Psychotropic Drug Usage and Human Behaviour During Fire Emergencies

Submitted by

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This thesis is submitted in total fulfilment
of the requirements for the degree of
Doctor of Philosophy

School of Psychology
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December 2014

Declaration

I, Chris Lykiardopoulos, declare that the PhD titled "Psychotropic Drug Usage and Human Behaviour During Fire Emergencies" is no more than 100,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

Signature: **Date:**

Abstract

Relatively little is known about the impact psychotropic drugs, particularly sedatives, have on human behaviour in a residential fire emergency. Two separate but related avenues of investigation were conducted to explore human behaviour in fire when under the influence of psychotropic drugs. In Study One the efficacy of current and alternative smoke alarm signals was tested after the consumption of hypnotics. A supplementary objective was to examine the relationship between sleep quality and arousal thresholds. Study Two retrospectively analysed an Australian database of fire fatalities using advanced algorithmic modelling techniques to determine: (a) if users of psychotropics and hypnotics were overrepresented in the Australian fire fatality statistics; and (b) the relationship between psychotropic drug consumption and a number of behavioural, environmental, and demographic risk factors. The research provided new, important evidence revealing an uncomfortably high level of risk to users of psychotropic drugs, particularly hypnotics, in a residential fire context. This was the first time psychoactive drug usage had been considered in such detail in the international literature. The major findings of the research centred around five key areas:

1. **Smoke alarms and a safety culture**

   The 520 Hz square significantly outperformed the 3100 Hz sine wave in users of hypnotics. A strong case is building challenging the effectiveness of the existing smoke alarm to alert vulnerable people from fire, and the results provide additional support for the continued adoption of the 520 Hz square wave. However, a fatality may not be avoided even when a potential victim has been alerted to danger. Therefore, a preventative focus facilitated by the adoption of a safety culture provides a more satisfactory answer to the fire threat.

2. **Psychoactive drugs and the fire response**

   The results indicate that psychotropic drugs and especially sedatives have a profound impact on behaviour when facing a fire emergency, particularly amongst middle aged adults. Alarm responsiveness was significantly attenuated after the consumption of a hypnotic and users of psychotropic drugs are greatly overrepresented in the Australian fire fatality statistics compared to population statistics. A key risk is the impact sedatives

may have on human error, evidenced by the relationship between time of fire and hypnotic consumption, which was largely responsible for most accidents.

3. **Complexity and research design**

   Fires are complex events. Algorithmic modelling was found to be better suited to analysing fire fatality statistics or other intricate data sets produced from fire incidents compared to traditional forms of regression. Furthermore, it is argued that a greater balance in research design that considers internal and external validity is necessary when investigating residential fires. This will produce more persuasive and realistic research outcomes that can educate and inform decision makers in the fire industry.

4. **Sleep quality and arousal thresholds**

   There is a possible relationship between sleep quality and arousal thresholds, which would challenge existing work that claims there is no difference in arousal thresholds between good and poor sleepers. However, no conclusive evidence was found regarding the relationship between sleep quality and arousal thresholds due to issues of power. A difference in arousability during the night is only predicted in patients who suffer from sleep maintenance issues, which is an insomnia subtype notably absent in previous research.

5. **Embracing a multi-disciplinary approach**

   The broader significance of this project is the contribution made to a shift in the approach to understanding fire accidents from one that focuses on organisational accidents analysed from technologically driven perspectives to a concentration on individual accidents examined from a psychological perspective.

Acknowledgements

I would like to convey my special thanks to my supervisors, Professor Dorothy Bruck and Dr. Michelle Ball for their unyielding support, wisdom and insightful feedback. I also owe a debt of gratitude to the many research assistants who have contributed to the Victoria University Coroners' Database, including Erin Read, Lynette Walpole, Kara Dadswell and Lin Xiong. Thanks also to Samara Neilson for your assistance with the arousal program, Dr. Simon Frenkel for your guidance on sedatives and to Neil Diamond for your advice on algorithmic modelling.

I would also like to thank my family, particularly my wife, Michelle and my son, Isaac. I would not have started nor finished were it not for you both.

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Introduction

Most fatal fires occur in the home. Domestic fire fatalities in Australia number approximately one hundred annually. Despite being a significant community hazard, relatively meagre attention is afforded to understanding the risk factors associated with residential fires. Research that has attempted to investigate the problem has often taken a technologically driven approach even though human error and involvement is frequently responsible for fire accidents in the home. As a result, human factors and characteristics that may impair human behaviour have received relatively modest attention in the literature up to this point. A pertinent case is psychotropic drugs, where comparatively little is known about the impact these drugs may have on the fire response, despite their well-documented negative effect on cognition. Psychoactive drug usage has been implicated in the accidental death literature, but rarely analysed directly or in relation to a fire emergency. This research attempted to redress this imbalance by analysing the influence of psychoactive drug usage on human behaviour in a fire emergency. The overall aim of this research was to determine the impact psychotropic drugs might have on human behaviour during fire emergencies. The focus of this research was on hypnotics as the effects associated with this class of drugs were expected to be especially damaging to an emergency response. A greater understanding of the role of psychoactive drug consumption in fire emergencies is likely to inform preventative strategies and potentially reduce the risk of death and injury from fire. The research comprised two separate but related avenues of investigation.

A limited collection of work does exist. Barillo and Goode (1996a) identified illicit recreational drugs as a potential risk in fire fatalities; however, other psychoactive drugs have not been examined. Given the differences in impairment profiles between different psychoactive drugs, this remains an important area of clarification. Psychoactive drug usage has also been implicated in the accidental death literature. A number of studies have investigated mental illness and accidental death and found that not only are people suffering from a mental illness more likely to die in an accidental death, but they are more likely to have drugs in their system at the time of death compared with people who are not mentally ill (Bayard-Burfield, Sundquist, & Johansson, 1998; Gau & Cheng, 2004; Holding & Barraclough, 1975, 1977; Watts-Hampton, Bruck, & Ball, 2006). However, psychoactive drug usage has never been analysed directly as a key risk factor

in fire fatalities in an Australian sample. Moreover, the specific impact of sedatives on fire fatalities has never been directly investigated before.

The first part of the research involved an evaluation of the reaction to fire detection equipment, which is critical in order to understand the broader response to a fire emergency. Smoke alarms are the dominant fire detection technology used in Australian homes, and can only be effective if they are audible and processed by victims of fire (e.g., by waking up). The successful widespread adoption of smoke alarms is partly due to government regulation and consistent messaging that has created the perception that smoke alarms are a cost effective means of saving lives. However, the effectiveness of traditional smoke alarms (3100 Hz sine wave) in waking certain groups in the population is increasingly being challenged, and a new frequency (520 Hz square wave) has been shown to be significantly more successful at arousing groups at-risk of not waking to the existing alarm. The new alarm has been tested in a number of vulnerable groups including older adults, children, people with a hearing loss, and the alcohol impaired. The 520 Hz square has not been tested in users of hypnotics or any psychoactive drugs (excluding alcohol), and there exists only limited empirical research on the potential dulling effect hypnotics may have on our ability to wake to a standard (3100 Hz sine wave) smoke alarm. Only one study has evaluated the response to a standard smoke alarm when under the influence of hypnotics and this was conducted more than a quarter of a century ago and was methodologically limited. Therefore, this study first aimed to assess the viability of both current and alternative smoke alarm technology in users of hypnotics. Older adults were the focus of the research since this demographic are more likely to be using sedatives and be victims of fire compared to younger adults.

A supplementary objective was to examine the relationship between sleep quality and arousal thresholds. Previous research has not supported a difference in arousal thresholds between good and poor sleepers, but the hyperarousal model of insomnia predicts a relationship. Hyperarousal theory suggests that cortical arousal is higher in people suffering from insomnia than in normal sleepers (even when asleep) and has received considerable support in the literature. Finding a relationship between sleep quality and arousability would have implications when making comparisons with previous research, and designing arousal studies involving poor sleepers.

The second component of the research analysed Australian coronial files. Psychoactive drugs are often cited as a risk factor in residential fires, but this has only been established by a small number of studies. None of this research has reviewed Australian data or specifically analysed hypnotics, the latter point being an important oversight given the large differences in impairment profiles between different psychoactive drugs. Consequently, the coronial research aimed to clarify the level of risk associated with different categories of psychoactive drugs in an Australian sample.

The detailed coronial data also permitted an exploration of the role of psychoactive drug consumption in relation to other documented risk factors. Fires often involve numerous risk factors and are multi-faceted. Little is currently known about how psychoactive drug consumption may influence or be related to other variables. A close examination of the circumstances of fire victims can inform the relationship between psychoactive drug consumption and other risk factors, which provides a more complete understanding of the role of psychoactive drugs in fire emergencies.

This thesis is organised into four major chapters. The first chapter is devoted to a review of the literature relevant to psychotropic drug usage and human behaviour during a fire emergency. The literature review begins by examining the development and prevailing attitudes and standards with regard to smoke alarms, which sets the scene for a more detailed examination of the areas that can impact our response to smoke alarms and fire in general. This chapter concludes with a rationale for the basis of this research. The next two chapters are devoted to the two separate studies conducted as part of this research. Each chapter describes the methods employed and discusses the results obtained. Chapter Two examines the impact of hypnotics on arousal thresholds in response to two different alarm signals. An evaluation of the level of risk attached to psychoactive drug consumption, and the relationship between psychoactive drug usage and other risk factors is investigated in Chapter Three. Chapter Four provides a broad discussion on the implications, limitations, directions for future work, and conclusions of the research.

CHAPTER 1 – Literature Review

Smoke Alarm Development and Current Standards

This section provides a brief history of smoke alarms, focusing on the reasons for their development and mainstream adoption. A detailed evaluation of current standards worldwide and in Australia follows. A critical evaluation of smoke alarm performance then closes the section. It is argued that smoke alarm effectiveness may be overstated.

Brief History

An American physicist, Francis Robbins Upton, was responsible for the invention of the first electric fire alarm and detector in 1890. A commercially viable residential smoke alarm was not developed until 1965. Initial sales of the smoke alarm were poor due to its relatively high expense and limited availability. It was estimated that in 1970, less than five per cent of American households had installed detectors (McLoughlin, Marchone, Hanger, German, & Baker, 1985). It was not until a tragedy of nationwide significance during the early 1970s that residential fire alarms became widely distributed (Giffen, Haro, Letho, & Papastavrou, 1996).

In 1972, Hurricane Agnes, one of the largest hurricanes on record, ravaged northeastern United States. As part of the disaster relief response, the United States Government built thousands of mobile homes equipped with fire alarms. This was the first wide-scale installation of smoke alarms anywhere in the world (Hall, 2001). A subsequent very positive review of the fire safety statistics surprised many and drew attention to the smoke alarm. While the expected number of fires occurred since the development of the mobile homes, there were no fire deaths or injuries (Bukowski, 2001). The results were credited to the installation of fire alarms (Bukowski, 2001).

This early success was followed by increasingly more detailed tests. While the results from the mobile homes were impressive, several operational defects in the alarms had been noted (Cote, 2008). In the early to mid 1970s, a collection of experiments suitably named the Indiana Dunes Tests were conducted in a lakeshore located in northwest Indiana. The experiments involved

placing commercially available smoke alarms in homes that were to be demolished. The homes were fitted with common residential furniture and trimmings and the time to escape was monitored in a variety of different settings. A review of the results by a fire safety panel found that in order to achieve an arbitrary three minute escape time, smoke detectors would be required on every level (McLoughlin, et al., 1985)

The Indiana Dunes Tests had an immediate regulatory impact (Hall, 2001). Laws were adopted across the United States that required the installation of smoke alarms on every level of new residential homes. Other areas also enforced this condition in existing homes. Montgomery County Maryland was one of the first jurisdictions to adopt this stance in 1975, with encouraging results. Successes like Montgomery County led to the rapid adoption of mandatory smoke detectors in most state or provincial building codes in the United States and Canada (Cote, 2008).

By 1975 the U.S. Building Officials and Code Administrators International (BOCA) building code was revised so that it became a requirement to install smoke alarms in all residential homes, both new and existing (McLoughlin, et al., 1985). Similar results have been achieved in Australia. In the 1990s, the Building Code of Australia (BCA) also amended its building code to make the installation of mains powered smoke alarms mandatory in new properties and any existing properties that have undergone significant renovations.

Current Standards

The world standards market for the protection of life and property from fire has historically been comprised of three main segments, the National Fire Protection Authority (NFPA) based in the United States, CEN (European Committee for Standardisation), and local standards from other parts of the world, such as Australia (Efstratiadis, Karirti, & Arvanitoyannis, 2000). Over the last decade, there has been an increasing demand for consensus based international standards, primarily because these standards reduce the technical barriers to trade and are based on best practice (Drahos, Lokuge, Faunce, Goddard, & Henry, 2004). Global standards on fire protection are produced by the International Organisation for Standardisation (ISO), with the

exception of Europe where equivalence with CEN is sought whenever possible via the Vienna agreement.

ISO is a worldwide federation of national standards bodies comprised of 163 country members, including Australia. The work of preparing international standards is normally carried out through expert technical committees. The ISO committee on fire protection was formed in the mid 1970s. The committee has published a total of 64 standards to date. The most relevant standard to this study is ISO 8201, Acoustics – Audible emergency evacuation signal (International Organisation for Standardisation, 1987). ISO 8201 specifies two parameters, the temporal pattern and the required sound pressure level at all places within the intended reception area (International Organisation for Standardisation, 1987).

The temporal pattern prescribed in ISO 8201 is known as the Temporal-Three (T-3). The T-3 is a unique three-pulse sound pattern comprised of a repeating four second cycle of three beeps and a pause (see Figure 1). Single or multiple frequency tones are permitted (International Organisation for Standardisation, 1987). This includes two-step tones and tones that shift from one pitch to another (International Organisation for Standardisation, 1987). In all cases, the temporal pattern is the key parameter (International Organisation for Standardisation, 1987). The temporal pattern can be supplemented by inserting a voice message in the pause period between the blocks of three tone bursts. Longer messages can be inserted during pauses in the tone pattern if desired (International Organisation for Standardisation, 1987). The standard demands that the signal be repeated for a period of time not less than 180 seconds (International Organisation for Standardisation, 1987). The ISO 8201 standard is primarily used for fire signals, and a separate ISO standard (ISO 7731) exists for auditory alarms that are used to *alert* occupants to investigate the area rather than immediately *evacuate* (International Organisation for Standardisation, 2003). In order to avoid confusion, the ISO 7731 signal is required to be significantly different from the "three-pulse" character of the ISO 8201 evacuation signal.

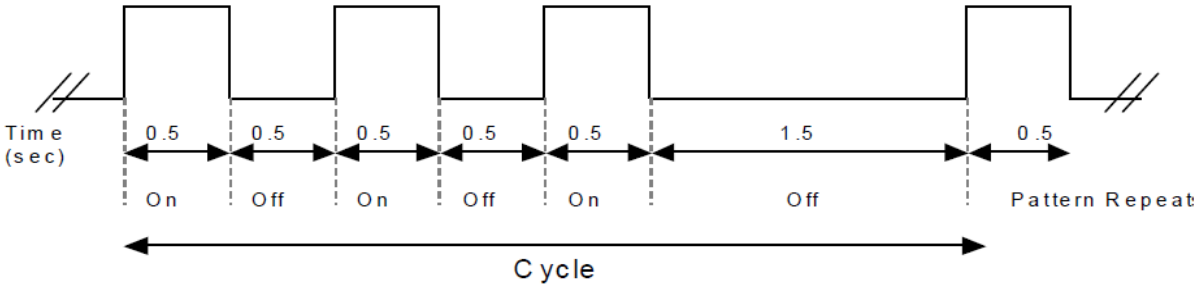

Figure 1. Temporal-Three signal pattern (Ball & Bruck, 2004b).

The second parameter established by ISO 8201 is sound pressure. The A-weighted sound pressure level must exceed the level of background noise within the intended reception area, and must be received at no less than 65 dBA (International Organisation for Standardisation, 1987). The A-weighted sound pressure is the adjusted sound level as heard by the human ear (Parker, 2003). This correction is made because the human ear is less sensitive at different frequencies (Parker, 2003). The minimum required sound pressure raises to 75 dBA at the pillow with all doors closed if the device is intended to awake sleeping occupants (International Organisation for Standardisation, 1987).

The current Australian standard for smoke alarms is consistent with the international ISO 8201 standard in most respects (Standards Australia, 2004). The second and current edition of the AS 3786 standard was published in 1993, and re-issued a further four times with minor amendments (Standards Australia, 2004). This document outlines all aspects of smoke alarm manufacture and installation. The T-3 pattern is adopted but the local standard differs slightly from the international version on the subject of sound pressure. The AS 3786 standard only specifies that the sound level should be 75 dBA at three metres. There are no occupant dependant variations (Standards Australia, 2004). For buildings that are constructed entirely of timber or have elements of timber in either the external or internal construction and are over two storeys, or buildings constructed entirely of masonry or concrete and are three storeys or less, a more sophisticated smoke detection system known as AS 1670 is required (Australian Standard 1670, 2004). A key requirement of the AS 1670 standard is that smoke alarms be located not more than 5.1 meters from any wall and not more than 10.2 meters apart (Australian Standard 1670, 2004).

It is hoped that the universal adoption of a single sound pattern will minimise potential failures to recognise the fire alarm signal, which have been evidenced in the past. Proulx and Laroche (2003) set out to assess people's recollection and identification of the T-3, as well as how urgent the signal was perceived to be in a North American sample. Results showed the T-3 was rarely identified as a smoke alarm or evacuation signal and was not judged as conveying urgency. The T-3 was usually judged to be a domestic signal, such as a busy phone tone. These findings have since been replicated in an Australian sample (Farley & Ball, 2012).

The ISO has a number of new projects being prepared. Any possible changes internationally will most likely be replicated in Australia. For example, the current overall ISO standard on smoke alarms (ISO 12239) is being revised to include items related to different types of smoke alarms. Once this standard is published it is intended for the ISO 12239 to replace the existing AS 3786 standard (International Organisation for Standardisation, 2010).

Smoke Alarm Performance

Despite the exponential growth of smoke alarm adoption, no experimental evidence exists on whether they actually reduce the prospect of death and injury from fire. Causal claims have largely been made on the basis of retrospective data trends, where cause and effect is extremely difficult to establish due to confounding factors (Hess, 2004). Furthermore, the use of these statistics to establish the credentials of smoke alarms has often been misleading.

The NFPA has been the foremost defender of smoke alarm effectiveness since their introduction (Ahrens, 2004, 2007, 2011a, 2011b). The agency is a worldwide authority on fire safety and maintains a database comprising 75% of all reported fires that occur annually in the United States. The database is the largest of its kind anywhere in the world. According to the NFPA, the rapid implementation of smoke alarms over the last two decades has corresponded with a significant decrease in the number of residential fire deaths, the corollary being that the smoke alarm alone has been responsible for the improved level of fire protection (Ahrens, 2011b). However, this statistic fails to acknowledge the large decline in the total number of fires during

the same period (Crapo, 2000). The actual number of deaths per 1,000 fires has only gently decreased during this time (Crapo, 2000). Furthermore, this decreasing trend began prior to the adoption of smoke alarms.

Crapo (2000) also noted differences in fatality rates when comparing households without smoke alarms to households with smoke alarms that were inoperable. Fatality and injury rates were lower in households with a smoke alarm, even if that alarm was not functioning. Crapo (2000) explained this non-intuitive result by arguing that individual pre-existing differences in fire safety consciousness may have influenced the decision to purchase and install a smoke alarm.

The NFPA data has been questioned in other areas. The inclusion of confined fires in the NFPA statistics may distort the real benefit of smoke alarms (International Association of Fire Fighters, 2008). Confined fires are fires that are restricted to the object of origin, such as cooking fires confined to the vessel of origin (Ahrens, 2011a). The likelihood of death in a confined fire is significantly reduced, yet the NFPA conclude that being alerted to the fire by a smoke alarm was lifesaving in these circumstances. Furthermore, the presence or absence of smoke alarms in confined fires was only known in two percent of cases (Ahrens, 2011a), and rather than exclude data where this information was missing, it appears that the small proportion of valid data was used to generalise to the 98% of missing data. The validity of this type of analysis is questionable.

Similarly, after analysing the circumstances of Australian fire fatalities, Bruck and Thomas (2010) concluded that many of the fatalities were difficult to mitigate and the presence of a working smoke alarm would most likely have been irrelevant in many cases to whether or not the person died in the fire. An explanation for this trend from the NFPA is that working smoke alarms were overrepresented in victims where smoke alarm effectiveness was made redundant because of victim impairment (Ahrens, 2011b). The implication being that smoke alarms are largely effective in saving lives except when there are other impediments that impact a victim's ability to escape. However, this is an admission that in many cases the current smoke alarm is not effective in the groups likely to die in a fire, when people most at-risk must surely be a priority when evaluating any protective measure.

It should also be noted that Australian bodies in the fire industry adopt a similar position to the NFPA. The Australian Fire Authorities Council (AFAC) and major state fire brigades, such as the Metropolitan Fire Brigade, Melbourne (MFB) regularly champion the life saving benefits of smoke alarms without justifying these claims (AFAC, 2006; MFB, 2014). The AFAC state that it is their belief that smoke alarms save lives, but this belief is not supported or justified with evidence (AFAC, 2006). Similarly, the MFB claim that "only smoke alarms save lives" and "you are more than four times more likely to die without a working smoke alarm", without citing any evidence to reinforce these claims (MFB, 2014).

The effectiveness of smoke alarms has also been questioned in the arousal literature. In order for smoke alarms to be effective they must be heard. In the last decade, research suggests that the current smoke alarm may fail to awaken a number of vulnerable groups in the population, including children (Bruck, 1999; Bruck & Bliss, 2000; Bruck, Reid, Kouzma, & Ball, 2004), those aged over 65 years (Bruck & Thomas, 2007a), people with hearing loss (Bruck & Thomas, 2007c), and the alcohol impaired (Ball & Bruck, 2004b; Bruck, Thomas, & Ball, 2007). This led to the development of the 520 Hz square wave, which has been shown to be more effective compared with the current alarm in a number of at-risk groups. For details of the new signal review the section beginning on page 27 devoted to auditory arousal thresholds (AATs).

Overall, work from a number of sources highlights growing scepticism surrounding the true level of smoke alarm effectiveness. There is a large investment in smoke alarms, but not the associated evidence to suggest this technology is successful in reducing the human cost of residential fire. Empirical research is needed to validate the many claims made with respect to smoke alarm performance.

Sleep and Insomnia

This section introduces the concept of sleep and its architecture. The various methods of measuring sleep are evaluated. This is followed by a review of classification and aetiological based issues in our understanding of insomnia. The association between age and sleep is a particular focus throughout the section.

Sleep Architecture

The average person will spend a third of their life sleeping. Sleep is a reversible behavioural state of perceptual disengagement from and unresponsiveness to the environment (Kryger, Roth, & Dement, 2005). Sleep is accompanied by a number of physiological and behavioural processes, such as immobility, closed eyes, and greatly reduced cognitive function (Tobler, 1995).

Sleep architecture refers to the amount and distribution of specific sleep stages (Szuba, Kloss, & Dinges, 2003). Sleep in humans and most mammals and birds is comprised of two distinct states: non-rapid eye movement (NREM); and rapid eye movement (REM). NREM sleep accounts for the vast majority (70-80%) of sleep time. The electroencephalography (EEG) features of sleep were used to separate NREM sleep into four distinct stages. In stage 1 sleep, alpha activity is present and wakefulness slowly diminishes. Stage 1 represents 3-8% of total sleep time. In stage 2 sleep, overall brain activity slows but K-complexes and sleep spindles are present in the EEG. A K-complex is a brief negative high voltage peak followed by a slower positive. Sleep spindles are a short burst of brain activity. Stage 2 begins after approximately 10-12 minutes of stage 1 sleep and comprises 45-55% of total sleep time. Stage 3 or 4 is deep or slow wave sleep (SWS). Stage 3 sleep is characterised by moderate amounts of SWS, while stage 4 includes large amounts of SWS. Approximately 15-20% of total sleep time occurs in SWS (Lee-Chiong, 2006).

In contrast to NREM sleep, the brain is quite active in REM sleep (Kryger, et al., 2005). Slow wave alpha and theta waves are usually present in the EEG during REM sleep. Throughout

REM sleep there is a burst of rapid eye movement, which is associated with dreaming (Lee-Chiong, 2006). REM sleep is associated with muscle atonia (paralysis of voluntary musculature), gating of sensory input, rapid eye and middle ear movements, as well as heart rate and respiration changes (Gagnon et al., 2002). Muscle atonia prevents us from moving when we are dreaming. REM sleep accounts for 20-25% of total sleep time (Lee-Chiong, 2006).

NREM and REM sleep alternate throughout the night in sequence. This process can be depicted graphically through a hypnogram (see Figure 2). Hypnograms were developed to summarise the huge amount of data produced in an overnight sleep study. One complete NREM-REM cycle occurs about every 90 minutes and there are approximately four to six cycles each sleeping period (Zillmer & Spiers, 2001). The distribution of NREM-REM is uneven during the sleeping period. There is a larger proportion of SWS during the initial cycles and additional REM sleep during the later cycles of the sleeping period (Kryger, et al., 2005).

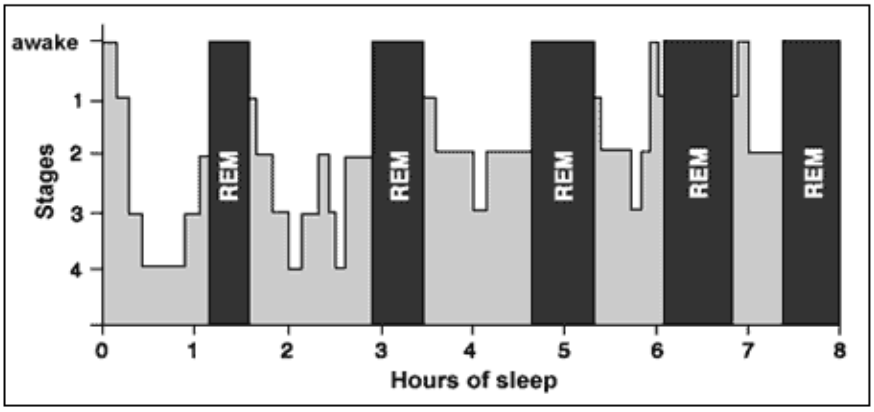

Figure 2. A typical hypnogram from a young, healthy adult (sourced from Kryger, et al., 2005). Light-gray areas represent NREM sleep.

Sleep Measurement

Sleep can be measured in various ways. Polysomnography (PSG) has long been considered the most accurate method of measuring sleep. This technique combines the measurement of brain activity (electroencephalogram or EEG), eye movements (electro-occulography), and muscle

tone (electromyography) to produce an objective record of sleep that can accurately reveal sleep architecture in addition to determinations of overall sleep or wake. However, PSG can be expensive and inconvenient when monitoring sleep over an extended period (Ancoli-Israel et al., 2003). For these reasons, actigraphy is emerging as popular alternative (C. McCall & McCall, 2012). An actigraph is a device, similar to a watch in appearance, used to measure arm movement. An algorithm interprets the arm movement recorded via the actigraph as a proxy for sleep and wake. Recordings can be conducted for days or weeks in a participant's home with minimal disturbance, which allows for a more naturalistic setting than can be achieved via PSG in a sleep laboratory. Actigraphy is also less expensive than PSG, and the number of nights to be recorded can be increased with minimal cost. Actigraphy has achieved favourable comparisons to PSG in a number of validation studies critically reviewed by Tyron (1996); however, the populations, scoring algorithms, and devices in the selected studies often varied widely. Furthermore, many of the studies were performed in a sleep laboratory, which presents a threat to external validity, by contrast actigraphs are intended to be used in a participant's home (Morgenthaler, Alessi et al., 2007).

A key limitation of actigraphy relates to precision. Actigraphy is most accurate when used to measure major periods of sleep and waking, and is much less accurate when used to identify short periods of sleep or wakefulness (Standards of Practice Committee, 1995). Body movement is only an indirect measure of sleep and individual differences can vary widely, particularly amongst sufferers of disorders that may impact nocturnal body movement, such as Parkinson's disease or restless leg syndrome (Morgenthaler, Alessi et al., 2007). For this reason, the accuracy of actigraphic measurements in people suffering from insomnia is considerably worse than it is in good sleepers (Morgenthaler, Alessi, et al., 2007). However, a number of authors have demonstrated the validity of actigraphy in a population of people suffering from depression (Coffield & Tryon, 2004; C. McCall & McCall, 2012), which suggests that actigraphy may have a greater role to play in some clinical settings (Morgenthaler, Alessi, et al., 2007).

A third method of sleep measurement is the sleep diary. Sleep diaries are widely used and often viewed as a useful adjunct to other sleep measures in many sleep studies (Standards of Practice Committee, 1995). Sleep diaries allow a day-to-day evaluation of a variety of sleep variables,

such as bedtime, rising time, or sleep onset latency, which is the amount of time to fall asleep after the lights have been turned off. Sleep diaries have many advantages, including price, ease of use, and flexibility (Standards of Practice Committee, 1995). In many cases sleep diaries are also more accurate than other methodologies. Sleep diaries have proven to be more precise when assessing the sleep-wake patterns of participants with body movement disorders (Morgenthaler, Lee-Chiong et al., 2007). A major disadvantage of the sleep diary is that the information is subjectively reported, which impacts the accuracy of the account (Libman, Fichten, Bailes, & Amsel, 2000). The combination of a sleep diary and actigraphy is often advocated in the literature due to their complementary aspects (Carney, Lajos, & Waters, 2004; Van Den Berg et al., 2008). For example, the use of actigraphy has been shown to improve the accuracy of reporting in sleep diaries (Carney, Lajos, & Waters, 2004).

Insomnia

Insomnia is the experience of inadequate or nonrestorative sleep despite sufficient time in bed (Roth, 2008). Insomnia is a frequent disorder reported globally (Ohayon & Partinen, 2002; Terzano et al., 2005). Approximately one in three of the general population in the United States suffer from insomnia symptoms of various degrees of severity (Roth, 2001; Terzano et al., 2005). However, the prevalence of individuals meeting the diagnostic criteria of the most widely used classification system (see below for details) for insomnia is approximately 10% of the general population in the United States (Ohayon & Partinen, 2002; Passarella & Duong, 2008; Roth, 2001; Terzano, et al., 2005). Similar results have been achieved in other countries (Ohayon & Partinen, 2002; Terzano, et al., 2005). There is some evidence that the incidence of insomnia is increasing (McCall, 2001; Roth, 2001). However, the various classification systems and increased public awareness may obscure these results.

There are currently four major diagnostic classification systems available for sleep disorders: (1) the Diagnostic Classification of Sleep and Arousal Disorders (DCSAD; Association of Sleep Disorders, 1979); (2) the International Classification of Sleep Disorders, which effectively was developed as an update to the DCSAD (ICSD; American Sleep Disorders Association, 2005); (3) the International Classification of Diseases (ICD-10; World Health Organisation, 2007); and (4)

the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). A detailed analysis of each system will not be offered because of their similarity. Instead, the DSM-5 will be reviewed in isolation since it is the most widely used and accepted classification system by the psychiatric community (Szuba, et al., 2003). The following diagnostic criteria are relevant to a diagnosis of insomnia disorder in the DSM-5:

A. A predominant complaint of dissatisfaction with sleep quantity or quality, associated with one (or more) of the following symptoms:

1. Sleep onset insomnia or a difficulty initiating sleep.
2. Sleep maintenance insomnia or a difficulty maintaining sleep, characterised by frequent awakenings or problems returning to sleep after awakenings.
3. Terminal insomnia or early-morning awakening with inability to return to sleep.

These symptoms may vary over time (Hohagen et al., 1994) and commonly co-occur (Rajput & Bromley, 1999).

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

C. The sleep difficulty occurs at least 3 nights per week.

D. The sleep difficulty is present for at least 3 months.

E. The sleep difficulty occurs despite adequate opportunity for sleep.

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

G. The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

H. Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia.

Sleep maintenance symptoms are more common than sleep onset issues particularly in groups vulnerable to insomnia (Baker, Wolfson, & Lee, 2009; Rosenberg, 2006; Shochat, Umphress, Israel, & Ancoli-Israel, 1999). Older adults, sufferers of depression, and the medically ill are all much more likely to complain about sleep maintenance issues (Foley et al., 1995; Gislason &

Almqvist, 1987; Webb, 1982; Webb & Campbell, 1980) compared to the average. Despite this, sleep onset problems have traditionally been the focus of both research and treatment (Rosenberg, 2006). This imbalance has a number of implications. Most importantly, the understanding of the problem and ultimate solution will not be representative or maximised in people suffering from sleep maintenance insomnia because the focus has been on patients experiencing sleep onset insomnia.

There exists support for various models in the aetiology of insomnia. Psychological models include the concepts of conditioning, stress response, predisposing personality traits, and attitudes and beliefs about sleep. Perhaps the most compelling explanation of insomnia that has been endorsed at the National Institute of Mental Health (NIMH) State of the Science Conference on the nature of insomnia and insomnia therapeutics is hyperarousal (NIH, 2005) and has also received a great deal of empirical support (Bonnet & Arand, 1995, 1998b; Freedman, 1986; Freedman & Sattler, 1982; Hajak et al., 1995; Haynes, Adams, & Franzen, 1981; Irwin, Clark, Kennedy, Gillin, & Ziegler, 2003; Lack, Gradisar, Van Someren, Wright, & Lushington, 2008; Merica, Blois, & Gaillard, 1998; Nofzinger et al., 2004; Perlis, Smith, Andrews, Orff, & Giles, 2001; Riemann et al., 2002; Stepanski, Glinn, Zorick, & Roehrs, 1994). Hyperarousal suggests that inappropriate physiological arousal in people suffering from insomnia is responsible for their difficulty in falling asleep and staying asleep (Bonnet & Arand, 1996, 1998a). Patients suffering from insomnia have been shown to exhibit increased activity in a range of relevant physiological parameters, such as brain metabolism, autonomic nervous system activity, and EEG spectral analyses when compared to controls (Bonnet & Arand, 1996, 1997, 1998a, 2010; Drake, Roehrs, & Roth, 2003; Kronholm et al., 2007; Regestein, Dambrosia, Hallett, Murawski, & Paine, 1993; Stepanski, Zorick, Roehrs, Young, & Roth, 1988). Importantly, increases in arousal have been demonstrated not only when patients are awake trying to sleep, but also when patients are asleep.

The timing of this endogenous arousal is important and believed to help explain the various insomnia symptoms. Evidence from the temperature regulation literature has been particularly illuminating in this area. It has been shown that there are specific irregularities in core body temperature when comparing across insomnia subtypes (e.g., sleep onset cf. sleep maintenance

insomnia). Core body temperature is typically inversely proportional to sleep propensity (Akerstedt, Fraberg, Friberg, & Wetterberg, 1979; Lack & Lushington, 1996). For example, lower body temperatures usually coincide with a greater propensity for sleep. In people experiencing sleep onset insomnia, the reduction in core body temperature common prior to sleep is delayed and not synchronised with their habitual sleep time (Morris, Lack, & Dawson, 1990). In contrast, people suffering from sleep maintenance insomnia experience elevated core body temperature throughout the night (Lack, et al., 2008). This evidence suggests that although all people suffering from insomnia will experience inappropriate cortical arousal at some point, only a difficulty maintaining sleep is associated with nocturnal awakenings as a function of heightened cortical arousal during the early part of the morning after the onset of sleep.

Age and Sleep

Age is a critical factor when discussing sleep. The most important variable affecting sleep stage distribution is age (Roth & Roehrs, 2000). Newborns spend the majority of the day asleep, frequently napping during the day (Chokroverty, 2010). The biggest changes occur in REM sleep. REM sleep occupies 50% of total sleep time in newborns compared to just 25% in young adults (Bonnet & Arand, 1989). Sleep quality diminishes with age and over time there is a decrease in the amount of SWS and increase in the number of awakenings (Lee-Chiong, 2006). As a result of these changes in addition to other issues, such as a greater propensity for medical problems, older adults are significantly more likely to be suffering from insomnia (McCall, 2005), particularly sleep maintenance insomnia (Foley, et al., 1995) compared to younger adults.

Sedatives and Other Psychotropic Drugs

This section provides an overview of psychoactive drug classes before leading into a more detailed discussion on drugs used to treat insomnia. The greatest attention is given to those psychotropic drugs with sedative properties that are most commonly prescribed to sufferers of insomnia in Australia. The mechanism of action of each drug and the impact on both sleep and the body is considered. A review of prescription patterns is offered, and the section concludes by examining the likely profile of users of hypnotics.

Psychotropic Drugs

There are six major classes of psychiatric medication: (1) antidepressants; (2) anxiolytics; (3) antipsychotics; (4) mood stabilisers; (5) stimulants; and (6) hypnotics (Stahl, 1996). It should also be noted that illicit drugs or narcotics also constitute a class of psychotropic drugs that are illegal and used for non-medical purposes (e.g., heroin or marijuana). Antidepressants are a class of drugs that reduce symptoms of depressive disorders by changing chemical imbalances of neurotransmitters in the brain. Anxiolytics are a type of medication used to treat anxiety. Antipsychotics are a class of drugs used to treat psychosis. Mood stabilisers are used to treat mood disorders characterised by powerful shifts in affect. Stimulants are psychotropic drugs that raise the level of mental or physical functioning, which are often used in the treatment of attention deficit-hyperactivity disorder and disorders of excessive daytime sleepiness. Finally, hypnotics are typically used in the treatment of insomnia. The focus of this study was on hypnotics as the decrease in mental and physical functioning associated with hypnotics is possibly more damaging to an emergency response than other types of drugs that may stimulate or amplify consciousness (Koelega, 1993; Oster, Huse, Adams, Imbimbo, & Russell, 1990; Pickworth, Rohrer, & Fant, 1997). In addition, hypnotics are one of the most frequently prescribed psychotropic medications (Britt, Miller, & Charles, 2007; Smith & Tett, 2009). Benzodiazepines are the most commonly prescribed hypnotics in Australia (Britt, et al., 2007), with a new class of related drugs (Z-drugs), also gaining in popularity (Hollingworth & Siskind, 2010). In the United States, the Z-drugs are now prescribed more frequently than benzodiazepines (Roy & Smith, 2010). Drugs from other classes that have sedating properties

are also sometimes used to alleviate sleep dysfunction. These drugs include sedating antidepressants, and a variety of other drugs, such as anticonvulsants, antihistamines, antipsychotics, hormones, and natural substances. A brief review of the primary hypnotics follows.

Benzodiazepines

Benzodiazepines came to prominence in the 1960s and represented a major advance on existing medications that induce sleep, such as barbiturates. Initial excitement in the drugs eased in the ensuing decades as a variety of negative side effects became apparent (e.g., tolerance and dependence issues). In spite of these problems, benzodiazepines remain popular. Approximately 1 in 20 Australians have taken benzodiazepines in the last two weeks and almost half of these pills were of the benzodiazepine, temazepam (Australian Bureau of Statistics, 2010a). Other popular benzodiazepines in Australia include oxazepam, nitrazepam, and diazepam, although the latter is more commonly prescribed for anxiety rather than sleep problems (Barker, Jackson, Greenwood, & Crowe, 2003).

Benzodiazepines are central nervous system depressants and enhance the actions of the chief inhibitory neurotransmitter gamma-aminobutyric acid (GABA). GABA controls the excitability of neurons by binding to the GABA-A receptor site (Kryger, et al., 2005). GABA-A is a protein complex located in the synapses of neurons that comprises three moieties, a chloride ionophore, a GABA-A recognition site, and a benzodiazepine recognition site. When the benzodiazepine binds to the receptor in combination with GABA, this process increases the flow of negatively charged chloride ions into the neuron, which changes the neuron's charge and effectively stops the neuron from firing (Kryger, et al., 2005). This can have a potent impact because of the abundance of GABA-A receptors in the central nervous system (Stahl, 1996).

