neuropsychological Assessment (4th edn)* (pp. 157-285). New York: Oxford University Press.

Hartman, B. O., & Langdon, D. E. (1965). *A second study on performance upon sudden awakening* (School of Aerospace Medicine Report No. TR-65-61). Brooks, TX: US Air Force.

Haslam, D. R. (1982). Sleep loss, recovery sleep, and military performance. *Ergonomics, 25,* 163-187.

Haslam, D. R. (1985). Sleep deprivation and naps. *Behavior Research Methods, Instruments & Computers, 17,* 46-54.

Hinton-Bayre, A. D., & Geffen, G. (2005). Comparability, reliability, and practice effects on alternate forms of the Digit Symbol Substitution and Symbol Digit Modalities Tests. *Psychological Assessment, 17,* 237-241.

Hinton-Bayre, A. D., Geffen, G., & McFarland, K. (1997). Mild head injury and speed of information processing: A prospective study of professional rugby league players. *Journal of Clinical and Experimental Neuropsychology, 21,* 70-86.

Hinton-Bayre, A. D., Geffen, G., Geffen, L. B., McFarland, K. A., & Friis, P. (1999). Concussion in contact sports: Reliable change indices of impairment and recovery. *Journal of Clinical and Experimental Neuropsychology, 21,* 70-86.

Horne, J.A., & Pettitt, A.N. (1985). High incentive effects on vigilance performance during 72 hours. *Acta Psychologica, 58,* 123-139.

Hofer-Tinguely, G., Achermann, P., Landolt, H-P., Regel, S. J., Rétey, J. V., Dürr, R., Borbély, A. A., & Gottselig, J. M. (2005). Sleep inertia: Performance changes after sleep, rest and active waking. *Cognitive Brain Research, 22,* 323-331.

Hood B. & Bruck D. (1996). Sleepiness and performance in narcolepsy. *Journal of Sleep Research*, 5, 128-134.

Horne, J. A., & Ostberg, O. (1977). Individual differences in human circadian rhythms. *Biological Psychology, 5,* 179-190.

Hou, Y., Huangfu, E., Zhang, L., & Miao, D. (2007). Changes in cognition and mood due to sleep inertia after 30-hour sleep deprivation. *Internet Journal of Mental Health, 4*.

Howard, M. E., Jackson, M. L., Kennedy, G. A., Swann, P., Barnes, M., & Pierce, R. J. (2007). The interactive effects of extended wakefulness and low-dose alcohol on simulated driving and vigilance. *Sleep, 30*, 1334-1340.

Hurst, P. M., & Bagley, S. K. (1972). Acute adaptation to the effects of alcohol. *Quarterly Journal of Studies on Alcohol, 33*, 358-378.

Jääskeläinnen, I. P., Schröger, E., & Näätänen, R. (1999). Electrophysiological indices or acute effects of ethanol on involuntary attention shifting. *Psychopharmacology, 141*, 16-21.

Jeanneret, P. R., & Webb, W. B. (1963). Strength of grip on arousal from full night's sleep. *Perceptual and Motor Skills, 17*, 759-761.

Jewett., M. E., & Kronauer, R. E. (1999). Interactive mathematical models of subjective alertness and cognitive throughput in humans. *Journal of Biological Rhythms, 14*, 588-597.

Jewett, M. E., Wyatt, J. K., Ritz-de Cecco, A., Bir Khalsa, S., Dijk, D-J., & Czeisler, C. A. (1999). Time course of sleep inertia dissipation in human performance. *Journal of Sleep Research, 8*, 1-8.

Johnson, M. P., Duffy, J. F., Dijk, D. J., Ronda, J. M., Dyal, C. M., & Czeisler, C. A. (1992). Short-term memory, alertness and performance: A reappraisal of their relationship to body temperature. *Journal of Sleep Research, 1*, 24-29.

Jones, B. M. (1973). Memory impairment on the ascending and descending limbs of the blood alcohol curve. *Journal of Abnormal Psychology, 82*, 24-32.

Jones, J. C. (1983). Multiple death fires in the United States. *Fire Journal, 77*, 10-25.

Kaida, K., Takahashi, M., Åkerstedt, T., Nakata, A., Otsuka, Y., Haratani, T., & Fukasawa, K. (2006). Validation of the Karolinska sleepiness scale against performance and EEG variables. *Clinical Neurophysiology, 117*, 1574-1581.

Kalat, J. W. (2001). *Biological psychology (7th edn.)*. Belmont, CA: Wadsworth/Thompson.

Karlamangla, A. S., Singer, B. H., Chodosh, J., McEwen, B. S., & Seeman, T. E. (2005). Urinary cortisol excretion as a predictor of incident cognitive impairment. *Neurobiology of Aging, 26*, 80-84.

Karter, M. J. (1986). Patterns of fire deaths among the elderly in the home. *Fire Journal, March,* 19-22.

Klerman, E. B., & St. Hilaire, M. (2007). On mathematical modeling of circadian rhythms, performance, and alertness. *Journal of Biological Rhythms*, *22*, 91-102.

Knowles, J. B., Laverty, S. G., & Kuechler, H. A. (1968). Effects of alcohol on REM sleep. *Quarterly Journal of Studies on Alcohol*, *29*, 342.

Koelega, H. S., & Brinkman, J. A. (1986). Noise and vigilance: An evaluative review. *Human Factors*, *28*, 465-481.

Kolff, M., Hofman, W., Kerkhof, G., & Coenen, A. (2003). The time course of sleep inertia in a semantic priming paradigm. *Sleep and Hypnosis*, *5*, 78-82.

Koulack, D., & Shultz, K. J. (1974). Task performance after awakenings from different sleep stages. *Perceptual and Motor Skills, 39*, 792-794.

Kräuchi, K., Cajochen, C., & Wirz-Justice, A. (2004). Waking up properly: Is there a role of thermoregulation in sleep inertia? *Journal of Sleep Research, 13*, 121-127.

Krull, K. R., Smith, L. T., Sinha, R., & Parsons, O. (1993). Simple reaction time event-related potentials: Effects of alcohol and sleep deprivation. *Alcoholism: Clinical and Experimental Research, 17*, 771-777.

Kuboyama, T., Hori, A., Sato, T., Mikami, T., Yamaki, T., & Ueda, S. (1997). Changes in cerebral blood flow velocity in healthy young men during overnight sleep and while awake. *Electroencephalography and Clinical Neurophysiology, 102*, 125-131.

Langdon, D. E., & Hartman, B. O. (1961). *Performance upon sudden awakening* (School of Aerospace Medicine Report No. 62-17). Brooks, TX: US Air Force.