The body's response to a drug is impacted by the drug's pharmacokinetic profile (Stahl, 1996). Pharmacokinetic principles address the absorption, distribution, metabolism, and excretion of drugs and their metabolites (Keltner & Folks, 2005). Absorption is defined as the movement of the drug from its administrative site to the bloodstream (Stahl, 1996). Distribution refers to the

movement of the drug from the blood to the rest of the body (Keltner & Folks, 2005). Metabolism is defined as the enzyme-catalysed alteration of drugs by the living organism (Keltner & Folks, 2005), while excretion refers to the removal of the drug from the body (Keltner & Folks, 2005). Pharmacokinetic properties are particularly important in determining the effectiveness, tolerability and safety of sleep medications (Drover, 2004).

The vast majority of hypnotics are rapidly and completely absorbed, with most achieving peak plasma levels in one to one and a half hours (Stahl, 1996). However, the elimination half-lives vary widely (Kryger, et al., 2005). The elimination half-life refers to the period of time it takes for the concentration of the drug to be reduced by one-half (Keltner & Folks, 2005). Hypnotics are typically sorted into short (1-5 hours), medium (6-19 hours), or longer (greater than 19 hours) acting agents based on their elimination half-life. A hypnotic with a relatively long elimination half-life will accumulate during nightly use, due to the addition of new hypnotics before the existing hypontics have been completely eliminated, which may result in daytime residual sedation (Stahl, 1996).

Age has an influence on the absorption, distribution, metabolism, and excretion of drugs (Keltner & Folks, 2005; Woodward, 1999). Age related decreases in gastrointestinal functioning can have a minor impact on absorption (Stahl, 1996). Increased body fat, decreased total body water, decreased albumin, and increased alpha-1-acid glycoprotein can also influence the way drugs distribute in older adults (Keltner & Folks, 2005). Overall, an increase in distribution in older adults is likely, which will result in a longer half-life (Kryger, et al., 2005). Also, decreases in the size of the liver and blood flow reduce the body's ability to metabolise the drug (Keltner & Folks, 2005). Finally, age related declines in renal function are likely to decrease the rate of excretion (Stahl, 1996). Consequently, age related declines in body functioning hamper the body's ability to respond to drugs.

Research on benzodiazepines indicate that sleep latency and wake time after sleep onset are usually reduced, and total sleep time increased (Mendelson, 1987). Spindle activity, which is a burst of brain activity during stage two sleep, may be increased (Mendelson, 1987). REM sleep is reduced and suppression of slow wave sleep (stages three and four) is usually evident (Pandi-

Perumal & Monti, 2006). The reduction of REM sleep can lead to REM rebound when hypnotics are withdrawn, which is the lengthening and increasing frequency and depth of REM sleep, which occurs after periods of REM sleep deprivation (Lee-Chiong, 2006).

Z-Drugs

Despite their effectiveness and continued prescription, regular consumption of benzodiazepines has been shown to lead to abuse, dependence, and adverse side-effects in many cases (Sim, Khong, & Wain, 2007). In response to these issues, a new class of nonbenzodiazepine drugs was introduced in the 1990s (Goa & Heel, 1986). Pharmacologically, nonbenzodiazepines operate in much the same way as traditional benzodiazepines, acting at various regions of the GABA-A benzodiazepine receptor complex; however, chemically, they are different (Kryger, et al., 2005). There are currently three major classes of nonbenzodiazepines: (1) imidazopyridines; (2) pyrazolopyrimidines; and (3) cyclopyrrolones. The best known derivatives of these three drug classes are zolpidem, zaleplon, and zopiclone respectively. Together, they are known colloquially as "Z-drugs".

Zolpidem (common trade names include Stilnox, Zolpibell, Dormizol, Somidem, and Stildem) is an imidazopyridine compound that binds in a relatively selective manner to the central benzodiazepine receptor (Kryger, et al., 2005). Zolpidem is the most widely prescribed Z-drug in Australia and its use is growing (Hollingworth & Siskind, 2010). Peak concentrations are reached after one and a half hours, while the half-life is short at just two to three hours (Kryger, et al., 2005). Zolpidem has not proven effective in maintaining sleep and is used more for sleep initiation problems because it is short acting (Calamaro, 2008; Moen & Plosker, 2006). As a result, zolpidem extended-release was developed to ensure that the patient remains asleep through the middle of the night without increasing the risk of next-day residual sedation (Calamaro, 2008; Moen & Plosker, 2006). There is limited research verifying the viability of the extended-release formulation, but what research exists is generally supportive of the longer acting variation (Calamaro, 2008; Moen & Plosker, 2006). Unlike benzodiazepines, Zolpidem does not impact slow wave or REM sleep (Hollingworth & Siskind, 2010).

Zaleplon (common trade names include Sonata and Starnoc) was the most recently developed Z-drug and also binds selectively to the benzodiazepine receptor (Calamaro, 2008; Moen & Plosker, 2006). Zaleplon is not currently available in Australia. Zaleplon reaches peak concentration in just one hour and has an elimination half-life of also one hour (Calamaro, 2008; Moen & Plosker, 2006). Because of zaleplon's very short half-life, zaleplon appears to be the best tolerated of the three Z-drugs in terms of next-day effects and adverse reactions (Terzano, Rossi, Palomba, Smerieri, & Parrino, 2003). Zaleplon has been shown to improve sleep induction, but there is little evidence of impact to any other sleep parameters, including sleep architecture (Hollingworth & Siskind, 2010).

Zopiclone (common trade names include Imovane and Zimovane) is a cyclopyrrolone that acts at the type GABA-A receptor, to which benzodiazepines also bind (Kryger, et al., 2005). Zopiclone is available in Australia, but not prescribed to the same levels as Zolpidem. Zopiclone reaches peak plasma concentrations in 30 minutes to two hours and has a half-life of approximately six hours (Kryger, et al., 2005). The impact of Zopiclone on sleep parameters is similar to Zolpidem, where there is evidence of improvements to sleep induction and sleep maintenance (when compared specifically with the Zolpidem controlled release formula), and no change to slow wave or REM sleep (Hollingworth & Siskind, 2010).

Sedating Antidepressants

Certain antidepressants are also prescribed for sleep issues (Kryger, et al., 2005). Antidepressants are often the second choice in treating patients who are unable to sleep and are sometimes used to avoid the negative side effects of benzodiazepines (Holshoe, 2009). The classification of sedating properties in the literature is not always clear. Some authors only consider the tricyclics antidepressants (TCAs) and related compounds mianserin and mirtazapin to be sedating (Ramaekers, 2003), while there is also evidence to suggest selective serotonin-reuptake inhibitors (SSRIs), serotonin-2 receptor antagonists/reuptake inhibitors (SARIs), and atypical agents also impact sleep parameters (Gursky & Krahn, 2000). Monoamine-oxidase inhibitors (MAOIs) are also believed to have some sedating properties but are rarely prescribed because of potentially lethal dietary and drug interactions (Holshoe, 2009). Consistent with the

comprehensive approach adopted throughout this project, all antidepressant drugs linked with sleep will be reviewed.

TCAs were introduced in the 1960s and have largely been superseded by newer antidepressants (Kryger, et al., 2005). TCAs are traditionally used to treat mood disorders, such as obsessive–compulsive disorder, panic disorders, and generalized anxiety disorder (DeMartinis & Winokur, 2007). Dothiepin and amitriptyline are the two most common TCAs currently prescribed (Smith & Tett, 2009). The majority of TCAs act by inhibiting both serotonin and norepinephrine reuptake transporters (Kryger, et al., 2005). TCAs are rapidly absorbed and reach maximum concentrations two to six hours postdose (Kryger, et al., 2005). TCAs have long half-lives of approximately 15-20 hours (Kryger, et al., 2005). TCAs improve sleep architecture by shortening sleep latency, increasing total sleep time, decreasing awakening after sleep onset, suppressing REM sleep, and improving overall sleep efficiency (DeMartinis & Winokur, 2007).

In comparison to TCAs, SSRIs have much milder side effects and a higher toxic dose (Kryger, et al., 2005). As a result, SSRIs are now the most widely prescribed antidepressant in many parts of the world (Holshoe, 2009). SSRIs are primarily prescribed for clinical depression (Kryger, et al., 2005). Citalopram, venlafaxine, and paroxetine are commonly prescribed SSRIs (Kryger, et al., 2005). SSRIs are believed to act by inhibiting the reuptake of serotonin after being released in synapses (Kryger, et al., 2005). SSRIs reach maximum concentration approximately two hours postdose and have a half-life of 30-40 hours (Kryger, et al., 2005). SSRIs have generally not been associated with sleep improvements, and have also been shown to actually deteriorate sleep quality (Wilson & Argyropoulos, 2005); however, some SSRIs have demonstrated an ability to enhance sleep. Paroxetine has been shown to improve sleep outcomes in both depressed (Montgomery, 1992) and non-depressed patients with insomnia (Nowell, Reynolds, Buysse, Dew, & Kupfer, 1999), and increase SWS when given to healthy controls in the morning (Oswald & Adam, 1986). Similarly, escitalopram appears to be beneficial for the treatment of sleep problems in patients experiencing depression (Stein & Lopez, 2012).

SARIs are another class of antidepressants that modulate serotonin activity (Kryger, et al., 2005). Trazodone and nefazodone are the two most commonly prescribed drugs in this class, but these

drugs are rarely prescribed in Australia (Kryger, et al., 2005). SARIs act by antagonising serotonin receptors and inhibiting the reuptake of serotonin, norepinephrine, and/or dopamine (Kryger, et al., 2005). SARIs reach maximum concentrations one to two hours postdose and have relatively short half-lives of two to nine hours (Kryger, et al., 2005). SARIs have been shown to improve sleep efficiency, decreases awakenings, and enhance REM sleep (DeMartinis & Winokur, 2007).

Other antidepressants from various classes that are sometimes used for sedation have been listed here under atypical agents. Mirtazapine, mianserin, and bupropion are three drugs that fall into this category. Mirtazapine and mianserin are tetracyclic antidepressants (TeCA) closely related to TCAs that are primarily used to treat major depressive disorder (DeMartinis & Winokur, 2007). Mirtazapine is capable of improving sleep continuity and efficiency without altering REM sleep, but at a cost of significant daytime sleepiness and possible weight gain (DeMartinis & Winokur, 2007). In contrast, Mianserin has been shown to suppress REM sleep (Maeda et al., 1990). Bupropion is non-tricyclic antidepressant and its primary pharmacological action is norepinephrine-dopamine reuptake inhibition (DeMartinis & Winokur, 2007). At present, its effects on sleep are somewhat unclear. Bupropion does not suppress REM, but decreases REM latency and increases REM time and percentage (DeMartinis & Winokur, 2007).

Other Drugs Used to Treat Insomnia

In addition to hypnotics and antidepressants, there remains a variety of other drugs, such as anticonvulsants, antipsychotics, hormones, and natural substances that may be used to treat insomnia. Melatonin, diphenhydramine, doxylamine, valerian, gabapentin, tiagabine, choralhydrate, olanzapine, gamma-hydroxy-butyrate, and quetiapine are all used to induce sedation (Kryger, et al., 2005). These drugs represent a wide variety of drug classes and differ in their mechanism of action and pharmacokinetics. With the exception of melatonin (Wade et al., 2010) and gamma-hydroxy-butyrate (Mamelak, Escriu, & Stokan, 1977), currently, very little data exists on the efficacy of many of these treatments (Kryger, et al., 2005).

Patterns of Prescription

Over the last 15 years gradual changes in the prescription patterns of sleep medication have emerged. First, benzodiazepine usage has decreased and antidepressant usage has increased (Walsh & Scweitzer, 1999). There is evidence of a definite shift towards the use of antidepressants in the treatment of insomnia (Vermeeren, 2004). Second, there has been a move toward shorter acting agents, which decrease the likelihood of residual effects (Hollingworth & Siskind, 2010; Vermeeren, 2004). While shorter acting agents, particularly the Z-drugs, enjoy a more favourable safety profile, they have not completely eliminated issues of tolerance and dependence (Hallfors & Saxe, 1993; Terzano, et al., 2003) and there have also been reports of other unintended side effects, such as somnambulism (sleep walking) reported (Hoque & Chesson, 2009) . Finally, there is consensus that hypnotics should be used at their lowest possible dose for limited durations (Doghramji, 2000). Higher dosages not only increase the intensity of action but also its duration (Lader, 1998). Dosing levels of antidepressants for sleep problems are unclear. The majority of data regarding antidepressant drugs on sleep has come from studies in patients with depression, where drug dosage is typically higher than it would be for treatment of a sleep disorder (Kryger, et al., 2005). Research has indicated that doses used to treat depression are not needed to improve sleep, but less is known about the most effective dosing ranges (DeMartinis & Winokur, 2007).

User Profile

There are several groups within the population who are more likely to be using benzodiazepines. These include older adults, females, patients suffering from physical or mental ailments, drug abusers, and children with anxiety conditions (American Psychiatric Association, 1990; Coffey, 1993; Makkai, 2001; Ross & Darke, 2000; Schuckit, Smith, Kramer, Danko, & Volpe, 2002; Schweizer, 1995; Smith & Tett, 2009). A particularly strong trend is visible amongst older adults. Consumption of hypnotics generally increases with age, and is most likely in older adults (Smith & Tett, 2009). Not only are older adults more likely to be taking hypnotics, but they are also more likely to be using hypnotics long term, particularly those older adults suffering from physical or mental problems (Bartlett et al., 2004; Llorente, David, Golden, & Silverman, 2000;

Shorr & Robin, 1994). Consequently, there is a greater risk of withdrawal or other health complications, such as the risk from falls, amongst older adults (Frey, Ortega, Wiseman, Farley, & Wright, 2011).

Auditory Arousal Thresholds

This section first provides a brief historical account of the research on arousal thresholds, which develops into a review of a specific frequency at the forefront of signal performance technology. Following this, an account of the key factors that affect auditory arousal thresholds is given. It is then argued that hypnotic consumption is also likely to be an important factor, but little work currently exists in this area, and research that has been conducted is hampered by methodological limitations. Differences in arousability between good and poor sleepers are also considered.

Brief History

Research on arousal thresholds during sleep began in the 1960s and was primarily concerned with the relationship between arousal thresholds and characteristics such as sleep stage, time of night, and performance (Bonnet, 1982). Drawing on Gibsonian theory (Gibson, 1979) that suggests people perceive stimuli more readily if the stimulus itself conveys meaning, early findings in the area highlighted the role of signal significance in modulating arousal. In 1960, Oswald and colleagues (1960) found that participants were significantly more likely to respond during sleep to their own name rather than an alternative name. Similar results have been replicated in varying formats (LeVere, Davis, Mills, & Berger, 1976; McDonald, Schicht, Fiuzler, Shallenberger, & Edwards, 1975). A tidy explanation for these findings comes from neurologists in the United Kingdom who have suggested that affective content stimulates regions of the brain that activate the prefrontal cortex and induce arousal (Portas et al., 2000).

In the 1980s work on arousal thresholds began in the area of human behaviour in fire (Bonnet, 1982). Early research focused on the response of normal people to a smoke alarm (Bruck, 1998; Bruck & Horasan, 1995; Nober, Peirce, & Well, 1981). It was found that normal adults tend to awaken to the standard 3100 Hz sine wave smoke alarm under typical conditions (Nober, et al., 1981); however, researchers at the time recognised that conditions are rarely ideal during a fire emergency and normal adults are unlikely to be representative of those most at risk (Bruck, 2001). The first study to consider this issue was by Nober et al. (1981). This study found that

while all 30 young adult men participating in the research were able to awaken quickly (within 21 seconds) to a high frequency alarm presented in their homes at levels ranging from 55 to 85 dBA at the pillow, only 70% of the men awoke when the alarm was presented at the hallway volume of 55 dBA when presented with background noise of 53 dBA from an air conditioner. As early as 1978, Berry (1978) recognised that a signal of over 100 dBA may be required to successfully wake some people in certain circumstances (e.g., when hearing is impaired). Then in 2001 Bruck (2001) drew together the then available research on the ability of smoke alarms to wake sleeping people and the sleep research on arousal thresholds and concluded that people in many groups in the population would not awaken to a 75 dBA (at the pillow) high-pitched alarm. This review was then expanded and updated by Bruck and Ball (2007). Since the initial review a number of at risk groups have been identified, including children, those aged over 65 years, people with hearing loss, and the alcohol impaired (Thomas & Bruck, 2008).

Guided by earlier research on signal salience, work began on finding a more effective signal at waking people at risk of dying in a fire. The majority of research has focused on auditory signals, due to their low cost (Bruck & Thomas, 2008a). In 2004, Ball and Bruck (2004a) explored the arousal thresholds of 14 adults aged 18-26 years who were self declared deep sleepers in response to a range of signals. Each participant received three signals: a female voice alarm, a standard high pitched smoke alarm, and the 520 Hz square wave T-3 alarm. There were no silences between the sounds in this study beyond that in the T-3 pattern. At the benchmark level of 75 dBA, the 520 Hz square wave slightly outperformed the female voice alarm and was approximately six times better than the standard smoke alarm (7% did not respond compared with 43% non-response for the standard smoke alarm).

Since then developments in the literature on alarms over the past decade have shown that the 520 Hz square wave performs significantly better than the standard high pitched 3100 Hz sine wave alarm (Bruck, Ball, Thomas, & Rouillard, 2009). Bruck and Thomas' (2007a) study involving older adults aged between 65 to 85 years found that, when the lower frequency 520 Hz square wave was presented together with the 3100 Hz sine wave during stage 3 or 4 sleep the mean arousal threshold for the 3100 Hz sine wave signal was 64 dBA, while it was just 48 dBA for the 520 Hz square wave. Similarly impressive results have been achieved when waking children

(Bruck, Reid, Kouzma, & Ball, 2004), people with hearing loss (Bruck & Thomas, 2007c), and the alcohol impaired (Ball & Bruck, 2004a; Bruck, et al., 2007).

The greater effectiveness of the 520 Hz square wave has been explained by signal advantages at the sensory and perceptual processing stages (Bruck, et al., 2009). The response to auditory stimuli is commonly conceptualised as a two stage process. First, sensory processing of the physical attributes of the sound, such as the loudness, pitch, and perceived duration take place within the auditory periphery (Yost, Popper, & Fay, 2007). Loudness is a particularly important physical characteristic at this level (Coenen & Drinkenburg, 2002). However, recognition of the sound requires a higher level of cognitive processing. The auditory stimulus then travels to auditory structures in the cortex where a conceptual interpretation of the sound is developed (Moore, 2007). At the cognitive level, the relevance of the auditory stimulus drives recognition (Langford, Meddis, & Pearson, 1974). Therefore, both sensory and perceptual processes are required in order for a sound to be perceived and different sound characteristics are important at each level. Sensory processing of the 520 Hz square wave is enhanced due to the effect of bandwidth on loudness perception. A square wave consists of a fundamental (f) and an infinitude of odd only harmonics of overall decreasing volume level. Thus the frequency peaks are at f, 3f, 5f, 7f, 9f etc. The various frequency peaks of the wave pattern lie more than a critical band width apart and this creates a loudness summation, giving the impression that the sound is louder, although this is not reflected in sound meter levels (Zwicker, Flottorp, & Stevens, 1957). This arises because the different frequencies activate different parts of the basilar membrane (Zwicker, et al., 1957). Perceptual processing is improved due to the pitch of the signal. Low frequency sounds, such as the human voice, are more meaningful because greater exposure to the human voice creates more opportunities to create meaning (Gibson, 1979).

The 520 Hz square wave has three other important advantages over high frequency pure tones. First, low frequencies travel through doors and walls more effectively (Quirt, 1985). This occurs because energy from high frequencies are more likely to be reflected rather than transmitted (Quirt, 1985). Second, older adults will have more trouble hearing a high frequency sound due to high frequency hearing loss typical of normal aging (Cruickshanks et al., 1998). Finally, a signal with multiple frequency components, such as a square wave, is less likely to be masked

than one with fewer or one frequency component, such as a pure tone (Lawrence, 1970). Sound masking occurs when the perception of one sound is affected by the presence of another, often rendering one of the sounds inaudible (Yost, et al., 2007).

The 520 Hz square wave is currently mandated in the United States for all sleeping rooms occupied by people with mild to severe hearing loss (NFPA Code 72, 2010; NFPA Technical Committee, 2009). From 2014 the National Fire Protection Authority (NFPA) code will require it in all commercial sleeping rooms. Given the success of the 520 Hz square wave there exists an argument to change the smoke alarm signal in residential buildings as well; however, the cost involved in widespread retrospective change would require greater literature support in two areas: (1) replication of the relatively small number of studies completed to this point; and (2) research on other potentially at-risk groups, such as older adults who take sleeping medication.

Factors That Affect Auditory Arousal Thresholds

There are a wide range of factors that affect the auditory threshold of a person while asleep, including alcohol, sleep deprivation, hearing loss, sex differences, depression, or the sound environment. A thorough appraisal of these variables has been made in two earlier papers (Bonnet, 1982; Bruck, 2001). This discussion will be limited to arguably the three most critical factors, which are, in order of importance, individual differences, age, and sleep stage (Bruck, 2001).

Individual differences play a leading role in explaining differences in arousal thresholds, possibly more significant than sleep stage or age (Bruck, 2001). In a reliability study of 35 participants from two independent studies, a strong level of consistency in auditory arousal thresholds was found within sleep stage, within night, between sleep stages, and between nights (Bonnet, Johnson, & Webb, 1978). However, arousal thresholds when asleep were not correlated with auditory thresholds when awake, which suggests that individual differences in sleep depth not associated with hearing ability may explain the discrepancy. Other authors have also implicated this distinction (light compared with deep sleepers) between participants

(Johnson, Church, Seales, & Rossiter, 1979; Mendelson, James, Garnett, Sack, & Rosenthal, 1986; Mendelson, Martin, Stephens, & Giesen, 1988).

Age related changes explain less of the variance in arousal thresholds than individual differences but are more important than variations in sleep stage (Bruck, 2001). The research has found that the probability of responding to auditory signals increases with advancing age (Zepelin, McDonald, & Zammit, 1984). Age related changes have been shown to be most prominent in stage 4 sleep awakenings, and become apparent at the beginning of the fifth decade (Zepelin, et al., 1984).

The third most important variable is sleep stage. Research has consistently shown that arousability decreases with increasing sleep depth (Bonnet, 1982, 1986; Portas, et al., 2000; Rechtschaffen, Hauri, & Zeitlin, 1966). Since there is a larger proportion of SWS during the initial cycles of the sleeping period (Kryger, et al., 2005), arousal thresholds are likely to be higher during the first part of the night (Bruck, 2001).

Arousal Thresholds and Hypnotics

The consumption of hypnotics is also likely to be an important factor modulating arousal thresholds due to their stated goal of reducing responsiveness to external stimuli, such as alarm signals. Hypnotics influence auditory recognition at both the sensory and perceptual levels, which may explain any hypnotic effect in arousal thresholds. Benzodiazepines impede sensory processing by enhancing the actions of the chief inhibitory neurotransmitter, gamma-aminobutyric acid (GABA), which controls the excitability of neurons, and is present in a range of auditory structures, including the auditory cortex (Clark, 1996). Perceptual processing is then moderated via changes in attention. In determining the relevance of a stimulus, the brain must process some information whilst ignoring other interfering information. This process of selective attention has been shown to be sensitive to neural inhibition (Clark, 1996; Walley & Weiden, 1973), which is altered after the consumption of benzodiazepines.

Only a small number of authors have investigated the effects of hypnotics on arousal thresholds (Hartse, Thornby, Karacan, & Williams, 1983; Johnson, et al., 1979; Spinweber & Johnson, 1982), and just one study has examined these effects in response to a standard smoke alarm signal (3100 Hz sine wave). Johnson, Spinweber, Webb, & Muzet (1987) considered the sedative effects of a benzodiazepine (triazolam, 0.25 or 0.5 mg) on the ability to wake sleepers suffering from sleep onset insomnia to the sound of a standard smoke alarm (3100 Hz sine wave). Three one-minute 78 dBA alarms were presented during deep sleep, about two hours after drug intake, and it was found that 50% failed to respond to any of the three alarm presentations, compared to 100% positive response after a placebo.

The work of Johnson et al. (1987) and other authors in this area was pioneering, but the methodology applied in these experiments was potentially problematic in four areas. First, a critical variation relates to sample selection. Inclusion criteria have been inconsistent. Two studies excluded poor sleepers (Bonnet, Webb, & Barnard, 1979; Hartse, et al., 1983), while the remaining four studies (Johnson, et al., 1979; Johnson, et al., 1987; Mendelson, et al., 1988; Spinweber & Johnson, 1982) included poor sleepers. Where poor sleepers were recruited, there was an inconsistent operationalisation of what constituted a poor sleeper. The pattern and duration of sleep complaints was not consistent across studies. For example, in one study sleep complaints for a period of 12 months was a pre-requisite (Mendelson, et al., 1988), while in another a period of only six months was required (Johnson, et al., 1987). It is possible that this was due to the absence of explicit operational criteria in the diagnostic manuals at the time these studies were conducted (Edinger et al., 2004). The consistent definition of insomnia has improved immeasurably since. The larger sample selection problem was the lack of generalisability to users of hypnotics. None of the previous studies recruited samples that were representative of users of hypnotics. The demographic profile of users of hypnotics is quite different from the samples that were typically recruited. Participants were usually younger males, while users of hypnotics are characteristically older females (Woodward, 1999). In fact, participants with a history of hypnotic usage were screened out in the vast majority of cases. This is potentially damaging because the reaction to naive benzodiazepine ingestion would likely be more potent (Stahl, 1996) resulting in higher arousal thresholds. A point acknowledged by Johnson et al. (1987) when explaining the marked reduction in failed awakenings from the first

arousal night to the second (50% cf. 37.5%). There is also evidence that long term benzodiazepine usage may have a deleterious impact on the auditory system (Gallager, Rauch, & Malcolm, 1984; Morand-Villeneuve et al., 2003).

Second, the hypnotics used in previous research are not necessarily representative of contemporary prescription practices. Earlier work on hypnotics and their impact on arousal thresholds was conducted at a time when the pattern of benzodiazepine prescription was turbulent and evolving. During the early 1980s widespread benzodiazepine usage was garnering public attention after an increasing number of documented cases of abuse and misuse (Tone, 2005). Over time the varied safety profile of different hypnotics was instructive in changing prescription practices. For example, flurazepam and triazolam were the two most frequently tested drugs in the six studies cited earlier investigating hypnotics and arousal thresholds. At the time of publication these two drugs were commonly prescribed, but are now rarely used because of a range of health concerns (American Psychiatric Association, 1990; Lader & Russell, 1993). However, triazolam is still used in some cases.

A third problem related to situational context. All of the previous work was conducted in a sleep laboratory. Participants usually slept in sound proof, air conditioned rooms, hooked up to an EEG machine. They were typically asked to refrain from napping or drinking alcohol. A major benefit of this method is the control of variables that may influence arousal. This may explain the large differences cited in Johnson et al.'s (1987) research. However, an artificial environment may elicit a response from participants different to what would be expected in a more naturalistic setting.

Finally, all of the previous work on arousal thresholds and hypnotics has been conducted on younger adults. The absence of older adults, which is a demographic principally at risk due to their higher likelihood of both consuming sedatives and being involved in a fire emergency (Hall, 2005; Smith & Tett, 2009), also limits generality claims. In particular, older adults are significantly more likely to be suffering from sleep maintenance insomnia (Dijk, Duffy, & Czeisler, 2001; Webb & Campbell, 1980), which may have special implications in the context of waking to an alarm during the night and taking more, longer acting hypnotics. Emerging

etiological explanations of insomnia predict a difference in baseline arousal thresholds between the various subtypes of insomnia. Hyperarousal theory suggests that cortical arousal is higher early in the morning after sleep onset in people suffering from sleep maintenance insomnia (Bonnet & Arand, 1997; Lack, et al., 2008). Greater levels of cortical arousal are associated with lower waking arousal thresholds (Bonnet, 1986; Portas, et al., 2000; Rechtschaffen, et al., 1966). Therefore, more research is required on the impact of hypnotics on arousal thresholds that addresses these issues.

Good and Poor Sleepers

The theoretical objections discussed above can also be extended to other arousal studies that have attempted to distinguish good and poor sleepers from one another. Prior research has often found no significant differences in arousal thresholds between people suffering from insomnia and normal sleepers (Johnson, et al., 1979; Mendelson, et al., 1986; Zimmerman, 1968); however, this research has also typically recruited younger adults experiencing sleep onset difficulties, rather than older adults suffering from sleep maintenance insomnia, who may be more vulnerable to nocturnal arousal due to a predisposition to arousal when asleep early in the morning. Hyperarousal does not predict a difference in the ability to wake to an alarm after sleep onset when comparing normal sleepers and patients troubled by sleep onset insomnia. However, an important avenue of investigation would involve comparing the arousal thresholds of older adults with sleep maintenance insomnia with normal sleepers.

Fire Fatality Statistics

This section examines a broad range of documented risk factors in accidental residential fires. These factors include, age, sex, smoke related materials, people presence in home, sleep/wake, location, conditions preventing escape, smoke alarm presence/operation, drug and alcohol intake, mental illness, physical illness, and time of fire. A gap is identified in the level of research devoted to understanding psychoactive drug consumption as a risk factor, and its relationship with other risk factors. It is highlighted that little is effectively known about the role of drugs in a fire emergency despite their ability to significantly impair cognitions. Particular attention is given to coronial data as it is the primary method of data collection in this study.

Age

We require less sleep as we age, but our risk of dying in a fire changes with age. In fact, age is a leading risk factor in fire fatality. Victims are more likely to be under five or over 65 years of age, and there is often increasing risk with increasing age in the senior years (Barillo & Goode, 1996a, 1996b; Brennan, 1998; Copeland, 1985; Gormsen, Jeppesen, & Lund, 1984; Karter, 1986; Marshall et al., 1998; Runyan, Bangdiwala, Linzer, Sacks, & Butts, 1992; Wasiak et al., 2009). Barillo and Goode (1996a) analysed 727 fire fatalities occurring within the State of New Jersey and noted that victims under the age of 11 years or over the age of 70 years constituted 22.1 per cent of the state population but 39.5 per cent of all fire fatalities. Children between the ages of two and four had the highest fatality rate (Barillo & Goode, 1996a). Similar results with respect to older adults have been recorded in Australian coronial files (Graesser, Ball, & Bruck, 2009). The pattern of risk at different life stages is presumably a consequence of age related physical or cognitive limitations/impairments that are common in young children or older adults (e.g., immobility, limited appreciation of fire hazards, or restricted sensory responsiveness).

Sex

Another important demographic risk factor is sex. The frequency of male fire fatalities is consistently reported to be between 50-100% higher than female fire fatalities (Brennan, 1998;

Buyuk & Kocak, 2009; Chien & Wu, 2008; Graesser, et al., 2009; Holborn, Nolan, & Golt, 2003; Marshall, et al., 1998; Rogde & Olving, 1996; Ronald & Jerry, 1982; U.S. Fire Administration, 1999). Data from the National Fire Incident Reporting System (NFIRS), which represent the world's largest, national, annual database of fire incident information, indicate that over the past two decades in the United States males have continued to have almost twice as many fire deaths as females in nearly all age groups (U.S. Fire Administration, 1999). Sex differences in risk taking behaviour provide a compelling explanation for this trend. Males typically engage in more high risk behaviours than females, which may lead to death more often (Redeker, Smeltzer, Kirkpatrick, & Parchment, 1995). Results from other geographical regions are consistent with this interpretation. The male bias is accentuated in certain areas such as the Scandinavian region, that exhibit excessive male risk taking behaviour, notably high alcohol use (Honkanen, Koivumaa-Honkanen, & Smith, 1990).

Smoking Related Materials

Together with demographic factors, misuse of smoking related materials, such as discarded cigarettes, lighters, or matches, contribute to fire fatalities (Diekman, Ballesteros, Berger, Caraballo, & Kegler, 2008; FEMA, 1999; Hall, 2005; Holborn, et al., 2003; Newton, 2003). In the United States, smoking is the fifth most frequent cause of residential fires, the leading cause of fire fatalities, and the second most common cause of fire-related injuries (U.S. Fire Administration, 1999). Similar trends exist around the developed world (Miller, 2005). A persuasive explanation for these results is the influence of human error (Holleyhead, 1999; Miller, 2005). For example, the careless disposal of a lit cigarette will often occur when the victim is sleeping or intoxicated.

Recent societal and legislative changes may diminish the role of smoking related materials in fire ignition. There has been a general decline in the incidence of smoking in Australia since 1945 (Winstanley, Woodward, & Walker, 1995). Currently, less than one in five Australians smoke (Australian Institute of Health and Welfare, 2011). The decrease in the number of smokers is likely to impact positively on smoking related fire fatalities. In the United States, it has been estimated that a one percent reduction in the prevalence of current smokers would result in a

seven percent decrease in fire related fatalities from smoking (Diekman, et al., 2008). Similarly, self-extinguishing cigarettes are now mandatory in Australia, many states in the United States and in many other parts of the world. However, at this stage no evidence currently exists on the efficacy of this change and this will not alter the underlying behaviour patterns that lead people to smoke carelessly, which may result in the misuse of other smoking related materials, such as matches. Overall, while smoking related materials remain the leading cause of fatal residential fires, there is a reasonable expectation of change in the future.

People Presence in Home

The presence of other people during a fire emergency can greatly enhance your chances of survival. The vast majority of residential fires involve individual fatalities (Brennan & Thomas, 2001). Living alone is a significant risk factor of fire fatality (Leth, Gregersen, & Sabroe, 1998; Marshall, et al., 1998; Newton, 1998; Warda, Tenenbein, & Moffatt, 1999). In Denmark, Leth et al. (1998) found that the majority (57%) of victims in fatal fires lived alone. Similarly, Fahy and Molis (2004) reviewed 72 fatal fires where a smoke alarm operated and observed that 47% were home alone when the fire started. The primary reason for this risk is that people living alone, typically older adults, have less resources to deal with a fire emergency (Brodzka, Thornhill, & Howard, 1985).

There are times when additional people can be counter-productive in a fire emergency. Multiple fatality fires often involve children, who are unable to help themselves and depend upon their parents for escaping a fire (Runyan, et al., 1992). Parents often misjudge the speed at which fires develop and do not have enough time to evacuate their children, which may compromise their decision making ability in an emergency (e.g., retreating back into the fire to rescue a child when it is too dangerous to engage in a rescue attempt), or simply reduce the amount of time or energy they have to effect their own escape (Newton, 1998). Furthermore, children often respond to a fire by hiding, particularly when they have been involved in the fire, decreasing the likelihood of being saved (Miller, 2005). Certain parents are more at risk than others. Single parents, parents with a disability, or parents who are intoxicated during the fire are particularly vulnerable (Holborn, et al., 2003; Runyan, et al., 1992; Scholer, Hickson, Mitchel, & Ray, 1998). This is

likely due to their compromised capacity to respond to a fire emergency (Holborn, et al., 2003). In the case of single parents, they are compromised by a lack of support when compared with dual parents when dealing with their child or children. Consequently, the number of people on location at the time of fire onset can help or hinder an effective response to a fire emergency.

Sleep/wake

Sleeping during a fire is a well known risk factor (Barillo & Goode, 1996a; Brennan, 1998; Karter, 1986; Runyan, et al., 1992). Brennan (1998) analysed coronial reports on 114 fire fatalities and found that victims were two times more likely to be asleep (67% cf. 33%). Moreover, almost nine out of ten (86%) victims who died in a fire at night (8pm to 8am) were reported to be sleeping (Brennan, 1998). The majority (75%) of sleeping victims did not move from their original location, which suggests they either did not respond to the emergency or were not given enough time to respond adequately (Brennan, 1998). Sleeping during the day also presented a risk. Thirty-one percent of victims who died during daytime hours (8am to 8pm) were asleep (Brennan, 1998). So being asleep impacts our responsiveness to a fire emergency, which likely leads to fire fatalities, at any time of day.

Location

The location of the victim at the time of fire onset is an important risk factor in fire fatalities. Kitchens, bedrooms and lounge areas are the most likely locations for a fire to originate (Ahrens, 2007; Baker & Adams, 1993; Bounagui, Benechou, & Victor, 2004; Newton, 2003). Ahrens (2007) noted that more than six out of ten fatal fires in the United States during 2000 to 2004 occurred in the lounge area (24%), bedroom (23%) or kitchen (15%). Additionally, an analysis of Victorian coronial files found that almost eight out of ten victims were located in the room of fire origin at fire start (Bruck & Thomas, 2010). Behaviour prior to ignition and unique to the various rooms is commonly used to explain these results. For example, victims may ignite their clothing when cooking in the kitchen. Over half of the fire fatalities in London between 1996 and 2000 originating in a living room or bedroom were due to smoking related materials

(Holborn, et al., 2003). Therefore, the location at the time of fire onset is an important risk factor that relates strongly to behaviour prior to ignition.

Conditions Preventing Escape

An environmental factor or the very rapid progress of the fire may prevent an easy escape and increase the risk of a fatality. The physical characteristics of a construction provide the setting for a fire emergency. Unlike commercial developments, most residential buildings have not been engineered for a fire emergency (Parker, Sklar, Tandberg, Hauswald, & Zumwalt, 1993). Consequently, barred windows, locked doors, or other impediments often encumber escape attempts (Brennan, 1998; Rhodes & Reinholdt, 1996).

Fire progress, which is often a function of building characteristics, is another factor limiting escape. The spread of fire beyond the room of origin is a key element in fire fatality (Newton, 1998). In approximately eight out of ten cases this occurs after flashover (Newton, 1998). A flashover is the near simultaneous ignition of all combustible material in an enclosed area (Newton, 1998). It is exposure to this violent and destructive event that hinders many escape attempts. Flashover is more likely to occur late at night or early in the morning when fires are allowed to smoulder for hours unattended (U.S. Fire Administration, 1999). In additional to building characteristics, a fire can also progress quickly because the fire has started on the victim (Bruck & Thomas, 2010). This may occur due to the ignition of flammable materials on the person, such as clothing or cleaning fluids. Generally, environmental factors and fire progress are important risk factors in residential fire fatalities.

Smoke Alarm Presence/Operation

Smoke alarms are the most common safety precaution adopted by Australian households (Australian Bureau of Statistics, 2007). The absence of a working smoke alarm has often been highlighted as an important risk factor in fatal fires (Barillo & Goode, 1996a; Marshall, et al., 1998; Newton, 1998; U.S. Fire Administration, 1999), although the veracity of this assertion is currently being tested. In a major nationwide review of fire fatality statistics in Australia under

the auspices of high level government bodies, Newton (1998) found that where the presence or absence of a smoke alarm had been recorded, the vast majority (97%) of structures did not have a smoke alarm at the time of a fatal fire. However, the presence or absence of a smoke alarm was missing from almost two thirds of the sample and a range of structural fires where a smoke alarm would not be expected to be installed were included in the analysis (e.g., caravans and campervans). Similarly, flaws in other supporting evidence have also surfaced (Crapo, 2000; International Association of Fire Fighters, 2008), and experimental testing has shown that the current standard smoke alarm is ineffective in certain vulnerable groups within the population. See the section on smoke alarm development and current standards for a more complete review of smoke alarm performance issues.