Lavie, P., & Weler, B. (1989). Timing of naps: Effects on post-nap sleepiness levels. *Electroencephalography and Clinical Neurophysiology, 72*, 218-224.

Leistikow, B. N., Martin, D. C., & Milano, C. E. (2000). Fire injuries, disasters, and costs from cigarettes and cigarette lights: A global overview. *Preventive Medicine, 31*, 91-99.

Lex, B. W., Rhoades, E. M., Teoh, S. K., & Mendelson, J. H. (1994). Divided attention task performance and subjective effects following alcohol and placebo: Differences between women with and without a family history of alcoholism. *Drug & Alcohol Dependence, 35*, 95-105.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment (4th edn.)*. New York: Oxford University Press.

Lorenzo, J-L., & Barbanoj, M-J. (2002). Variability of sleep parameters across multiple laboratory sessions in healthy young subjects: The "very first night effect". *Psychophysiology, 39*, 409-413.

Lubin, A., Hord, D., Tracy, M. L., & Johnson, L. C. (1976). Effects of exercise, bedrest and napping on performance decrement during 40 hours. *Psychophysiology, 13*, 334-339.

Lupien, S. J., de Leon, M., de Santi, S., Convit, A., Tarshish, C., Nair, N. P., et al. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nature Neuroscience, 1*, 69-73.

Lukas, S. E., Mendelson, J. H., Kouri, E., Bolduc, M., & Amass, L. (1990). Ethanol-induced alterations in EEG alpha activity and apparent source of the auditory P300 evoked response potential. *Alcohol, 7*, 471-477.

Mallis, M. M., Banks, S., & Dinges, D. F. (2007). Sleep and circadian control of neurobehavioral functions. In R. Parasuraman and M. Rizzo (Eds.), *Neuroergonomics: The brain at work* (pp. 207- 220). New York: Oxford University Press.

Maloney, T. R. (2001). Electrophysiological, behavioral and subjective assessments of slow-wave sleep inertia after forced awakenings from the first two NREM-REM cycles. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 62*, 595.

Marshall, S.W., Runyan, C. W., Bangdiwala, S. I., Linzer, M. A., Sacks, J. J. & Butts, J. (1998). Fatal residential fires. Who dies and who survives? *Journal of the American Medical Association, 279*, 1633-1637.

Matchock, R. L., & Mordkoff, J. T. (2007). Visual attention, reaction time, and self-reported alertness upon awakening from sleep bouts of varying lengths. *Experimental Brain Research, 178,* 228-239.

McEwen, B. S., Weiss, J. M., & Schwartz, L. S. (1968). Selective retention of corticosterone by limbic structures in rat brain. *Nature, 220,* 911-912.

Merica, H., & Fortune, R. D. (2004). State transitions between wake and sleep, and within the ultradian cycle, with focus on the link to neuronal activity. *Sleep Medicine Reviews, 8,* 473-485.

Meyer, J., Ishikawa, Y., Hata, T., & Karacan, I. (1987). Cerebral blood flow in normal and abnormal sleep and dreaming. *Brain Cognition, 6,* 266-294.

Miccoli, L., Versace, F., Koterle, S., & Cavallero, C. (2008). Comparing sleep-loss sleepiness and sleep inertia: Lapses make the difference. *Chronobiology International, 25,* 725-744.

Mullin, F. J., Kleitman, N., & Cooperman, N. R. (1933). Studies on the physiology of sleep: The effect of alcohol and caffeine on motility and body temperature during sleep. *American Journal of Physiology, 106,* 478–487.

Mullington, J., & Broughton, R. (1994). Daytime sleep inertia in narcolepsy-cataplexy. *Sleep, 17,* 69-76.

Mumenthaler, M. S., Taylor, J. L., O'Hara, R., & Yesavage, J. A. (1999). Gender differences in moderate drinking effects. *Alcohol Research and Health, 23,* 55-61.

Naitoh, P. (1981). Circadian cycles and restorative power of naps. In L. C. Johnson, D. I. Tepas, and P. Colquhoun (Eds.), *Biological rhythms, sleep and shiftwork* (pp. 553-580). New York: Spectrum.

Naitoh, P. (1992). Minimal sleep to maintain performance: The search for sleep quantum in sustained operations. In C. Stampi (Ed.), *Why we nap* (pp. 199-216). Boston: Birkhäuser.

Naitoh, P., Kelly, T., & Babkoff, H. (1993). Sleep inertia: Best time not to wake up? *Chronobiology International, 10*, 109-118.

National Institute on Alcohol Abuse and Alcoholism (1997). *Alcohol alert: Alcohol metabolism*. No. 35, PH 371. Bethesda, MD: The Institute.

Newcomer, J. W., Selke, G., Melson, A. K., Hershey, T., Craft, S., Richards, K., et al. (1999). Decreased memory performance in healthy humans induced by stress level cortisol treatment. *Archives of General Psychiatry, 56*, 527-533.

Nicholson, M. E., Wang, M., Airhihenbuwa, C. O., Mahoney, B. S., Christina, R., & Maney, D. W. (1992). Variability in behavioral impairment involved in the rising and falling BAC curve. *Journal of Studies on Alcohol, 53*, 349-356.

Nolte, J. (2002). *The human brain: An introduction to its functional anatomy (5th edn.)*. St. Louis, Missouri: Mosby.

Ogilvie, R. D., & Simons, I. (1992). Falling asleep and waking up: A comparison of EEG spectra. In R. J. Broughton and R. D. Ogilvie (Eds.), *Sleep, arousal and performance* (pp. 164-175). Boston: Birkhauser.

Patetta, M. J. & Cole T. B., (1990). A population-based descriptive study of house fire deaths in North Carolina. *American Journal of Public Health, 80,* 1116-1117.

Peeke, S. C., Callaway, E., Jones, R. T., Stone, G. C., & Doyle, J. (1980). Combined effects of alcohol and sleep deprivation in normal young adults. *Psychopharmacology*, *67*, 279-287.

Petros, T. V. (1985). The effects of alcohol on prose memory. *Physiology and Behavior*, *35*, 43-46.

Pihl, R. O., Paylan, S. S., Gentes-Hawn, A., & Hoaken, P. N. S. (2003). Alcohol affects executive functioning differentially on the ascending versus descending limb of the blood alcohol concentration curve. *Alcoholism: Clinical and Experimental Research*, *27*, 773-779.

Ponsford, J., & Kinsella, G. (1992). Attentional deficits following closed head injury. *Journal of Clinical and Experimental Neuropsychology*, *14*, 822-838.

Porter, N. M., & Landfield, P. W. (1998). Stress hormones and brain aging: Adding injury to insult. *Nature Neuroscience*, *1*, 3-4.