Drug and Alcohol Intake

Psychoactive drug and alcohol usage may also impair a person's ability to escape from a fire emergency, and represent conditions that prevent escape that exist in the person rather than the environmental factors discussed earlier in this section. Alcohol impairment in fire fatalities has been the subject of intense research (Ball & Bruck, 2004a; Barillo & Goode, 1996a; Berl & Halpin, 1978; Bruck, Ball, & Thomas, 2011; Marshall, et al., 1998). A study of fires in predominantly rural areas in the United States found that the presence of an alcohol-impaired person was the strongest independent risk factor for death (Runyan, et al., 1992). Similarly, the fire mortality risk for alcoholics is 9.7 times higher than the rest of the population (Schmidt & De Lint, 1972). Alcohol intake has also been shown to have an impact on other behavioural and environmental risk factors, such as age, sex, smoking, and conditions preventing escape (Bruck, et al., 2011). The role of alcohol has been attributed to its effects on physical coordination, cognitive impairment, impaired sensory perception, and risk taking behaviours (Miller, 2005).

While alcohol is implicated in fire deaths, psychoactive drug consumption may also be a potential risk factor, but this has not yet been established with certainty. Psychoactive drugs alter moods or perceptions, which can impair a person's ability to respond to a fire emergency (Stahl, 1996). Psychoactive drugs are frequently prescribed for medical disorders, such as insomnia, but are also used illicitly for recreation. However, there is a paucity of data on

psychoactive drug usage in fire fatalities, and certainly very little data, if any, on how psychoactive drugs work in conjunction with other fire factors. This is often because illegal drugs are not openly discussed with authorities, toxicology tests are not conducted, or are conducted after the administration of treatment for burns or other fire related injuries (Bruck, 2001).

A limited collection of work does exist. Barillo and Goode (1996a) identified illicit recreational drugs as a potential risk in fire fatalities; however, other psychoactive drugs have not been examined. Given the differences in impairment profiles between different psychoactive drugs, this remains an important area of clarification. Psychoactive drug usage has also been implicated in the accidental death literature. A number of studies have investigated mental illness and accidental death and found that not only are people suffering from a mental illness more likely to die in an accidental death, but they are more likely to have drugs in their system at the time of death compared with people who are not mentally ill (Bayard-Burfield, et al., 1998; Gau & Cheng, 2004; Holding & Barraclough, 1975, 1977; Watts-Hampton, et al., 2006). However, psychoactive drug usage has never been analysed directly as a key risk factor in fire fatalities in an Australian sample. Moreover, the specific impact of sedatives on fire fatalities has never been directly investigated before.

Any potential risk from taking drugs or alcohol is also likely to increase when substances are taken together. Drug and alcohol usage commonly co-occurs with problematic consequences. A recent study in the United States found the 12-month prevalence of alcohol use disorders among those with 12-month drug use disorders to be as high as 55 percent (Stinson, Grant, & Dawson, 2005). Co-morbid users of drugs and alcohol are at risk of the typical complications from the individual substances, but may also be presented with possibly more devastating interactive effects. For example, the use of alcohol with sleeping pills or sedatives may increase the risk of fatal poisoning (Koski, Ojanpera, & Vuori, 2002).

Mental Illness

Mental illness is a construct that is complex to define. One definitional approach that balances flexibility and precision comes from an article written by Beumont and Carney (2004), where mental illness is defined as a condition that seriously impairs, either temporarily or permanently, the mental functioning of a person both in thoughts and or behaviour. Mental illness has consistently been linked with premature accidental death (Eastwood, Stiasny, Meier, & Woogh, 1982; Holding & Barraclough, 1975, 1977); however, little research has been conducted in the area of fire fatality. A recent Australian study examined coronial data in the state of Victoria for the period from February 1998 to June 2005 and observed that more than half (55%) of victims were suffering (definitely or probably) from a mental illness prior to their death (Watts-Hampton, et al., 2006). This result may be a consequence of deficient coping skills that may lead to irrational actions and behaviours during the fire (Watts-Hampton, et al., 2006). It should be noted that in Watts-Hampton et al.'s (2006) study alcohol dependency was considered a mental illness, consistent with the latest version of the DSM (American Psychiatric Association, 2013), and this was a contributing factor to a significant number of deaths. The link between alcohol usage and fire fatalities is well established and has been discussed in more detail earlier on in the section.

The above results present a strong case for mental illness having a role in many fire fatalities, but more research is required to determine the magnitude of effect. Opposing evidence exists. An analysis of 117 unintentional dwelling fire deaths in London showed that just five percent of the victims suffered from mental illness or handicap (Holborn, et al., 2003) – a result significantly below the aforementioned 55%. This study has been found wanting methodologically. The data is sourced from the London Fire Brigade, which relies on investigators unlikely to have access to the victims' medical records to produce the reports. In this situation, the victims' personal circumstances are only noted at the investigators' discretion, making a true assessment of incidence difficult. The many definitions of mental illness would only make this process more demanding. Furthermore, only those victims with a medical history of a diagnosed mental illness would have been included in the group defined as mentally ill. Considering these limitations, the incidence of mental illness among fire victims is likely to be closer to the

assessment made by Watts-Hampton (2006). Nevertheless, while mental illness is likely to be important as a cause of fire risk, the small number of studies in the area and imprecise operationalisation of important terms promotes caution when trying to understand how pervasive mental illness is in fire fatalities.

Physical Illness

A physical disability can be a significant impairment during a fire emergency (Marshall, et al., 1998; Miller, 2005). The level of impairment is often a function of the type of physical disability. Speed of action is imperative when responding to a fire. Therefore, mobility impairment would significantly prevent the ability to escape from a fire emergency. Furthermore, a respiratory problem may hasten the time taken to succumb to smoke inhalation.

Time of Fire

Non-fatal fires are more probable during the daytime, while it is more likely that a fatal fire will develop during the early morning sleeping period (Ahrens, 2011b; Miller, 2005; Newton, 1998). A disproportionate number of fires occur between midnight and 8:00 am (Newton, 1998). The primary explanation for this finding is that victims are more likely to be asleep, reducing the time to escape and increasing the chances of exposure to a high concentration of carbon monoxide, which is often the cause of death (Ahrens, 2011b; Miller, 2005; Newton, 1998). The relationship between the time of day and hypnotics, which are normally taken prior to bedtime, makes a more detailed investigation of the impact of hypnotics on the response to a fire emergency particularly relevant and important.

Accident Causation and Human Error

This section examines the different approaches to explaining accidents, with a particular emphasis on human error. The difference between organisational and domestic accidents is explored and it is postulated that fire accidents are somewhat different and more complex compared with regular accidents in the home. Attention is drawn to the scarcity of research addressing both domestic accidents and using a human factors approach in the fire literature. Finally, the section concludes by providing a contemporary review of the way human error can be managed.

Accidents are generally defined as unfortunate incidents that happen unexpectedly and unintentionally, typically resulting in damage or injury (The Oxford dictionary, 1997). This type of definition presents a number of problems. First, intentionality is a philosophically loaded concept that can be difficult to establish (Searle, 1983). Second, accidents are often very predictable despite the implication of randomness (Quinlan, Bohle, & Lamm, 2010). It is precisely the ability to anticipate accidents that allows models of accident causation to be developed (Quinlan, et al., 2010).

The emphasis placed on various sources of injury has changed over time as new theories of accident causation have been developed. The disciplines of ergonomics, engineering, sociology, and psychology have all made significant contributions to models of injury causation. In the area of fire investigation, the ergonomic and safety engineering fields have traditionally been active contributors (Miller, 2005). The primary concern of these technical areas has been with potential sources of injury in the physical environment (Quinlan, et al., 2010). This position places less emphasis on human activity in the ignition and growth of fires. In isolation, this narrow view of injury provides an incomplete understanding of accidents, which are often a result of a combination of causes, whether human or technological (Brennan & Thomas, 2001; Rasmussen, 1985). Increasingly more importance in the literature is placed on psychological explanations that investigate individual behaviour and human error (Brennan & Thomas, 2001; Bryan, 2003).

The law of unintended consequences states that almost all human activity has at least one unintended consequence (Merton, 1936). Human errors can be defined as any human action that in failing to achieve the desired result leads to unwanted consequences (Salvendy, 1987). Error in human activity is believed to explain the vast majority of accidents (Quinlan, et al., 2010).

Two theoretical approaches to understanding human error are the systems approach and the person approach. The systems approach considers error to be a function of the conditions residing within the system (Reason, 2000b). Human error is not treated as the primary cause of the accident but simply a consequence of the circumstances. In contrast, the person approach holds individuals responsible for their unsafe acts and views human error to be a function of distorted cognitive processes, such as inattention, negligence, or forgetfulness (Reason, 2000b).

During the past several decades there has been a shift away from the person approach towards a more systems approach that focuses on collective responsibility rather than individual responsibility (Quinlan, et al., 2010). A systems approach implicates upstream factors. Upstream factors are preceding variables in the casual chain of events that may have led to an accident. Upstream factors at increasingly broader levels are now implicated in accidents, but this can create a problem. Reason (2000b) contends that remote factors, such as society at large, lack causal specificity, are generally outside the control of relevant stakeholders to the accident, and their impact can be shared by many systems. For these reasons, the approach adopted in this research was primarily person centred, but not to the exclusion of system inspired insights where they seemed appropriate.

The most significant model adopting the person approach is Reason's (1990) influential taxonomy of human error, the generic error-modelling system (GEMS). GEMS proposes three fundamental types of error: (a) skill-based slips or lapses; (b) rule-based mistakes; and (c) knowledge-based mistakes. This model is an extension of Rasmussen's (1979) earlier work, and relates the error types back to their cognitive origins while also explaining the mechanisms behind switching between levels. Skill-based errors are the most common and occur when executing familiar, automatic, or procedural tasks (e.g., a typing error). These errors can be slips or lapses. Slips are defined as errors where the intention is correct but there is a failure to

execute the required activity, while lapses are monitoring failures that implicate memory (Reason, 1990). Rule-based mistakes involve a failure to follow guidelines learnt either implicitly or explicitly, such as incorrectly following a checklist (Reason, 1990). When rules or experience do not apply, knowledge-based mistakes can arise when improvisation is required in novel circumstances (e.g., driving a car for the first time). Both types of mistakes (rule or knowledge-based) are seen to be cognitive planning failures (Reason, 1990).

Skill-based mistakes typically occur prior to the detection of a problem, while rule-based and knowledge-based errors often take place during subsequent attempts to solve the problem (Reason, 1990). The GEMS framework proposes that solutions to all problems will first be sought at the rule-based level. The pursuit of a rule-based solution is often perpetuated by an underestimation of the danger of the hazard (Breakwell, 2007). Only if the rule-based answer is insufficient will an attempt be made to find a solution at the more taxing knowledge-based level. Although errors at the skill-based level are more numerous, the opportunities for error are greatest at the knowledge-based level as these tasks are more challenging and difficult to correct (Reason, 1990). In this sense, a risky situation that is created after a simple skill-based error may encourage further mistakes when attempting to solve the problem at a higher level. Consistent with this approach, individuals performing in novel situations have been shown to be error prone (Dietz & Thoms, 1991; Grasso, Rothschild, Jordan, & Jayaram, 2005).

Reason (1990) also discussed a second level of human failure distinguished from error, termed violation. A violation is defined as any behaviour that deviates from accepted norms, rules, or procedures (Reason, 1990). Violations can be either deliberate or unintentional.

Accidents in the Home

Domestic accidents, such as residential fires, have attracted considerably less attention in the literature compared with organisational accidents despite approximately one third of all adult injuries occurring in the home (World Health Organisation, 2005). This may be due to organisational financing of research projects driven by legal liability issues. Another explanation is that organisational accidents are typically larger with respect to the injury or death toll and

receive greater coverage, such as the partial nuclear meltdown that occurred at the Three Mile Island power plant in Pennsylvania in 1979, which was said to inspire Perrow's (1984) theory on normal accidents in complex systems several years later.

Individual accidents that typically occur in the home often have distinguishing characteristics from organisational accidents. Reason (2007) argued that individual accidents normally occur in circumstances where the victim or group of victims are intimately involved in the creation of the hazard. The damage to the parties involved may be terminal, but there are not usually widespread consequences. Most individual accidents are deemed to be errors, frequent in nature, at the skill-based level with limited causes. Due to the proximity between the hazard and the victim the defences normally are limited or non-existent. In contrast, organisational accidents usually occur within complex systems as a result of multiple causes (Reason, 2007). These types of accidents are often rare but there can be widespread consequences.

Reason's (2007) contention that domestic accidents are less complex than organisational accidents is possibly less applicable to residential fire accidents for three reasons. First, the most common accidents in the home are falls (Devroey, Van Casteren, & Walckiers, 2002). A fall would be classified as a slip that does not involve higher order thinking after being detected as it occurs instantaneously and is normally unable to be corrected. On the contrary, during a residential fire, with a few exceptions, there is often a limited opportunity to escape that typically requires rule or knowledge-based decision making under duress. Complexity emerges in this brief period after the detection of a fire that is not characteristic of many other individual accidents, particularly if the victim is impaired. Second, although the cause of a fire may be rudimentary compared to a nuclear meltdown or other catastrophic organisational accidents, the necessary ingredients for a fire are a step above the cause of most domestic accidents. In order to ignite, a fire requires oxygen, a fuel source, and an ignition source, while a fall or a poisoning are arguably much simpler accidents to create. Finally, a simple skill-based error may have created the error, but often there is a complex set of circumstances that created the conditions for error to manifest itself, consistent with a more systems perspective approach to accident causation. Compared to other domestic accidents, victims of fire seem to be exposed to a greater number of risk factors than victims of other accidents (Devroey, et al., 2002). For example,

mobility loss appears to play a critical role in most falls, which may be why functional mobility screening tools have been shown to have such a high degree of predictive validity (Tiedemann, Shimada, Sherrington, Murray, & Lord, 2008). However, it is relatively more difficult to attribute fire fatalities to one factor or category of factors. It is also important to consider that even if an environment is comparatively low risk, the resultant carelessness and inattention to the environment may create an even more dangerous or complex scenario compared to a high risk organisational environment where there is a heightened awareness of potential dangers (Strohschneider & Gerdes, 2004). Overall, residential fires should be treated as a relatively complex type of domestic accident.

As most domestic accidents occur during motion (Devroey, et al., 2002; Reason, 1990), trends in human activity patterns are an important consideration. Fire fatalities during periods of sleep are a notable exception already identified, but even in many of these cases, the actual error that caused the accident may have occurred during movement prior to sleep (e.g., smoking materials left unattended). Human activity largely takes place during the daytime, around feeding or work routines, because of various social and physiological influences (Hawley, 1950). Traditionally, women have had greater social and domestic demands placed on them, which has meant they are more likely to be at home engaging in activity (Quinlan, et al., 2010). However, there is now a dispersion of activities away from the household, driven largely by the integration of women into the workforce. Those people that do remain at home during the day are usually the most vulnerable in the community (e.g., retirees or the unemployed). Often this level of disadvantage grows as more time is spent at home (Alwash & McCarthy, 1988).

Error Management and Safety Culture

Part of the problem is that it is more difficult to legislate safety in the home compared to public places (World Health Organisation, 1965). However, methods of managing error that have been devised for organisational settings can be applied to a domestic environment. Error management involves reducing the causal factors that induce error and minimising error when it does occur (Helmreich, 2000). This involves error prevention and error containment. Error prevention is the use of strategies to identify and prevent the incidence of errors (Reason, 2000a). Error

containment acknowledges that some errors are unavoidable and provides strategies to detect and correct errors, including the design of flexible systems that are tolerant of error (Reason, 2000a). The inability to prevent errors and their containment is the major premise in many accident causation models, such as Normal Accident Theory (Perrow, 1984).

Error management is the primary goal of a safety culture, which has received considerable support in the literature (Arden, 1993; Geller, 1994; Reason, 2007). Culture is defined as the system of shared beliefs and values that develops within a group and guides the behaviour of its members (Schein, 1990). Safety culture then refers to these attitudes, beliefs, perceptions, and values that the group may share in relation to safety (Arden, 1993). Errors in human behaviour are believed to be responsible for a sizeable number of accidents (Helmreich, 2000). Culture is very adept at controlling behavioural norms (Schein, 1990). Therefore, the creation of a positive safety culture can have a lasting impact on the attitudes, beliefs, perceptions, and values that the community share in relation to safety (Reason, 2007), which then would be expected to guide behaviour that is less likely to cause a fire emergency.

Research Aims

A paucity of research exists on the influence drugs that alter our cognitions may have in a fire emergency. Therefore, the overall aim of this research was to determine the impact psychotropic drugs may have on human behaviour during fire emergencies, with a particular focus on hypnotics due to their frequent use and potentially damaging impact on cognition in a fire context. This involved two related studies. In Study One (Chapter Two) the efficacy of current and alternative smoke alarm signals was tested after the consumption of hypnotics in older adults who are regular hypnotic users. This analysis revealed the extent of risk posed to users of hypnotics in responding to smoke alarms, which are currently the primary means of defence from a fire event. A supplementary objective was to examine the relationship between sleep quality and arousal thresholds. Study Two (Chapter Three) retrospectively analysed an Australian database of fire fatalities to determine: (a) if users of psychotropics and hypnotics were overrepresented in the Australian fire fatality statistics; and (b) the relationship between psychotropic drug consumption and a number of behavioural, environmental, and demographic risk factors previously identified in the literature or added if the variable was complementary to the analysis. The specific association between sedatives and the same behavioural, environmental, and demographic risk factors identified above was also analysed**.** The relevant objectives and hypotheses are included in the relevant chapters.

CHAPTER 2 – STUDY ONE – Arousal Thresholds

Brief Introduction

As smoke alarms are the most prevalent form of fire safety equipment and certain groups have been shown to be at risk of non-awakening (Thomas & Bruck, 2008), the impact of hypnotics on arousability was assessed in Study One against the current smoke alarm standards. Only one study, conducted more than two decades ago, has investigated the response to a 3100 Hz sine wave smoke alarm after ingestion of a hypnotic. Three one-minute 78 dBA alarms were presented during deep sleep, about two hours after intake of triazolam, and it was found that 50% of participants failed to respond to any of the three alarm presentations, compared to 100% positive response after a placebo (Johnson, et al., 1987). Consumers of any type of sedatives or other drugs were excluded from the study and the participants, all young males, were asked to refrain from napping and drinking alcohol. The study was conducted in a laboratory where participants slept in an air conditioned, sound proof room, and sleep was monitored using EEG. The methodology applied in this experiment was potentially problematic in four areas. First, the recruited demographic were not representative of the population group most likely to either perish in a residential fire or consume sedatives. The exclusion of users of hypnotics is particularly troubling given the reaction to naive benzodiazepine ingestion is often more potent (Stahl, 1996), resulting in higher arousal thresholds. Second, the data are less relevant in Australia. Triazolam is rarely prescribed in Australia due to possible amnestic complications and there are important differences in the sensory deficits produced by different sedatives. Third, the data were collected in an artificial sleep laboratory environment that may be expected to evoke responses that are very different from those elicited in a home context. This presents a threat to external validity. Finally, all of the previous work on arousal thresholds and hypnotics has been conducted on younger adults. The absence of older adults, which is a demographic principally at risk due to their higher likelihood of both consuming sedatives and being involved in a fire emergency (Hall, 2005; Smith & Tett, 2009), also limits claims of generalisability. In particular, older adults are significantly more likely to be suffering from sleep maintenance insomnia (Foley, et al., 1995), which may have special implications in the context of waking to an alarm during the night and taking more longer acting hypnotics. Emerging etiological explanations of

insomnia predict a difference in baseline arousal thresholds between the various subtypes of insomnia. Hyperarousal theory suggests that cortical arousal is higher in the second half of the sleep period in people suffering from sleep maintenance insomnia (Bonnet & Arand, 1997; Lack, et al., 2008). In contrast, in patients who have difficult falling asleep, elevated levels of arousal are present prior to the sleep period beginning (Bonnet & Arand, 1997; Lack, et al., 2008). Therefore, the first objective was to determine the extent to which hypnotic ingestion increased auditory arousal thresholds to the current smoke alarm signal (3100 Hz sine wave) in a representative group of users of hypnotics, who were older adults, using sedatives they would typically consume, in a naturalistic home environment. **It was hypothesised that hypnotic ingestion would significantly increase arousal thresholds compared to the no hypnotic condition (Hypothesis One).**

Since there is growing scepticism with regard to the current smoke alarm performance and new findings suggest that the 520 Hz square wave may usurp the existing equipment, arousal thresholds in users of hypnotics were tested against the 520 Hz square wave, and the results were compared against the current standards. A number of sources have provided evidence that the current smoke alarm is not effective as previously believed (Bruck & Thomas, 2010; Crapo, 2000; Thomas & Bruck, 2008). Research has raised the possibility that an alarm of a different frequency (520 Hz square wave) may be more effective for waking sleeping individuals than the current high pitched (3100 Hz sine wave) alarm (Bruck, et al., 2009; Bruck & Thomas, 2007b). The 520 Hz square wave is currently mandated in the United States for all sleeping rooms occupied by people with mild to severe hearing loss (NFPA Code 72, 2010; NFPA Technical Committee, 2009). From 2014, the NFPA code will require it in all commercial sleeping rooms. The new low frequency alarm has been successfully tested in a range of vulnerable groups (Ball & Bruck, 2004a; Bruck, 1999; Bruck, et al., 2004; Bruck & Thomas, 2007c; Bruck, et al., 2007), but no tests have been conducted on users of hypnotics. Given the widespread usage of hypnotics and their stated goal to make sleepers less responsive to both external (e.g., alarms) and internal stimuli (e.g., worrying thoughts), users of hypnotics are an important risk group to investigate. Therefore, the second objective was to establish if there were lower arousal thresholds to the 520 Hz square wave alarm compared to the current (3100 Hz sine wave) smoke alarm in older adults who consume sedatives when under the influence of their usual hypnotic.

It was hypothesised that there would be lower arousal thresholds to the 520 Hz square wave alarm in the hypnotic condition compared to the 3100 Hz sine wave alarm (Hypothesis Two).

In order to quantify the risk posed to users of hypnotics, the exact likelihood of waking to a 3100 Hz sine wave fire alarm at the Australian standard alarm intensity level of 75 dBA when under the influence of a hypnotic was tested as a third objective. These figures were then compared to the results for the 520 Hz square wave alarm in order to evaluate the relative risk of failing to wake to the Australian standard for both alarms. **It was hypothesised that hypnotic ingestion would increase arousal thresholds to above the standard alarm intensity (75 dBA) for the 3100 Hz pure tone for a proportion of participants, but no participants were expected to sleep through the 75 dBA standard alarm intensity for the 520 Hz square wave (Hypothesis Three).**

The fourth and final objective involved investigating sleep quality and arousal thresholds. Poor sleepers often report a sensitivity to external stimuli during sleep that disrupts their sleep patterns (Monroe, 1967). In spite of these claims, researchers have regularly failed to corroborate the subjective reports of poor sleepers with objective data from arousal studies (Bonnet, et al., 1978; Johnson, et al., 1979; Mendelson, et al., 1986). The previous empirical work was not guided by a detailed theoretical understanding of the problem. This limitation led to certain research design flaws, such as the recruitment of poor sleepers only suffering from sleep onset insomnia. This research now proposes a mechanism that may support differences in responsiveness as a function of sleep quality. Lower arousal thresholds amongst people suffering from sleep problems are directly implicated in hyperarousal theory (Bonnet & Arand, 1997), which attempts to explain insomnia pathophysiologically. Due to a heightened state of cortical arousal during sleep, poor sleepers who are suffering from sleep maintenance insomnia could reasonably be expected to be more responsive to auditory cues during the night.

Beyond establishing a tentative link between poor sleep and light sleep, it was hoped that this analysis would ascertain if sleep problems were a factor that may have contributed to inconsistency in arousal thresholds in previous research. Such a determination would highlight

the importance of the clear and consistent operationalisation of insomnia and its subtypes when measuring arousal thresholds in the future, particularly given the prevalence of sleep maintenance insomnia in the community.

This objective was tested using two different methods. The first method was a correlation analysis investigating the relationship between arousal thresholds and the self-reported severity of insomnia symptoms in intermittent hypnotic users, and to determine whether such a relationship may exist when they were on or off their medication, or both. **It was hypothesised that as self-reported insomnia symptoms increased arousal thresholds would decrease for both alarm signals (Hypothesis Four)**.

The second method was to compare the auditory arousal thresholds (for both types of alarm signals) of the poor sleepers recruited in this study when not consuming their hypnotic with the auditory arousal thresholds of approximate age matched good sleepers (data from Bruck and Thomas 2008a). Participants in Bruck and Thomas' (2008a) study were tested with the same signals under conditions of EEG determined deep sleep. **It was hypothesised that poor sleepers, when not under the influence of their hypnotic, would have significantly lower arousal thresholds compared with good sleepers for both alarm signals (Hypothesis Five).**

The decision to answer this objective with two methods was made in order to increase the confidence in the overall analytical outcome due to the respective methodological weaknesses of each alternative. The first method used a research design that is less robust than experimental designs because research variables were not controlled or manipulated (Klein, 1992). Similarly, the between-group design used in the second method was limited due to the methodological differences between the two groups being tested. The availability of data where the participants were matched approximately on age, tested with the same signals, and awoken during the first third of the night meant a comparison across studies would be a useful and justifiable exercise. Nevertheless, a number methodological differences between the studies rendered such an analysis potentially problematic. Both groups differed with respect to the inclusion of silences between signals, the control of sleep stage, the removal of naps, and the level of priming, which are all variables that would be expected to influence arousal thresholds. Therefore, the

combination of two distinct approaches to answering this problem was viewed as a useful way of increasing the level of confidence in the results.

Method

Participants

The experiment involved remotely monitoring participants' sleep patterns and arousal thresholds over 11 nights in their own home primarily to maximise the nights studied and increase the external validity of the study. The experimental method will be discussed in more detail later in the chapter. Ten females and two males aged between 65 to 80 years with an average age of 71 (standard deviation = 4.0) participated in the study. This compared favourably with the demographic age profile from the control study (data from Bruck & Thomas, 2008a; relevant to Hypothesis Five), where the mean age of the 45 adults aged between 65 to 83 was 73.1 years (standard deviation = 5.6). The two samples did differ in reference to sex. Bruck and Thomas' (2008a) research sample was comprised of 50% females, significantly below the 83% recorded in this study. Since females typically are more responsive to alarms when asleep, this may have lowered the arousal thresholds observed in this study compared to Bruck and Thomas' (2008a) research (Bonnet, 1982; Bruck, 2001).

The vast majority of the participants in this study were using temazepam. One participant was using the non-benzodiazpine, zolpidem and another participant was using nitrazepam. Most participants' self-rating of insomnia would be defined as sub-threshold insomnia.

Given the additional variability often present in small samples, a larger number of participants to maximize the power of the analysis had originally been targeted. However, a small sample size was evidently adequate as the effect size was sufficient to achieve significance at .05. A sample size of 12 is generally considered adequate for a repeated measures design (Tabachnick & Fidell, 2007). A larger sample size would have provided additional confidence in the results and allowed for comparisons across sub groups, such as sex, but was abandoned due to the difficulty in sourcing participants.

Three recruitment methods were employed. The first method involved placing advertisements in local newspapers. Details of the advertisement are attached as Appendix A. Participants were invited to contact a land-line number, which diverted to an answering machine. A brief message thanked participants for their interest in the study and requested that they leave their personal details in order for the researcher to contact them at a later date. Unfortunately, this method only yielded a very small number of responses. The second method involved cold calling older adult groups in order to arrange the presentation of a small report on sleep hygiene (attached as Appendix B). At this presentation interest in participating in the study was also canvassed. Project pamphlets were also distributed at these meetings (attached as Appendix C). The third recruitment method involved word of mouth through friends and family. In practice, this final method was the most successful, possibly due to the reciprocal nature of personal relationships. Recruitment was quite arduous and complicated by the high refusal rate. The prospect of waking someone with a history of poor sleep during the middle of the night proved too demanding for many potential participants. All participants were offered the option of one free consultation with a qualified sleep psychologist over the phone as compensation for participating (only one participant followed up on this offer).

The following inclusion criteria were used to select participants for the study. Participants were included in the study if they were: (a) aged between 65 to 85 years; and (b) intermittent users (two to six nights per week) of a hypnotic/sedative on our medication list (see Appendix D). The vast majority of the participants in this study were using temazepam (10 of 12). One participant was using nitrazepam. The remaining participant was using the non-benzodiazpine, zolpidem.

Participants were excluded from the study if they were: (a) unable to understand the risks and procedures of the study due to insufficient comprehension of the English language; (b) diagnosed with a serious neurological or psychiatric disorder; (c) reporting any hearing difficulty, which was subsequently objectively confirmed via a hearing test; (d) regularly taking any sedative drug not on the list of approved hypnotics/sedatives relevant to this study (see Appendix D); or (e) regularly taking any sedating antidepressant on the list of disapproved antidepressants relevant to this study (see Appendix E).

Self-reported ‘normal’ hearing ability was confirmed via an auditory examination after recruitment to ensure hearing thresholds were no lower than one standard deviation below normal hearing levels for participant age and sex. The American Speech-Hearing-Language Association (ASHA) guidelines were followed to ascertain hearing thresholds (American Speech-Language-Hearing Association, 2005). Details of the instructions followed are attached as Appendix F.

The study was approved by the Victoria University Human Research Ethics Committee (attached as Appendix U).

Medication Issues

Drug exclusion and inclusion criteria followed a systematic process and were developed with the aim of creating a list of drugs that were representative of current prescription patterns for the treatment of insomnia in Australia. The achievement of this goal was designed to increase the external validity of the study. Drugs that were primarily used for other purposes, such as antidepressants, but included sedative effects were excluded to avoid any confounding effects. In practice, no participant was regularly using both hypnotics and sedating antidepressants at the same time. This trend was anticipated. As both hypnotics and antidepressants are often indicated in similar conditions, it is unlikely they would be prescribed simultaneously in normal circumstances.

The list of drugs to be included was constructed in two stages. First, a complete list of all drugs that are primarily used as hypnotics and sedatives was formed. This list was created using the Anatomical Therapeutic Chemical (ATC) classification system (WHO Collaborating Centre for Drug Statistics Methodology, 2000). The ATC is recommended by the World Health Organisation and categorises drugs into different classes according to their therapeutic and chemical characteristics. This list was then cross-referenced against drugs commonly used in Australia for the treatment of insomnia (Morgan et al., 2012; Sim, et al., 2007; Smith & Tett,

2009). Hypnotics that were not typically prescribed in Australia were then filtered out. The final list of hypnotics is attached as Appendix D.

A similar process was used to establish a list of excluded drugs. Significant sedation is generally not associated with modern day psychotropic drugs in a number of classes (Bourin & Briley, 2004). Therefore, the focus of exclusion was on sedating antidepressants, as these drugs represented the most likely source of producing confounding effects. First, a number of antidepressants were collated from the ATC. With the assistance of the literature (Holshoe, 2009; Ramaekers, 2003), this list was then shortened to only include antidepressants with sedative properties. A conservatively large selection of antidepressants, many of which may only cause sedation in high doses or where there was a suggestion of paradoxical sedation in the literature, was used in order to greatly reduce the chances of confounding. Again, this record was cross-referenced with drugs normally prescribed in Australia to avoid lengthy and time consuming questionnaire lists (Smith & Tett, 2009). The final list of sedating antidepressants that were excluded is attached as Appendix E. In addition to this procedure, all participants were asked to disclose the use of any psychotropic drugs. This allowed for the identification of unusual or rare drugs that could then be considered against the study objectives. The advice of an accredited sleep physician, Dr. Simon Frenkel, was also sought early in the study formulation stage to assist in identifying prescription patterns.

Materials

Awakenings occurred via a sound delivery computer program that was operated automatically, without the need for onsite monitoring. This had the advantage of eliminating the need to have a stranger in the house while participants slept, thereby reducing disturbance and increasing the external validity of results. Furthermore it meant that more nights could be included in the overall protocol. This also allowed for a more naturalistic assessment of auditory arousal. The computer program was developed in-house at Victoria University and designed specifically for this research. The computer program ran on a small laptop placed in an adjacent room or hallway to the participant's bedroom, which was then connected to a bedside button located within reach of the participant's bed. The remote location of the laptop ensured noise and light

from the device did not interfere with the participant's sleep and that they could easily signal when they had awoken.

For the sound delivery computer program it was necessary to have sound files of each signal at levels from 35 dBA to 95 dBA in 5 dBA increments. Sound files from a preexisting study (Ball & Bruck, 2004a) were utilised. The sound files were created in a sound attenuated TV studio at a Victoria University campus. Once a signal was available at a particular volume it was played through the speakers to be used in the study and the decibel level adjusted using acoustic software (Sound Forge 6) so that it was measured to be received at a particular volume (e.g., 75 dBA) as assessed by the sound meter. A tolerance range of plus or minus 1 dBA was allowed. Thirteen sound files (one sound file at each 5 dBA increment from 35 dBA to 95 dBA) were created for each of the two sound types (520 Hz square wave and 3100 Hz sine wave). A spectral analysis of the 520 Hz square wave can be found in Appendix G. All sound files for both signals were played in the standard T-3 configuration.

An ascending method of limits procedure adapted from Bonnet (1982) guided signal presentation. The method of limits requires that an auditory stimulus be commenced at a low sound intensity level and be increased across discrete increments of strength over predetermined periods of time until the participant responds. This involved presenting a signal at 35 dBA, which was then increased by 5 dBA every 30 seconds until the participant woke up and pressed the button. This presentation sequence was used to explore all hypotheses highlighted in Study One. It should be noted that arousal studies that have tested sounds commencing from silence have generally noted slightly greater waking effectiveness (i.e., lower AATs) than the same sounds with no silence between the increments, presumably as a result of habituation to the signal (Bruck & Thomas, 2007c; Bruck, et al., 2007). Following this methodology allowed for easy comparison with previous research, which had used a similar procedure. There was a silence of approximately 10-20 seconds between each signal level presentation, which has demonstrated positive methodological properties (Bruck, et al., 2009). Intervening silences varied due to differences in processing power between the computers used in the study. The time on each laptop was synchronised via the internet to ensure the timing on each of the

experimental laptops were aligned with the timing on the research laptop housing the actigraph software (detailed below). A small tolerance of plus or minus three seconds was allowed.

Participants' arousal thresholds were the sound level (dBA) being presented at the time each participant pressed the bedside button. The bedside button needed to be pressed three times within a ten-second interval to register as an arousal; however, the time of arousal was recorded as the time of the first button press. Acknowledgment beeps between 40-45 dBA in volume were played after the respondent successfully pressed the bedside button three times. If the respondent pressed the button during the mediating silences or up to 30 seconds post the entire sound sequence their response was coded as the preceding sound level. If the respondent pressed the button more than 30 seconds after the final sound sequence had elapsed then arousal was not recorded by the software. Although this was a feature of the software, in practice this never eventuated.

Sounds were presented via a speaker attached to the laptop through an amplifier, and the sound level at the pillow was calibrated using a sound level meter applying A-weighting. Technical details of the sound delivery equipment used in the study can be found in Appendix H. The sound meter was professionally recalibrated immediately prior to the study.

Sleep and wake was measured via actigraphy, which is a device, similar to a watch in appearance, used to measure arm movement. An algorithm interpreted the arm movement recorded via the actigraph as a proxy for sleep and wake. The actigraph used was the Actiwatch 2 produced by Philips Respironics. Epoch length, which is the period of time the actiwatch accumulates activity data, was set at 30 seconds and default sensitivity settings were applied. Actigraphy was used to confirm bedtime and to have some objective record of participants' activity levels at the time of signal presentation. This was important because it was imperative participants were asleep when the signals were presented. In order to achieve this objective a sleep/wake decision making matrix was developed, which is discussed in more detail later on in this section. Participants were instructed to wear the actigraph continuously at night for the duration of the study on their non-dominant wrist. Use of the actigraph complied with the

current standards released by the American Sleep Disorders Association in order to achieve reliable results (Standards of Practice Committee, 1995).

A recruitment interview was developed by the research team and included detailed information on the study and questions regarding participants' treatment plan, standard demographics, and subjective sleep history (see Appendix I). The aim of the interview was primarily to inform participants of the study and apply the exclusion criteria to willing participants. The interview was between five and 10 minutes in duration.

Two in-home questionnaires were used to include information relevant to the study (see Appendix J). The first questionnaire was a two-week sleep diary and the second questionnaire was the Insomnia Severity Index (ISI). The sleep diary included questions relating to the project and was required to be completed each day. The ISI is a 7-item instrument designed to briefly evaluate and screen for insomnia in the general population following criteria from the DSM-5 (American Psychiatric Association, 2013) and the ICSD (ICSD; American Sleep Disorders Association, 1997). The ISI provided a global index of the severity of insomnia, which was used to categorised participants into one of four different severity profiles: (1) no clinically significant insomnia; (2) sub-threshold insomnia; (3) clinical insomnia (moderate severity); and (4) clinical insomnia (severe). The ISI has been found to be a reliable and valid instrument to assess primary insomnia (Bastien, Valli"eres, & Morin, 2001). The ISI was used to determine if insomnia severity moderates arousal thresholds. No cut-off score was used as an inclusion criteria as the level of insomnia severity would have been influenced by the intermittent hypnotic usage. The ISI questionnaire took approximately five to 10 minutes to complete.

Procedure

All participants who had made themselves available for the research were contacted over the phone and completed the recruitment interview. In-home appointments were arranged with participants who successfully completed the interview. Participants were sent the information and consent forms at this time. Shortly after the recruitment call, participants were contacted to

confirm their in-home appointment and their willingness to participate in the research having read through the information and consent forms.

Prior to attending the in-home appointment, the appropriate time for alarm presentation was determined. Alarm presentation time was individualised across participants and was calculated based upon information from the recruitment interview, including each participant's typical bed time, their prescribed hypnotic medication, and usual ingestion time. The goal was for the alarm signal to be presented when the medication was at peak concentration and the participants were asleep. Alarm presentation was at the same time on the hypnotic and hypnotic-free night for each condition to ensure consistency. Further details on the drug condition and general experimental design will be discussed later in this section. In view of the decision to present the alarm signal at a time dependent on peak hypnotic concentration (rather than say, during a particular stage of sleep), PSG recording was not undertaken. Instead, considerable effort was made to determine sleep/wake status at the time of the alarm.

Decision rules were created around the self-report information and the movement profiles yielded by the actigraphy to ensure data was not included where participants appeared to be awake before the alarm sounded. Subjective sleep or wake at alarm presentation was indicated on the sleep diary, which was usually completed each morning, via one of three options (yes/no/not sure). Self-report data was available for each data point, although sometimes this was completed several days after the night in question. With regard to the actigraph data, the number of activity counts was used to define wake. The activity count was set at the recommended value of 40, which represents medium sensitivity. If the sum total of activity in each epoch (together with a diminishing proportion of activity from surrounding epochs) was above this threshold the epoch was coded as awake. This means that activity surrounding each epoch is considered in the sleep/wake calculation, but activity in close time proximity to the epoch being calculated is given a higher weighting than activity recorded relatively later or earlier from the epoch being calculated. Five minutes of actiwatch coded sleep prior to the alarm sounding was required in order for a person to be considered asleep prior to alarm presentation. In addition to the classification of sleep already determined by the actigraph algorithm, this additional time period was deemed to be sufficient to ensure the transition from full wakefulness

to sleep had been made if wake was to be coded. Actigraph data was missing for a very small number of data points (approximately 2%). This was due to participant non-compliance.