Poulton, E. C. (1976). Arousing environmental stresses can improve performance, whatever people say. *Aviation, Space & Environmental Medicine*, *47*, 1193-1204.

Rabbitt., P., Diggle, P., Smith, D., Holland, F., & McInnes L. (2001). Identifying and separating the effects of practice and of cognitive ageing during a large longitudinal study of elderly community residents. *Neuropsychologia*, *39*, 532-543.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects.* Bethesda: U.S. National Institute of Neurological Diseases and Blindness Neurological Information Network.

Ribak, J., Ashkenazi, I. E., Klepfish, A., Avgar, D., Tall, J., Kallner, B., & Noyman, Y. (1983). Diurnal rhythmicity and air force flight accidents due to pilot error. *Aviation, Space & Environmental Medicine*, *54*, 1096-1099.

Roehrs, T., Burduvali, E., Bonahoom, A., Drake, C., & Roth, T. (2003). Ethanol and sleep loss: a "dose" comparison of impairing effects. *Sleep*, *26*, 981-985.

Rosa, R. R., & Bonnet, M. H. (1985). Sleep stages, auditory arousal threshold, and body temperature as predictors of behavior upon awakening. *International Journal of Neurosciences*, *27*, 73-83.

Rosa, R. R. , Bonnet, M. H., & Warm, J. S. (1983). Recovery of performance during sleep following sleep deprivation. *Psychophysiology*, *20*, 152-159.

Rosenwasser, A. M. (2001). Alcohol, antidepressants and circadian rhythms: Human and animal models. *Alcohol Research and Health*, *25*, 126-135.

Rundell, O. H., Lester, B. K., Griffiths, W. J., & Williams, H. J. (1972). Alcohol and sleep in young adults. *Psychopharmacologia*, *26*, 201-218.

Runyan, C. W., Bangdiwala, S. I., Linzer, M. A., Sacks, J. J. & Butts, J. (1992). Risk factors for fatal residential fires. *The New England Journal of Medicine, 327*, 859-863.

Salamé, P., Otzenberger, H., Ehrhart, J., Asmes, G. D., Nicolas, A., Tassi, P., Libert, J-P., & Muzet, A. (1995). Effects of sleep inertia on cognitive performance following a 1-hour nap. *Work & Stress, 9*, 528-539.

Sallinen, M., Härmä, M., Åkerstedt, T., Rosa, R., & Lillqvist, O. (1998). Promoting alertness with a short nap during a night shift. *Journal of Sleep Research, 7,* 240-247.

Sano, M., Wendt, P. E., Wirsén, A., Stenberg, G., Risberg, J., & Ingvar, D. H. (1993). Acute effects of alcohol on regional cerebral blood flow in man. *Journal of Studies on Alcohol, 54,* 369-376.

Scheer, F. A. J. L., Shea, T. J., Hilton, M. F., & Shea, S. A. (2008). An endogenous circadian rhythm in sleep inertia results in greatest cognitive impairment upon awakening during the biological night. *Journal of Biological Rhythms, 23,* 353-361.

Schulz, H., & Salzarulo, P. (1997). The relationship between sleep and cognitive functions during wakefulness: An introduction. *Acta Neurologica Belgica, 97,* 93-95.

Schweizer, T. A., Jolicœur, P., Vogel-Sprott, M., & Dixon, M. T. (2004). Fast, but error prone responses, during acute alcohol intoxication: Effects of stimulus response mapping complexity. *Alcoholism: Clinical & Experimental Research, 28,* 643-649.

Schweizer, T. A., & Vogel-Sprott, M. (2008). Alcohol-impaired speed and accuracy of cognitive functions: A review of acute tolerance and recovery of cognitive performance. *Experimental and Clinical Psychopharmacology, 16,* 240-250.

Schweizer, T. A., Vogel-Sprott, M., Danckert, J., Roy, E. A., Skakum, A., & Broderick, C. E. (2006). Neuropsychological profile of acute alcohol intoxication during ascending and descending blood alcohol concentrations. *Neuropsychopharmacology, 31,* 1301-1309.

Sekizawa, A. (1991). Statistical analyses on fatalities characteristics of residential fires. In Fire Safety Science- *Proceedings of the Third International Symposium*. London: Elsiever.

Seminara, J. L., & Shavelson, R. J. (1969). Effectiveness of space crew performance subsequent to sudden sleep arousal. *Aerospace Medicine, 40*, 723-727.

Sher, K. J. (1985). Subjective effects of alcohol: the influence of setting and individual differences in alcohol expectancies. *Journal of Studies on Alcohol, 46*, 137-146.

Silva, E. J., & Duffy, J. F. (2008). Sleep inertia varies with circadian phase and sleep stage in older adults. *Behavioral Neuroscience, 122*, 928-935.

Skosnik, P. D., Chatterton, R. T., Swisher, T., & Parks, S. (2000). Modulation of attentional inhibition by norepinephrine and cortisol after psychological stress. *International Journal of Psychophysiology, 36*, 59-68.

Sjögren, H., Eriksson, A., & Ahlm, K. (2000). Role of alcohol in unnatural deaths: A study of all deaths in Sweden. *Alcoholism in Clinical and Experimental Research, 24,* 1050-1056.

Squires, T. & Busuttil, A. (1997). Alcohol and house fire fatalities in Scotland, 1980 – 1990. *Medicine, Science & Law, 37,* 321-325.

Stampi, C., & Davis, B. (1991). Forty-eight days on the "Leonardo da Vinci" strategy for sleep reduction: Performance behaviour with three hours polyphasic sleep per day. *Sleep Research, 20,* 471.

Stampi, C., Mullington, J., Rivers, M., Campos, J., & Broughton, R. (1990). Ultrashort sleep schedules: Sleep architecture and recuperative value of 80-, 50- and 20-min naps. In J. Horne (Ed.), *Sleep '90* (pp. 71-74). Bochum: Pontenagel Press.

Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia: it's prized and dangerous effects. *American Psychologist, 45,* 921-933.

Stones, M. J. (1977). Memory performance after arousal from different sleep stages. *British Journal of Psychology, 68,* 177-181.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary (3rd ed.).* New York: Oxford University Press.

Tabachnick, B. G., & Fidell, L. S. (2001). *Using multivariate statistics* (4th ed.). Boston, MA: Allyn & Bacon.

Taffinder, N. J., McManus, I. C., Gul, Y., Russel, R. C., & Darzi, A. (1998). Effect of sleep deprivation on surgeons' dexterity on laparoscopy simulator. *Lancet, 352,* 1191.

Tamaki, M., Nittono, H., Hayashi, M., & Hori, T. (2005). Examination of the first-night effect during the sleep-onset period. *Sleep, 28,* 192-202.

Tassi, P., Bonnefond, A., Engasser, O., Hoeft, A., Eschenlauer, R., & Muzet, A. (2006). EEG spectral power and cognitive performance during sleep inertia: The effect of normal sleep duration and partial sleep deprivation. *Physiology & Behavior, 87,* 177-184.

Tassi, P., Bonnefond, A., Hoeft, A., Eschenlauer, R., & Muzetand, A. (2003). Arousal and vigilance: Do they differ? Study in a sleep inertia paradigm. *Sleep Research Online, 5,* 83-87.

Tassi, P., & Muzet, A. (2000). Sleep inertia. *Sleep Medicine Reviews, 4,* 341-353.

Tassi, P., Nicolas, A., Dewasmes, G., Eschenlauer, R., Ehrhart, J., Salame, P., Muzet, A., & Libert, J. P. (1992). Effects of noise on sleep inertia as a function of circadian placement of a one-hour nap. *Perceptual and Motor Skills, 75*, 291-302.

Tebbs, R. B., & Foulkes, D. (1966). Strength of grip following different stages of sleep. *Perceptual and Motor Skills, 23*, 827-834.

Thorne, O. R., Genser, S. G., Sing, H. C., & Hegge, F. W. (1985). The Walter Reed performance assessment battery. *Neurobehavioral Toxtcology & Teratology,* 7, 415-418.

Tietzel, A. J., & Lack, L. C. (2001). The short-term benefits of brief and long naps following nocturnal sleep restriction. *Sleep, 24*, 293-300.

Uchiyama, C. L., D'Elia, L. F., Delinger, A. M., Selnes, O. A., Becker, J. T., Wesch, J. E., Chen, B. B., Satz, P., Van Gorp, W., & Miller, E. N. (1994). Longitudinal comparisons of alternate versions of the Symbol Digit Modalities Test: Issues of form comparability and moderating demographic variables. *The Clinical Neuropsychologist, 8*, 209-218.

Vakulin, A., Baulk, S. D., Catcheside, P. G., Anderson, R., van den Heuvel, C. J., Banks, S., & McEvoy, R. D. (2007). Effects of moderate sleep deprivation and low-dose alcohol on driving simulator performance and perception in young men. *Sleep, 30*, 1327-1333.

Van, F., O'Boyle, D. J., & Hume, K. I. (1995). Effects of alcohol on the sleep-stage structure of a nap in the afternoon. *Biological Psychology, 41*, 55-59.

Van Reen, E., Jenni, O. G., & Carskadon, M. A. (2006). Effects of alcohol on sleep and the sleep electroencephalogram in healthy young women. *Alcoholism: Clinical and Experimental Research, 30*, 974-981.

Vedhara, K., Hyde, J., Gilchrist, I. D., Tytherleigh, M., & Plummer, S. (2000). Acute stress, memory, attention and cortisol. *Psychoneuroendocrinology, 25*, 535-549.

Wall, T. L., & Ehlers, C. L. (1995). Acute effects of alcohol on P300 in Asians with different ALDH2 genotypes. *Alcoholism: Clinical and Experimental Research, 19*, 617-622.

Watts-Hampton, T., Bruck, D., & Ball, M. (2007). Examination of risk factors and mental health status in an adult accidental fire death population 1998 – 2005. In Fire Safety Science- *Proceedings of the 7th Asia-Oceania Symposium*. Hong Kong: Asia-Oceania Association for Fire and Technology.

Webb, W. B., & Agnew, H. (1964). Reaction time and serial response efficiency on arousal from sleep. *Perceptual and Motor Skills, 18*, 783-784.

Wertz, A. T., Wright, K. P., Ronda, J. M., & Czeisler, C. A. (2006). Effects of sleep inertia on cognition. *Journal of the American Medical Association, 295*, 163-164.

Wilkinson, R. T., & Colquhoun, W. P. (1968). Interaction of alcohol with incentive and with sleep deprivation. *Journal of Experimental Psychology, 76*, 623-629.

Wilkinson, R. T., & Stretton, M. (1971). Performance after awakening at different times of night. *Psychonom. Science, 23*, 283-285.

Williams, N. L., & Lubin, A. (1967). Speeded addition and sleep loss. *Journal of Experimental Psychology,* 73, 313-317.

Williams, H., & Salamy, A. (1972). Alcohol and sleep. In B. Kissin and H. Begleiter (Eds.), *The Biology of Alcoholism* (pp. 435-483). New York: Plenum Press.

Young, J. R. (1970). Blood alcohol concentration and reaction time. *Quarterly Journal of Studies on Alcohol, 31*, 823-831.

Yules, R. B., Freedman, D. X., & Chandler, K. A. (1966). The effect of ethyl alcohol on man's electroencephalographic sleep cycle. *Electroencephalography & Clinical Neurophysiology, 20*, 109-111.

Yules, R. B., Lippman, M. E., & Freedman, D. X. (1967). Alcohol administration prior to sleep: The effect on EEG sleep stages. *Archives of General Psychiatry, 19*, 94-97.

Zavada, A., Strijkstra, A. M., Boerema, A. S., Daan, S., & Beersma, D. G. M. (2009). Evidence for differential human slow-wave activity regulation across the brain. *Journal of Sleep Research, 18*, 3-10.

Appendix A: Instructions for the Descending Subtraction Task and Examiners Record Form

TASK ONE: DESCENDING SUBTRACTION TASK

MATERIALS: *STOP WATCH, EXAMINERS RECORD FORM, PENCIL, MP3 RECORDER*
TIMING: 3 MINUTES

Say..
"You may remember the subtraction task that you have performed a few times before. I will give you a three-digit number which you are to repeat aloud as your first response. Then I would like you to subtract 9 from this number, performing the calculation in your mind, but saying the answer out loud. Then subtract 8 from this answer, then subtract 7 from the next answer, then subtract 6, etc., until you are subtracting the number 2, and then go back to subtracting 9 and repeat the sequence again. Each time you give me an answer, that is the number that you will be subtracting from."

"Would you like an example?"*

"It is important that you work as fast as possible and keep a steady pace, but try to remain as accurate as possible. Do you have any questions?"

"Okay, ready? The first number is _____, begin by repeating _____"

**If the participant asks for an example, say the following:*
"If the first number is 450, you will start by repeating the number 450 aloud. You will then subtract 9 from this number and give the response → 441. You will then subtract 8 from 441, which is 433. *[pause]* 433 minus 7 is 426, *[pause]* 426 minus 6 is 420, and so on. Once you have progressed down to subtracting the number 2, you then go back to subtracting the number 9 again. This sequence will be repeated for a period of 3 minutes."
Begin timing now. Begin recording responses with MP3 now. Begin recording responses on examiners record form now.
Stop recording/timing after 3 minutes.