People suffering from insomnia are more likely to underestimate their sleep or engage in negative sleep state misperception (Means, Edinger, Glenn, & Fins, 2003). They are also more likely to be restless during the night, which would increase the level of activity recorded by the actigraph (Morgenthaler, Alessi, et al., 2007). This suggests that in this population the accuracy of self-reported 'sleep' is likely to be higher, while the precision of the actigraphically defined sleep may be lower. Therefore, subjective sleep was unilaterally defined as sleep, despite actiwatch evidence to the contrary. Subjective measures were also unilaterally applied when participants self-reported to be awake. Research suggests that the greatest false negatives (thinking you are awake when in fact asleep) occur when the participant is in stage 1 or 2 sleep (Mendelson, 1998). For the purposes of the research, a more conservative definition of sleep, consistent with possible transition to stage 3 or 4 was preferable. In addition, people suffering from insomnia may also lie in bed motionless for long periods (Sadeh, Hauri, Kripke, & Lavie, 1995), which would undermine a classification of sleep generated by the actigraph. Actiwatch data was only used when the participant was uncertain about their sleep status (i.e., they indicated they were *not sure* of their sleep status on the sleep diary). In the absence of actiwatch data, the data point was conservatively coded as awake.

The final decision rules are presented in

Table 1. Despite the possible options, the two measures of actigraph and self-report were in agreement 89.6% of time. Only 10.4% of cases of alarm presentation were deleted due to being awake, and most of these cases were coded as awake on both subjective and objective measures. While the proportion of missing cases was relatively small, a method for dealing with these cases was needed and is described later in this section when dealing with analytical considerations.

Table 1

Sleep/wake decision matrix presenting the decision made under different self-reports and actigraph profiles at the time of alarm presentation

Self-report	Actigraph		
	(1) Asleep	(2) Awake	(3) Absent
(A) Asleep	Asleep	Asleep	Asleep
(B) Awake	Awake	Awake	Awake
(C) Uncertain	Asleep	Awake	Awake

Signed consent forms were collected upon arrival at the participants' homes. The participants were then briefed on all materials used in the study and given a Project Pack (see Appendix K), which included; the sleep diary, one Actiwatch, a prescription plan (with instructions on which days to consume their usual hypnotic), handy hints and reminders (e.g., advising participants that an alarm would not be presented on Friday and Saturday night), and contact details of the research team. The auditory examination was then performed. Results from the test as a function of age and sex are attached as Appendix F. Participants were then required to listen to both sounds used in the research to ensure there were no confounding effects due to differential familiarity with the alarm sounds. Participants were then given the ISI to complete. Finally, the sound delivery equipment was setup and the speaker calibrated to play the sounds at the appropriate sound levels. The speaker was positioned approximately one meter from the pillow and presented sound to the participant's side when lying in the supine position. A pre-recorded sound file of the 520 Hz square wave at 75 dBA was used for the speaker calibration at the pillow. Contact was made with participants on the third day of the experiment to verify there were no problems. Besides gently requesting participants not deviate from their normal routine too much, participants were otherwise free to engage in tasks they normally would (e.g., napping or drinking alcohol) in order to enhance the external validity of the study. In practice, no

evidence of excessive drinking (e.g., over seven standard drinks) was recorded in the sleep diaries for any participants.

Experimental design. The experiment involved monitoring the participants' sleep patterns and arousal thresholds over 11 nights, modulating hypnotic intake (using their usual hypnotic) and alarm presentation (the current 3100 Hz sine wave compared with the 520 Hz square wave) to ensure all independent variable combinations occurred. The type and dose of hypnotic used for each participant is recorded in Appendix L. Table 2 presents the enumerated design.

Table 2

Experimental design

Night		Alarm	Hypnotic	Row
N1	Mon	No alarm	No drug	A
N2	Tue	Alarm 1	No drug	B
N3	Wed	No alarm	Drug	C
N4	Thu	Alarm 2	Drug	D
N5	Fri	No alarm	No drug	E
N6	Sat	No alarm	No drug	F
N7	Sun	No alarm	Drug	G
N8	Mon	Alarm 1	Drug	H
N9	Tue	No alarm	No drug	I
N10	Wed	Alarm 2	No drug	J
N11	Thu	No alarm	Drug	K

Presentation of the two alarm signals (the current high pitched 3100 Hz sine wave signal and the 520 Hz square wave) was counterbalanced and presented over four nights, one night for each treatment combination. Thus for half the sample Alarm 1 was the 3100 Hz sine wave signal and for the other half it was the 520 Hz square wave. Participants were advised that the alarm would not be presented on Friday and Saturday to allow the participants to enjoy irregular bedtimes on

traditional leisure days. To minimise priming effects participants were told that the alarm may be presented between three to five times without knowing which specific nights the alarm would be presented. In addition, the alarms were presented in a pseudorandom arrangement, occurring on the second, fourth, eighth, and tenth night (rows B, D, H, and J respectively in Table 2).

Hypnotic intake occurred in blocks every third night, ensuring some wash-out time and the alarm was always presented on the second night. There were two blocks of two nights of hypnotic intake followed by a block of one night, totalling five nights. The hypnotic medication was the participants' normal dose and type taken at their usual time prior to bed (details in Appendix L).

Analytical considerations. A repeated measures analysis of variance (ANOVA) was utilised to analyse the arousal data (Hypothesis One to Hypothesis Three). The dependent variable was mean Auditory Arousal Thresholds (AAT). The within-subjects independent variables were alarm frequency (3100 Hz sine wave cf. 520 Hz square wave), and hypnotic consumption (no hypnotic taken cf. hypnotic taken). Two significant advantages of within-subjects designs compared to between-subjects designs are: (a) fewer participants are required as each participant is tested on all levels of each factor; and (b) the variance between individuals is eliminated increasing the power of the test (Tabachnick & Fidell, 2007). A univariate approach was favoured over multivariate ANOVA for two reasons: (a) the multivariate test should generally not be used when dealing with very small sample sizes (Maxwell & Delaney, 1990); and (b) if the univariate assumptions are met, the univariate ANOVA is more powerful. Further to the second item, the assumption of normality was upheld and Mauchly's test of sphericity was not needed because only two repeated measures were employed (Tabachnick & Fidell, 2007).

A disadvantage of this approach is that repeated measures are vulnerable to practice effects. Practice effects occur when a participant performs a test multiple times and this can sometimes have either a positive or negative effects on their response (Tabachnick & Fidell, 2007). These effects were minimised by counterbalancing the order of the signal presented across participants (Warner, 2008). The absence of any significant trial effects was confirmed via a paired samples *t*-test comparing the night of first alarm presentation with the second night of presentation in both the hypnotic and non-hypnotic conditions. This meant that in the no hypnotic taken

condition the first night of alarm presentation (N2) was compared with the fourth night of alarm presentation (N10), with $t(11) = .296$, $p = .772$. In the hypnotic taken condition the second night of alarm presentation (N4) was compared with the third night of alarm presentation (N8), with $t(11) = .275$, $p = .789$ also not being significant.

As ad hoc methods such as listwise and pairwise deletion are considered the least desirable or inadequate methods to deal with missing values (Wilkinson & The APA Task Force on Statistical Inference, 2003), the Expectation–Maximization (EM) algorithm, which is a model based approach that has demonstrated good performance against other methods was utilised to impute missing values (Musil, Warner, Yobas, & Jones, 2002; Zhou, 2001). A preliminary analysis of the data prior to imputation confirmed a manageable fraction of data (10.4% of values) was missing and that these missing values were Missing Completely at Random (MCAR). This means that the events that lead to the data points being missing were independent of both observable and unobservable variables, which is an important assumption in many imputation techniques, including the EM method (Musil, et al., 2002). Therefore, imputation could proceed satisfying the assumptions of the EM algorithm.

Two methods were used to analyse the supplementary and fourth objective related to sleep quality and arousal thresholds. The first method investigated the association between the ISI and arousal thresholds (Hypothesis Four) using a correlation technique. This method is less robust than experimental designs (Klein, 1992), but was used in order to increase the confidence in the overall analytical outcome. Supporting evidence was important because of the weaknesses inherent in the alternative analysis into objective four (Hypothesis Five). In addition, the small sample size meant a between group comparison was not achievable. Furthermore, all participants that were recruited were suffering from some form of sleep complaint, which meant the group could not be split accurately into good and poor sleepers.

The final analysis (Hypothesis Five) involved comparing arousal data in this study to approximate age matched healthy controls (data from Bruck and Thomas 2008a) in a between-group design. The large discrepancy in sample size between this study and the control study (approximately $n = 30$) was potentially problematic. Normally a *t*-test would be used to examine

this type of problem. The *t*-test is generally robust against violations to the assumption of homogeneity of variance, except in cases of unequal sample sizes (Zimmerman, 1987). In these cases, inequality of variance can have a profound impact on the significance levels (Zimmerman, 1987). Significance is less likely to be achieved when the smaller sample is less variable than the larger sample, while significance is more likely when the reverse is true (Hsu & Feldt, 1969; Scheffe, 1959). Therefore, significance levels would be decreased in this case as the sample from this study was small and less variable, evidenced by a narrower range and standard deviation in arousal thresholds. It is common to replace the *t*-test with a nonparametic alternative, such as the Mann-Whitney *U*-test, when posed with unequal sample sizes, particularly when the smaller sample is less variable (Zimmerman, 1987). The *U*-test does not require homogeneity of variance or normal distributions and simply assumes the dependent variable is continuous and the data are independent from one another (Tabachnick & Fidell, 2007). These assumptions were satisfied thus the Mann-Whitney *U*-test was used to investigate Hypothesis Five.

Results

The first objective was to determine the extent to which hypnotic ingestion increased auditory arousal thresholds (AATs) to the current smoke alarm signal (3100 Hz sine wave). It was hypothesised that hypnotic ingestion would significantly increase arousal thresholds even amongst people who intermittently use hypnotics (Hypothesis One). A two factor (2 x 2) ANOVA was performed using mean AATs as the dependent variable. The within-subjects independent variables were alarm frequency (3100 Hz sine wave cf. 520 Hz square wave), and hypnotic consumption (no hypnotic taken cf. hypnotic taken).

There was a significant main effect for hypnotic consumption, with $F(1, 11) = 28.04$, $p < .001$, $\eta^2 = .718$. Hypnotic ingestion increased AATs by approximately 9 dBA and this effect was consistent at either alarm frequency. This finding is depicted graphically in Figure 3, which displays the mean AATs across levels of both alarm frequency and hypnotic consumption.

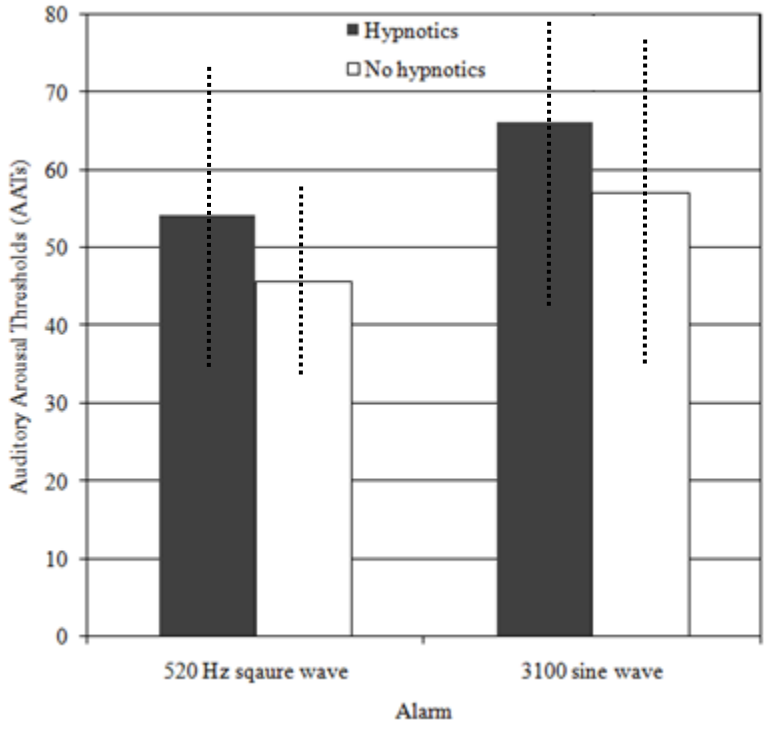

Figure 3. Mean auditory arousal thresholds (dBA) across levels of both alarm frequency and hypnotic consumption (n = 12). The dotted lines indicate the 95% confidence interval of each mean.

A second objective was to establish if there were significantly lower arousal thresholds to the 520 Hz square wave alarm compared to the current (3100 Hz sine wave) smoke alarm when under the influence of hypnotics. It was hypothesised that there would be lower arousal thresholds to the 520 Hz square wave alarm in the hypnotic condition compared to the 3100 Hz sine wave alarm (Hypothesis Two). There was a main effect for alarm frequency, and significantly lower AATs for the 520 Hz alarm in both the drug or no drug condition, $F(1, 11) = 88.11$, $p < .001$, $\eta^2 = .889$. The 520 Hz square wave reduced AATs by approximately 12 dBA. While both main effects were significant, the main effect for alarm frequency was marginally stronger than for hypnotics.

The third objective was to verify if the current recommended Australian standard alarm intensity level of 75 dBA at the pillow was adequate for waking intermittent users of hypnotics under the influence of their usual hypnotic. It was hypothesised that hypnotic ingestion would increase arousal thresholds for a proportion of participants above the standard alarm intensity of 75 dBA for the 3100 Hz pure tone but not for the 520 Hz square wave (Hypothesis Three). Figure 4 presents the cumulative proportion of participants asleep at each AAT for each of the four different alarm and drug conditions. All participants were awoken by the time the 520 square wave was played at 75 dBA, even after taking their usual hypnotic. Even more impressively, all participants were awoken by the time the 520 Hz square was played at 55 dBA when not taking their usual hypnotic. In contrast, during the 3100 Hz sine wave alarm approximately one in 12 (8%) participants failed to wake at 75 dBA, and this proportion effectively doubled (17%) after the consumption of a hypnotic. The greatest disparity between the different alarm frequencies and drug conditions occurred in the 50 to 70 dBA range.

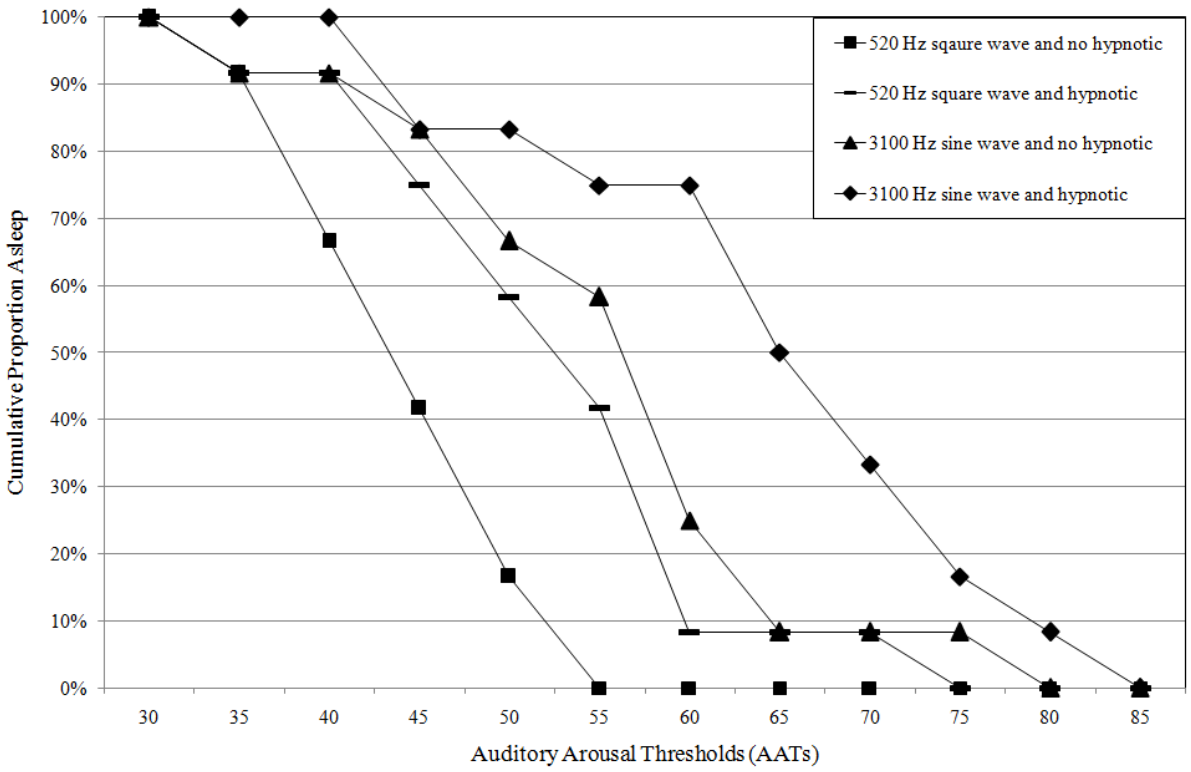

Figure 4. Cumulative proportion of participants asleep at each auditory arousal threshold for each of the four different alarm and drug conditions (n = 12). Imputed missing data was rounded to the nearest multiple of five for the purposes of this graph.

The fourth and final objective involved investigating sleep quality and arousal thresholds. This was analysed via two separate methods. The first method involved a correlation analysis between arousal thresholds and the self-reported severity of insomnia symptoms in intermittent hypnotic users, and to determine whether such a relationship may exist when they were on or off their medication, or both. It was hypothesised that as insomnia symptoms increase arousal thresholds will decrease for both alarm signals (Hypothesis Four). The mean ISI was 14.8 with a standard deviation of 4.0, which would be defined as sub-threshold insomnia (Bastien, et al., 2001). The correlation between the ISI and AATs was determined for each of the four alarm and drug conditions, which were in order of magnitude: (1) 3100 Hz and hypnotic = -.533; (2) 520 Hz and hypnotic = -.410; (3) 3100 Hz and no hypnotic = -.330; and (4) 520 Hz and no hypnotic = -.218. *Figure 5* displays the correlations in a scatter plot. A linear regression line was overlaid (R^2 = .284) on the strongest relationship (the 3100 Hz and hypnotic condition).

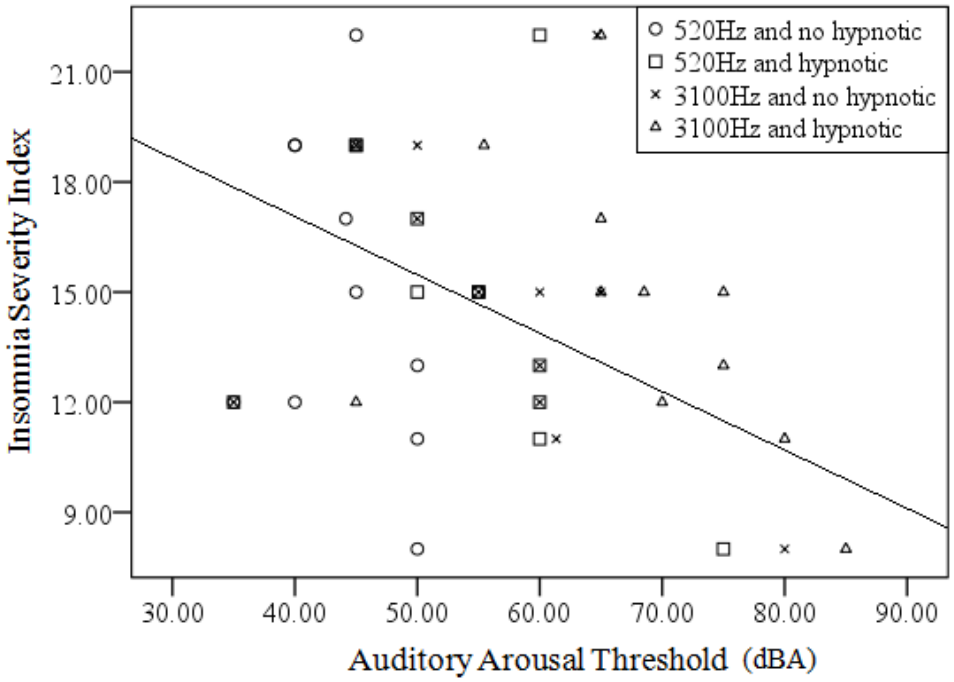

Figure 5. Scatter plot displaying the Insomnia Severity Index on the *y* axis as a function of auditory arousal thresholds (dBA) on the *x* axis for each of the four different alarm and drug conditions (n = 12). A linear regression line has been overlaid (R^2 = .284) on the strongest relationship (the 3100 Hz sine wave and hypnotic condition), which shows a large negative correlation.

The correlations were all negative and of moderate to large strength. None of the correlations were significant but the strength of the relationships suggested a significant result would have been achieved had the power of the test been greater. The strongest correlation between the 3100 Hz sine wave and the hypnotic condition was above .50, which is considered large in the social sciences (Cohen, 1992). Furthermore, a posteriori power analysis using G*power (Faul, Erdfelder, Buchner, & Lang, 2009), indicated almost twice the number of participants would have been required to achieve a sample size necessary for 80% power to detect this effect, despite the effect being large. This level of power is regarded as appropriate since levels lower than 80% would unduly increase the risk of committing a type II error, which occurs when the null hypothesis is not rejected in spite of there being a real effect (Cohen, 1992). An interpretation of statistical difference even though the result was non-significant is also consistent with the view of the American Psychological Association (APA), which published

their findings on the lengthy debate surrounding the use of null hypothesis significance testing and determined that effect sizes and confidence intervals be given greater prominence with less weight attached to *p*-values (Balluerka, Gomez, & Hidalgo, 2005; Fraley & Marks, 2007). Overall, the weight of evidence, while thought provoking, is not conclusive and only partially supports the hypothesis.

The second method was to compare the auditory arousal thresholds (for both types of alarm signals) of the poor sleepers recruited in this study when not consuming their hypnotic with the auditory arousal thresholds of approximate age matched good sleepers (data from Bruck and Thomas 2008a). It was hypothesised that poor sleepers would have significantly lower arousal thresholds compared with good sleepers for both alarm signals (Hypothesis Five). A summary of statistics comparing the results of the poor sleepers used in this study against the good sleepers from Bruck and Thomas' (2008a) research for each alarm is detailed in Table 3. The good sleepers from Bruck and Thomas' (2008a) study were healthy older adults awoken during SWS.

Table 3

Summary of descriptive statistics at each alarm frequency for the group of poor sleepers (on and off hypnotics) and healthy, deep sleep established good sleepers[a]

AAT (dBA)	520 Hz square wave			3100 Hz sine wave		
	Poor sleepers		Good sleepers[a]	Poor sleepers		Good sleepers[a]
	Hypnotics (n = 12)	No hypnotics (n = 12)	(n = 43)	Hypnotics (n = 12)	No hypnotics (n = 12)	(n = 44)
Mean	54.17*	45.76	48.02	66.16	57.17	63.75
SD	10.19	6.35	13.28	12.57	11.39	15.33
Range	35-75	35-55	35-85	45-85	35-80	35-105
Median	55.00	45.00	45.00	66.76	60.00	65.00
% slept through 75 dBA	0.00	0.00	4.60	16.67	8.33	18.30

a Data displaying good sleepers from Bruck and Thomas (2008a) were tested with the same signals under conditions of electroencephalography (EEG) determined deep sleep (stages 3/4) and no hypnotics. The sample size differs across signals due to missing data.

A Mann Whitney *U*-test was performed on the data and no significant difference was apparent between good sleepers and poor sleepers in the no hypnotic condition; however, there was

evidence of a result approaching a trend that suggested poor sleepers may indeed have significantly lower arousal thresholds compared to good sleepers with the 3100 Hz sine wave alarm (see Table 4 for Mann Whitney *U*-test results).

For completeness, significance testing was conducted on both conditions. After consuming a hypnotic, poor sleepers were significantly less responsive to the 520 Hz square wave compared with good sleepers (Table 4). However, since the impact of poor sleep and hypnotic consumption on arousal thresholds is in many ways counterbalanced the usefulness of this result is questionable.

Table 4

Summary of Mann Whitney U-test results at each alarm frequency for the group of poor sleepers (on and off hypnotics) and healthy, deep sleep established good sleepers[a]

Alarm	Good sleepers (no hypnotic) vs. poor sleepers (no hypnotic)	Good sleepers (no hypnotic) vs. poor sleepers (hypnotic)
3100 Hz sine wave	$U = 184$ $p = .108$	$U = 229$ $p = .475$
520 Hz square wave	$U = 255$ $p = .95$	$U = 162$ $p < .05$

a Data from good sleepers from Bruck and Thomas (2008a) were tested with the same signals under conditions of electroencephalography (EEG) determined deep sleep (stages 3/4) and no hypnotics.

Discussion

The major aim in Study One was to determine the utility of smoke alarms, with both incumbent and alternative technology, in a population of older adults who intermittently use hypnotics. This was accomplished by first testing the ability of the current smoke alarm (3100 Hz sine wave) to wake users of hypnotics under the influence of their usual hypnotic, then comparing this performance with the new signal (520 Hz square wave). An additional and supplementary line of enquiry sought to explore the potential relationship between arousal thresholds and sleep quality, as this would have interesting implications, particularly for the primary objectives. For example, this analysis would ascertain if sleep problems were a factor that may have contributed to inconsistency in arousal thresholds in previous research.

Objective one. The first objective was to evaluate the impact of hypnotic consumption on the ability to arouse to the current smoke alarm (3100 Hz sine wave). It was hypothesised that the presence of hypnotics would significantly increase arousal thresholds at the time of peak action of the hypnotic ingested (Hypothesis One). This hypothesis was supported and hypnotic ingestion was found to increase arousal thresholds in intermittent users of hypnotics to both the 3100 Hz sine wave and the 520 Hz square wave. This finding corroborates earlier work with electronic tones generally (Bonnet, et al., 1979; Hartse, et al., 1983; Johnson, et al., 1979; Mendelson, et al., 1988; Spinweber & Johnson, 1982), and the single study using the standard smoke alarm frequency (3100 Hz sine wave) more specifically (Johnson, et al., 1987). A strong hypnotic effect is also consistent with sensory and perceptual deficiencies in the auditory system as a result of hypnotic ingestion as one possible theoretical explanation (Clark, 1996; Walley & Weiden, 1973). Given the time and methodological differences between the current and past studies, the replication of this finding provides good support for a hypnotic effect. However, there was less agreement on the size of this effect.

The precise size of the increase in arousal thresholds is a critical factor in alarms research. The production of a significant effect does not necessarily imply that hypnotics will prevent awakenings at the Australian standard of 75 dBA. This will depend on the size of the effect and the threshold with no hypnotic. A 9 dBA increase in mean arousal thresholds was achieved after

the consumption of a hypnotic in this study, but similar measures have varied widely in the literature. Results have fluctuated from an increase under 10 dB to gains of over 40 dB (Bonnet, et al., 1979; Hartse, et al., 1983; Johnson, et al., 1979; Mendelson, et al., 1988; Spinweber & Johnson, 1982). Note the use of the dB symbol when citing previous research as these studies did not use A-weighted sound pressure (dBA); however, this would have minimal impact on comparisons made at these frequencies, as the weighting fractions are largest at very low or high frequencies (Parker, 2003). Returning to the issue of variation in arousal thresholds, given these inconsistencies, this research attempted to provide a clearer picture of risk.

It is argued that this research provides a much more realistic picture of the danger of sleeping through the Australian standard at the pillow than previous studies because methodological changes have been made to maximise external validity. Previous research typically employed a between-group design and was performed in sleep laboratories using strict protocols and targeted samples. Large effect sizes and highly significant results characterised much of this preceding work. However, that research was carried out more than two decades ago and was encumbered by design limitations exposed by the passage of time that limited generality claims. This study increased the external validity of the findings by implementing a number of design changes to the sample selection, drug selection, and situational context.

The priority of this study was to select a sample that was representative of users of hypnotics in Australia who were consuming typical hypnotics. The non-probability sampling methods employed and small sample size may not have satisfied the strict conditions necessary for representativeness, but every effort was made to approximate a sample reflective of the target population. Non-random selection need not necessarily greatly impact the generality of results in cases where it is reasonable to assume that the sample would not significantly differentiate on the responses (Chow, 2002). The judgement of the researcher and relevant literature would be expected to be critical in making this determination. Having reviewed these issues, two requirements were put in place to ensure the sample better reflected users of hypnotics. First, participants were not excluded from the study if they did not meet certain thresholds of sleep dysfunction, as had been the case in previous empirical work. The sleep pattern of users of hypnotics varies enormously. Some suffer from chronic insomnia while others may only

experience insomnia subjectively. The sample needed to reflect this heterogeneity, even though this additional variability would decrease the probability of finding a significant result considering the size of the sample. Consequently, some of the participants involved in this study may not strictly satisfy the criteria for a diagnosis of insomnia according to the current edition of the DSM (American Psychiatric Association, 2013). This was in fact borne out in the data where the average ISI in the sample was 14.8, which would be defined as sub-threshold insomnia (Bastien, et al., 2001). Levels of sleep disruption are important because evidence from this study suggests a link between sleep quality and arousal thresholds. Second, older adults were recruited, whereas only younger adults had been selected in the past. Older adults are significantly more likely to die in a fire than their younger counterparts (Hall, 2005), represent the largest consumers of hypnotics (Woodward, 1999), and age influences arousal thresholds (Bruck & Thomas, 2008a). Other issues may impact generalisability across the population, such as the exclusion of the hearing impaired. In summary then, the sample selected for this study was designed to reflect users of hypnotics rather than poor sleepers, which provided a more externally valid representation of smoke alarm performance after the consumption of a hypnotic.

The schedule of sedatives included in this research were more representative of contemporary prescribing practices compared to previous research. Since benzodiazepines differ in the potency and duration of their therapeutic effects, it is reasonable to expect individual drug differences in arousal thresholds. For example, a number of hypnotics are far more effective at promoting sleep compared to muscle relaxation, even allowing for equivalent doses. While sleep promotion is an effect that would likely be of more relevance to sensory sensitivity. The number of hypnotics that have been tested in arousal thresholds is underwhelming and not reflective of current consumption trends. Prior to this study, just three different sedatives (triazolam, brotizolam, and flurazepam) had been investigated from a significantly larger collection of hypnotics now currently available, and these sedatives, with the exception of Triazolam, are rarely prescribed. Triazolam and brotizolam are both short acting agents that are similar in effect, while flurazepam is a longer acting benzodiazepine (Mandrioli, Mercolini, & Raggi, 2008). While all three drugs are potent hypnotics, there are documented differences in the impact they may have on a number of performance measures (Langley & Clissold, 1988). In contrast, in agreement with reports in the literature, the vast majority of the participants in this

study were using temazepam. One participant was using the non-benzodiazpine, zolpidem and another participant was using nitrazepam. The use of a non-benzodiazpine is particularly noteworthy. This class of drugs was generally not available or rarely prescribed at the time most of the literature was carried out. Drugs in different drug classes are even more likely to possess dissimilar effects than different drugs within the same class (Keltner & Folks, 2005). Therefore, it is imperative that the results reflect current consumer purchasing habits, especially as new chemically diverse hypnotics come on to the market.

It is tempting to agree with Johnson et al.'s (1987) conclusion, that an increase in arousal thresholds would be likely after consuming any sedative. However, given the differences between hypnotics, the small number of drugs tested so far may not be large enough to produce a reliable sample from which to draw inferences about other drugs, even within the same drug class. Certainly, not large enough to suggest that all drugs would impede the response specifically to a smoke alarm. While this study adds to the number of hypnotics that have now been tested, more work is required before a judgment can confidently be extended to all hypnotics. However, this was not the present study's goal. Rather, the objective was to be representative of hypnotics usually consumed by users of hypnotics in Australia, not representative of all hypnotics.

This research was also a field experiment conducted in the participants' homes rather than a sleep laboratory. Participants were not asked to significantly alter their routines and alarm times were scheduled around the participants' self-reported normal sleeping schedules. While additional variability was introduced into the experiment, a major benefit is that field experiments have greater ecological validity (Maxwell & Delaney, 1990). This method also placed less demand on the participants compared with previous work.

Sleep stage was another area of interest that was monitored in all of the previous work and results of those studies have shown that there were significant variations as a function of sleep stage (Bonnet, et al., 1979; Hartse, et al., 1983; Johnson, et al., 1979; Mendelson, et al., 1988). Generally, deeper levels of sleep required higher volumes to successfully wake the participants. In contrast, sleep stage was not controlled in this study. Sleep stage was not controlled in this

study because deep sleep did not necessarily coincide with peak hypnotic concentration, which was the central point of interest. Both hypnotics and age reduce slow wave sleep, such that older adults experience only relatively short periods of slow wave sleep so it may have been logistically difficult to achieve both SWS and peak hypnotic concentration simultaneously. The decision not to monitor sleep stage did however, introduce a major problem. It was possible the alarms may have sounded when the participant was either awake or had only achieved a very light level of sleep (i.e., stage 1). Research that has studied errors in performance testing after the consumption of sedatives, has demonstrated that it is possible to stay awake, albeit in a slowed dissociative state, even when the sedative is at peak concentration (Tiplady, Hiroz, Holmes, & Drummond, 2003). The results would be completely inaccurate if the participant was awake and distorted in the event that the participant had just fallen asleep. A range of techniques were recruited to combat these eventualities. Participants were required to complete a sleep diary each morning and also wear an actigraph each night. It was hoped that collecting sleep information from a range of sources, both subjective and objective, would provide greater confidence in the result, as the strengths of one method often subsidises for the weaknesses of another. Subjective reports of sleep have often disagreed with more objective measures (Agnew & Webb, 1972; Bonnet, Bootzin, Kihlstrom, & Schacter, 1990; Rechtschaffen, Ogilvie, & Harsh, 1994). This is particularly evident in people who suffer from sleep problems or take sleep medication (Bonato, 1998; Bonnet, et al., 1990). Despite these findings, the measures were in agreement approximately 90% of time. One explanation for this congruence is that the degree of difficulty in determining sleep/wake status at one particular point early in the morning is much easier than estimating total sleep time, which is a measure often used in the literature when comparing subjective and objective measures of sleep (Van Den Berg et al., 2008). Nevertheless, this level of consistency provided confidence that the alarms were activated during sleep. In addition, only 10.4% of cases were deleted due to being awake, and this was not considered especially high given the fractured nature of sleep typically experienced by participants with symptoms of sleep maintenance insomnia. The sleep diaries also suggested that participants faithfully followed their predicted bed times and schedule of drug consumption, with only a few exceptions; however, waking after their planned bedtime was an issue for some participants involved in the project. This means that some participants when to bed at the

prescribed time but then woke after initially falling asleep, but in most of these cases the participants fell back to sleep prior to the alarm sounding.

Overall, this study has extended previous work on the impact hypnotics have on the response to electronic tones. This suggests that the consumption of hypnotics may increase the chances of perishing in a fire due to a reduced ability to respond to a fire alarm, which is the most widely used type of fire protective equipment. Hypnotics do undermine the response to current residential fire alerting equipment.

Objective two. The second objective was to test the difference in performance between the 520 Hz square wave and the 3100 Hz sine wave to confirm any signal advantages in an untested vulnerable population. It was hypothesised that there would be lower arousal thresholds to the 520 Hz square wave alarm in either condition compared to the 3100 Hz sine wave alarm (Hypothesis Two). This hypothesis was supported and it was found that the 520 Hz square wave alarm performed significantly better than the 3100 Hz sine wave frequency in a group of intermittent users of hypnotics, both when on and off their hypnotics. This finding further reinforces previous research conducted with other vulnerable groups that made similar comparisons (Ball & Bruck, 2004a; Bruck, 1999; Bruck, et al., 2004; Bruck & Thomas, 2007c; Bruck, et al., 2007), and is also consistent with existing theoretical explanations that implicate sensory and perceptual signal advantages (Gibson, 1979; Zwicker, et al., 1957).

Although the direction of the effect was comparable to previous research there was again a noted variance in the size of arousal threshold differences. The 520 Hz square wave has generally outperformed the 3100 Hz pure tone by approximately 20 dBA in previous studies (Ball & Bruck, 2004a; Bruck, 1999; Bruck, et al., 2004; Bruck & Thomas, 2007c; Bruck, et al., 2007). This is almost double the effectiveness observed in this research, where an approximate decrease of 12 dBA was recorded for AATs for the 520 Hz square wave. In the comparison study of healthy older adults tested with the same signals (Bruck & Thomas, 2008a), the mean arousal threshold for the 3100 Hz pure tone signal was 64 dBA, while it was 48 dBA for the 520 Hz square wave, which is a marginally smaller difference of 16 dBA. In all likelihood the divergent

methodological procedures that separated this study from the previous research can explain this inconsistency.

This study was intended to be more naturalistic and representative of a real world setting than previous research, where a greater emphasis was placed on internal validity. There have only been a relatively small number of experimental studies investigating the 520 Hz square wave. These studies have all been methodologically similar, employing a repeated measures design, utilising a highly specific sample, and operating in a tightly controlled field environment where sleep was measured via EEG. A sensible attempt was generally made to remove extraneous variables from the experimental design. A different approach was taken in Bruck et. al.'s (2004) study, which tested the 520 Hz square wave in children aged 6 to 10 during a three night experiment in the child's home. Sleep was monitored via wrist actigraphy. While Bruck et. al.'s (2004) study is most similar to this study in design, children's waking profiles are significantly different from adults, which makes a methodological comparison difficult. Therefore, this is the first experiment to analyse the performance of the 520 Hz square wave after making a significant departure from many of the procedures that characterised the previous work in a sample of adults.

Nevertheless, these methodological differences do complicate comparisons between this study and the literature. In most of the previous research testing the 520 Hz square wave the participants were necessarily informed of which night the alarm would be presented because of the need to monitor EEG for arousal during deep sleep. This knowledge may have primed participants to respond to the alarm signal. Priming occurs when exposure to one stimulus impacts the response to a later stimulus (Matsumoto, 2009), and has been shown to significantly decrease arousal thresholds in specific circumstances (Wilson & Zung, 1966). Even in Bruck et. al.'s (2004) study where actigraphy was used, the level of priming would be likely to be higher since there were only three experimental nights and the first night was always an adaptation night and the alarms were not activated, which meant the probability of the alarm sounding on one of the remaining nights was high (50% likely). If the alarm did not sound on the second night it was certain to sound (100% likely) on the final night. Furthermore, the researchers observed that most children in the study were highly motivated to anticipate the alarm. There are

other possible complicating factors. It is also probable that parental supervision influenced expectation levels, or since priming is a learned activity children may be at a cognitive disadvantage because the ability to learn has been shown to be a function of your stage of cognitive development (Nakagaki, 2011).

In contrast, participants in this research were also primed to react to a fire cue, but the degree of priming was less acute. To minimise priming effects participants in this study were unaware of which nights they would be awoken, and only told that the alarm may be presented between three to five times over the 11 nights. Priming was also reduced by automating the collection of data, which lengthened the time between the initial stimuli (research briefing on day one of study) and subsequent responses to the alarm. In most of the previous research the immediacy of the testing environment where the researcher was sitting in the hall outside the bedroom, may have added to the level of participant expectation. Overall, the level of priming in this study could be categorised as low compared to other work in this area, and this may have increased arousal thresholds.

There was also substantial disparity in the control of extraneous variables. In all of the earlier work participants were asked not to indulge in any behaviour that may have unduly impacted their arousal thresholds. For example, participants were generally not allowed to drink alcohol or nap before the experimental night. No such mandate was given in this study in order to increase generalisability at the expense of experimental control. It is not clear what impact this had on arousal thresholds. In some instances, such as alcohol ingestion, the outcome would have been an increase in arousal thresholds in the first third/half of the night (Bruck, et al., 2007). While in others, such as excessive napping prior to bedtime, the opposite may have been true. Consequently, the lack of control of confounding factors in this study likely had both a positive and negative impact on the level of arousability.

The difference in the level of environmental artificiality may also be of importance. A member of the research team was often present in the house in previous research that monitored sleep via EEG. The data collection procedure was automated in this study so no member of the research team was onsite, and sleep was logged via actigraphy, which is non-intrusive. The presence of a

researcher at the time of data collection in the previous research may have resulted in a number of biases (Matsumoto, 2009). Furthermore, the attachment of EEG electrodes is an unfamiliar and sometimes discomforting process that is not conducive to sleep, which may have made the participant anxious or unsettled. Overall, the artificiality of the testing environment may have elicited responses from the participants that were not consistent with their everyday behaviour. While anxiety has been shown to impact the quality of sleep (Fuller, Waters, Binks, & Anderson, 1997) it is important to note that previous research had established deep sleep via EEG prior to the presentation of sounds. Therefore, the impact of environment artificiality is likely to be less of a factor compared to other methodological differences.

There are also unique population factors at play. Poor sleepers and users of hypnotics have always been excluded from previous similar research. This research has provided tentative evidence of a relationship between sleep quality and arousal thresholds that suggests that poor sleepers may have lower baseline arousal thresholds compared to good sleepers. Furthermore, the sample in this research was disproportionally female compared to other studies. Females have been shown to be significantly more sensitive to alarm signals (Hasofer & Bruck, 2004). Together, these population factors combine to decrease arousal thresholds reported in this study.

As previously discussed, the sleep stage of participants at the time of awakening is also influential in arousal studies. With the exception of the studies into children's arousal thresholds (Bruck & Bliss, 2000; Bruck, et al., 2004), all other studies presented the alarm signals during deep sleep. Therefore, the absence of this control would be expected to result in a decrease to arousal thresholds in the current study.

Finally, the decision to include or exclude silences between the different incremental levels of the sounds is also a factor. This study included a silence of between 10-20 seconds between each 30 second signal presentation. Intervening silences varied due to differences in processing power between the computers used in the study. Silences were inserted in order to replicate the conditions present prior to a smoke alarm sounding in an authentic fire emergency, and silences of approximately 10-20 seconds between each signal presentation have demonstrated positive methodological properties (Bruck, et al., 2009). Arousal studies that have tested sounds

commencing from silence have generally noted slightly greater waking effectiveness (i.e., lower AATs) than the same sounds with no silence between the increments, presumably as a result of habituation to the signal (Bruck & Thomas, 2007c; Bruck, et al., 2007). The majority of studies investigating the 520 Hz square wave used a presentation procedure that did not include silences (e.g., Ball & Bruck, 2004a; Bruck & Thomas, 2008b). Arousal thresholds in this study would be expected to be slightly lower than studies that did not include silences as result of sensory adaptation.

Overall, there are a range of factors influencing arousal thresholds that make comparisons between studies difficult, but may explain inconsistencies in differences in arousal thresholds and data variability. The decreased degree of priming and level of environmental artificiality used in this study would be expected to increase arousal thresholds. Conversely, the population, possible sleep stage, and decreased level of sensory adaptation that characterise this study would be expected to decrease arousal thresholds. It is not clear what impact the control of extraneous variables may have produced. A final determination on the impact of these competing variables is further complicated by the inability to quantify the relative weight of each factor in influencing arousal thresholds. Therefore, there is enough evidence to suggest methodological differences account for changes in differences in arousal thresholds, but the complexity of these variations means that care is required when making comparisons across studies.

The finding of a robust alarm effect despite these methodological variations provides strong support for the 520 Hz square wave. This support has two aspects. First, the results were able to be replicated in a new population. Second, a strong effect was found even in a more realistic context that introduced greater variability into the experiment, which provides greater external validity. Therefore, this study extends the small but growing repository of experimental work on the 520 Hz square wave, by assessing the alarm in a vulnerable group not previously tested and utilising a more realistic design. This provides increasing confidence that the alarm is likely to perform better than the current standard in all populations and in real life conditions. Outside of fire alarms, there are a number of areas where a more effective night time alarm would be advantageous. These areas include common alarm clocks and bed wetting alarms. Of course, a different sound pattern may need to be used to avoid confusion with an evacuation signal.

Objective three. The third objective was to determine the actual risk of sleeping through both the 3100 Hz sine wave and the 520 Hz square wave at the standard alarm intensity of 75 dBA. It was hypothesised that hypnotic ingestion would increase arousal thresholds to above the standard alarm intensity (75 dBA) for the 3100 Hz pure tone for a proportion of participants, but no participants were expected to sleep through the standard alarm intensity for the 520 Hz square wave (Hypothesis Three). This hypothesis was supported for the 3100 Hz pure tone, where 17% of older adults slept through a 75 dBA signal after consuming their usual hypnotic. Similarly, the hypothesis was also supported for the 520 Hz square wave, where no participants slept through the alarm at this intensity even after consuming a sedative.

These findings are consistent with previous research. Bruck and Thomas (2008b) reviewed the existing literature and compared the performance of the 520 Hz square wave against the 3100 Hz pure tone at the 75 dBA standard. The ratio of the likelihood of sleeping through the 3100 Hz pure tone to the 520 Hz square wave at 75 dBA was reported to be between 4:1 and 12:1 in the studies conducted to date depending on the population being tested (Bruck & Thomas, 2008b). A ratio was unable to be calculated in two cases, both involving young adults but one sample was in a sober condition and the other sample was under the influence of alcohol, due to no participants sleeping through the 520 Hz square wave (Bruck & Thomas, 2008b; Bruck, et al., 2007). On this evidence, the 3100 Hz pure tone has consistently been shown to be the least effective signal at waking people at all dBA levels, including the current standard of 75 dBA.

The ability of the 520 Hz square wave to awaken all participants in both drug conditions is especially encouraging. Although previous research has always shown a significant improvement in arousal associated with the 520 Hz square wave, the threat of sleeping through the low frequency alarm has only been completely eliminated at the 75 dBA level in young adults, either in a sober or intoxicated condition (Bruck & Thomas, 2008b; Bruck, et al., 2007). Even at higher volumes there have often been a small number of participants that have slept through the entire sequence of 520 Hz square wave signals. The better performance of the 520 Hz square wave in a number of studies has been strong and promising but the inability of the 520 Hz square wave alarm to wake all participants in many cases does not represent an entirely

satisfying fire alerting answer. Smoke alarms are the most popular fire alerting tool in Australia (Australian Bureau of Statistics, 2007). The goal of any smoke alarm includes maximising waking potential. For every person where this aim is not achieved, the likelihood of dying in a fire may be significantly higher. Therefore, even if the theoretical number of deaths can be reduced by changing the alarm signal to the 520 Hz square wave (as intimated by Bruck and Thomas (2008a) in their particular sample of older adults), any number of casualities could be argued to still represent an unacceptable level of risk despite being an improvement from previous technology. Introducing an arbitrary threshold beyond which represents an unacceptable number of possible casualities would be a philosophically daunting task beyond the scope of this research. Instead, by removing all risk of sleeping through the alarm in the selected sample, this study has reinforced the credentials of the 520 Hz square wave alarm in this particular population, but different options may be required in other populations. Possible solutions are addressed in the General Discussion.

It should be noted that these figures may not precisely reflect the actual risk to users of hypnotics. While the current research benefited from the attempt to improve external validity, there were elements where this was not always possible. Smoke alarm location is a relevant example. The results are based on the unlikely prospect of minimal variation between the emitted and received sound volume. This might be a reasonable assumption if the smoke alarm was located in the same room as the occupant at the time of the fire, but this eventuality is not typical. During the early morning period people are largely sleeping in their bedrooms, yet most smoke alarms are installed in the hallway or kitchen (Ahrens, 2011b). Unlike the NFPA in the United States, the Building Codes Board in Australia (ABCB, 2008) does not require smoke alarms in bedrooms. The location of the smoke alarm can significantly impact the intensity of the signal. Using full scale model houses, Thomas and Bruck (2011) demonstrated substantial reductions of between 30 to 40 dBA depending on whether the alarm was located in the hallway or another room or if the doors were closed or not. A larger reduction (6 dBA or greater) was evident between rooms for the 3100 Hz sine wave alarm when compared with the 520 Hz square wave alarm. This is attributed to the ability of low frequencies to travel through doors and walls more effectively (Quirt, 1985). Applying these assumptions to the results would appreciably alter the waking outcomes. Even a conservative reduction of just 35 dBA (as a result of closed

doors and a hallway alarm) to the 3100 Hz sine wave alarm, which would mean that 75 dBA would be received at 40 dBA at the pillow, would result in 92% not being notified and remaining asleep. All participants would remain asleep under these conditions during the 3100 Hz sine wave alarm after taking their usual hypnotic. A similar but less severe decline is evident for the 520 Hz square wave alarm. A reduction of 30 dBA to the 520 Hz alarm, which would mean that 75 dBA would be received at 45 dBA at the pillow, would result in 42% not being notified if not taking their hypnotic, and 75% remaining asleep after consuming their hypnotic. Therefore, the research may significantly underestimate the risk to users of hypnotics due to the likely reduction in sound intensity when a smoke alarm is not located in the room of a potential victim. However, even after considering these issues the 520 Hz square wave still demonstrates greater arousability, and even extends its signal advantages over the 3100 Hz sine wave due to better penetration through walls and other barriers in the home.

Objective four. The fourth and final objective involved investigating sleep quality and arousal thresholds due to the inconsistency of predictions made by hyperarousal theory and existing empirical work. This was analysed via two separate methods. The results of each method will be briefly discussed before the theoretical implications of both approaches are considered together. The first method involved examining the relationship between sleep quality and arousal thresholds using a correlation design. It was hypothesised that as self-reported insomnia symptoms increase arousal thresholds would decrease for both alarm signals (Hypothesis Four). A moderate to large negative relationship between the ISI and arousal thresholds was found but this result was not significant. Given the size and direction of the effect and the lack of the sensitivity of the test and the small sample size, it is reasonable to conclude that this result does at least provide partial support for the hypothesis despite being non-significant, especially given that more recent considerations give *p*-values less weight (Balluerka, et al., 2005; Fraley & Marks, 2007)

The second method utilised a between-group design comparing the auditory arousal thresholds (for both types of alarm signals) of the poor sleepers recruited in this study when not consuming their hypnotic with the auditory arousal thresholds of approximate age matched good sleepers (data from Bruck and Thomas 2008a). It was hypothesised that poor sleepers when not under the

influence of their hypnotic would have significantly lower arousal thresholds compared with good sleepers for both alarm signals (Hypothesis Five). This difference was only established tentatively (i.e., significant at a confidence level of close to 90%) at the 3100 Hz sine wave frequency, but was not significant for the 520 Hz square wave. It is possible that a floor effect was responsible for this latter finding. For the poor sleepers to have significantly lower arousal thresholds compared to the good sleepers in response to the 520 Hz square wave, their arousal thresholds would have needed to average around 35 dBA. This is close to or lower than most adults' hearing threshold when asleep (Bruck, 2001) and the equivalent to background noise. It may be that there is a difference in arousal thresholds between good and poor sleepers across both signals but the 520 Hz square wave equalised this difference. Another possibility is that there may only be a difference between good and poor sleepers at certain frequencies, due to an underlying variation in the way sound is processed and interpreted. A final consideration is that due to the additional variability at the higher frequency a greater degree of difference between the groups could be expected, particularly at these small sample sizes, although greater variability would also reduce the likelihood of achieving significance.

The partial support for both Hypothesis Four and Five leaves objective four in somewhat of a quandary, which raises more questions than it answers. The current findings are arguably inconsistent with previous work (Johnson, et al., 1979; Mendelson, et al., 1986), which has found no difference in arousal thresholds between good and poor sleepers. It is likely that the result may not conform to the literature due to differences in sample selection relevant to the theory of hyperarousal and design issues.

Previous research that has investigated hypnotics and arousal thresholds (Bonnet, et al., 1979; Johnson, et al., 1987; Spinweber & Johnson, 1982) has typically recruited poor sleepers who may be less likely to be responsive to external stimuli during sleep. Most research recruited younger adults experiencing sleep onset difficulties. It is possible that the participants recruited for the current study that were suffering from sleep maintenance problems may have been more vulnerable to nocturnal arousal than people suffering primarily from sleep onset issues because the former group have more of a predisposition to arousal when asleep. Lower arousal thresholds amongst people suffering from sleep maintenance insomnia are directly implicated in

hyperarousal theory (Bonnet & Arand, 1997), which attempts to explain insomnia pathophysiologically. This theory suggests that cortical arousal is higher in people suffering from insomnia than in normal sleepers even when asleep (Bonnet & Arand, 1997), and has received considerable support (Bonnet & Arand, 1995, 1998b; Freedman, 1986; Freedman & Sattler, 1982; Hajak, et al., 1995; Haynes, et al., 1981; Irwin, et al., 2003; Lack, et al., 2008; Merica, et al., 1998; Nofzinger, et al., 2004; Perlis, et al., 2001; Riemann, et al., 2002; Stepanski, et al., 1994).

The timing of this endogenous arousal is important and believed to help explain the various insomnia symptoms. Evidence from the temperature regulation literature has been particularly illuminating in this area. It has been shown that there are specific irregularities in core body temperature when comparing across insomnia subtypes (e.g., sleep onset cf. sleep maintenance insomnia). Core body temperature is typically inversely proportional to sleep propensity (Akerstedt, et al., 1979; Lack & Lushington, 1996). That is, lower body temperatures usually coincide with a greater propensity for sleep. In people experiencing sleep onset insomnia, the reduction in core body temperature common prior to sleep is delayed and not synchronised with their habitual sleep time (Morris, et al., 1990). In contrast, people suffering from sleep maintenance insomnia experience elevated core body temperature throughout the night (Lack, et al., 2008). This evidence suggests that although all people suffering from insomnia will experience inappropriate cortical arousal at some point, only a difficulty maintaining sleep is associated with nocturnal awakenings as a function of heightened cortical arousal during the early part of the morning after the onset of sleep. Consequently, the participants in this study, all of whom were experiencing sleep maintenance insomnia, were more likely to experience higher levels of endogenous arousal during the night when the alarms were scheduled to sound, and this would have led to a greater responsiveness to the environment and lower waking arousal thresholds compared to the participants experiencing sleep onset insomnia in previous research.

In providing an explanation for the possible differences between good and poor sleepers on measures of arousal threshold, the current research has produced a novel means of demonstrating support for hyperarousal as a theoretical position. This is the first time that support, albeit somewhat tentative, for hyperarousal has been demonstrated in the field of alarms research and

arousal thresholds. The accumulation of a larger body of evidence is important in supporting hyperarousal, which remains contentious with some researchers (Varkevisser, Van Dongen, & Kerkhof, 2005) despite strong empirical support (Bonnet & Arand, 1997).

Another interesting trend in the data was that correlations between the ISI and arousal thresholds seemed to be stronger after the consumption of a hypnotic (3100 Hz and hypnotic = -.533; 520 Hz and hypnotic = -.410; 3100 Hz and no hypnotic = -.330; 520 Hz and no hypnotic = -.218). This was somewhat unexpected. Participants experiencing the more severe symptoms might be expected to consume larger doses of hypnotics, which would exert a stronger sedative effect and then moderate the relationship between the ISI and arousal thresholds. However, a review of the drug dosages by ISI was not supportive of this interpretation. There was excellent consistency in the type and dosage of drugs consumed across all participants irrespective of the severity of their sleep problems. Although it is unclear how much drug dosage disclosure was contaminated, if at all, by a social desirability bias. This refers to the tendency of participants to answer questions in a manner that will be viewed favourably by others (Edwards, 1957).

Both analytical methods employed in answering the fourth objective were subject to certain limitations that may also have contributed to the inconsistency with the literature. A correlational method was utilised to test Hypothesis Four compared with the experimental designs employed by Johnson et al. (1979) and Mendelson et al. (1986). These studies were measuring group differences while this analysis was merely testing relationships. The reason why an experimental method was not adopted here was that the sample was not large enough or defined with enough precision to conduct this analysis. These issues were not accommodated at the design stage because the objective was exploratory and secondary to other study aims. However, the correlational method did demonstrate that there is potentially a relationship between sleep quality and arousal thresholds, which is worthy of further investigation. Of course, this does not provide any evidence for a causal interpretation (Klein, 1992).

The addition of the between-group experimental design added rigour to the analysis, but in practice, the differences between the two studies made a comparison difficult. Bruck and Thomas' (2008a) research enjoyed a greater sex balance, enforced stricter behavioural protocols,

did not insert silences into the sound delivery program, was subject to different levels of priming, and presented signals during PSG deep sleep. As previously discussed, all of these factors have been shown to impact arousal thresholds. Overall, while the adoption of two approaches increased the level of confidence in the results and future research is warranted in this area, the limitations of each approach mean that care must be taken when reviewing the findings against the prevailing literature.

CHAPTER 3 – STUDY TWO – Coronial Investigation

Brief Introduction

The second study involved a retrospective analysis of Australian coronial data in order to achieve two objectives. The first objective was to determine if users of psychotropics and hypnotics were overrepresented in the Australian fire fatality statistics. Please note that hypnotics are a subgroup of psychotropics, and the terms hypnotics and sedatives are synonymous with one another. This provided an indication of the extent to which consumption of different psychotropic drugs places a person at risk from dying in an Australian residential fire, which was previously not known. **It was hypothesised that users of psychoactive drugs and sedatives would be overrepresented in the coronial database (Hypothesis Six).** The second objective was to determine the relationship between psychotropic drug consumption and a number of behavioural, environmental, and demographic risk factors previously identified in the literature, or added if the variable was complementary to the analysis. In order to understand the role of psychoactive drugs, it is important to have knowledge of how the consumption of psychotropic drugs is related to other documented risk factors. This has been absent in the literature. A multi-factorial investigation of numerous factors is also important due to the complexity of a fire emergency, where a combination of risk factors is often more likely and lethal than any single risk factor on its own (Brennan, 1998). **It was hypothesised that there would be an association between the detection of psychotropic drug usage and selected behavioural, environmental, and demographic risk factors (Hypothesis Seven).** Specifically, it was predicted that where psychotropic drug usage was detected in toxicology analyses of fire fatality victims the following risk factors would be more likely (compared to those with no psychotropic drug use detected): being a middle aged adult (compared to being an adult of other ages); being male (compared to being female); the involvement of smoking materials (compared to no involvement of smoking materials); being alone (compared to not being alone); being asleep (compared to being awake); being in the RFO (room of fire origin) at ignition (compared to being elsewhere at fire start); alcohol intake (compared to no alcohol intake); having no conditions preventing escape (compared to where there were conditions preventing escape); where a smoke alarm was active or present (compared to all other possibilities when a smoke

alarm was not active or present); the presence of a mental illness (compared to the absence of a mental illness); the presence of a physical illness (compared to the absence of a physical illness); and the fire starting during waking hours (compared to the fire starting during resting hours). The above demographic, environmental and behavioural variables were largely the same as those investigated in a study of alcohol and fire risk by Bruck, Ball, and Thomas (2011). Mental and physical illness were added based on literature support. As this study is dealing with drugs affecting both wake and sleeping behaviour, it was also considered prudent to include waking versus resting hours as an independent variable.

The specific association between sedatives and the same behavioural, environmental, and demographic risk factors identified above was also analysed**. It was hypothesised that the association between the risk factors and sedatives would be stronger than the association between the risk factors and the detection of any psychotropic drugs (Hypothesis Eight).** The decrease in mental and physical functioning associated with sedatives is possibly more damaging to an emergency response than other types of drugs that may stimulate or amplify consciousness (Koelega, 1993; Oster, et al., 1990; Pickworth, et al., 1997).

Method

Participants

Over the past several years the team at Victoria University has methodically coded hundreds of detailed coronial files and entered these details into a database following a manual developed at Victoria University for this purpose. The subsequent Victoria University Coroners' Database (VUCD) contains comprehensive information on 376 fatalities that occurred in the three mainland east coast states of Australia (Victoria, New South Wales, and Queensland) from 1998 to 2007 in circumstances of fire. These states represent approximately 80% of the Australian population (Australian Bureau of Statistics, 2011). The database currently includes 210 variables based on information comprehensively compiled in the coronial file, allowing detailed examination of the circumstances of each death.

The database was filtered to only include cases of adult fire fatalities from accidental residential fires who were tested for drugs. Of the initial 376 fatalities, 274 (73%) occurred in residential buildings. A large proportion of cases that were removed (19%) occurred on mobile properties, such as cars. Non-residential fires were excluded because the focus of the study is on residential fires. Of the 274 residential fire fatalities, 175 (64%) were accidental. Murder or suicides were also removed. Of the 175 residential fire fatalities that were accidental, 164 (94%) were adults 18 years or over. Children were removed because the focus is on the adult response to a fire and children are much less likely to be taking psychotropic drugs. Of the 164 residential fire fatalities that were accidental and involved adults, 108 (66%) were tested for drugs prior to any possible hospital treatment. In a small number of cases (n = 17), drug testing was performed after hospital treatment. These cases were removed from the analysis as it could not be determined which drugs had been administered prior to the fire. Therefore, a final sample of 108 residential fire fatalities that were accidental and involved adults who were tested for drugs was used for analysis.

The study was approved by the Victoria University Human Research Ethics Committee.

Materials and Procedure

Objective one. Two lists were required to assess over-representation. The first was a list of actual observed values from the VUCD data, while the second was a list of expected population values obtained from several governmental sources. Data manipulation was required in order to create two data sets that were comparable and easy to interpret.

Provision is made in the VUCD for up to three drugs to be entered. The chemical substance is typically reported. There are currently 40 different drugs or drug categories across the three variables in the VUCD. This list of drugs was sorted into three categories: (a) sedatives; (b) antidepressants; or (c) illicit drugs (see Appendix M for a complete list of classified drugs). The list of sedatives largely included benzodiazepines and zolpidem (a nonbenzodiazepine drug with similar effects). While these three drug classes are mutually exclusive, the categories per victim are not, as various combinations of the drugs may have been detected in the victims. For

example, a drug is not able to be defined as both a sedative and antidepressant, but both of these drugs may be present in a victim. As there are three categories in the database, it is possible for a victim to be positive in all three drug classes. This classification was made for two reasons: (a) sedatives are the focus of the research; and (b) comparable population data on drug usage was only available at these drug class subgroups.

Population data was referenced from two national governmental reports, the: (a) 2010 National Drug Strategy Household (NDSH) survey (Australian Institute of Health and Welfare, 2011); and (b) 2007 National Health (NH) survey (Australian Bureau of Statistics, 2010b). The 2010 NDSH survey was the 10th survey in a series which began in 1985. The survey investigated knowledge of and attitudes toward drugs, drug consumption histories, and related behaviours. The survey also recorded usage in the past week. The 2007 NH survey provided information on the prevalence of mental health conditions, and the types of support received by people suffering from mental health problems. The survey recorded usage in the past two weeks amongst people suffering from distress or a mental health condition. In order to reflect prevalence in the broader Australian population, the figures from the NH survey were assessed as a proportion of the 18 years and over 2007 population statistics (see Appendix N for calculations).

Objective two. Two types of statistical analysis were utilised: an unadjusted Odds Ratio (OR) analysis; and a form of algorithmic modelling discussed in more detail later in this section. This combination of analytical methods was chosen in order to provide an avenue for comparison with prior research, address many of the limitations inherent in the dataset, gain an understanding of the relative importance of the independent variables, and take advantage of emerging predictive modelling techniques suitable for research in this area. The results were explained in the context of a psychological model of accident causation.

Alcohol intake has been considered extensively elsewhere (e.g., Bruck, et al., 2011; FEMA, 1999; Runyan, et al., 1992) and was only documented here as a function of the overall sample and in terms of age and sex. An analysis of combined alcohol and drug intake in relation to the selected risk factors is beyond the scope of this paper; however, a descriptive data review of the association between alcohol and different drug subgroups is briefly explored.

Variable definition. The independent variables (e.g., age, sex and conditions preventing escape) were largely re-coded into binary variables for the analysis. Where this coding process was non-intuitive it was explained together with the results in the following section. In some instances, continuous versions of the variables were used where available for modelling purposes. A complete description of the independent variables is available in Appendix O. Two dichotomous dependent variables were created: (a) psychotropics detected; and (b) sedatives detected. In group (a), only drugs that were considered likely to affect mood, emotions, sleep or arousal were included. Other drugs, such as paracetamol, were ignored. Group (b) included the same list of drugs defined as sedatives in objective one (see Appendix P for a complete list of drugs in both classifications). As sedatives are also psychotropic drugs, group (b) is a subgroup of group (a) and thus the data presented for psychotropic drugs includes sedative use. The concentration of drugs was not listed in the available coroners' data, so only the presence of drugs was presented here.

It should be noted that even the presence of drugs may be inaccurate or underestimated in the VUCD. Differences in post-mortem drug redistribution, the half-life of the drug, or the time between drug consumption and testing can influence testing results. Similarly, toxicology records may underestimate the role of drug presence in fatal fires because they are only requested of victims who have died in a fire, even if others (survivors) were involved in the fire and may have played a role (e.g., they were involved in ignition).

Analytical considerations. Three main types of analyses were performed on the coronial data. First, a Pearson's chi-square goodness of fit test complete with a residual analysis was completed to investigate the level of over-representation of certain groups within the VUCD. This test is suited to problems where frequency data from a single sample is assessed to see how well it fits the population distribution (Tabachnick & Fidell, 2007). One disadvantage is the test has historically been reported to be sensitive to small sample sizes (e.g., below five in each cell) (Warner, 2008). In these cases, Yates' (1934) correction factor has traditionally been recommended, which makes the test more conservative. However, research has emerged over the last several decades that has shown that the chi-square test without correction is in fact very

robust to small cell sizes (Camilli & Hopkins, 1978; Haviland, 1990). Furthermore, these authors recommend against using Yates' correction factor due to an unnecessary loss of power. Therefore, the correction factor was not applied since it is generally recommended to select the most powerful statistical test when testing significance (Tabachnick & Fidell, 2007). Given the size of the disparity, it is unlikely to have made any material difference in any case to the outcome of the analysis.

The second analysis explored the relationship between a number of risk factors and sedative usage using odds ratios with confidence intervals. The odds ratio is a measure of effect size, describing the strength of association or non-independence between two binary data values (Warner, 2008). An advantage of the odds ratio is that it is invariable across different study designs making it easily comparable to previous research (Tabachnick & Fidell, 2007). This analysis also provided a means of validating the algorithmic modelling results discussed in the following paragraph.

Statistical testing (alpha was set at .05) for the first and second analysis was conducted with the software SPSS 20 for Windows (Chicago, Illinois). The SPSS output is available at Appendix Q.

Finally, machine learning techniques were used to investigate the relative importance of associations between sedative usage and other risk factors. Machine learning involves algorithms or techniques that strive to learn from the data. Learning refers to the ability to review the data then extract the important common features that characterise the cases (Hastie, Tibshirani, & Friedman, 2009). The machine learning paradigm is conceptually dissimilar from conventional probabilistic data modelling techniques (Breiman, 2001). In classical statistics the focus is on hypothesis testing and model choice is based on parameter significance and goodness-of-fit statistics. However, in machine learning the emphasis is on predictive accuracy and models are primarily selected using cross validation (Breiman, 2001). More information on cross validation is available in Appendix R.

Boosted Regression Trees (BRT) are a relatively new machine learning technique that combines regression tree and boosting methods to model predictive performance (Elith, Leathwick, & Hastie, 2008; Friedman, 2001; Hastie, et al., 2009). BRTs were used to construct the model using the gbm package in R (Ridgeway, 2007) plus additional custom code created by Elith et. al. (2008). The R syntax is available in Appendix S. The selection of key parameters to optimise the BRT solution was guided by general recommendations made in Elith, et al. (2008).

The key parameters for optimising BRTs are the tree complexity, learning rate, and bag fraction (Elith, et al., 2008). The tree complexity refers to the interaction depth for each tree (Elith, et al., 2008). An interaction depth of one would be a main effects model. The learning rate, which is also known as the shrinkage parameter, regulates the amount of learning possible in each tree (Elith, et al., 2008). While the bag fraction specifies the amount of stochasticity or randomness in the modelling procedure (Elith, et al., 2008). For example, a bag fraction of 0.5 indicates at each iteration the models were built from a random sample of 50% of the raw data. Detailed information on other elements of the BRT method are available in Appendix S.

BRTs have a number of advantages over other forms of regression that compensated for the limitations of the dataset in this study. A key issue was the relatively small sample size and large number of predictors, many of which contained missing data. BRT is well equipped to handle these constraints (Friedman & Meulman, 2003). Furthermore, the work was exploratory and many of the independent variables displayed non-linear patterns. This is a particular strength of BRT models. No assumptions are made with regard to the underlying distribution of independent variables in BRT, which means non-linear functions can be easily fitted. Interactions are also easily managed and BRT is immune to the effects of irrelevant predictors (Friedman & Meulman, 2003). These models are also able to handle a mix of variables (e.g., continuous and categorical), eliminating the need to recode variables and remove variance. Finally, the use of traditional statistical inference testing here was debateable because the sample (coronial files) was the entire population (Bennett, 1985; Western & Jackman, 1994). A major assumption when using statistical inference is that the data are subject to random variation, which is not true of the coronial data which represents all of the available information from the population of interest.

Data limitations. Comparability between the observed VUCD data and the expected population statistics was limited in four key areas. First, the usage profile in each data set varied. Prevalence is a function of the usage range captured. The population statistics reflected usage in the past week or fortnight and likely overestimated prevalence when compared to data captured by the VUCD. This limitation would impact some psychotropic drugs more than others. For example, the prevalence of psychotropic drugs that are consumed less frequently, such as sedatives, would be disproportionally inflated compared with the prevalence of psychotropic drugs, such as antidepressants, that are often consumed daily. In the latter case, prevalence in the past day or in past fortnight would be similar, while in the former case, prevalence in the past fortnight would be substantially higher than prevalence in the past day.

Second, as drug usage figures were only collected from respondents self-reporting distress or a mental health condition in the NH survey, the expected data may underestimate total usage as there may be users of psychotropic drugs who are not suffering from a mental health condition or using pharmaceutical drugs for non-medical reasons. In addition, data quality may have been impacted by self-report bias as respondents may view a discussion of their mental health conditions as very personal or sensitive and thus under-reported.

Third, comparability was also limited by time period. The VUCD covers a time period ranging from 1998 to 2007 and this is not consistent with the time periods in the corresponding governmental data from the NH or NDSH surveys (for which the closest available periods were from 2007-2008, and the 2010 period respectively). Given the large differences between the expected and actual figures in Table 5 and the small likelihood of significant change in the time periods in question (Australian Institute of Health and Welfare, 2011; Gorevski, Bian, Kelton, Martin Boone, & Guo, 2012; Sonnenberg et al., 2011) this limitation was tolerated, as it was not expected to significantly vary the outcome of the analysis.

Finally, the removal of a large proportion of fire fatality cases (n = 56) from the final sample that were not tested for drugs is a threat to external validity. There may have been a selective bias in deciding to conduct a toxicology report. It is reasonable to conclude that toxicology reports were

more likely to be ordered in cases where drug involvement was suspected. However, other unknown biases may also have operated.

Results

The coronial database was filtered to only include a sample of 108 residential fire fatalities that were accidental and involved adults who were tested for drugs of any kind. Of the 108 residential fire fatalities, slightly less than one half (n = 48) tested positive for any type of drug (e.g., including analgesics, such as paracetamol). Of those who tested positive for any type of drug, 65% tested positive for sedatives, 23% tested positive for antidepressants, 8% tested positive for illicit drugs, and 48% tested positive for drugs that were not considered likely to affect mood, emotions, sleep, or arousal, such as paracetamol. At least one psychoactive drug was detected in approximately four out of ten (41/108, 38%) of the total sample. Since a drug of any type (e.g., including analgesics, such as paracetamol) was detected in 48 cases and approximately 41 cases were using at least one psychoactive drug, then when any type of drug was detected, it was almost always psychoactive in nature (41/48, 85%).

On average, there were 1.5 psychotropic drugs detected per fatality amongst those who tested positive for a psychotropic drug (n = 41). As the database only records up to three different drugs from the toxicology report, this figure may slightly underestimate the number of psychotropic drugs per fire fatality; however, most victims had not consumed more than three drugs. Figure 6 displays the number of psychotropic drugs detected in the victim by key demographic splits.

In the following graph and throughout this chapter age was not analysed with orthodox breaks. Age was first examined using sensible linear divisions that were supported by the literature. For example, since older adults have a lower tolerance to sedatives (Wortelboer, Cohrs, Rodenbeck, & Rather, 2002) a comparison was first made between younger adults (under 65) and older adults (65 and over); however, this relationship was not significant. A post hoc exploration of the data revealed that the relationship between age and psychotropic/sedative usage was curvilinear and peaked during middle age. Therefore the decision was made to compare a middle age group (30-60) to an age group including all other adult ages (81% of whom were above 60 and the rest under 30). This decision was entirely data driven.

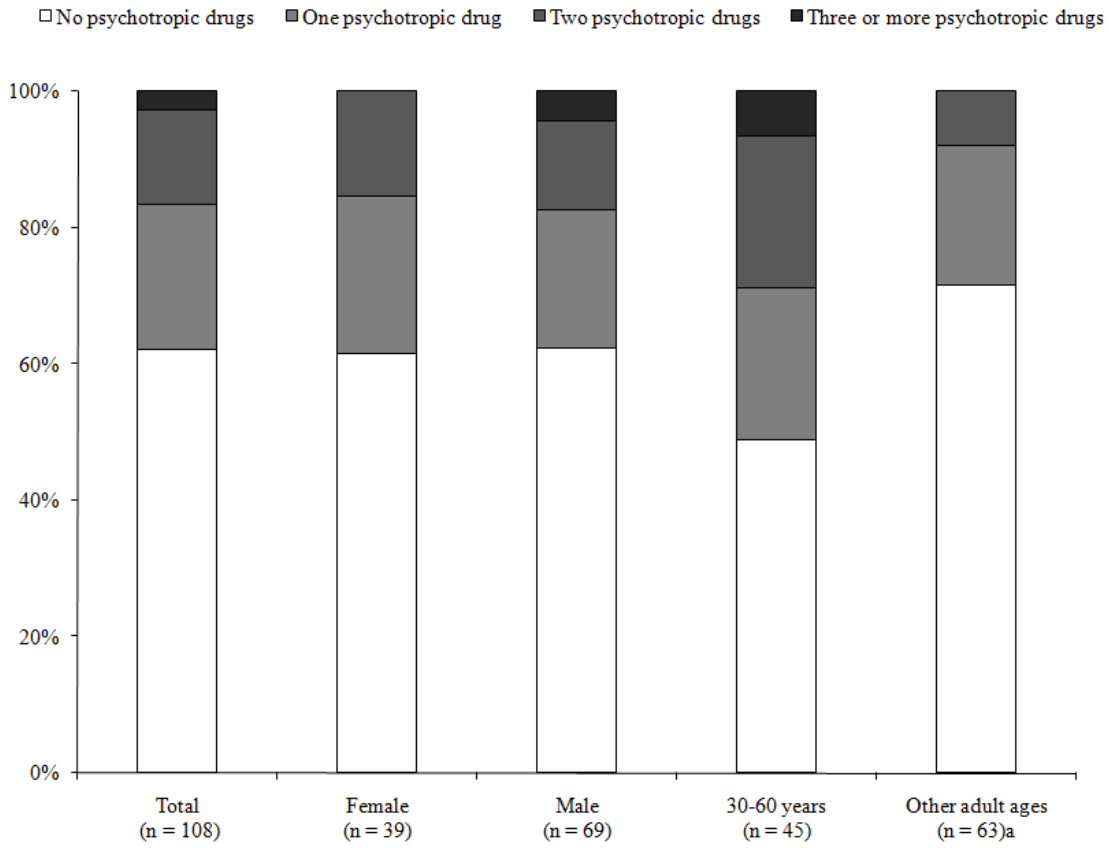

Figure 6. Number of psychotropic drugs detected by sex and age.

a. The other adult age group is comprised primarily of older adults over the age of 60 (18-29 = 19%; 61-99 = 81%; mean = 67 years).

In Figure 6, the middle aged group were more likely to be using an increasing number of psychotropic drugs when compared to the group including other adult ages (1.7 cf. 1.3 drugs per fatality respectively). There was little difference in the number of drugs detected between males and females.

The first objective was to determine whether users of psychoactive drugs were overrepresented, as a group, in the Australian fire fatality statistics compared to the general population. It was hypothesised that users of psychoactive drugs and sedatives would be overrepresented in the

coronial database (Hypothesis Six). Table 5 compares the observed prevalence of three categories of psychoactive drugs in the VUCD with the expected prevalence in comparable adult population statistics. The chi-square test was performed and followed by a complete residual analysis to indicate the strength of any deviation. A *z*-score over two indicates the observed values were much greater than we would expect by chance. The results from Table 5 are presented as a function of sex in Table 6.

Table 5

Observed prevalence (%) of psychoactive drug usage in coronial data (n = 108 observed fire fatalities) compared with the expected prevalence (%) in adult population statistics

Psychotropic Drug	*n*	Expected	Observedb	χ^2	%Δ	*z*
Sedatives	108	1.05a	28.70	794.92***	+2633.33	+28.04
Antidepressants	108	3.43a	10.20	14.88***	+197.38	+3.80
Illicit drugs	108	5.50c	3.70	0.67	-32.73	-0.80

Note. χ^2 = chi-square test (df = 1); %Δ = percentage deviation from expected to observed ((observed – expected / expected) x 100); *z* = standardised residuals ((observed count – expected count / sqrt(expected count)); *p<.05; **p<.01; ***p<.001; drug usage is not mutually exclusive, and the proportion present was grouped against the proportion absent for each drug.

a. Expected values (%s) were sourced from the 2007 NH survey and reflect usage in the past two weeks. These figures were re-proportioned to the general population (see Appendix N for workings).

b. Observed values (%s) were sourced from the coronial data files.

c. Expected value was sourced from the 2010 NDSH survey and reflects usage in the past week of any of one of 13 illicit drugs (listed in Appendix T).

The results from Table 5 provided very strong evidence of a significant difference between psychotropic pharmaceutical drug prevalence in the VUCD compared with the general population. The difference between the prevalence of users of sedatives who were victims of fire (28.7%) and users in the general population (1.05%) was 26.33 times greater than what we would have expected, and this result was highly significant. Antidepressants were also significantly overrepresented in the coronial data, but standardised scores suggested the

difference was much stronger for users of sedatives (note the data limitations discussed in the Method section).

Table 6

Observed prevalence (%) of psychoactive drug usage in coronial data (n = 108 observed fire fatalities) compared with the expected prevalence (%) in adult population statistics as a function of sex

Psychotropic Drug	*n*	Expected	Observedb	χ^2	%Δ	*z*
Females						
Sedatives	39	1.39 a	33.30	288.10***	+2296.19	+16.90
Antidepressants	39	4.20 a	15.40	12.13**	+266.63	+3.41
Illicit drugs	na					
Males						
Sedatives	69	0.69 a	26.10	639.77***	+3662.90	+25.34
Antidepressants	69	2.63 a	7.20	5.88*	+173.33	+2.34
Illicit drugs	69	6.60 c	5.80	0.07	-12.12	-0.26

Note. χ^2 = chi-square test (df = 1); %Δ = percentage deviation from expected to observed ((observed – expected / expected) x 100); *z* = standardised residuals ((observed count – expected count / sqrt(expected count)); *p<.05; **p<.01; ***p<.001; drug usage is not mutually exclusive, and the proportion present was grouped against the proportion absent for each drug; na = test unable to be performed due to missing data.

a. Expected values (%s) were sourced from the 2007 NH survey and reflect usage in the past two weeks. These figures were re-proportioned to the general population (see Appendix N for workings).

b. Observed values (%s) were sourced from the coronial data files.

c. Expected value was sourced from the 2010 NDSH survey and reflects usage in the past week of any of one of 13 illicit drugs (listed in Appendix T).