If the participant becomes lost in a sequence or does not respond for 20 seconds (whichever occurs first) say:
"Please continue, guess if you have to"
and urge them to continue every few seconds if they do not respond.

If participant self-corrects a wrong response, record both answers on the examiners record form, indicating that it is a self-correction by placing an 'x' next to the initial wrong response. Do not correct wrong responses.

DESCENDING SUBTRACTION TASK: EXAMINERS RECORD FORM

TIMING: 3 MINUTES *MP3 PLAYER/SET (PLEASE CIRCLE): A B MP3 file #:* _____

Participant ID: _____ ****** please specify on MP3 audio recording the participant's ID, the night, and the condition (e.g., baseline, baseline 0.05, or sleep inertia)

_____	↓	_____	_____	_____
_____	↓	_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____
_____		_____	_____	_____

Please write any relevant behavioural observations in the space below.

Appendix B: Modified Descending Subtraction Task Instructions

TASK ONE: DESCENDING SUBTRACTION TASK

MATERIALS: *STOP WATCH, EXAMINERS RECORD FORM, PENCIL, MP3 RECORDER*

TIMING: 3 MINUTES

Once the participant has indicated wakefulness by pressing the bedside button, walk into the room and leave the door open to allow some illumination of light from the adjacent room. Allow the participant to remain supine in bed. Say..

"I would like you to do the subtraction task again. The first number is _____, begin by repeating _____"

Begin timing now. Begin recording responses with MP3 now. Begin recording responses on examiners record form now.
Stop recording/timing after 3 minutes.

If necessary, remind the participant that the subtraction sequence goes from 9 to 2, and then back to 9 again. Say "Remember that the number which you are to subtract decreases by 1 until you reach the number 2, then go back to subtracting 9 and repeat the sequence again. Each time you give me an answer, that is the number that you will be subtracting from."

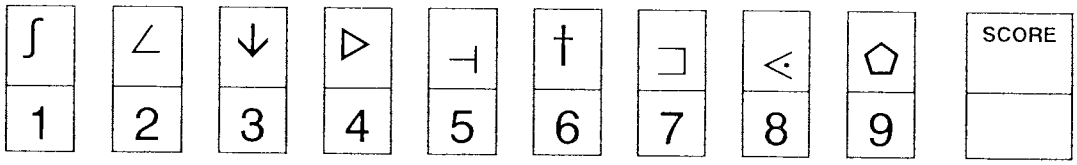
If the participant becomes lost in a sequence or does not respond for 20 seconds (whichever occurs first) say:
"Please continue, guess if you have to"
and urge them to continue every few seconds if they do not respond.

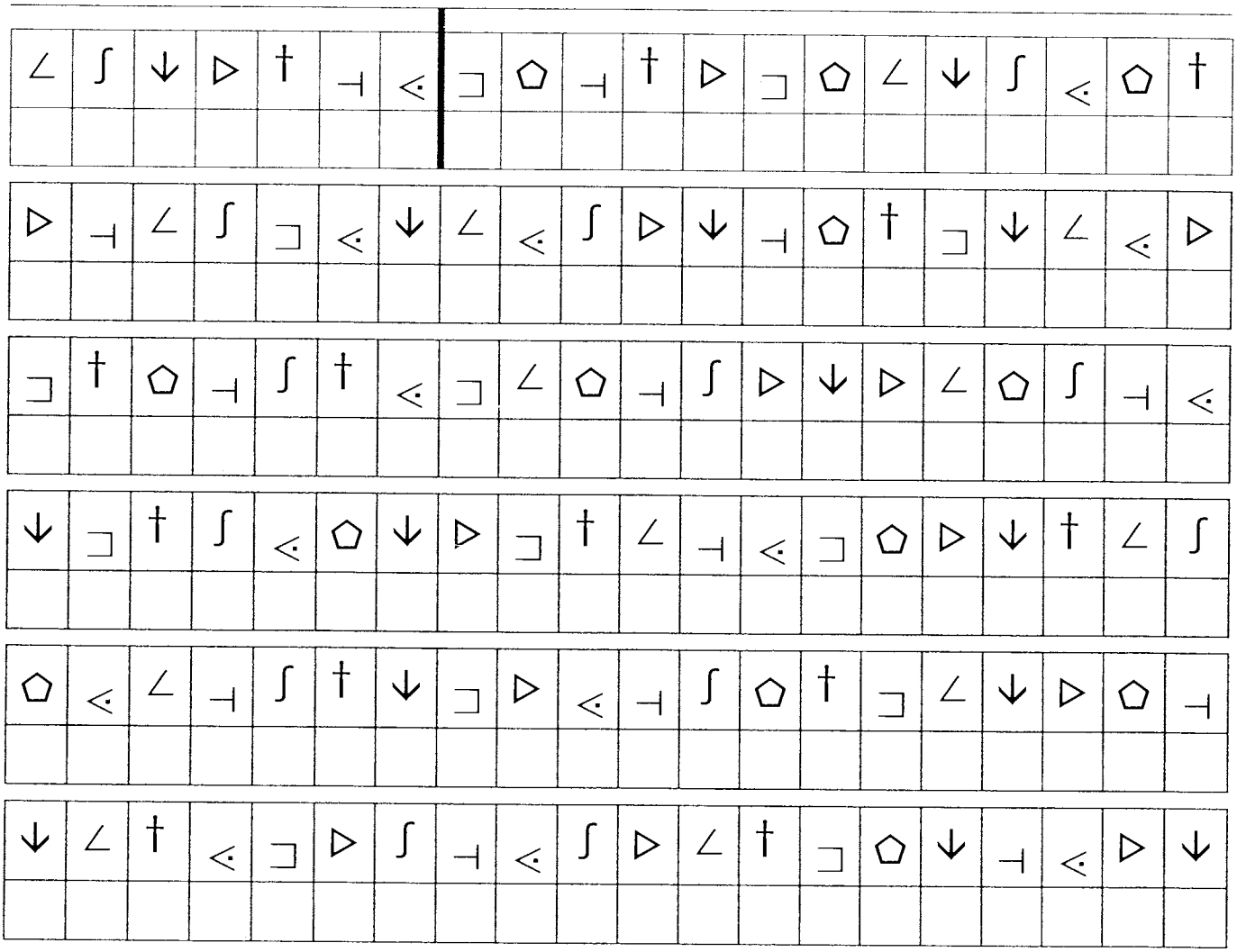
If participant self-corrects a wrong response, record both answers on the examiners record form, indicating that it is a self-correction by placing an 'x' next to the initial wrong response. Do not correct wrong responses.

Please indicate the time the Descending Subtraction Task/the testing block began (see response sheet), so that the 10-minute period for the first block can be accurately timed.