When analysing the observed versus the expected figures by sex, the results from Table 6 suggest that male and female users of both sedatives and antidepressants were significantly overrepresented in the coronial data. The deviations vary by sex. Male sedative users were slightly more overrepresented than female sedative users. Male victims of fire were 36.63 times more likely to be consuming sedatives compared with the general population while female victims of fire were 22.96 times more likely to be consuming sedatives compared with the

general population. In contrast, male antidepressant users were slightly less overrepresented than female antidepressant users (victims were 2.67 cf. 1.73 times more likely than the general population respectively).

The second objective was to determine the relationship between psychotropic drug consumption and a number of behavioural, environmental, and demographic risk factors. It was hypothesised that there would be an association between the detection of psychotropic drug usage and selected behavioural, environmental, and demographic risk factors (Hypothesis Seven). A review of these relationships was first analysed via unadjusted odds ratios in order to produce results that were comparable to previous research and not clouded by the presence of other variables. Table 7 presents the odds ratios and confidence intervals calculated for each independent variable as a function of psychotropic drug usage. This table is sorted by magnitude of effect. Table 8 presents a similar analysis amongst victims where only sedatives were detected. It was hypothesised that the association between the risk factors and sedatives would be stronger than the association between the risk factors and the detection of any psychotropic drugs partially due to the greater depressive effects of sedative drugs (Hypothesis Eight).

Please note that some risk factors achieved significance despite having a lower odds ratio than other measures where no significance was noted. For example, smoke alarm status recorded an odds ratio of 2.98 and was not significant but age was significant with an odds ratio of just 2.56 (see Table 8). This is because significance is a function of more than simply the effect size. As with all tests of statistical inference, the probability of achieving a significant result or the power is determined by the sample size, alpha level, effect size, and the variability inherent in the data (Cohen, 1992). Variability in this case is expressed via the proportion of cases exhibiting the relevant feature. Significance levels are enhanced by the size of the effect and a larger sample size, while significance is less likely with greater variability or a more stringent alpha level (Cohen, 1992). In the above example, smoke alarm status contained fewer cases than the age factor (81 cf. 108). A significant ($p = < .05$) result would have been achieved with a comparable sample size, all other parameters being equal.

Table 7

Relationship between behavioural, environmental, and demographic risk factors and the detection of psychotropics (PD)

Risk Factor	*n*	No PD	PD	OR	95% CI
Mental illness	90a			5.17***	[2.03,13.14]
Present		20	30		
Absent		31	9		
Physical illness	107a			3.04*	[1.22,7.58]
Present		38	33		
Absent		28	8		
Location	92a			2.69	[0.96,7.54]
In RFO at fire start		37	26		
Elsewhere at fire start		23	6		
Age	108			2.61*	[1.17,5.82]
30-60		22	23		
Other adult ages		45	18		
Smoking related materials	108			2.27*	[1.02,5.05]
Involved		29	26		
Not involved		38	15		
Smoke alarm	81a			2.14	[0.69,6.64]
Active		7	8		
All other possibilities		43	23		
Conditions preventing escape	108			2.13	[0.94,4.84]
Present		18	18		
Absent		49	23		
People presence in home	108			2.07	[0.79,5.44]
Alone		47	34		
Not alone		20	7		
Smoke alarm	81a			1.82	[0.72,4.57]
Present		25	20		
Not present		25	11		
Time of fire	100a			1.59	[0.68,3.73]
10:00 to 22:00 hours		18	15		
22:01 to 09:59 hours		44	23		
Alcohol intake (BAC > 0)	106a			1.55	[0.69,3.48]
Positive		36	26		
Negative		30	14		
Sleep/wake	108			1.47	[0.66,3.30]
Awake		38	27		
Asleep		29	14		
Sex	108			0.97	[0.43,2.17]
Male		43	26		
Female		24	15		

Note. OR = odds ratio; CI = confidence interval; RFO = room of fire origin; *p<.05; **p<.01; ***p<.001; for each variable the ratio of the latter to the former was estimated; a$N \neq 108$ as a result of insufficient available information on some fatalities; Pearson chi-square tests were used to calculate all *p*-values.

Table 8

Relationship between behavioural, environmental, and demographic risk factors and the detection of sedatives (SD)

Risk Factor	*n*	No SD	SD	OR	95% CI
Mental illness	90a			7.00***	[2.36,20.79]
Present		25	25		
Absent		35	5		
Physical illness	107a			3.58*	[1.24,10.35]
Present		45	26		
Absent		31	5		
Smoke alarm	81a			2.98	[0.93,9.55]
Active		8	7		
All other possibilities		51	15		
Location	92a			2.91	[0.89,9.47]
In RFO at fire start		43	20		
Elsewhere at fire start		25	4		
People presence in home	108			2.88	[0.90,9.15]
Alone		54	27		
Not alone		23	4		
Time of fire	100a			2.79*	[1.13,6.91]
10:00 to 22:00 hours		19	14		
22:01 to 09:59 hours		53	14		
Age	108			2.56*	[1.09,6.02]
30-60		27	18		
Other adult ages		50	13		
Smoking related materials	108			2.18	[0.92,5.17]
Involved		35	20		
Not involved		42	11		
Smoke alarm	81a			2.07	[0.74,5.81]
Present		30	15		
Not present		29	7		
Alcohol intake (BAC > 0)	106a			1.99	[0.81,4.91]
Positive		41	21		
Negative		35	9		
Conditions preventing escape	108			1.40	[0.59,3.33]
Present		24	12		
Absent		53	19		
Sleep/wake	108			1.29	[0.55,3.07]
Awake		45	20		
Asleep		32	11		
Sex	108			0.71	[0.30,1.66]
Male		51	18		
Female		26	13		

Note. OR = odds ratio; CI = confidence interval; RFO = room of fire origin; *p<.05; **p<.01; ***p<.001; for each variable the ratio of the latter to the former was estimated; a$N \neq 108$ as a result of insufficient available information on some fatalities; Pearson chi-square tests were used to calculate all *p*-values.

The following discussion begins by addressing the odds ratio analysis for each independent variable in dedicated subsections, prior to a review of the results from the algorithmic modelling. The presentation of the independent variables is identical to the order in which the variables have been raised throughout the thesis. The results in each subsection will be dealt with in order of magnitude.

Age/sex. Age was a predictor of positive psychotropic drug detection. The odds of detecting psychotropics were 2.61 higher (see Table 7) when aged between 30 and 60 years of age when compared to adults of other ages, and this result was significant ($\chi^2(1, N = 108) = 5.663, p < .05$). A comparable and significant trend was also evident when detecting sedatives (see Table 8). There was no association between sex and either psychotropic drug or sedative detection.

Smoking related materials. Smoking materials were defined in the database as "discarded cigarette or other smoking materials". These included fires that may have been started by matches and lighters where there was evidence that smoking activity was most likely to have been happening at the time of fire onset. In this subset the involvement of smoking materials for each victim was rated as: (i) definite; (ii) most likely; (iii) possible/speculated; or (iv) not involved. For the purposes of the analyses, the first two options were considered as indicative of smoking materials being involved, and the last two as no smoking materials involved.

Smoking materials were involved in slightly more than half (51%) of all cases, and approximately two-thirds of cases where psychotropic drugs were implicated. The odds of psychotropics being detected were 2.27 times greater when smoking materials were involved compared with occasions when smoking materials were not involved. This result was significant, with $\chi^2(1, N = 108) = 4.125, p < .05$. As can be seen in Table 8, the relationship between smoking materials and the detection of sedatives was less convincing (OR = 2.18; $\chi^2(1, N = 108) = 3.213, p = .073$); however, a *p*-value between .05 and .10 suggested there was some evidence of an effect (Warner, 2008).

It was suspected that the relationship between sedatives/psychotropic drugs and smoking materials may have been impacted by the sleep/wake status of victims. The results from this analysis were compared against a similar analysis comparing alcohol and the sleep/wake status of victims, given the only comparable literature involved alcohol. A post-hoc exploration of data from the VUCD showed that in a large number of fires (51%) that involved smoking materials the victim was asleep at the time of fire start. Victims where alcohol was involved were more likely to be asleep (50%) and perish during resting hours (69%), compared with victims where sedatives had been detected, who were more likely to be awake (65%) and perish during waking hours (50%).

People presence in home. The large majority (75%) of fire victims were alone at home at fire start, and many of these victims were living alone at the time. In fact, only one victim of the 68 living alone was not alone at fire start. An odds ratio of 2.88 between people presence in home and the detection of sedatives was not significant, though there was a trend toward significance, with $\chi^2(1, N = 108) = 3.393$, $p = .065$. Similarly, being alone was not a significant driver of psychotropic drug detection in victims (OR = 2.07; $\chi^2(1, N = 108) = 2.215$, $p = .137$).

It was suspected that the relationship between sedative consumption and people presence in the home may have been impacted by whether the victim lived alone. A post-hoc exploration of data from the VUCD showed that victims where sedatives had been detected were more likely (87%) to live alone when compared to victims where no sedatives had been consumed (70%).

Sleep/wake. It is likely that the number of people coded as asleep were underestimated, as sleeping was only mentioned in the coroners' files if there was some reason to believe that this was the case. Where it was not known, the default option would be left as "wake". A fatality was coded as asleep if there was evidence of sleep before ignition, at ignition, or during the fire. Again, in this subset the likelihood of each victim being asleep was rated as: (i) definite; (ii) most likely; (iii) possible/speculated; or (iv) not involved. For the purposes of the analyses, the first two options were considered as indicative of sleep, and the last two as awake. There was no evidence of an association between drug usage and being awake or asleep. The majority of victims (60%) were awake at the time of fire start.

Location. Of the 63 victims (68%) who were in the RFO at fire start, 17 (27%) were involved in ignition either by their own direct actions, or by inaction (e.g., they had left a burning cigarette on a blanket). Location at ignition was unable to be determined in a small number of cases (*n*=16). In an unknown number of cases the burnt condition of the human remains for those in the RFO precluded accurate drug testing. This may have lead to an underestimation of the number in the RFO and drug affected.

The odds of psychotropics being detected were 2.69 times greater when in the RFO at fire start compared to occasions when they were not in the RFO at fire start. While the association between psychotropic drug usage and location was not significant, the difference approached significance, with $\chi^2(1, N = 92) = 3.708$, $p = .054$. A similar pattern was evident for victims where only sedatives were detected, with $\chi^2(1, N = 92) = 3.320$, $p = .068$ and an odds ratio of 2.91.

It was suspected that the relationship between sedative consumption and location may have been impacted by whether the victim was involved in starting the fire at the point of ignition and the type of this involvement (i.e., how the fire was started). Again, the results from this analysis were compared against a similar analysis comparing alcohol and victim involvement at the point of ignition, given the only comparable literature involved alcohol. A post-hoc exploration of data from the VUCD suggested that victims where sedatives had been detected were more likely than victims who had not consumed sedatives or consumed alcohol to be involved in starting the fire at the exact point of ignition (23% cf. 13% respectively).

Conditions preventing escape. In many cases the fire victims were prevented from escaping by an environmental factor or the very rapid progress of the fire. A summation of all the cases where there was a condition preventing escape was made where this included the following: no time as the fire progressed too rapidly (e.g., explosion, accelerant); clothing on casualty burning; blocked due to fire between casualty and exit; doors locked; or windows barred. Other circumstances which were not included in this variable included where the victim was overcome with smoke, was asleep, or suffered poor visibility due to smoke. The rationale

for not including such factors was that such circumstances typically arose from responding too slowly to the presence of the fire, and drug usage was likely to affect such response time. The focus was on more impeding environmental circumstances once the fire had started. Again, in this subset the involvement of conditions preventing escape for each victim was rated as: (i) definite; (ii) most likely; (iii) possible/speculated; or (iv) not involved. For the purposes of the analyses the first two options were considered as indicative of conditions preventing escape being involved, and the last to as not.

Environmental circumstances prevented escape in approximately one third of cases. The relationship between conditions preventing escape and the detection of psychotropics approached significance (OR = 2.13; $\chi^2(1, N = 108) = 3.322$, $p = .068$). There was no significant association between conditions preventing escape and the detection of sedatives.

Smoke alarm presence/operation. The impact of a smoke alarm was analysed in two variables. One variable simply recorded smoke alarm presence and the other indicated smoke alarm presence and operation. A smoke alarm was present in slightly more than half (56%) of cases, but was known to have operated on just one out of three (19%) occasions. This distinction was made in an attempt to delineate behavioural safety predispositions associated with smoke alarm ownership with the actual usefulness of a smoke alarm when it was known to have operated. In 27 (25%) of cases it was not known or otherwise unspecified whether or not a smoke alarm was present so they were removed from both the analysis of smoke alarm presence and smoke alarm operation.

There was no significant association between smoke alarm presence and the detection of drugs. There was some evidence of an effect when smoke alarms were present and operated and only sedatives were involved. The odds of sedatives being detected were 2.98 times greater when a smoke alarm was operating in comparison to when sedatives were detected and a smoke alarm was not operating, with $\chi^2(1, N = 81) = 3.541$, $p = .060$ (see Table 8).

It was suspected that the relationship between sedative consumption and smoke alarm presence/operation may have been impacted by whether the victim was living in an apartment

block. A post-hoc exploration of data from the VUCD showed that victims that achieved a positive sedative test were 4.57 times more likely to be residing in apartment blocks compared to victims where sedatives were not detected (32% cf. 7% respectively). Smoke alarms were more likely in apartment blocks and were present in nine out of 11 multiple storey units.

Additional post-hoc analysis looked at the circumstances of victims who died with sedatives in their system when a smoke alarm was known to have operated. First, it was evaluated whether victims with sedatives in their system were awake and in the RFO at fire start. Data from the VUCD showed that five of the seven fatalities with sedatives in their system who died despite a working smoke alarm were awake and in the RFO at fire start. Second, the upkeep of homes of victims who had an operating smoke alarm were evaluated to determine possible favourable pre-existing safety attitudes. Data from the VUCD showed that victims who had an operating smoke alarm were more likely to have lived in a clean and tidy house.

Alcohol intake. A positive (greater than zero) Blood Alcohol Concentration (BAC) was recorded in almost six out of ten cases (58%). BAC refers to the amount of alcohol in the body measured in grams of alcohol per 100 millilitres of blood (g%). Almost all (92%) of the victims with alcohol in their system recorded a BAC above .10 g%. The mean BAC was .20 g% with a range from .014 g% to .370 g%. The legal limit in Australia for driving is .05 g%. BAC information was not available for two fatalities and these cases were removed from the analysis.

Alcohol intake was also evaluated as a function of sex and age (see Table 9). Examination of the data from Table 9 revealed that alcohol was significantly more likely to be detected in the middle aged group than other adult ages, with $\chi^2(1, N = 106) = 7.560$, $p < .01$. This age group was also more likely to have a higher concentration of alcohol in their blood when compared to other adult ages (0.24 g% cf. 0.17 g% respectively).

Table 9

Alcohol intake as a function of sex and age

		Sex		Age	
	Total	Female	Male	30-60 years	Other ages
	(*n* = 106)	(*n* = 38)	(*n* = 68)	(*n* = 43)	(*n* = 63)
Positive alcohol intake (%)	58	50	63	74**	48
Mean BAC (g%)a	0.20	0.21	0.20	0.24	0.17

Note. *p<.05; **p<.01; ***p<.001; significance tests compared non-overlapping splits within each demographic (e.g., female cf. male, rather than female cf. total).

a. Based on victims who registered a positive BAC only.

Alcohol and drug intake. An association with testing positive for alcohol was evident amongst victims where sedatives were detected and with victims where any psychotropic was detected (OR = 1.99 cf. 1.55 respectively); however, the association for either psychotropic drug category was not significant. Nevertheless, when psychotropic drugs were present in the system, alcohol was usually involved.

The relationship between alcohol and the two other main psychotropic drug subgroups was reviewed and is displayed in a Venn diagram in Figure 7. The three-way Venn diagram shows all the possible logical relations between each of the three drug subgroups. For example, sedatives, antidepressants, and alcohol were all detected in 7% of victims. Figure 7 is based on victims where sedatives, antidepressants, and/or alcohol were detected (75/108, 69%). The remainder of the sample were either not using any drugs or alcohol (27/108, 25%), or only using drugs that were not defined as psychoactive (e.g., paracetamol).

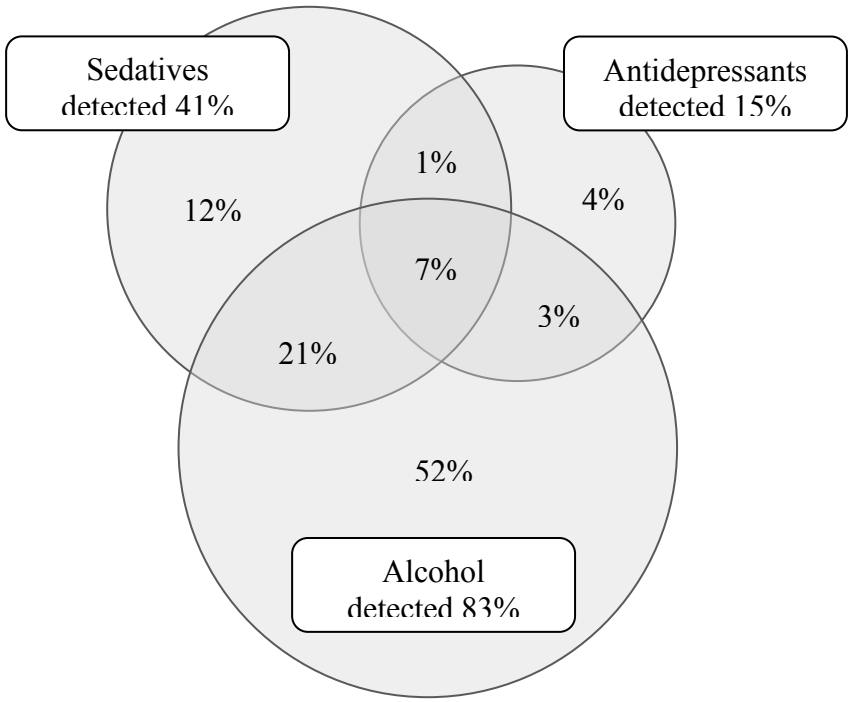

Figure 7. Venn diagram depicting the relationship between drug types.

Note: *n* = 75 (sedatives, antidepressants, or alcohol detected).

As Figure 7 shows, there is a great deal of crossover between the three drug subgroups. Two or more drug subgroups were detected in almost one out of three (32%) victims. Users of sedatives and users of antidepressants were more likely to be using drugs in one or more of the other two psychotropic drug categories in the Venn diagram compared with users of alcohol. Sedatives and alcohol were detected in more than one in four (28%) cases - although one in four of these victims were also using antidepressants (7%/28%, 25%). Interestingly, middle aged adults were more likely to be using multiple drug types, such as alcohol and sedatives, compared to adults of other ages.

Mental illness. The vast majority of sedative or psychoactive drug users were suffering from a mental illness (83% and 77% respectively). A significant difference was recorded for both drug groupings, but sedative consumption presented an even greater risk for mental illness.

The odds of detecting sedatives in people with a mental illness were seven times higher when compared to victims without a mental illness ($\chi^2(1, N = 90) = 14.063, p < .001$). The two most prevalent mental disorders were depression or substance abuse disorder.

A number of factors may have decreased the prevalence of mental illness in the sample. First, the mental condition of victims was only entered when a formal diagnosis was documented. Second, a number of cases where the mental illness status of victims was unknown ($n = 18$) were removed from the analysis. Finally, despite insomnia and other sleep disorders being recognised as types of mental illness in the DSM (American Psychiatric Association, 2013) they do not feature in the codeframe on the database. This is because the codeframe was built off the coronial data, and the coroners' files were less likely to mention or emphasise mental illnesses that are less obvious or not on the public agenda, such as sleep disorders. It is perhaps likely that a diagnosis of some form of insomnia would precede a sedative prescription. Although, given the increasing non-medical usage of prescription drugs (Ross & Darke, 2000; Schuckit, et al., 2002), this may be an unqualified assumption. Overall, it is difficult with the data available to disentangle any possible insomnia as an essential or associated feature of another mental disorder. Nevertheless, this information does present a picture of mental illness if sleeping issues are to some extent assumed in the victims who had consumed sedatives.

Physical illness. Victims who had suffered from a physical disability were significantly more likely to have consumed any type of psychotropic drug compared with victims who were not suffering from a physical disability. A significant odds ratio of 3.04 between physical illness and psychotropic drug consumption was evident. A slightly stronger risk was associated with the consumption of sedatives, where the odds of sedative consumption where 3.58 times higher when physical disability had been implicated in the fatality compared with occasions when physical disability had not been implicated in the fatality. Heart disease, respiratory disease, and diabetes featured prominently in the list of physical ailments relative to other types.

Time of fire. While there was a variable specifying the time of fire in the database, the precise time of fire start was unknown in a substantial number of cases. However, this information can be reasonably inferred from other time related variables. The final time of fire

variable was created by first establishing a hierarchy of time related variables sorted on their time proximity to the actual time of fire. The four variables in order were: (1) time of fire start; (2) time first person alerted; (3) time of notification of fire services; and (4) time of arrival of fire services. The time at variable one was entered, but if this cell was empty, the time at variable two was entered, and this process was repeated until a valid time reference was recorded. A small number of cells (n = 8) were removed due to either an invalid time reference or lack of confidence in the accuracy of the time. In most of these cases the fire had self-extinguished prior to discovery.

In the large number of cases where the exact time of fire was not known there was always likely to be some time delay reflected in the figures, even if only a matter of minutes. This bias might also have disproportionately impacted certain fires (e.g., smouldering fires) more than others (e.g., explosive fires). Even so, given the focus in this analysis was primarily comparing day and night time, two large time intervals, this limitation was not viewed as a major impediment and was not expected to change the outcome of the analysis. Furthermore, it would be unusual for a fire of a sufficient size and ferocity to inflict casualities to go unnoticed for too long.

The results point to a strong time related effect amongst victims who had consumed sedatives. The odds of detecting sedatives were 2.79 times higher when perishing in a fire during the waking hours (10:00 to 22:00 hours) compared to the odds of a fire death during resting hours (22:01 to 09:59 hours) when sedatives were detected ($\chi^2(1, N = 100) = 5.083$, $p < .05$). This result appeared to be counter-intuitive. Since sedatives are typically consumed prior to bed time at night, it would be normal to expect an association between sedative detection and night time fires rather than day time fires. However, many popular sedatives have relatively long half-lives that would ensure the drug's presence remains at levels that may still produce impairment after 24 hours (Gray et al., 2006; Kool, Ameratunga, & Robinson, 2011; MacDonald, 1999; Rosenberg, 2006). It is also possible the sedative was taken during the daytime, particularly for non-medical purposes. Interestingly, this effect did not hold for the broader psychoactive group.

It should also be noted that the effect of time of fire may be confounded by time spent at home. It may be that victims who had consumed sedatives were more likely to spend time at home

during the daytime, thereby increasing their chances of falling victim to fire. Of course, a similar criticism could also be made of the association between early morning and fire fatalities. After all, this is a time interval when almost all individuals can be found at home. To test this possibility the relationship between sedative consumption and time of day was observed after controlling for time spent at home. It is difficult to ascertain time spent at home from the available data, but this can be inferred from indirect sources. Since occupational status is a marker of daytime activity in the home, this variable was used for this purpose and retirees and the unemployed were defined as occupations with relatively high potential for home activity and other types of occupation (e.g., employed, student) were defined as occupations with relatively low potential for home activity. A logistic regression was run with sedative consumption as the dependent variable and time of fire as the independent variable. As per Table 8, prior to controlling for home activity, the odds of detecting sedatives were significantly (2.79 times) higher when perishing in a fire during the waking hours (10:00 to 22:00 hours) compared to resting hours (22:01 to 09:59 hours). After adding home activity as an independent variable into the regression, the odds of detecting sedatives remained significantly higher, but the odds ratio was smaller (2.41 times greater after controlling for home activity). Therefore, it was found that the relationship between sedative consumption and time of fire still held even after controlling for occupations associated with home activity during the day. This suggests any impact from time at home is unlikely to significantly alter the outcome of the analysis; however, it is important to recognise that a more robust measure of time spent at home is needed to add rigour to this analysis.

Even if this analysis underestimates the importance of time spent at home, it does not invalidate the hypothesis that increased and impaired activity during the waking hours is placing vulnerable individuals at risk. In order to provide more clarity on this issue, a frequency count analysis of the cause of fire by time of fire was carried out, even though this was not specifically hypothesised. There were clear differences in the cause of fires between night and day that implicate human involvement. For example, daytime fires were three times more likely to be cooking fires, and 40% less likely to be caused by an electrical failure than night time fires. This supports the notion that erroneous human involvement was more likely during the day and was responsible for the fire.

Algorithmic modelling (BRT). The unadjusted OR analysis provided an indication of the association between psychotropic/sedative consumption and a number of behavioural, environmental, and demographic risk factors. This allowed a comparison against previous research using similar analytical approaches, and was an analysis not influenced by other variables. Algorithmic modelling was then used to determine the relative importance of these same associations and their level of interdependence. Only sedative usage was predicted in the modelling since this was a focus of the study.

The BRT and unadjusted odds ratio analyses used largely the same variables with some minor variations. In order to increase data richness the continuous versions of age, blood alcohol content, and time of fire were used. Data complexity was also minimised by merging the alarm presence and operation variables into one variable, alarm operation. A complete description of all the variables used in both analyses is available in Appendix O.

Two BRT models were run. A base model including all predictors was initially fitted, and then this model was simplified. The simplified model involved dropping unimportant variables using a method similar to backward elimination. This involves starting with all the predictors then deleting candidate variables that improve the model against some criterion (in this case where the reduction in predictive performance exceeds some threshold) until no further improvement is possible (Elith, et al., 2008). This process yielded four variables for removal. Model simplification is particularly useful for small data sets where redundant predictors may degrade performance by increasing variance (Elith, et al., 2008). Overall, the simplification resulted in a more parsimonious model without significantly eroding model fit (see Table 10).

The area under the Receiver Operating Characteristic (ROC) curve (AUC) and deviance were used to measure model performance (Table 10). These complementary measures have been used extensively in the literature to measure predictive performance (Elith, et al., 2008; Hastie, et al., 2009; Ridgeway, 2007). A brief description of each now follows.

The AUC is a measure of discrimination and can be interpreted as the probability that a classifier will rank a randomly chosen positive instance (sedative detected) higher than a negative instance (sedative not detected) that is randomly chosen (Hanley & McNeil, 1982). AUC ranges from 0 to 1, with values below 0.6 indicating a performance no better than random, values between 0.7 to 0.9 considered as useful, and values greater than 0.9 as excellent (Hanley & McNeil, 1982).

Deviance (*D*) is a goodness of fit measure. The residual deviance is equivalent to the residual sum of squares in traditional regression and indicates how much of the variation in sedative usage the model did not explain (Zuur, Leno, Walker, Saveliev, & Smith, 2009). A smaller residual deviance will result in a better model. The null deviance is analogous to the total sum of squares and signifies the total variation in sedative usage in the model (Zuur, et al., 2009). The proportion of deviance explained (*D* = null deviance – residual deviance / null deviance) is a measure of overall model accuracy, analogous to the R^2 in linear regression (Yee & Mitchell, 1991).

Table 10

Evaluation statistics of the base and simplified models

Model	No. Predictors	No. Trees	AUC	*D*
Base	12	1500	0.76 (0.06)	31 (0.53)
Simplified	8	1300	0.74 (0.05)	30 (0.47)

D = % deviance explained with standard errors in brackets; AUC = area under the Receiver Operating Characteristic (ROC) curve with standard errors in brackets.

For the base model, where the number of predictors = 12, the learning rate = .001, the interaction depth = 4, and the bag fraction = .5, the optimal number of trees was reached at 1500 (Table 10). The base model accounted for 31% of the total deviance and an AUC of .76 was achieved. These performance measures suggest the model performed well. The simplified model reached the optimal number of trees at 1300 with largely the same settings and four fewer variables. Despite a smaller quantity of predictors being included, the simplified model performed at parity with the base model.

Compared with conventional regression, there are no *p*-values in BRT to indicate the significance of model coefficients. Instead, the relative importance of each predictor was calculated using a formula developed by Friedman (2001) for both the base and simplified models for comparison (Figure 8). This technique is considered to be a robust alternative to traditional methods (Whittingham, Stephens, Bradbury, & Freckleton, 2006). The measure is based on a predictor's contribution to model fit (Friedman, 2001). Relative importance scores range from 0-100 with higher numbers indicating greater importance.

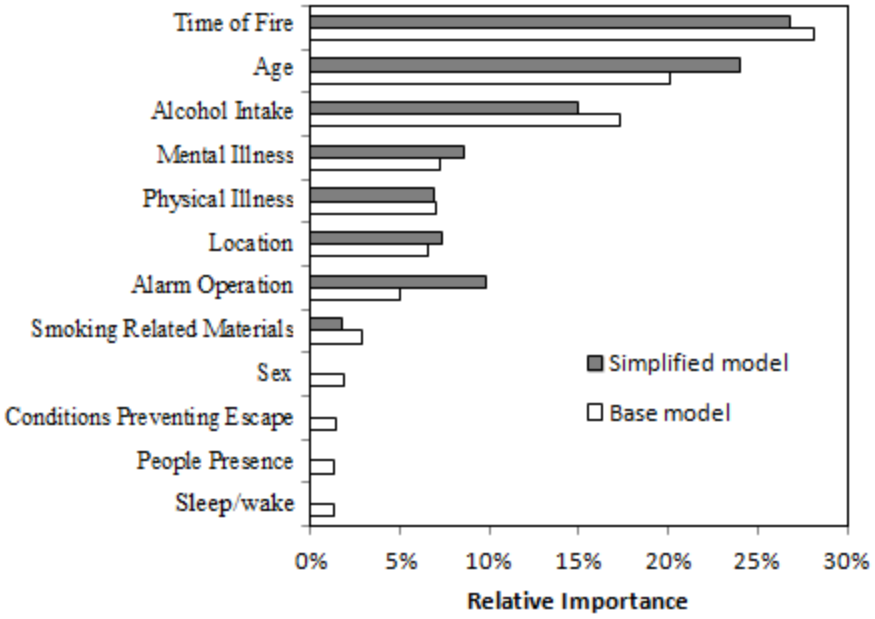

Figure 8. Summary of the relative contributions (%) of variables predicting sedative consumption using a boosted regression tree model first developed with all predictors, then simplified by removing unimportant predictors.

For the simplified model, the five most influential (important) variables were: (1) time of fire; (2) age; (3) alcohol intake; (4) alarm operation; and (5) mental illness (see Figure 8). Sex, conditions preventing escape, people presence in home, and sleep/wake were removed during simplification indicating these variables were of limited importance in fitting the model. Alarm operation's importance almost doubled after the expulsion of these variables.

Partial dependence plots were then created for the most influential predictors of sedative usage fitted in the simplified model. Partial dependence plots are similar to a partial residual plot in conventional regression, and attempt to show the nature of the dependence between sedative consumption and the response variable given the effects of other variables in the model. Specifically, partial dependence plots showed the effect of a variable on sedative consumption after accounting for the average effects of all other variables in the model (Hastie, et al., 2009).

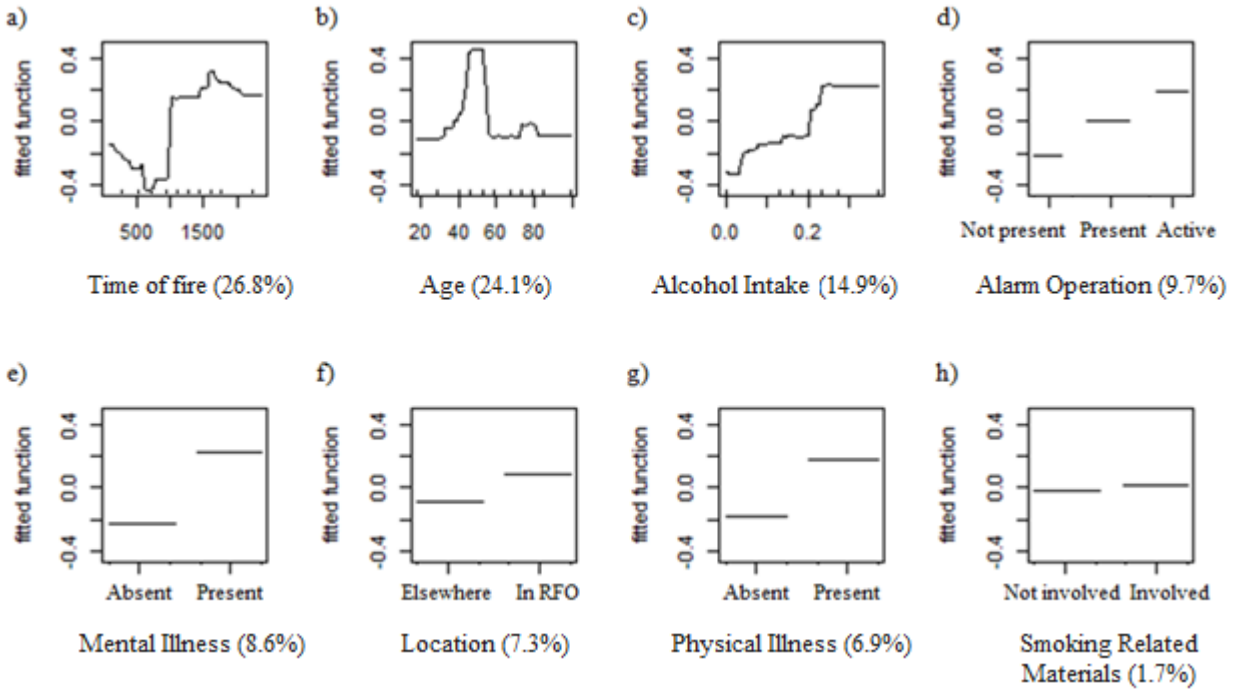

Figure 9. Partial dependence plots for the most influential variables in the simplified boosted regression tree model that show the effect of a variable on sedative consumption after accounting for the average effects of all other variables in the model. The plots are sorted by their related influence, in parentheses. *Y* axes are on the logit scale and are centred to have a zero mean over the data distribution. Rug plots at inside bottom of plots show the distribution across that variable, in deciles. Proportions in brackets reflect relative importance in the simplified model.

Figure 9 shows the partial dependence plots for the most influential variables in the simplified boosted regression tree model. The plots are sorted by their related influence, in parentheses. Y axes are on the logit scale and are centred to have a zero mean over the data distribution. Rug plots at inside bottom of plots show the distribution across that variable, in deciles. Also note

that the plots on the continuous data are not strictly smooth. This is a result of using tree based models, where smoothness is not a constraint imposed on the fitting procedure (Hastie, et al., 2009).

The partial dependence of sedative usage and time of fire was non-linear and fluctuated between day and night hours (Figure 9a). During the early hours of the morning the response declined before dramatically increasing at around 10:00 hrs. The curve then plateaued, peaking shortly after 15:00 hrs.

Sedative consumption was heavily dependent on age. The slender peak in some ways resembled a leptokurtic distribution (Figure 9b). The marginal effect increased substantially throughout middle age (40-60 years), while there was also evidence of weak dependence around 80 years of age. Data density, indicated by the concentration in the rug plots, also increased around these two peaks.

The dependence between sedative usage and alcohol consumption was largely positive; however, when alcohol consumption exceeded 0.2, sedative consumption increased sharply (Figure 9c). Similarly, the partial responses for mental and physical illness indicated that users of sedatives were more likely to have suffered from these ailments. Users of sedatives were also more likely to have lived in a dwelling with a working smoke alarm than no working smoke alarm. Somewhat unexpectedly given their importance in earlier analyses when the variable was analysed without interdependencies, sedative consumption appeared to have weak partial dependence on smoking related materials and location.

In particular, the weak partials with location (Figure 9f) appeared to be inconsistent with its relative importance rating from Figure 8. This suggested that an interaction with other predictors may be masking the effect of location on sedative consumption. Table 11 presents the top five most important pair-wise interactions for the simplified model. Age and time of fire emerged as important interactors. Interestingly, location's interaction with age was the highest pair-wise interaction detected for the simplified model, which was consistent with location's relatively lofty position in the odds ratio analysis of importance relative to the evidence from the partials.

Table 11

Top five most important pair-wise interactions for the simplified model

Rank	Variable 1	Variable 2	Interaction Size*
1	Location	Age	6.49
2	Time of Fire	Alcohol Intake	5.60
3	Physical Illness	Age	4.45
4	Mental Illness	Age	4.00
5	Alarm Operation	Time of Fire	3.16

*Interaction size indicates the relative degree of departure from a purely additive effect, with a value of zero indicating that no interaction was present.

The joint dependence between location and age is depicted graphically in Figure 10. All other variables except those graphed remained held at their means. Sedative consumption was higher when in the RFO, but this relation was stronger during middle age (40-60 years) compared with other age groups.

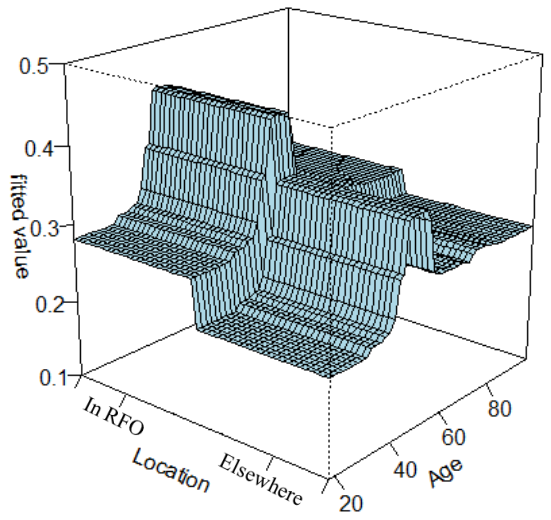

Figure 10. Three-dimensional partial dependence plot for the strongest interaction in the boosted regression model after simplification. All variables except those graphed are held at their means.

Discussion

The present study examined the effects of psychoactive drug usage on human behaviour in fire. This was accomplished by first determining whether users of psychoactive drugs were over-represented in the VUCD compared to the general population, and then by investigating the relationship between psychoactive drug usage and other risk factors. The relative importance of these risk factors was analysed via BRT, which is a new and promising algorithmic modelling technique. This is the first time BRTs have been used to interrogate Australian coronial data. Given their adverse effects in the context of a fire emergency, particular attention was given to sedatives as a drug class in the analysis.

The results indicate that users of sedatives and psychoactive drugs more broadly represent a small proportion of the Australian population but a substantial share of victims of unintentional residential fires. Such over-representation was particularly pronounced amongst victims where sedatives were detected. While the impact of drugs has often been indirectly implicated in the fire literature, this is the first time psychoactive drug usage has been directly linked with fire fatalities in an Australian sample and the first time psychotropic drug usage has been considered in such detail in the international literature. The substantial increase in risk is likely to be a function of common drug side effects and the interaction of drug usage with other known risk factors.

All psychoactive drugs affect brain function, which can have negative impact on mobility, judgement, and stamina in a fire emergency. Drugs that produce greater cognitive impairment would then be expected to be associated with higher levels of risk. The extremely strong risk associated with sedatives, relative to antidepressants or illicit drugs, supports this interpretation. The CNS depressant effects of sedatives have been shown to impair cognition to a greater degree than other drugs (Koelega, 1993; Oster, et al., 1990; Pickworth, et al., 1997).