Appendix C: Symbol Digit Substitution Task Example Form

SYMBOL-DIGIT SUBSTITUTION – 3a





ID:_____

Date:_____

Condition:_____

Trial:_____

Appendix D: Letter Cancellation Test Form

LETTER CANCELLATION TASK

Below is a matrix of random capital letters. Search for the letters ..... and ....., and mark these letters by drawing a line through them. Work as quickly and as accurately as possible, making sure that you search sequentially from left to right and from top to bottom.

D H A T Z J L U X S P F T N H O G B P X L O J S H A F U C B G Z
P L S R U T G B Z A H X U H D O N C Z A P D O B R J T S N F C L
C Z R L B U D H F J T A N S P R O X C U H X B R J T G F L P D Z
N A X J S B H C L U P G R T O D N Z F O T P U J B X Z R D L C G
A L S B F J Z U G X C P T H D O R X N G J B U Z F P A D O C R N
B C P U G F A H N Z R X D S O N L T O D L A R S F J G B X P N T
Z R J F S X L N G D O U P T H U B S H F U X D Z J A C S N B L G
U Z D J P H S T R F O L G X B A J C X U H R S F B N P L O D G T
F H S C X P J L A Z B D T G R N U O P F L O S G X Z H A R A N J
O H D S P C Z B J T A X N R C U G F C X B U N Z O F L J A G D C
A P C S U G T O R B F Z D X N J L H F N Z G A S B X S D O U L T
H U Z X F R P D A L J G T L C T S O N G A J X O R C T F D P H S
F G H C A N U O S Z P F J Z H D R T L R A T G C X S P O N U B J
N G J B U Z F P A D X C R N T S L H S H Z J C U B O X L G A T R
T B Z G P O D A Z R U N C H J L F X T O B F P J C S A X Z U R H
X R N L S T P Z B A J C O F H G D U S H J F U X L N G D O C P T
A S T N P Z G C H B U X J L O F R N G D O U R Z P X S F B D J T
P T L O J S H A F U C B G Z N R X D C S F R G D N O P A J U L B
B C T L Z P F H U N J F R A O G D S R X T N J S H B A Z C L O D
S H Z J C U B O X L G A T R H D N P A P C S U G T L R B F Z D X
Z G X U T L F R H A N C O B S D P J L J R C H X F Z N P D B H A
L R A T G C X P D O N U B J S H Z F A C R B L H T N P G F D O J
T O B D P J C S A X Z L R H L G N D P F L O S G X Z H A R U N J
U D O X G Z N C A H P S B R F J T L F G H C A N U X S B P O J Z
C S Z R G D N O P A J U O D F X T H S X G F C A O R B T J Z P U
O T R J F G D U S P X L N C B H Z A D G B F O N R H L C Z U A X
N G A J X Z R F L F D P H S Z B U T A S L N P Z G C H T U X J D
R X C N J S H B A F D L O G P Z T U S L C R J T G B O P H X U F
F B H D N U C O G T S R Z P X J A L B J X L Z P F H U N C T R A
G N O J R Z P U S F B D C T L A U H X R N L S J A Z B T J C O F
A C R B L H T N P G F D O X J P D Z C U Z X F R U D A B J H N L
B A S G Z H O X S P F C R U T L N J T B Z G P O D A S R U N C H
L J R U H X T Z N P D B G A F C S O Z G X D T L F R H U N C O B
S T G F C A O R X T L Z P U D N H J U D O X G B N C A H P S B R
C D Z T P X A L R N J H O F S B G U S G Z J C U B O X L N A T H
H O N R P Z C G U L S X F D A T J X O T R J F G D U S P B L N C
D G B F O S R H L C Z P A X S J T N A B X L Z F T G P O H S D N
P F L O S H X Z H A R U T J D B L C F G D P B T H C N X S R J A
J P S N B O G R U X T F Z H L D X T P Z L T A U A B G N C S D F
F B H D T U L O G T S R Z P X J A L G N O J R Z P X S F B D C T
R X C N J S H B H F D L O G P Z T U C S Z R G D N O P A J U L B
U D O X U Z N C A H P S B R F J T L R Z C N J S H B A F D L O G
T B Z G P O D A S R U N C H J L F X B A D G Z H O X S P F C R U
B J X L Z P F H U N C T R A O G D S A C U B L H T N P G F D J O
A S L N P Z G C H T U X J D O F R B L R A T G C X P D O N U R J

ID:_____

Date:_____

Condition:_____

Trial:_____

Appendix E: Karolinska Sleepiness Scale

KAROLINSKA SLEEPINESS SCALE

10 MINUTE TRIAL **20 MINUTE TRIAL**

HOW DO YOU FEEL RIGHT NOW?

Tick one box.

☐ Extremely sleepy (fighting sleep)

☐

☐ Sleepy but no difficulty remaining awake

☐

☐ Neither alert nor sleepy

☐

☐ Alert

☐

☐ Extremely alert

Appendix F: The Clearheadedness Rating Scale

CLEARHEADEDNESS RATING SCALE

10 MINUTE TRIAL **20 MINUTE TRIAL**

HOW CLEARHEADED DO YOU FEEL RIGHT NOW?

Tick one box.

☐ Extremely

☐ Quite a bit

☐ Moderately

☐ A little

☐ Not at all

Appendix G: Recruitment Advertisement

Victoria University is seeking volunteers for a study on sleep and fire safety.

Our research team has, for several years now, been looking at the question of what smoke alarm signal is the best for waking up people. We have tested children, young people and older people. The results have suggested that the current signal may not be as good as some alternative signals.

We now want to test how well *alcohol impaired individuals* will wake to fire alarm signals.

What's involved?

- **A member of the research team will assess your hearing of various sounds.**
- **A three-night sleep study that will test the ability of various signals (e.g. a range of auditory, visual and tactile signals) to wake you up after having consumed a set amount of alcohol. The sleep study will be conducted in your home or the VU Sleep Laboratory at St Albans campus with a sleep technologist (who has undergone a Police Check).**

Selection criteria for volunteers:

- **Be between 18 and 26 years.**
- **Usually do not have a lot of difficulty getting to sleep.**
- **Not be taking any medication that affects your sleep (e.g. anti-depressants, sleeping tablets).**

Why participate?

- **You will be contributing to research that will develop international standards for emergency notification devices and this will help reduce home fire deaths and injuries.**
- **Generous financial rewards to participants (total of $315).**

How?