Interestingly, illicit substance usage was not significantly different to the population baseline. This was in contrast to the significant differences observed for other psychoactive drugs, such as sedatives and antidepressants. This was surprising since illicit drugs could be expected to

produce an even more profound impact on cognition compared to some other psychotropic drugs, such as anti-depressants (Pickworth, et al., 1997). The failure to identify illicit drug usage as a risk factor when other psychoactive drugs were identified as a risk factor is likely due to data limitations. The expected proportions or population data were based on broad definitions of drug usage that were difficult to compare to the VUCD. For example, expected proportions of illicit drug usage in the population data included a number of commonly used medications that were being used for non-medical purposes (e.g., analgesics). It was not possible to separate out these influences with the available data.

The inability to find a difference between the incidence of illicit substance usage in the population and fire victims was in disagreement with evidence from the literature. Barillo and Goode (1996b) retrospectively reviewed the incidence of substance abuse in fire fatalities and found that illicit drug abuse increased the risk of fire related injury or death. The differences between this study and Barillo and Goode's (1996b) findings are likely to be a function of sample selection. Barillo and Goode (1996b) investigated all fires, even if they were not accidental. It is possible that illicit drugs are more likely to be detected in intentional fire fatalities as both illicit drug consumption and deliberate fire starting could be considered types of anti-social behaviour, and if you commit one type of anti-social behaviour you are more likely to commit another type of anti-social behaviour (Hovey, 1942). Furthermore, Barillo and Goode's (1996b) data may have also included quite a few suicides by self-immolation as well. Overall, while the current data suggested illicit drug usage was not overrepresented in Australian fire fatalities, data limitations make any final determination premature at this stage.

Age/sex

A review of the risk rates by sex is also instructive. Both male and female victims of fire were both significantly more likely to be using sedatives compared to the general population, but this over-representation was greater for males than females when compared to the population data. One explanation for this finding is sex differences in risk taking behaviours. Males are significantly more likely to engage in risk taking behaviours that could lead to a fatality (Iwamoto, Cheng, Lee, Takamatsu, & Gordon, 2011) and consistent with the literature (Brennan,

1998; Buyuk & Kocak, 2009; Chien & Wu, 2008; Graesser, et al., 2009; Holborn, et al., 2003; Marshall, et al., 1998; Rogde & Olving, 1996; Ronald & Jerry, 1982; U.S. Fire Administration, 1999) there were more males in the VUCD compared with females.

Sex differences in levels of sedation may also explain the above result. Despite body composition advantages in drug metabolism, males have been shown to achieve a deeper level of sedation than females (Sun, Hsu, Chia, Chen, & Shaw, 2008). The reasons for this are unclear. It is possible that hormonal differences may affect GABA-A receptor functioning, which is a receptor important in the action of sedatives (Bajaj, Raiger, Jain, & Kumar, 2007).

Sex differences in drug response may also explain the finding that female users of antidepressants were slightly more over-represented (compared to population figures) in fire fatality numbers compared to male users. While there is still some debate in the literature (Morishita, Kinoshita, Arita, Bancroft, & Ardley, 2008), sex differences in drug metabolizing enzymes have been shown to result in a larger treatment effect in females for antidepressants (Keers & Aitchison, 2010). A greater treatment effect might be responsible for the relatively larger over-representation of females compared to males in the VUCD on antidepressant usage, assuming a larger anti-depressant effect is associated with more impaired behaviour.

Age also played a role in risk rates, and an important finding relates to middle aged adults. Middle aged adults who had perished in a fire were more likely to have been using sedatives and psychoactive drugs in general compared to adults of other ages. It was originally anticipated that older adults would be at more risk of dying in a fire after the consumption of psychoactive drugs since older adults have a lower tolerance to sedatives (Wortelboer, et al., 2002), and sedatives have been associated with a number of adverse effects in older individuals (Smith & Tett, 2009; Woodward, 1999). Despite being a relatively unexplored area of investigation, there is strong support for an association between middle age and psychoactive drug consumption in victims of fire. Barillo and Goode (1996b) also noted a higher risk of death in a fire amongst substance abusers in middle age.

The relationship between middle age and psychoactive drug consumption may be explained by drug crossover. Middle aged fire victims were much more likely to use both alcohol and sedatives compared to older adults (see Figure 7). Alcohol consumption is known to generally decline with advancing age (Eigenbrodt et al., 2001; Moore et al., 2005), while sedative usage typically increases over time (Woodward, 1999). So a gradual transition away from alcohol and towards sedatives and other medications eventually occurs (Woodward, 1999), but during this phase it is likely that psychotropic poly-drug usage exposes this age group to greater impairment and therefore increased risk of dying in a fire. There are likely to be a range of psychological stressors associated with middle age that may help explain this trend.

Middle age is an important transitional time where people reflect on their lives and prepare for their retirement years (O'Connor, 1981). It is a period of time where there are often challenges in one or more of the following areas; work or career, spousal relationships, maturation of children, aging or death of parents, and physical changes associated with aging (O'Connor, 1981). These issues are succinctly described by Erikson (1963) in his seminal text, which articulated his stages of psychosocial development. The theory outlines eight developmental stages in which psychological, social, and physical changes combine to trigger a crisis whose resolution will result in either psychosocial regression or growth. The seventh stage was most likely to be encountered during middle age. Erikson (1963) believed the primary developmental task during middle age was to contribute to society and help to guide future generations. A failure to make this contribution was believed to lead to intense personal stagnation (Erikson, 1963). Since psychoactive drugs and alcohol have commonly been used to dull the pain from these types of confronting life stressors (Gray & Moore, 1942), it is possible that many of the victims of middle age who had consumed both sedatives and alcohol were attempting to escape from the stagnation Erikson (1963) described when the psychosocial crisis in middle age was not resolved. The level of psychological distress may have been so severe in some victims that multiple drugs were required to mask the stress. This is consistent with evidence from the literature, where greater levels of psychological distress or mental illness are associated with psychotropic poly-drug usage (Malmberg et al., 2010). Overall, while there are likely to be a range of psychological stressors associated with middle age that may help explain greater psychotropic poly-drug usage amongst middle aged adult fire victims, the specific reasons remains unclear.

Consistent with the crossover hypothesis discussed above, the data also indicated that middle aged adults (30-60 years) were also more likely to be using a greater number of drugs compared to adults of other ages (a group comprised mostly of adults older than 60). Polypharmacy is positively correlated with poor health (Australian Bureau of Statistics, 1995; Morgan, et al., 2012). An additional factor may be that middle aged adults using drugs are at a higher risk of dying because of particularly poor health. Overall, a range of behavioural (e.g., psychotropic poly-drug usage) and psychological (e.g., personal stagnation) factors associated with middle aged victims of fire that can help explain their heightened risk. Interventions that can target these behavioural and psychological factors that may increase the risk of dying in a fire for middle aged adults should be given greater emphasis.

Smoking related materials. The literature points to a strong association between alcohol and smoking materials in fire victims (Bruck, et al., 2011). Similarly, smoking materials appear to be significantly more likely in fatalities involving psychoactive drugs, although the modelling suggested this effect was blunted (i.e., explained less of the variance in consumption) when considered with other risk factors. Since psychomotor activity, such as the appropriate disposal or use of smoking related materials may be compromised when under the influence of a psychotropic drug (Pickworth, et al., 1997), this relationship was anticipated.

Interestingly, the relationship between sedative consumption and smoking related materials (odds ratio of 2.18) was weaker when compared to the relationship between psychotropic drugs and smoking materials and indeed the relationship between alcohol and smoking related materials (odds ratio of 4.42) observed in the literature (Bruck, et al., 2011). The reason for the more moderate relationship between sedatives and smoking materials may be due to differences in the sleep/wake status of victims. Data from the VUCD showed that in a large number of fires that involved smoking materials the victim was asleep at the time of fire start. Victims where psychotropic drugs were involved or alcohol was involved were more likely to be asleep and perish during resting hours, compared with victims where sedatives had been detected. This finding is non-intuitive since sedative consumption is normally associated with sleep. However, differences in the sleep/wake status may explain why the relationship between smoking materials

and psychotropic drug usage or alcohol consumption was stronger than the association between smoking materials and sedatives. There appears to be less likelihood of allowing an unextinguished cigarette to smoulder for the necessary time to ignite if you are awake compared to when you are asleep.

People presence in home. The results indicate some evidence of a relationship between being alone at fire start and the consumption of sedatives. In contrast to alcohol and other recreational drugs, which are more likely to be consumed in social settings, sedatives are typically taken at times when people are more likely to not be in social settings. This may explain the weakening of the relationship (i.e., a smaller odds ratio) when compared to other psychoactive drugs in this study, and contrasting evidence in other comparable research that has investigated the relationship between alcohol and people presence in home, where no evidence of a relationship between being alone at fire start and the consumption of alcohol was found (Bruck, et al., 2011).

Data from the VUCD showed that victims where sedatives had been detected were also significantly more likely to live alone when compared to victims where no sedatives had been consumed. It stands to reason that living alone significantly increases the chances of being alone at fire start. The fact of living alone amongst victims who had consumed sedatives is likely to be multi-factorial, and may relate to a range factors that are consistent with the emerging picture of disadvantage and psychological vulnerability amongst users of psychoactive drugs.

Sleep/wake. Sleep/wake was not strongly associated with psychoactive drug consumption or sedative consumption. Given the sleep promoting effects of many psychoactive drugs, particularly sedatives, this finding was unexpected. One possible explanation for this finding is the time most of these fires were suspected to have occurred. Sleep is less likely during the day because sleep pressure (the tendency or likelihood to fall asleep) is normally at lower levels. Since victims who had consumed psychoactive drugs, particularly sedatives, were more likely to perish during the day, this may have reduced the likelihood of sleep because sleep pressure was at lower levels.

A trend approaching significance and supporting the relationship between sleep and alcohol, which also depresses brain function, exists in the literature (Bruck, et al., 2011). Similarly, the combination of the depressive effect associated with alcohol and the impact of sleep pressure associated with the time of day may help explain why those who had consumed alcohol in Bruck et al.'s (2011) study were more likely to be asleep despite alcohol's milder sedative effect (Koski, et al., 2002). Victims who had consumed alcohol in Bruck et al.'s (2011) study were more likely to be involved in a fire during resting hours when the impact of sleep pressure may have been heightened, while victims who had consumed sedatives were more likely to be involved in a fire during waking hours when sleep pressure may have been reduced (Table 8). So the impact of sleep pressure on consumers of alcohol may explain why they were more likely to be asleep despite consuming a milder sedative.

On the available evidence it is difficult to clarify the relationship between being asleep and the consumption of psychoactive drugs; however, sleep/wake may provide a solution to the problem of drug dosage. Despite being under the influence of a sedative, the majority of victims who had consumed sedatives were awake during the emergency (65%). As sedatives are designed to help people sleep or relax, being awake is unlikely to coincide with peak sedative concentration.

Other accounts are possible. It is also possible that the sedative concentration was high but the victim had built a tolerance to the drug over repeated use and remained lucid (American Psychiatric Association, 1990). This might be particularly relevant in cases where the sedatives were used for non-medical purposes. Even when tolerance has not been developed, research has demonstrated that it is possible to stay awake, albeit in a slowed dissociative state, even when the sedative is at peak concentration in naive populations (Tiplady, et al., 2003).

This evidence suggests that drug concentration was likely to be low during the emergency. Nevertheless, the results point to a strong overall drug effect. This lingering impact seems consistent with the literature, which suggests that those sedatives with established sleep maintenance efficacy are associated with next-day sedation (Rosenberg, 2006) and even modest levels of sedation can still produce impairments (Gray, et al., 2006; Kool, et al., 2011; MacDonald, 1999). In addition, the sedatives were rarely detected in isolation. Psychotropic

poly-drug usage was frequent, and even small concentrations of sedatives when combined with alcohol, would have had a deleterious impact on cognitive abilities.

Location. There was some evidence of an association between location and the detection of either psychoactive drugs or sedatives specifically. The pattern of results is in disagreement to similar work investigating the association between location and alcohol consumption (Bruck, et al., 2011) that found no evidence of a relationship between location and alcohol consumption.

The victim's involvement in starting the fire may explain these results. Data from the VUCD suggested that victims where sedatives had been detected were more likely than victims who had not consumed sedatives to be involved in starting the fire at the exact point of ignition. The ignition factor in cases involving sedatives was more likely to be combustibles too close to heat, and they were more likely to occur in the kitchen compared with other rooms. This suggests they were cooking fires. Victims who had not consumed sedatives may be less likely to die in cooking fires compared to victims where sedatives were involved. Given consumers of sedatives were more likely to be awake compared to victims who were not consuming sedatives, this may have increased the possibility of engaging in routine tasks that may have caused a fire. Similarly, victims in Bruck et al.'s (2011) study who had consumed alcohol and were less likely to be in the RFO at fire start may have been less likely to be involved in the fire because they were more likely to be asleep.

Conditions preventing escape. There was some evidence that psychotropic drug consumption was more likely to be detected when conditions preventing escape were present compared to occasions when there were no conditions preventing escape. The association was not significant when sedatives were detected. These findings are in strong disagreement with Bruck et al.'s (2011) study, where conditions preventing escape were in fact reduced, by more than a half, in victims who had consumed alcohol.

The literature suggests that conditions preventing escape are much more likely to be present if the victim is located in the RFO (Alarie, 2002). Given being in the RFO was more likely when

psychotropic drugs had been detected (compared to no psychotropic drugs), this may explain the inconsistent results. It may be a case of being in the wrong place at the wrong time.

Smoke alarm presence/operation. Smoke alarms are the most frequent safety precaution adopted by Australian households when preparing for an emergency (Australian Bureau of Statistics, 2007). Consistent with the literature, the presence of smoke alarms was significantly lower amongst victims of fire when compared to the general population (56% of fatalities cf. >90% of Australian households) (Australian Bureau of Statistics, 2007). However, results from the current study showed that prevalence did not vary significantly as a function of psychotropic drug detection (i.e., psychotropic drug victims compared to victims where psychotropic drugs were not detected), consistent with Bruck et al.'s (2011) study, which investigated the relationship between alcohol detection and smoke alarm prevalence. Despite being in agreement with previous research, this result is somewhat surprising as smoke alarm prevalence has been negatively associated with disadvantage and impairment in the literature (Sidman, Grossman, & Mueller, 2011), and many users of psychoactive drugs fall in this classification (Leeies, Pagura, Sareen, & Bolton, 2012).

The choice of housing might clarify this finding. Possibly due to affordability or ease of maintenance, data from the VUCD showed that victims that achieved a positive sedative test were 4.57 times more likely to be residing in apartment blocks compared to victims where sedatives were not detected (32% cf. 7% respectively). Smoke alarms were more likely in apartment blocks and were present in nine out of 11 multiple storey units (VUCD data). Apartment blocks are typically managed by a body corporate, which is responsible for a range of property issues, including building compliance with fire regulations. It is reasonable to expect higher levels of fire safety equipment adoption in such circumstances that is driven by the body corporate rather than individual. Of course, this is speculative and other explanations may be possible.

The data also indicate that the detection of sedatives in fire victims was more likely when a smoke alarm was operating (Table 8) (compared to non-detection of sedatives). An explanation for this trend is that working smoke alarms were overrepresented in victims where smoke alarm

effectiveness was made redundant because of victim impairment (Ahrens, 2011b). Five of the seven fatalities with sedatives in their system who died despite a working smoke alarm were awake and in the RFO at fire start. After applying Bruck and Thomas' (2010) event-tree analysis, the likelihood of being saved by the current smoke alarm in these circumstances would have been remote. The implication being that smoke alarms are largely effective in saving lives except when there are other impediments that impact a victim's ability to escape. This would explain why some people die in fires despite operational smoke alarms. However, the reliance on retrospective data in formulating a defence for smoke alarms limits the evidence.

Alternative explanations are possible. The presence of an operating smoke alarm may change the human reaction to a fire. For example, early detection as a result of a smoke alarm might impact the flight or fight response. Early investigation of a fire at the smouldering or flame stage may encourage victims to address a fire when immediate evacuation was the more sensible course of action, although this can only be speculated at this stage. The most pressing danger in a fire often comes from inhalation of smoke and fumes (Ahrens, 2011b). It is possible that smoke may be underestimated as a more benign risk. Errors of judgement are likely to be greater amongst victims who are impaired by psychotropic drugs. While focusing primarily on commercial buildings, Proulx (2003) outlined other human responses to an alarm that might decrease the chances of survival, such as the failure to recognise the signal as an alarm or a loss of confidence in the system due to nuisance alarms.

Pre-existing individual differences might also account for the data. Crapo's (2000) review suggests that people with working smoke alarms are more fire safety conscious and therefore less likely to experience a fatal fire in the first place. Crapo (2000) noted differences in fatality rates when comparing households without smoke alarms to households with smoke alarms that were inoperable. Fatality and injury rates were lower in households with a smoke alarm, even if that alarm was not functioning. Crapo (2000) explained this non-intuitive result by arguing that individual pre-existing differences in fire safety consciousness may have influenced the decision to purchase and install a smoke alarm.

However, legislative requirements are also likely to have strongly impacted on smoke alarm adoption. In addition, any relationship is likely to lessen in importance as the adoption of smoke alarms approaches its population zenith because even less safety conscious households will eventually install and maintain their smoke alarms due to legislative changes diluting the comparison with an increasingly smaller minority of non-compliers. Furthermore, pre-existing attitudinal differences are impossible to establish with data from the VUCD. However, it is interesting to note that, although not specifically investigated, victims who had an operating smoke alarm were also more likely to have lived in a clean and tidy house (compared to a cluttered house), which may also be symptomatic of fire safety conscious leanings or of greater levels of functioning in general.

Until more controlled research is conducted, the precise role of smoke alarms remains clouded and results must be interpreted cautiously. What is clear is that smoke alarms in their current guise fail to protect some people at high risk of dying in a fire. This does not mean smoke alarms should be dismissed. Alarms remain a promising, affordable, and easy to install method of delivering early notification. Nevertheless, improvements will be required to maximise the role of smoke alarms in residential fires.

Two changes to smoke alarm usage that may increase alarm effectiveness are: (a) interconnected alarms; and (b) the 520 Hz square wave. Thomas and Bruck (2011) demonstrated that interlinking smoke alarms, such that smoke detection in any one room will sound all smoke alarms, may provide a significant reduction in fatal residential fires. Similarly, changing the pitch of the alarm signal from the current 3000 Hz pure tone to a 520 Hz square wave has also been shown to be more effective at waking a number of groups at risk of dying in a fire (Ball & Bruck, 2004a; Bruck, 1999; Bruck, et al., 2004; Bruck & Thomas, 2007c; Bruck, et al., 2007), including users of sedatives (Study 1 of this research).

Beyond developing better alarm signals other well documented challenges remain. Despite the uptake (installation but not necessarily maintenance) of smoke alarms by a small majority of victims (56%), a large proportion of smoke alarms that were present did not operate (15/45, 33%). These results are consistent with the literature (Ahrens, 2011b; Douglas, Mallonee, &

Istre, 1999). Smoke alarms are most commonly inoperative because of missing or disconnected batteries in order to avoid nuisance alarms (Ahrens, 2011b). Strategies to address alarm desensitisation and programs to promote adequate maintenance of smoke alarms are essential. Simply increasing installation of smoke alarms alone will not solve the issue.

It is worth noting that the impact of smoke alarms may extend beyond occupant notification. The relationship between smoke alarms and emergency service response times is another important, but rarely discussed, area in the literature. While it remains speculative at this stage, smoke alarms may increase the likelihood of early notification of fire services (particularly when witnesses are involved in notifying fire services), and this may result in smaller overall response times when assessed from the time of fire. Lower response times have been shown to increase the chances of survival and decrease the severity of fire (Department for Communities and Local Government, 2009).

Overall, a significant quantity of financial and human capital has been and continues to be invested in smoke alarms (Ahrens, 2011b). It is difficult to calculate the opportunity cost of this investment if we are unsure what impact, if any, smoke alarms have in reducing the costs associated with fire. Methodologically sound experiments are required to provide a clearer picture of the role smoke alarms may play in residential fires, such as a naturalistic study of arousal thresholds; however, due to the risk involved in replicating fire emergencies (where actual fire sounds may awaken people before an alarm (Bruck & Brennan, 2001) or other differences that are difficult to replicate) this remains a difficult prospect.

Alcohol intake. Alcohol intake was not significantly associated with psychoactive drug usage or sedative usage in fire victims. Nevertheless, alcohol still has a role to play, particularly amongst users of sedatives. Alcohol was detected in 70% of victims who had consumed sedatives (Table 8) and the modelling indicated that when considered with other risk factors, alcohol plays a more active role that is not evident after a simple bivariate analysis.

Evidently psychotropic poly-drug usage is common in fire victims and the combination of alcohol and sedatives was the most prevalent psychotropic drug combination (see Figure 7). The

combination of sedatives and alcohol has generally produced an additive and decreasing effect on performance (Austroads, 2000; Maxwell, Dubois, Weaver, & Bacdard, 2010). Therefore, drug combinations are prevalent and increase impairment, which may further erode the ability of victims of fire to combat an emergency.

Mental illness. Psychoactive drugs are often used to treat mental illness. The vast majority of victims who had consumed sedatives prior to their death were likely to have experienced some sort of regular sleep disturbance, since they are generally prescribed after the reporting of regular sleep issues to a physician (Chokroverty, 2010). Sleeping problems are often a manifestation of a range of emotional and physical dilemma, and lengthy sleep disturbance that causes significant distress is itself recognised as a mental illness. Therefore, the association in this study between psychotropic drugs including sedatives alone, and mental illness is both intuitive and regularly replicated in the literature (Lin, Chen, Chen, Chung, & Lin, 2011; Toblin, Paulozzi, Lohaan, Hall, & Kaplan, 2012), including one study that specifically examined this relationship using Australian coronial records (Watts-Hampton, et al., 2006). However, this is the first study to examine the relationship between sedatives specifically and mental illness in the Australian fire fatality statistics. The relationship between psychotropic prescription medication and mental illness is a challenging one. Evidently, the treatment can paradoxically both help and hinder individuals. In many fire victims psychotropic drug usage appeared to have only worsened a cognitive malaise at a time when the importance of being alert could not have been overstated. This study demonstrates that a drug effect in concert with other risk factors is strongly associated with fire fatalities. In addition, data from the VUCD showed that a large number of victims were suffering from substance abuse disorders, which suggests that a proportion of these psychotropic drugs may have been consumed for non-medical purposes.

Physical illness. The relationship between physical illness and psychotropic drug usage replicates population trends (Gray, et al., 2006; Rayner et al., 2010; van Eijk, Bosma, Jonkers, Lamers, & Muijrers, 2010); although this link has not been described in the Australian coronial literature before. There are various possible reasons for the association. First, certain physical issues, such as respiratory disease and morbid obesity, are often a contributing source of poor

sleep (e.g., insomnia with or without sleep apnea), which may lead to a course of sedatives (American Psychiatric Association, 2013). Second, sedative consumption can by itself create mobility issues (Gray, et al., 2006).

Time of fire. Human error can be expressed as a function of temporal patterns in human performance and operation. Human activity largely takes place during the daytime, around feeding or work routines, because of various social and physiological influences (Hawley, 1950). Activity is a necessary precursor to error in most psychological models of accident causation (Quinlan, et al., 2010; Reason, 1990). Therefore, the diurnal cycle represents an interval in time and space where a confluence of risk factors specific to fires caused by human intervention has the greatest probability of occurring together.

The unexpected association of time of day and sedative consumption suggests that victims who had consumed sedatives were more likely to commit error while carrying out their everyday routines because their cognitive systems were less than optimal. Victims who had taken sedatives were often extremely vulnerable. A large proportion of these individuals suffered from mental and physical illness coupled with psychotropic poly-drug abuse. Many activities in these circumstances would have been dangerous, and the daytime represented a period of time of heightened mobility.

The temporal dispersion of non-fatal fires also supports this line of argument. Despite non-fatal fires being more probable during the daytime, it is more likely that a fatal fire will develop during the early morning (Ahrens, 2011b). It is possible that non-fatal fires do not progress into fatal accidents because the errors are largely detected and corrected. Regrettably, extremely vulnerable individuals may simply not possess the cognitive capacity to quickly and effectively make the right decisions, or mobility, to prevent a minor hazard from developing into a tragic accident, and some examples are outlined below.

Human error originates with a distortion to one or more of the three stages of cognitive processing for tasks: storage; execution; and planning (Reason, 1990). These biases are likely to be more common in victims who had consumed sedatives. There are numerous examples in the

coroners' files that illustrate how these cognitive impairments and their associated form of error combined to produce a tragic accident.

Cigarettes or candles left unattended whilst the victim was in another room or pursuing another activity are examples of lapses experienced by several victims who had consumed sedatives. These lapses are monitoring failures that implicate memory storage (Reason, 1990). Their latent nature means that lapses are sometimes immune to detection (Reason, 1990), particularly in the presence of memory impairments, which have been associated with the consumption of sedatives (Rummans, Davis, Morse, & Ivnik, 1993). This makes these types of error a prominent and regrettable element in the behaviour of people under the influence of a sedative. Therefore, an increase in the frequency of lapses and a decrease in the detection of lapses would be expected in people who have consumed sedatives.

Similar to lapses, slips are also failures of attentional checking (Reason, 1990). Slips are essentially errors of execution. An example of a slip from the data involved an elderly gentlemen who whilst cooking had attempted to open a cupboard door in close proximity to the stove. His clothing caught fire and he fell backwards and was immobilised. Although there were possibly other factors that influenced his equilibrium, it is likely that his sedative consumption contributed to his loss of balance. Even low doses of sedatives have been shown to impact motor control and mobility (Kool, et al., 2011).

The final cognitive stage relates to planning. Unlike slips and lapses, which are automatic and routine, mistakes in this cognitive mode relate to errors in judgement (Reason, 1990). Using an example, one unfortunate victim was cooking while using an oxygen unit, which was contrary to the instructions displayed on the unit, and the unit caught alight. This type of planning error is called a violation, and can be seen as a form of risk taking behaviour (Breakwell, 2007). The violation in this instance was likely to be routine and simply a matter of convenience, but the effects were devastating. In this area too, there is evidence of a sedative effect (Nikaido, Ellinwood, Heatherly, & Dubow, 1987).

An inappropriate response to an emergency is common to many of the above examples. In many cases in the data the fatal error was not the initial slip or lapse, but the rule or knowledge-based mistake that inevitably followed seeking a solution under duress. For example, one elderly woman who had consumed sedatives had caught fire while preparing her lunch. Rather than evacuate or immediately tend to her initial wounds, she simply stripped her clothes off and attempted to contact her son for assistance; a rule-based solution that she had likely relied on in the past. Unfortunately, on her way to the phone she lost consciousness. The reason for her reluctance to flee or immediately try and heal her wounds is possibly one of the major challenges in preventing deaths from fire. This is an extreme example, but people take refuge in the familiar and seek to project rules that served them in the past onto new situations that might demand a novel solution (Reason, 1990). This underlying premise is central in much of Reason's (1990) work. More specifically, it reflects a difficulty to move from the rule-based level to a knowledge-based level. Normally, a phone call to her son might have had some utility, but in these circumstances it was evidently not the appropriate course of action. In a fire emergency, if there are not established rules to deal with the hazard, by the time the individual recognises the importance to find a novel solution, it is often too late. This reinforces the need for existing fire safety rules to be in place to deal with a hazard to avoid the need to find new ones.

The pursuit of a rule-based solution is often perpetuated by an underestimation of the danger of the hazard. Risk perception can be an important ingredient in fire response (Breakwell, 2007). An assessment of risk based on the size of the fire can be misleading. As discussed earlier, most people succumb to the toxic fumes rather than the actual fire. An enclosed room can become unsafe in a matter of minutes (Alarie, 2002).

The time of fire did not have a similar impact on psychotropic drugs more broadly, in spite of victims who had consumed any psychotropic drugs being 38% more likely to have died during waking hours compared to victims who had not consumed psychotropic drugs. As both dependent variables are not mutually exclusive, much of this effect can be explained by the large sedative result. The small sample size meant an analysis by psychotropic drug type was not

possible, but the result points to drug differences in impairment profiles (Koelega, 1993; Oster, et al., 1990; Pickworth, et al., 1997) and lifestyle choices.

Since the effect of time of fire may be confounded by time spent at home, the relationship between sedative consumption and time spent at home was analysed. This analysis was limited because time spent at home was unknown and occupational status was used as a proxy for time spent at home. Nevertheless, the data suggested that any impact from time spent at home was unlikely to significantly alter the outcome of the analysis.

It is important to remember that even allowing for some of the variance to be an artefact of time spent at home, this finding in and of itself would not invalidate the contention that increased and impaired activity during the waking hours is placing vulnerable individuals at risk. There were clear differences in the cause of fires between night and day that suggested error laden activity during the waking hours were central in the development of the fatality. Together, the data present a compelling picture of the time of day's influence in fire emergencies in certain groups.

Overall, the time of fire result was an important although somewhat serendipitous finding. Fatal fires have traditionally been associated with the early morning (Ahrens, 2011b). This result emphasises the importance of other times of the day when dealing with users of sedatives, and possibly other vulnerable groups. Psychological models of accident causation seem to account for the findings and a range of empirical research verifies different aspects of the result. Once the importance of human agency in a fire is conceded, the critical role sedatives play in cognition and behaviour is perhaps not surprising given the depressive effect these drugs have on the entire central nervous system.

Algorithmic modelling. This is the first study to analyse retrospective coronial data in Australia with the BRT modelling procedure. Algorithmic modelling confirmed that the five most influential variables were, in order of predictive importance: (1) time of fire (waking hours versus sleeping hours); (2) age (middle age versus other adult ages); (3) alcohol intake (any consumption versus no consumption); (4) alarm operation (active versus being present or not present); and (5) mental illness (present or not present). The analysis confirmed the ability of

BRT models to consider a large number of predictors and explore possible nonlinear relationships and interactions. These advantages were particularly useful given the large and sometimes unwieldy dataset, and exploratory nature of the study.

The modelling highlighted the complexity associated with fire accidents. During the model building process tree complexity was trialled at various depths. Predictive performance improved steadily together with complexity before stabilising at the four-way interaction level. This suggested that the effect of certain variables is contingent on many other factors. This complexity would not have been captured in a main effects model.

Age and time of fire were two important interacters. The effect of many other predictors was magnified during the day, and in particular, during middle age. This partially explains why the importance of these two variables increased in the BRT model, when compared with their position after a simple rank of the unadjusted odds ratios.

As the model complexity increased, the type of sedative consumption patterns the model can describe also expanded (Chung, 2011). Therefore, predictors that are important in high dimensional models are effectively better at explaining more complicated circumstances than predictors that are important in low dimensional models. In this way, age and time of fire can be seen to be influential in defining the particularly intricate sets of circumstances involved in many fire fatalities where sedative consumption is implicated.

Alcohol intake was another variable that seemed to increase in importance after being subjected to the modelling process. Alcohol intake was ranked third most important in the BRT analysis, which was somewhat inconsistent with the OR analysis where it was ranked 10th. Of course, a primary reason for this related to the interdependencies between alcohol intake and the other predictors. Once other variables had been considered, alcohol intake rose in importance. At the same time, a change in the variable structure from binary to continuous most likely also contributed to this gain (see Appendix O for more details).

A ratio scaled version of alcohol intake reflecting the level of intoxication was used in the BRT model. This increase in variance undoubtedly had an inflationary impact on its importance (compared to the OR analysis), as both the presence and severity were considered. It should be noted that despite the continuous variable having a significantly larger number of levels compared to the dichotomous variable it replaced, the cross validation process should have ensured that this did not unduly increase its relative influence (Hastie, et al., 2009).

Overall, the model demonstrated the importance of considering risk factors together, including their interactions, when investigating fire fatalities. Predictors that interacted with other risk factors were more relevant, particularly when explaining difficult to classify observations. The model confirmed the relevance of human factors in explaining both sedative consumption, and fire fatalities that involved consumers of sedatives more generally. These findings build on existing work but also provide direction for future research. Given the complexity of fires, the proportion of deviance that remains unexplained in the model, and the advances in predictive modelling, more research is desirable that can examine an even larger number of predictors.

Overall, the consumption of a psychoactive drug, particularly a sedative, is evidently not a trivial matter in a fire context. It is marker of disadvantage and vulnerability, both physically and psychologically. Users of psychoactive drugs were more likely to be middle aged, which is an important transitional time that can often present many psychosocial stressors, such as career pressures, the maturation of children or the aging or death of parents. This life stage is characterised by a need to contribute to society and to help guide future generations (Erikson, 1963). It is likely that a failure to respond to these challenges may have led to intense personal distress (Erikson, 1963) and the need for a pharmacological solution to escape from these problems or help deal with them (Leeies, et al., 2012).

A recurrent theme throughout the chapter was the influence of human error on fire accidents. The association of time of day and sedative consumption suggests that victims who had consumed sedatives were more likely to commit error while carrying out their everyday routines because their cognitive systems were less than optimal. A large proportion of victims who had consumed psychoactive drugs suffered from mental and physical illness coupled with

psychotropic poly-drug abuse. Any activity in these circumstances may have been dangerous, and the daytime represented a period of time of heightened mobility.

CHAPTER 4 – General Discussion

This section provides a broader discussion of the findings and results from both studies and their significance in relation to the current state of knowledge in the field. The discussion is thematic in its approach and explores five key themes derived from the data: (1) smoke alarms and a safety culture; (2) psychoactive drugs and the fire response; (3) complexity and research design; (4) sleep quality and arousal thresholds; and (5) embracing a multi-disciplinary approach. The topics are addressed in order of importance and logical relevance. The goal is to present a compelling narrative that builds logically but also prioritises relatively more important themes early in the discussion. Research limitations are then acknowledged and concluding remarks are proffered.

Smoke Alarms and a Safety Culture

The overriding aim of this research was to examine the effects of psychoactive drug usage on human behaviour in fire. This first involved testing the utility of current and alternative smoke alarm signal technology in users of hypnotics. Establishing the level of risk posed to users of hypnotics in response to different alarm signals may inform smoke alarm standards and direct preventative strategies.

The NFPA is a worldwide authority on fire safety and has regularly championed the adoption of smoke alarms in the community, crediting the smoke alarm as the single most important fire safety strategy (Ahrens, 2004, 2011a). Thanks to a succession of consistent messaging from various stakeholders in the fire industry and government over the last decade, this view is also commonly held in the community. At this point in time smoke alarms are the primary warning in the event of a fire, and hold a preeminent position within the suite of fire detection equipment. However, evidence from both studies suggests that this equipment is failing users of hypnotics and psychotropics more broadly. In Study One, the consumption of hypnotics was shown to reduce the responsiveness to a fire alarm, and in Study Two users of hypnotics were more likely than non-users of hypnotics to have a working smoke alarm despite their demise, which could

suggest that smoke alarms are more likely to be ineffective amongst users of hypnotics who have been shown to be especially vulnerable to fire emergencies in this research. This research adds to a growing body of literature challenging the effectiveness of the current smoke alarms.

Counterevidence has come from various sources. Many with a proximity to the problem have highlighted discrepancies in the use of data to support the NFPA's position. Crapo (2000), a retired fire fighter and lecturer in fire science, indicated that the reduction in fatalities over the last several decades is approximately proportional to the decrease in the number of fires. The actual number of deaths per 1,000 fires has only gently decreased during this time. Furthermore, this decreasing trend began prior to the adoption of smoke alarms. Similarly, the International Association of Fire Fighters (IAFF), which is a body representing almost 300,000 fire fighters and paramedics in the US, has also disputed the NFPA figures. The IAFF, with the assistance of Joseph Fleming, the Deputy Chief of the Boston Fire Brigade, pointed out that the NFPA figures include information on confined fires (i.e., generally small fires restricted to the object of origin), which may distort the real benefit of smoke alarms (International Association of Fire Fighters, 2008). The likelihood of death in a confined fire is significantly reduced, yet the NFPA concluded that being alerted to the fire by a smoke alarm was always lifesaving in these circumstances. Furthermore, the presence or absence of smoke alarms in confined fires was only known in two percent of cases (Ahrens, 2011a), and rather than exclude data where this information was missing, it appears that the small proportion of valid data was used to generalise to the 98% of missing data. The validity of this type of analysis is questionable.

Evidence of smoke alarm ineffectiveness has also been produced after analysing coronial data. Bruck and Thomas (2010) reviewed the Australian fatality statistics and concluded that the presence of a working smoke alarm would have in many cases been irrelevant to whether or not the person died in the fire. This finding was supported by the current research, which found smoke alarm presence and operation was overrepresented in users of hypnotics, compared with victims where hypnotics had not been detected. This suggests that the presence of a smoke alarm, even if operational, was relatively less important when understanding the cause of the fatality amongst users of hypnotics. An explanation for this trend from the NFPA is that working smoke alarms were overrepresented in victims where smoke alarm effectiveness was

made redundant because of victim impairment (Ahrens, 2011b). The implication being that smoke alarms are largely effective in saving lives except when there are other impediments that impact a victim's ability to escape. However, even if this explains the data, this is effectively an admission that the current smoke alarm is not working in groups where protection from fire is most needed.

Another area critical of the current smoke alarm is arousal research. In agreement with the findings in this study, results from over a decade of research on smoke alarm effectiveness have demonstrated that many groups in the population are at risk of not waking up to the 3100 Hz sine wave currently mandated in Australia (Thomas & Bruck, 2008). Older users of hypnotics have now been added to this list. Overall, work from a number of sources suggests more action on smoke alarm signal technology may be needed to avoid fatalities and injury from fire.

Given the shortcomings of the current technology, the results from Study One support the implementation of the 520 Hz square wave. The low frequency alarm has been shown to be more effective compared with the current alarm in a number of vulnerable groups. The 520 Hz square wave is currently mandated in the United States for all sleeping rooms occupied by people with mild to severe hearing loss (NFPA Code 72, 2010; NFPA Technical Committee, 2009). From 2014, the NFPA code will require it in all commercial sleeping rooms. Demonstrating the improved effectiveness of the new signal in users of hypnotics adds significant weight to the argument for also changing the smoke alarm signal in residential buildings in the US and the adoption of the alarm in other countries.

There would be an expectation that an improved alarm would prevent death and injury from fire in the general public. However, it is difficult to be able to estimate the absolute impact any change in smoke alarm standards may yield. As pointed out in Thomas and Bruck's review of arousal research (2008), there is a great deal of missing information required to estimate this figure, which should be the subject of future research. A cost/benefit analysis is also compromised by the many practical issues that may need to be considered before embarking on this exercise.

There are enormous costs involved in refitting the broader community with new smoke alarms. Equipment and installation costs would be sizable. Aiming for a more gradual replacement where only new alarms are mandated to generate the 520 Hz square wave signal may be a more sensible option, but this is not the real challenge. Beyond the management of retrospective or even prospective installations, there are important social marketing challenges that would need to be considered. Social marketing is concerned with the application of marketing information to influence human behaviour in order to enhance social and economic outcomes (Bryant, Angus, & Hastings, 2011). The existing smoke alarm marketing messaging, particularly the dictum, "smoke alarms save lives", is firmly entrenched in the public consciousness. The introduction of a new smoke alarm would have implications for the incumbent messaging. Any departure from the existing messaging to accommodate new messaging associated with the rollout may confuse consumers and have a detrimental impact on the ability to change attitudes and behaviour (Bryant, et al., 2011). Specifically, a new alarm implies the existing alarm may not have been as effective at saving lives as previously thought. Messaging designed to address this inconsistency may erode credibility, which is central to the acceptance of any message (Bryant, et al., 2011).