- **Contact the project officer, Walter Pfister, on xxxx xxxx or email walter.pfister@students.vu.edu.au**

Appendix H: Participant Information Sheet

Title: Optimising fire alarm notification for individuals under the influence of alcohol

At Victoria University our research team has, for several years now, been looking at the question of what smoke alarm signal is the best for waking up people. We have tested children, young adults (both sober and under the influence of alcohol) and the elderly and the results suggest that the current signal may not be as good as some alternative signals. This is especially important as we know that most fatal fires occur during the time when people are asleep and one in four fatal fires occur despite the presence of an operating smoke alarm. Our most recent study was conducted with young adults and systematically varied the pitch and the pattern of signals in order to try and find the best possible alarm signal based upon what we now know. This process is ongoing, and we would now like to investigate different types of signals including lights or pads placed under the mattress or pillow that shake when there is a fire. We also need to test the best new signals drawn from the pitch and pattern study with people under the influence of alcohol, because drinking alcohol is the single most significant risk factor for death in a fire.

In this study we will be presenting some different signals to volunteers while they are asleep in their own home or the VU Sleep Laboratory at St Albans campus. Equipment will be set up in the bedroom including a pillow shaker, bed shaker, strobe light, and speakers. The signals will be presented softly at first and then getting stronger because we are interested to know how strong each would need to be to wake people up. The strongest signals are still within safe limits. Usually when the volunteer wakes up they will press a button by their bedside three times and then return to sleep. We will be presenting three signals a night and our previous experience suggests that people get very good at returning to sleep quite quickly. We want to always present the signals in the same type of sleep and because sleep changes across the night we will need to monitor the different stages of sleep of our volunteers. This is done by attaching ten small surface electrodes to the face and top of the head. A Sleep Technician (ST) is trained to do this and will present the signals from a hallway next to the bedroom. The gender of the ST will be matched with each participant for security purposes and all STs have passed a Police Check. The study will normally be conducted over three nights, with at least three nights between each individual study night to prevent volunteers being affected too much by sleep deprivation. On the first two nights volunteers will be provided with enough alcohol to obtain a blood alcohol content (BAC) of .05 in the form of vodka mixed with orange juice. Their BAC will be measured using a breathalyser obtained from Victoria Police for the study.

Each volunteer will receive a total of nine signals during their sleep, normally three each night, thus three nights of sleep testing are involved. However, if a person has trouble returning to sleep after the first awakening or some other problem arises, we may need an additional night.

As the study involves disruption to sleep, volunteers need to be aware that they may be sleepier than usual the next day and should be careful not to plan activities where sleepiness may be a problem. In particular the driving of a car should be avoided. This is especially important after nights when alcohol is taken in which case volunteers will be asked to sign an undertaking that they will not drive their car for a period of eight hours after their final drink.

We are also asking all volunteers to moderate their consumption of alcohol immediately prior to a night's testing and on the evening of testing. Also regular

sleep/wake patterns should be maintained at these times to avoid sleep deprivation on the night of testing. Volunteers need to sleep on their own during the testing nights and notify any other members of the household that it is possible their sleep may be disturbed by sounds during the night (ear plugs will be made available on request). The study can be conducted at the VU Sleep Laboratory at St Albans campus for any reason, e.g. if the volunteer or any members of their household are concerned about sleep disturbance to those not participating.

We are also interested in the issue of how groggy people are when they first wake up when they are under the influence of alcohol, compared to when they are sober. To this end we will be asking volunteers to complete a series of pencil and paper tests that measure thinking skills that might be useful in response to a smoke alarm, such as focused attention and problem-solving. They will do these tests before and after they have alcohol on the first night before sleep. They will then be asked to do them again after they wake up for the last time on each of the three nights of testing. At this time we will also ask if they remember incorporating any signals into their dreams and if so, which signal.

Because we realise that being part of our study involves some inconvenience we are paying each volunteer $80 for each night of sleep testing. Because the design of our study makes it important for the same volunteers to complete all three nights we will also be paying a $75 bonus on completion of all three nights. Thus the total payment for participation will be $315. For this project we need volunteers who meet our selection criteria. These are:

1. **Aged from 18 to 26 years (inclusive).**
2. **Believe that they have a normal hearing and pass a hearing screening test for both ears.**
3. **Do not regularly take medication to help them sleep.**
4. **Do not take medication that may interact with alcohol.**
5. **Report that they do not have a sleep disorder and pass some simple questions exploring this.**
6. **Report that they do not normally have difficulty falling asleep.**
7. **Report that they usually drink alcohol at least one night per week.**

Because hearing levels are so important to this study all volunteers are asked to undertake a free hearing screening test. We will arrange this at a time and place that is convenient for you (most likely at a campus of VU).

Your participation in this study will remain confidential and all data relating to your involvement will be identified by ID only. The cross-referencing of ID and name and address will be stored separately and securely.

Thank you for your interest in our research.

Contact regarding participation: XXXXXXXXXXXXXXXXXX

Appendix I: Prior Sleep and Alcohol Consumption Questionnaire

Prior Sleep and Alcohol Consumption Screening Questionnaire

Please complete this questionnaire prior to preparation for the sleep study.

ID Number _____

Please circle one: Night 1 Night 2 Night _____

1. Thinking about your sleep last night, compared to your usual sleep, was it: (please circle one of the options)

Much better than usual
A little better than usual
Same as usual
A little worse than usual
Much worse than usual

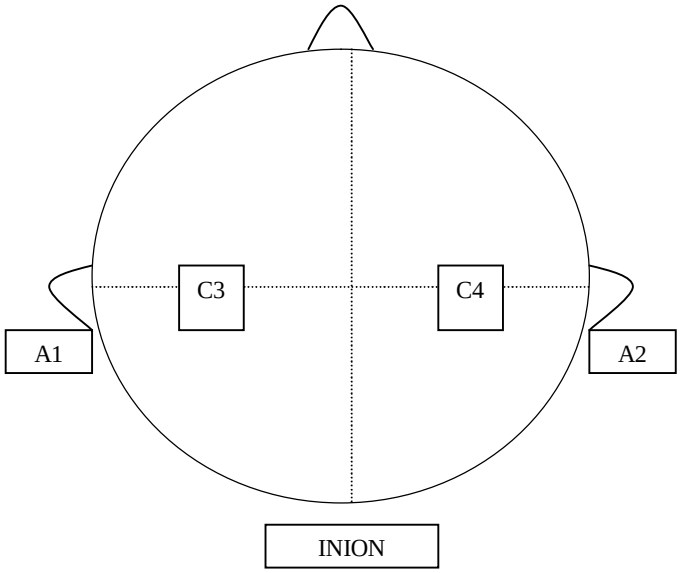
2. If you chose “much worse than usual”, please comment on why your sleep was much worse. (Otherwise leave blank)

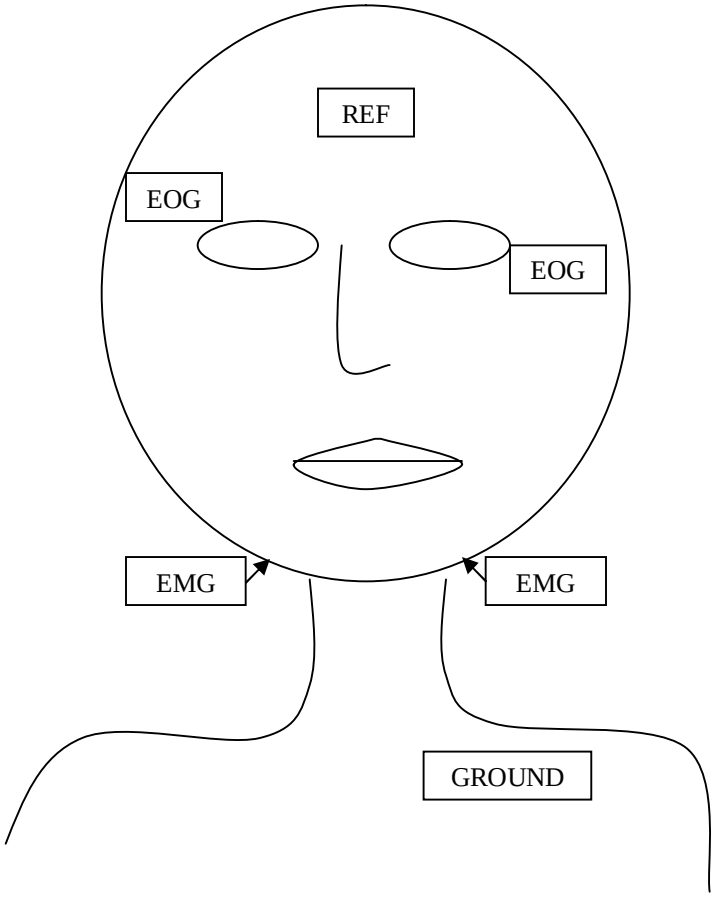
3. Have you consumed any alcohol since 4pm today? If so, please describe the type (beer, wine etc), the quantity and the time of day when it was consumed.

Type:
Quantity:
Time of Day:

In this research we are keen for your sleep to be as similar as possible on the different nights of the study. Two factors that can especially affect your ability to wake up are
If you are quite sleepy from having had poor sleep on the previous night, or, if you have consumed more than a glass or so of alcohol close to bedtime
If you think these may be of concern please discuss this with the Sleep Technician.
Thanks

Appendix J: Electrode Placement


C3
C4
A1
A2
INION


REF
EOG
EOG
EMG
EMG
GROUND

Appendix K: Details of the Blood Alcohol Concentration Testing Equipment and Procedure

Taken from Bruck, Thomas, & Ball (2007).

BAC is normally measured and reported as *milligrams* of alcohol per 1000 millilitres (1 litre) of blood (mg/1000 mL). The breathalyser devices (Lion Alcometer S-D2) were recalibrated every 3 months to ensure accuracy of measurement. Victoria Police advised that the Lion Alcometre was a preliminary breath testing unit only, and that a confirmatory evidentiary measure of BAC (e.g. via a blood sample) was also required when it was used by them.

The manual describes that the Lion Alcometer S-D2 measured the concentration of alcohol vapour in expired breath by using an electrochemical fuel cell which contained two platinum electrodes. This fuel cell generated a small voltage that was directly proportional to the amount of alcohol concentration present in breath that is drawn into the unit (Lion Laboratories, 1982). The exact specifications of the unit are reported below.

Model	Lion Alcometer S-D2
Detector	Electrochemical fuel cell
Specificity	Responds only to alcohol in breath and is unaffected by other possible contaminants, such as acetone
Accuracy	+/- 10mg per cent blood alcohol concentration around the calibrated level
Analysis time	Approximately one minute per test.
Dimensions	120 x 63 x 30mm

Adapted from Lion Alcometer Manual (Lion Laboratories, 1982)

Before breath testing a ‘ready check’ was performed to ensure the breathalyser fuel cell was completely free of alcohol. When a satisfactory ‘ready check’ had been completed, the researcher depressed the ‘set’ button on the breathalyser until it locked. A fresh mouthpiece was then attached to the sampling port of the unit and instructions were administered to the participant. A new mouthpiece was always used for each test. Participants were instructed to fill their lungs and blow into the lipped end of the mouthpiece tube strongly enough to illuminate light ‘A’,

and to then continue blowing long enough to illuminate light ‘B’ when they would be told to stop. The researcher depressed the ‘READ’ button immediately after instructing the participant to stop, and continued to hold it down until the display stopped changing (approximately 15 to 20 seconds). The BAC was recorded from the display. Testing was repeated if the participant failed to provide sufficient breath to illuminate both sampling lights. If the alcohol reading was below .05 more alcohol was administered, followed ten minutes later by further testing. The amount of alcohol administered was once again estimated, and was dependent upon the previous BAC reading.

If the amount of alcohol required was overestimated and overshooting occurred the ST was instructed to carry out testing every 20 minutes until the participant’s BAC fell to the level of .04. At this time another dose of alcohol was administered and the usual procedure for measuring BAC was followed. It is known that BAC continues to rise rapidly before peaking at 30 to 60 minutes after a person’s last alcoholic drink. The BAC level then slowly decreases in a linear fashion at an average rate of about .015 per hour. This meant that the BAC of participants who had consumed the right amount of alcohol to reach .05 without overshooting would continue to rise for about 30 minutes after they went to bed. If the procedure for overshooting simply required waiting until the person’s BAC fell to .05 without administering any additional alcohol, then the person would be going to sleep on the downward slope of the alcohol absorption curve, rather than continuing to rise. Because the added inconvenience to participants that occurred as a result of overshooting was considerable, a tolerance level of +.01 BAC was allowed. This meant that several participants were measured at .06 prior to lights out.

Appendix L: Raw BAC Data for the ‘Alcohol’ Night, Time 3 & 4

Participant ID	BAC night 2, time 3 (3-min post-arousal)	BAC night 2, time 4 (20-min post-arousal)
1	0.021	0.019
2	0.034	0.030
3	0.035	0.033
4	0.035	0.030
5	0.025	0.023
6	0.050	0.050
7	0.050	0.045
8	0.030	0.024
9	0.030	0.025
10	0.026	0.022
11	0.035	0.030
12	0.016	0.010
13	0.041	0.040
14	0.041	0.036
15	0.040	0.040
16	0.031	0.029
17	0.049	0.045
18	0.045	0.040
19	0.050	0.050
20	0.045	0.035
21	0.030	0.025
22	0.045	0.040
23	0.045	0.040
24	0.050	0.050