Factors that may favour the introduction of the new alarm also exist. It may be that the current messaging is tired and ineffective and this may provide an opportunity to address these issues. Evidence of this comes from the poor maintenance record of current smoke alarms (Barnett, Bruck, & Ball, 2011). If smoke alarms are assumed to save lives, then choosing not to maintain a smoke alarm, even considering the nuisance issues, represents message failure. It is also possible that people believe the alarm would save their life but are not convinced that their residence is at risk of a fire, rendering the alarm unnecessary or not worth the trouble. This would be an example of normalcy bias, which involves underestimating a potential danger (Yamori, 2009). Even if the existing message is working, there are other aspects that indicate the new alarm may be accepted and maintained without controversy. Research suggests that introducing a product that has clear benefits over what currently exists and is not too complex has an advantage over other products in terms of adoption (Bryant, et al., 2011). The 520 Hz square wave would seem to fit this description; however, there are additional costs associated with the 520 Hz square wave, such as the need for more power and a bigger speaker that are also important to note . Overall, given the number of issues facing the introduction of the new alarm

in the community, more research is desirable to first replicate the findings in more representative research and investigate many of the implementation issues.

The new alarm solution also omits an essential point. Even if a smoke alarm is successful in waking a potential victim of fire, being awake in and of itself is insufficient protection from fire. Once alerted to a fire, our response can also have a telling impact on survivability. Study Two highlighted the importance of the time of fire and the role of impaired activity during the daytime. An unusually large number of victims who had consumed sedatives were awake and perished during the daytime, despite the expectation that sedatives are normally consumed at night prior to sleep and exert their main effects during the sleep period. A closer analysis of the cases revealed that many fires were a result of human error, which has the greatest likelihood of occurring during the day when activity levels are at their highest. It was argued that this activity was then encumbered by the ongoing presence of a sedative in the victim's system resulting in a tragic accident. Importantly, it was not simply the greater prospect of committing simple errors associated with hypnotic consumption, but the diminished problem solving capacity that led to rule or knowledge based mistakes that thwarted attempts to escape and ultimately led to a person's demise. An influential psychological model of accident causation (Reason, 1990) was used to account for the findings and a range of empirical research verified different aspects of the result. The findings were in contrast to the literature, where fatal fires have traditionally been associated with the early morning (Ahrens, 2011b). This result emphasises the importance of other times of the day when dealing with users of sedatives, and possibly other vulnerable groups. In a fire emergency, if there are not established rules to deal with the hazard, by the time the individual recognises the importance to find a novel solution and can implement that solution, it is often too late. This reinforces the need for existing fire safety rules to be in place cognitively to deal with a hazard to avoid the need to find new ones. Further research on daytime fatal fires with other exposed groups may also be fruitful. Human factors seem to play a more expansive role during the day compared with early morning fires when people are asleep. This does present an opportunity. The greater availability of health and social resources during the daytime may produce a more compelling case for intervention not possible at other times.

Even the correct response to a fire is often insufficient to save a victim of fire. Bruck and Thomas (2010) outlined a number of plausible circumstances where the likelihood of surviving a fire would be remote irrespective of the circumstances, such as the case of victims who are located in the RFO at fire start. Although the development of new smoke alarms as suggested in Study One and the adoption of fire escape plans recommended in Study Two are important and would likely reduce both the physical injury and material damage from fire, the inevitability of a fatality in many cases suggests that fire prevention may be the strongest asset in providing a satisfactory answer to the risk from fire.

A preventative focus represents a substantial shift in thinking. There has traditionally been a concentration on external environmental causes (Miller, 2005), which place more emphasis on technological responses to fire emergencies (Rhodes & Reinholtd, 1998). Human agency is increasingly recognised as an important element in a fire emergency (Brennan & Thomas, 2001; Miller, 2005), where understanding the social and behavioural underpinnings of why groups become at risk can possibly prevent the emergency from ever occurring (Rhodes & Reinholtd, 1998).

Given the central causal influence of human factors in fire fatalities it is likely that prevention will require change at the cultural level. There is strong literature support for the adoption of a safety culture in managing human error. The creation of a positive safety culture can have a lasting impact on the attitudes, beliefs, perceptions, and values that the community share in relation to safety (Reason, 2007), which then would be expected to guide behaviour that is less likely to cause a fire emergency.

There is a great deal of evidence that safety is a social value that needs to be more influential. Many groups in the population and particularly victims of fire demonstrate a low level of fire safety consciousness and a poor safety culture. Most homes do not yet have the protection recommended in recent editions of the NFPA 72 (Ahrens, 2011b), do not have a pre-prepared escape plan, many are unlikely to be familiar with the human risk factors associated with fire, and there is still evidence of confusion surrounding what signal universally indicates the need to evacuate due to the presence of an emergency (Proulx, 2003).

The reasons for this lethargy are not always straight forward and in order to understand the causal chain of events properly a focus on upstream factors (e.g., regulatory environment, treatment of safety in the media) may be required. Proximal causes, such as human error, while they play a key role, often provide a narrow version of events when attempting to understand and change safety behaviours in the long-term. Unsafe acts are generally a product of various environmental, societal and technological influences. For example, a reliance on drug treatment is subsidised by a troubling prescription culture in many developed nations. Understanding the role these various environmental, societal and technological influences have on our attitude towards safety is critical. Rather than being a deterministic argument to remove accountability, this suggests that more needs to be done at a macro level to understand the broader issues and assist vulnerable people in society make decisions that will positively impact the safety of their lifestyle.

Many commentators have offered suggestions on how to improve the safety culture of an organisation (Arden, 1993; Geller, 1994; Reason, 2007). For example, educational campaigns targeting groups most at risk, the provision of adequate resources, or rewarding safe behaviour are but a few alternatives. There is a surplus of ideas. While the targeted audience for many of these suggestions were organisations, they can be easily extended to individual accidents, such as fire emergencies. The difficulty is more one of implementation. The onus is often placed on management to drive these initiatives in organisational settings. This model will not work in home accidents, where the large majority of victims lived alone. Broad direction could come from government, who have a vested interest in reducing the cost of hazards in the community. However, this vision would ultimately need to be disseminated at increasingly smaller cultural units. It may be that local councils are perfectly positioned to facilitate change. Community safety is a service that presently forms part of a council's brief. Safety culture teams could be appointed to introduce many of the suggestions discussed earlier in this paragraph in their local area.

An improvement in community safety culture might also increase the chances of individuals within a community working together to assist one another in a fire emergency. This may have

many benefits. For example, a greater emphasis on community safety culture may result in more attention to remove fire safety hazards in vulnerable neighbours (e.g., old, unsafe heaters).

Psychoactive Drugs and the Fire Response

Safety culture is a broad term encompassing many different behaviours and attitudes. A central theme in this research was to investigate the specific influence of hypnotics and other psychoactive drugs on the behavioural response to a fire emergency. Both studies highlight the considerable impact psychotropic drugs can have on a response to an emergency whether at peak intensity or even at more benign concentrations. A highly significant drug effect on the ability to awake to a fire alarm was evident in Study One when tested at peak concentration. This is only the second study to test a traditional fire alarm in this vulnerable population and overcame many of the limitations of the previous research that restricted the applicability of the results in a real life context. The current evidence from Study One suggests approximately one in six older adults who consume hypnotics would be expected to sleep through their 3100 Hz sine wave smoke alarms at the standard alarm intensity of 75 dBA after consuming their usual hypnotic. As sleep is a well documented risk factor in fire, it is possible that a significant proportion of older adults who do sleep through their fire alarm may perish, but this is difficult to quantify without more information and can only be speculated as the life saving potential of smoke alarms remains controversial and the results of the study must be generalised to the population with caution. A large drug effect was also apparent in Study Two, particularly in victims who had consumed hypnotics as opposed to other psychotropic drugs.

This was the first time victims of fire in Australia who had consumed hypnotics were analysed via retrospective coronial data. Approximately four out of ten victims of fire had consumed at least one psychotropic drug, which indicated that users of psychotropics were significantly overrepresented in fire statistics compared to their estimated population prevalence. Together, the results highlight the crucial role psychotropic drug usage plays in accidental fires.

The association between the time of fire and sedative consumption was an important finding that helped clarify the impact of hypnotics in the emergency. However, flawed cognitive and

behavioural processes are unlikely to be the only reason users of hypnotics are at additional risk. This requires a broader profile. The results from Study Two demonstrate that psychotropic usage is entangled with various traits that may augment the risk from fire. Drug consumption rarely occurs in isolation of other risk factors. Study Two established a number of pre-existing characteristics, such as mental and physical illness, associated with psychotropic usage. Many of these risk factors had never before been examined in a subset of fire victims where hypnotics had been detected. In Study One, the relationship between the Insomnia Severity Index (ISI) and Auditory Arousal Thresholds (AATs) provided soft evidence of endogenous differences in sensory sensitivity (i.e., differences in baseline arousal thresholds) that may paradoxically both help and hinder the emergency response. For example, earlier awareness of fire (due to a decrease in AATs) is only useful if that is combined with sensible decision making, which may also be impaired in individuals suffering from insomnia or depression and who choose to take hypnotics. Overall, both studies point to a variety of characteristics and lifestyle factors, some of which may be biologically driven, that separate users of hypnotics and psychotropics more broadly from people who are not using these drugs, and place these individuals at greater risk from fire.

Age was one particularly important area of divergence. The literature has traditionally highlighted that older and younger groups are at most risk from fire (Ball, Graesser, Bruck, & Thomas, 2009; Graesser, et al., 2009), but when psychoactive drugs are involved, middle aged adults appear to be extremely vulnerable. The identification of middle aged adults as a high risk group was somewhat unexpected. It was anticipated any variation by age would implicate older adults, as their responsiveness to alarm signals is significantly attenuated and they are more likely to consume hypnotics. A drug crossover hypothesis was suggested as one possible explanation for this trend. It was speculated that the likelihood of psychotropic poly-drug usage is enhanced during the transition with advancing age away from alcohol and towards sedatives and other medications (i.e., on average there is a greater chance of consuming both drugs at the same time when decreasing usage of one drug and increasing usage of another). It was also suggested that psychotropic drugs and alcohol consumption may have represented an attempt to escape from the unresolved psychosocial stressors commonly associated with this transitional time of middle age in most people's lives. This is consistent with evidence from the literature,

where greater levels of psychological distress or mental illness are associated with psychotropic poly-drug usage (Malmberg, et al., 2010).

A drug crossover hypothesis does not minimise the risk to older adults, who remain significantly more likely to die in a fire than their younger counterparts (Hall, 2005). Despite this risk relatively few studies have examined the reasons behind this over-representation. This is the first study to analyse arousal thresholds in older adults who are impaired. Together with the important work by Bruck and Thomas (2008a) on smoke alarm response in sleeping unimpaired older adults, the results from Study One may help explain why some older adults die in a fire. Their responsiveness to alarm signals is significantly attenuated, and this ability is further impacted by the consumption of sedatives, more commonly consumed at this life stage.

Managing the problem presented by psychotropic usage is difficult. Technological improvements are one possible solution to this problem. This study has confirmed that the 520 Hz square wave is successful at waking older adults, even after the consumption of hypnotics (all older adults participating in the study woke to the 520 Hz square wave alarm at 75 dBA). Introducing a mandate by the relevant authorities may further this aim, but this may be an overly simplistic response given that most died during waking hours if on hypnotics when being altered to the fire was not likely to be the critical factor. The cost involved in transitioning to the 520 Hz square wave also makes the requirement for greater evidence compelling. However, real progress will require management of the prescription of these drugs and the underlying reasons for their use in the community.

Most psychoactive drugs must be prescribed by a doctor. While there are many ethical and legal aspects to sedative prescription that can make it challenging (American Psychiatric Association, 1990), doctors are best placed to promote responsible usage and need to be proactive in redirecting particular patients to alternative services (e.g., substance abuse counselling) if required. Making consumers aware of the dangers of hypnotic usage and presenting viable non-medical options is an important first step. It is possible that many users of hypnotics are unaware of the extent of the risks associated with their usage. Presenting this information to users would facilitate a more informed decision on both the choice to take a hypnotic and the manner of their

consumption. This information could also include the myriad of options available to people experiencing sleep discomfort. For example, cognitive behavioural therapy (CBT) non-drug techniques for insomnia are known to be as effective as hypnotics in the short-term and more effective in the longer term (Mitchell, Gehrman, Perlis, & Umscheid, 2012).

In making decisions on the prescription of a sedative, GPs need to consider their knowledge of the patient and the risk of any drug abuse (Sim, et al., 2007). Generally, psychoactive drug manufacturers' product information statements would advise alcohol usage be eliminated or taken in small amounts in the presence of other psychoactive drugs, but particularly sedatives. Considering these warnings, the high rate of comorbid psychoactive drug usage apparent in the coronial data in Study Two was somewhat alarming and evidence of prescribing to psychotropic poly-drug users. It is unlikely that doctors would be comfortable prescribing to psychotropic poly-drug users, which may point to a lack of patient knowledge. Greater efforts need to be made to understand a patient's risk profile, and the dangers of mixing drugs need to be more clearly communicated to users of psychoactive drugs, especially those groups using sedatives, and victims who are at most risk of dying in a fire. Another option might be the creation of a central register detailing psychotropic drug prescriptions to help prevent psychotropic drug shopping.

Consumers of sedatives must also be aware of the length of time they are likely to be impaired or sedated. The indirect evidence in Study Two supporting low sedative concentrations in the toxicology reports of victims suggests that when the victim responded to the fire they were not experiencing the full effects of the sedative. Nonetheless, the victim's sedative consumption may still have contributed to their death. It is important for consumers of sedatives to understand that mild sedation can occur for a period of 12 hours or more after peak sedative concentration even in a typical short to medium acting compound (Rosenberg, 2006). The ongoing levels of sedation, while not enough to put the victim to sleep in most cases, contributed to their level of impairment. A better understanding of the risks involved in taking sedatives might dissuade potential victims of fire from carrying out seemingly routine and benign tasks (e.g., cooking) that can be potentially dangerous tasks without assistance until the drug is eliminated from their system.

Ultimately, benzodiazepines are the most widely prescribed medication in Australia (Britt, et al., 2007). Due to the ageing population, a demographic partial to hypnotics, this trend is likely to continue. It is possibly unrealistic to expect the vast number of users of hypnotics to cease their medication despite all of the risks. Therefore, the development of safer sedatives is paramount. New drugs are being developed. A recent example is suvorexant, which is currently seeking approval from the relevant bodies (Herring et al., 2012). Suvorexant is safer because it has a different mechanism of action, operating on the orexin system (a wakefulness promoting system) rather than the gamma-aminobutyric acid (GABA) system, which plays a role in sleep, wakefulness, memory, and locomotor function. Any major change in prescription habits would require further research on the impact, if any, these new drugs may have on arousability and skills relevant to accident prevention, particularly if the new hypnotic is chemically dissimilar to the current drugs being administered.

Complexity and Research Design

A central issue in designing arousal research must be to balance the need to maximise both internal and external validity (Cook & Campell, 1979). This is particularly relevant when attempting to generalise to fire emergencies, which are characteristically multi-faceted and complex. The situational context and interactive potential amongst key variables involved in a fire are difficult to approximate in a contrived setting. Addressing these concerns is not always straightforward. Often focusing on internal validity will come at the expense of external validity. For example, frequently only participants who were not suffering from sleep problems were recruited for similar experiments in the past when sleeping ailments may have been widely experienced in the target population (e.g., older adults). The need to demonstrate a causal connection between two variables and limit confounding factors is irrefutable; however, the requirement for scientific rigour is often overstated. The research must also educate and inform decision makers. In order to be useful to organisations that may coordinate and develop smoke alarm standards, the extent to which hypnotic ingestion might impede awakenings to smoke alarms in real life conditions is critical. Imprecision could have fatal consequences. A small

variation of only five decibels could mean the difference between waking or sleeping in response to a smoke alarm.

The departure from a more conventional and controlled design was intended to redress the issue of external validity in previous research. This meant that research took place in the participant's own home in their usual sleeping room, and the researcher was not present throughout the data collection period. Besides gently requesting participants not deviate from their normal routine too much, participants were otherwise free to engage in tasks they normally would (e.g., napping or drinking alcohol). The home environment was important in allowing participants to avail themselves to the contextual cues and reinforcers that are integral in facilitating daily rhythms in behaviour, such as sleep. These external cues are particularly important in people suffering from sleep problems. The equipment used in the study was also supportive of these aims. The sleep/wake status was determined using the innocuous wrist actigraphy technique rather than the demanding EEG equipment. Encouragingly, the introduction of greater variability did not obscure the significance of what was a strong hypnotic effect, but may have blunted the magnitude of the increase in arousal thresholds with hypnotics because greater variability decreases the chances of a significant result (Cohen, 1992). Rather than diminish the significance of the result, this provides a more realistic picture of the likely risk in an everyday home situation in this population and reinforces the value of testing alarms in conditions under which they would ordinarily be applied.

A more adaptable analytical technique was also implemented to deal with data complexity. Analysing multi-factorial problems, such as fire emergencies, has historically been difficult due to statistical considerations, such as the curse of dimensionality. This problem refers to the rapid decrease in space available when adding predictors to the analysis, which then impacts on the ability to achieve statistical significance (Bellman, 1957). However, these are issues machine learning techniques, such as BRT, are quite adept at solving. This research is the first of its kind to apply algorithmic modelling to coronial data. Given the suitability of this analytical procedure to fire problems, future research should be encouraged to utilise these new techniques, possibly in combination with existing methods as a means of validation.

Sleep Quality and Arousal Thresholds

Another aim of secondary importance was to investigate differences in arousability as a function of sleep quality. Poor sleepers often claim to awaken more easily at night (Monroe, 1967) and a main motivation for the preceding work was to reconcile the subjective reports of poor sleepers with objective data. However, numerous studies have consistently failed to find a significant difference in arousal thresholds between good and poor sleepers (Bonnet, et al., 1978; Johnson, et al., 1979; Mendelson, et al., 1986). This study is the first to challenge these findings. Partial support for a difference between good and poor sleepers came from two sources. First was the compelling moderate to large negative relationship that was found between the ISI and arousal thresholds that did not achieve significance, most likely due to the small sample size. Second, was the finding of a tentative difference (i.e., significant at a confidence level of close to 90%) between good and poor sleepers after responding to the 3100 Hz sine wave. Drawing on the theory of hyperarousal, this study now proposes a mechanism that may underpin any potential differences in responsiveness between good and poor sleepers; however, this proposition must be tempered by the study limitations. The theory of hyperarousal posits that the increased sensitivity to external stimuli, evidenced by lower arousal thresholds, is the result of heightened cortical arousal and this then results in sleep disruption (Bonnet & Arand, 2010). A theory of hyperarousal has received strong support in the literature (Bonnet & Arand, 1995, 1998b; Freedman, 1986; Freedman & Sattler, 1982; Hajak, et al., 1995; Haynes, et al., 1981; Irwin, et al., 2003; Lack, et al., 2008; Merica, et al., 1998; Nofzinger, et al., 2004; Perlis, et al., 2001; Riemann, et al., 2002; Stepanski, et al., 1994). This framework provides a good account of the new and tentative empirical findings, illustrating the importance of the type of insomnia when carrying out research in this area. The research on hyperarousal has shown that only people suffering from sleep maintenance insomnia are likely to experience inappropriate arousal during the night when an alarm is likely to sound (Lack, et al., 2008). Sufferers of this form of insomnia have never before been actively recruited, and there was a great deal of inconsistency in the definition of sleep onset insomnia amongst those participants that were recruited.

There are important implications should these findings be replicated in more robust designs. First, the theory of hyperarousal will receive support in arousal research for the first time, further

strengthening this theory's position, as the cause, or causes, of insomnia remains contentious (Varkevisser, et al., 2005). Second, sleep maintenance insomnia is the most common form of insomnia and this situation is likely to only worsen due to the ageing population (Australian Bureau of Statistics, 2011). Older adults are most likely to be suffering from sleep maintenance problems (Hoelscher & Edinger, 1988). Therefore, the proportion of poor sleepers who would be expected to awaken more easily to external stimuli is relatively large and increasing. This may reinforce the appropriateness of auditory alarms in notifying victims of a fire emergency in this population, particularly given that many older adults have trouble hearing higher pitched sounds (Bruck & Thomas, 2008a). Third, this study highlights the importance of a clear and consistent operationalisation of insomnia and its subtypes, especially in research contexts, as arousal thresholds may be expected to vary between different subtypes of insomnia or levels of severity. Finally, if arousal thresholds can capture differences in sleep quality, they may be used as one type of marker of insomnia. Currently, the use of expensive and time consuming EEG equipment is often relied upon to objectively identify faulty patterns in sleep. Actigraphy is increasingly being used but still has many shortcomings. The monitoring of arousal thresholds would be a much cheaper, quicker, and more readily available solution should it be perfected and norms developed.

Should there be no difference in arousal thresholds as a function of sleep quality, the distinction between good and poor sleepers remains important in a fire context. Many of the methodological considerations when assessing the differences between good and poor sleepers are less pertinent in the fire literature. Sleep stage is generally controlled when studying arousal thresholds, in order to limit the strongly supported inflationary impact of increasing sleep depth on arousal thresholds (Bonnet, 1986; Portas, et al., 2000; Rechtschaffen, et al., 1966). However, as has been suggested earlier, sleep stage is less relevant when discussing the response to a smoke alarm during sleep. Since a fire can start at any time of the night, the precise sleep depth of a potential victim is very difficult to predict. Of more importance is the probability of deep sleep occurring throughout the night. A greater distribution of slow wave sleep during the sleep period would increase the possibility of waking up to a fire alarm during a period of deep sleep, when waking from sleep is most difficult. Any factor that influences sleep structure would be liable in this way, such as psychotropic drugs. Given that insomnia has been associated with a

reduction in slow wave sleep, it follows that people suffering from insomnia not on hypnotics would be more likely to wake to a fire alarm, all other variables being equal. So even if there proves to be no difference in arousal thresholds between good and poor sleepers after controlling for sleep stage, there would still likely be a difference between these groups in the reaction to a smoke alarm during a real life fire emergency.

Overall, the current study suggests that there is a possible relationship between sleep quality and arousal thresholds, which would challenge existing work that claims there is no difference between good and poor sleepers. The current study suggests that a difference between good and poor sleepers is only predicted in poor sleepers who suffer from sleep maintenance insomnia. A key reason for the inability to find a difference between good and poor sleepers in previous research may be because poor sleepers who suffer from sleep maintenance insomnia have not been recruited in previous studies analysing arousal thresholds. More research is required to replicate the results recruiting sleepers who suffer from sleep maintenance insomnia.

Embracing a Multi-Disciplinary Approach

The broader significance of this project is the contribution made to a shift in the approach to understanding fire accidents. In general, there has been little focus on individual, small scale accidents (such as residential fire accidents) in the accident causation literature. Greater emphasis is afforded to organisational accidents, which are qualitatively different. In this latter area the disciplines of psychology, sociology, and engineering have been highly influential. In the specific area of fire emergencies, the engineering and ergonomic domains have traditionally been reliable and active contributors, despite the volume of work in other safety areas conducted by psychology or even more recently sociology. This has likely been a function of the technical nature of the fire industry. Overall, these trends have resulted in a modest contribution of psychological perspectives on safety applied in the often neglected but hugely important area of residential fire emergencies. However, there are an increasing number of papers dedicated to analysing residential fires from a psychological perspective. This research supports this trend.

Progress requires a multi-disciplinary approach, embracing the environment, technology, and the individual. This project places emphasis on various elements. Individual and environmental factors were investigated in Study Two, while technological improvements and sensory and perceptual modalities were the focus in Study One. Generally, the introduction of psychological models of accident causation and focus on human factors will contribute to a more balanced and informed disciplinary approach in the literature, which is necessary in order to better understand the complex pathways involved in fire accidents. In particular, there is an opportunity to develop a psychological model of accident causation tailored to home accidents. The relationships explored in Study Two could be considered a reference point for future theoretical work.

Limitations

There were a number of limitations inherent in both studies. In Study One, the small sample size restricted the ability to generalise to the population; however, there were evidently enough participants to detect a significant effect, which suggests there was adequate power for the analysis. The level of priming was also a concern in Study One. As is often the case in arousal research, participants in Study One were expecting to be aroused at some stage by a signal. Priming has been shown to significantly decrease arousal thresholds in specific circumstances (Wilson & Zung, 1966). The impact of priming on Study One was minimised by blinding the participant to the actual night of signal presentation, and by automating the collection of data, which weakened the prime by extending the time between stimulus influence (i.e., the setup of the project by the researcher on day one of the field experiment) and response (i.e., the automated testing of the alarm on subsequent nights). Nevertheless, future research in an unprimed home situation would be extremely valuable in clarifying the precise risk to older adults using hypnotics and other populations. It is worth adding that many people in the population will already be primed to wake to an emergency signal due to the efforts of existing safety messages and previous experiences with emergency alarms, and while this does not remove the problem of priming, it does attenuate it a little.

The investigation into the relationship between sleep quality and arousal thresholds was designed to take advantage of the available data and be exploratory in nature rather than a definitive and

robust analysis. Therefore, in certain areas the methodology is particularly vulnerable to additional criticism. The most important limitation was the use of the correlation method, which is less rigorous than experimental designs (Klein, 1992). Another area of concern related to the sample. The diversity of sleep problem severity within the sample did allow within group comparison and enhanced the likelihood of the sample reflecting population trends of users of hypnotics; however, these advantages did come at a cost. When turning our attention to poor sleepers rather than users of hypnotics, it is difficult to make claims about a population that is poorly defined. The goal of this research was to represent users of hypnotics, rather than poor sleepers. Participants did not have to pass criteria assessing their sleep. The resulting diversity of poor sleepers, evidenced by the wide range of scores on the Insomnia Severity Index, fragments the influence of sleep quality, makes comparisons with previous research difficult, and limits the conclusions that can be drawn with regard to poor sleepers. Altering the project to rectify these issues was not possible within the constraints of the main objectives, and this analysis was only designed to be preliminary. Having uncovered areas of contention, future research that recruits older adults suffering from sleep maintenance insomnia would be a sensible next step.

Study Two was a retrospective analysis of coronial data with its own challenges. First, there were generally quite large differences across most independent variables as a function of psychotropic or sedative consumption, which were not deemed significant; however, it is possible significant results would be achieved with a larger sample. The small sample size also limited the ability to investigate drug interactions. Second, as noted in detail earlier, comparability between the observed VUCD data and the expected population statistics was impacted by data limitations. Third, the study was retrospective, which made it difficult to control bias and confounders, such as the time of fire (Hess, 2004). Fourth, as previously discussed, drug presence was likely to be underestimated in the VUCD. Fifth, the VUCD only includes reported fire fatalities and it is possible that there is a difference between reported and non-reported fire events, particularly when analysing survivors of fires since it is likely that there would be a number of instances where people (who may or may not be under the influence of drugs) woke up in response to a smoke alarm and suppressed a fire without reporting the event. Finally, drugs were analysed in groupings, but the impairment profiles of drugs even in the same

chemical subgroup can vary, which may incorrectly include or exclude some psychoactive drugs in a documented effect (Kleykamp, Griffiths, & Mintzer, 2010).

Conclusions

Despite the limitations discussed in the previous section, the research provides new and importance evidence revealing an uncomfortably high level of risk to users of psychotropic drugs, particularly hypnotics, in a residential fire context. This danger stems from the impact psychotropic drugs have on the behavioural response to fire. Specifically, the following key conclusions can be drawn from the two studies:

- Hypnotic consumption will negatively impact the ability to awaken to a smoke alarm, which is currently the main fire safety technology in residential homes in Australia. This study extended the work of the solitary study in this area by addressing a number of methodological issues present in Johnson et al.'s (1987) research. More than double the proportion of participants slept through the 3100 Hz sine wave at 75 dBA when under the influence of their hypnotic compared to the no hypnotic condition (17% cf. 8% respectively). Together with the previous literature, this presents a major challenge to the effectiveness of existing equipment to protect vulnerable people from fire.
- The 520 Hz square wave is more effective than the 3100 Hz sine wave at waking older adults using hypnotics on an intermittent basis. This study represented the first time this frequency had been tested in users of prescription drugs of any kind. The 520 Hz square wave reduced AATs by approximately 12 dBA, and no participants slept through the 75 dBA standard in either the hypnotic or no hypnotic condition. This provides additional support for the continued adoption of the new technology.
- There is a possible relationship between sleep quality and arousal thresholds, which would challenge existing work that claims there is no difference between good and poor sleepers. However, no conclusive evidence was found regarding the relationship between sleep quality and arousal thresholds due to issues of power. A difference in arousability during the night is only predicted in patients who suffer from sleep maintenance issues, which is an insomnia subtype notably absent in previous research. More research is required to replicate the results using more robust designs.
- Users of sedatives are greatly overrepresented (26.33 times greater than expected) in the Australian fire fatality statistics, while the prevalence of antidepressant usage was also

high (19.7 times greater than expected) compared to population statistics. This is the first time users of psychoactive drugs have been analysed in an Australian dataset, and provides strong support for psychoactive drug usage as an important risk factor in residential fires.

- Hypnotic consumption and fire death is associated with a number of other risk factors. Algorithmic modelling confirmed that the five most influential variables were, in order of importance: (1) time of fire (waking hours versus sleeping hours); (2) age (middle age versus other adult ages); (3) alcohol intake (any consumption versus no consumption); (4) alarm operation (active versus being present or not present); and (5) mental illness (present or not present). In particular, daytime fires provide a medium where a confluence of risk factors specific to fires caused by human involvement has the greatest probability of occurring together. Sedative consumption in this context may first contribute to a larger number of slips and lapses. Critically these minor errors are then not corrected because the already arduous process of problem solving the original error is severely and fatally undermined by the presence of a sedative. Consequently, any initiative that reduces the cognitive stress involved in responding to a fire may increase the chances of survival, such as well understood fire plans.
- Middle age adults are particularly vulnerable in a drug context, which is a shift away from the focus on age ranges at either end of the adult age spectrum. A drug crossover hypothesis was proposed to explain this result, whereby the likelihood of psychotropic poly-drug usage is enhanced during the transition with advancing age away from alcohol and towards sedatives and other medications. The reasons for this remain unclear. Psychotropic poly-drug usage would result in a much higher degree of impairment, as the effects of many drugs are often potentiated during drug interaction.
- A fatality may not be avoided even when the response to the emergency is correct. Therefore, a preventative focus facilitated by the adoption of a safety culture may provide a more satisfactory answer to the fire threat. A focus on prevention and psychological constructs is juxtaposed to the traditional more technologically driven safety perspectives used in the fire industry.
- Fires are complex events. A greater balance in research design that considers internal and external validity is necessary when researching aspects relevant to residential fires.

This requires finding the best balance between real-life variability and controlling extraneous variables, taking into account available sample sizes. This will potentially produce more persuasive and realistic research outcomes that can educate and inform decision makers in the fire industry.

- Algorithmic modelling is better suited to analysing fire fatality statistics or other intricate data sets produced from fire incidents compared to traditional forms of regression. This is the first time this type of analysis has been performed on Australian coronial data. The technique used in this research (BRT) is capable of handling a large number of predictors, missing data, non-linear patterns, interactions, and different data scale types. Greater application of these techniques in fire problems is recommended, possibly used in conjunction with established methodologies for validation purposes.

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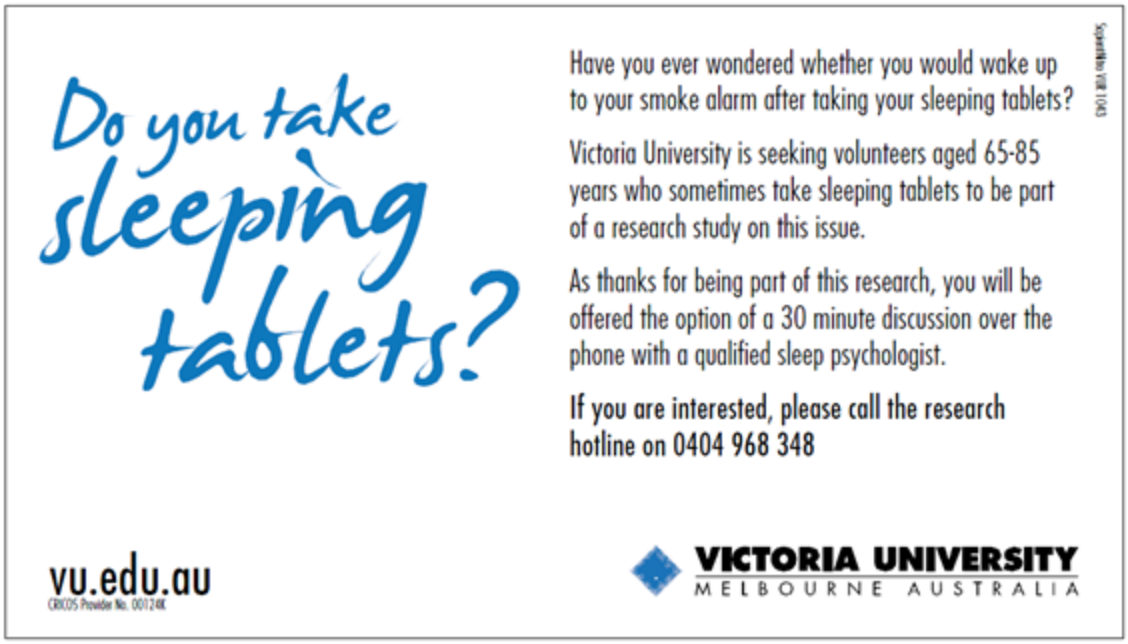
Appendices

Appendix A



Appendix B


Sleep Hygiene


What is Sleep Hygiene?
'Sleep hygiene' is the term used to describe good sleep habits
Medications tend to be only effective in the short-term


Why Does it Matter?
Over half of those over the age of 65 experience disturbed sleep


Snoring
Snoring may be harmless
Be concerned if experiencing very loud snoring, gasping, snorting, or waking up breathless
Talk to GP about possibility of sleep apnea (stopping breathing in night)
Sleep apnea increases risk of:
Daytime sleepiness
Cardio vascular problems


Sleep Hygiene Checklist - Bedroom
Cover the clock by your bedside
Have a comfortable mattress
Don't overheat with heavy bedclothes
Reduce noise
Ensure there are no disturbances such as pets
Feel secure (a night light might be helpful)


Sleep Hygiene Checklist - Daytime
Have predictable (routine) going to bed and getting up times
Don't sleep in long in the mornings (not even on weekends)
Have no naps – they take the edge off the sleep pressure you need (if you find you need a nap then set the alarm for JUST 20 minutes in bed)

Sleep Hygiene Checklist - Daytime

- Set aside a 'worry time' if you tend to take your worries to bed
- If you don't get much exercise try to get out and walk during the day
- Daylight exposure for one or more hours per day may be helpful (e.g., by a sunny window)

Sleep Hygiene Checklist - Evening

- Avoid all caffeine after 4pm
- Don't have a heavy meal late at night
- Any alcohol should be in moderate amounts only, preferably with food
- Don't do major exercise at this time of day
- Don't do activities that make it hard for you to unwind (e.g., nothing too exciting or upsetting)
- Don't fall asleep on the couch, not even for a minute

Sleep Hygiene Checklist - Bedtime

- Get ready for bed in good time, routines are good
- A warm shower can help with sleep onset
- Have a warm milk drink (it contains a substance that may make you drowsy) or camomile tea (takes a few weeks to work best)

Sleep Hygiene Checklist - Bedtime

- Restrict time in bed. Many poor sleepers and older people spend too much time in bed and this fragments sleep. Create a **7.5 or 8** hour *lights out* to alarm on window of time to sleep and stick to it

Where to Get Help

- Sleep disorder clinic
- Sleep psychologist
- Your doctor

Participants Required for Important Study

- Did you know that a 75 year old person is **three times** more likely to die in a home fire than a 35 year old?
- We are conducting research on different alarm signals to help increase the chances of older people, who take sleeping pills or tablets, waking up in a fire
- If you are 65-85 years old and also take sleeping pills or tablets please consider volunteering

VICTORIA UNIVERSITY
MELBOURNE AUSTRALIA

Appendix C

VICTORIA UNIVERSITY
MELBOURNE AUSTRALIA

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

Did you know that a 75 year old person is **three times** more likely to die in a home fire than a 35 year old?

We are conducting research to help increase the chances of older people, who take sleeping pills or tablets, waking up in a fire.

If you are 65-85 years old and also take sleeping pills or tablets please consider volunteering.

Why?

- Contribute to important research aiming to reduce fire deaths
- Determine if you are at risk of sleeping through your alarm
- Free 30 minute treatment session over the phone with a qualified sleep psychologist

How?

- Contact the student researcher, Chris Lykiardopoulos on **0404 968 348** or via email at chris.lykiardopoulos@live.vu.edu.au

Appendix D

Sedative	**Trade names**
Flunitrazepam	Hypnodorm
Midazolam	Hypnovel
Nitrazepam	Alodorm, Mogadon
Temazepam	Temaze, Euhypnos, Normison, Temtabs
Triazolam	Halcion
Zolpidem	Stilnox, Zolpibell, Dormizol, Somidem, Stildem
Zopiclone	Imovane, Imrest

Appendix E

Antidepressant	**Trade names**
Amitriptyline	Tryptanol, Endep
Citalopram	Celexa, Cipramil
Clomipramine	Anafranil
Dothiepin	Prothiaden, Dothep
Escitalopram	Esitalo
Fluoxetine	Prozac, Sarafem, Fontex
Fluvoxamine	Luvox
Imipramine	Tofranil
Mianserin	Bolvidon, Depnon, Norval, Tolvon
Mirtazapine	Remeron, Avanza, Zispin, Miro
Nortriptyline	Allegron
Paroxetine	Aropax, Paxil, Pexeva, Seroxat
Venlafaxine	Effexor or Efexor

Appendix F

Part 1: Hearing criteria guidelines

The American Speech-Hearing-Language Association (ASHA) guidelines were generally followed to formulate these instructions (American Speech-Language-Hearing Association, 2005). This involved three major steps. The equipment was first setup in a quiet room free from visual distractions (maximum permissible sound level was tested to ensure it was below the followings standards – 500hz – 40 dBA, 1000Hz – 40 dBA, 2000Hz – 47 dBA, 4000Hz – 57 dBA (American National Standards Institute, 2004)). The participant was then requested to place the earphones on and ensure there was no obstruction from hair, glasses, or other items. Then the purpose of the test was indicated, that was, to find the faintest tone that could be heard. The participant was then asked to respond whenever a tone was heard, by pressing the yellow button, no matter how faint it may have been. Following setup, the second step involved presenting participants with a familiarisation tone (1000Hz at 60 dBA). Measurement proceeded as the final step if there was a clear response. First, a frequency was presented at 5 dBA and this was then increased in 5 dBA increments until there was a response. Following this the volume was then dropped 10 dBA from the last sound presented and then again increased in 5 dBA until there was a response. This process was repeated until there were two responses at the same level, and then this dBA level was recorded as the arousal threshold for that frequency. This process was repeated at each frequency detailed below and for each ear. The thresholds required to pass replicated the procedure used in Bruck and Thomas' study (2008a). Mean pure tone air-conduction decibel hearing level (dBHL) by frequency, ear, sex and age, with the addition of one standard deviation are presented in the following tables for each sex. Normative values and standard deviations from data collected in 1993-1995 from Beaver, Wisconsin USA; n=3,753 aged 48-92 years (Cruickshanks, et al., 1998).

Pass = air conduction thresholds in dBHL ≤ the values below for each ear

Fail = air conduction thresholds in dBHL > the values below for either ear

Table 12

Minimum pure tone air conduction dBHL thresholds required to pass screening criteria for females across different frequencies

Age (years)	**500 Hz**	**1000 Hz**	**2000 Hz**	**3000 Hz**	**4000 Hz**
60 – 69	25	30	30	35	45
70 – 79	35	40	45	50	55
80 - 92	45	50	55	65	70

Table 13

Minimum pure tone air conduction dBHL thresholds required to pass screening criteria for males across different frequencies

Age (years)	**500 Hz**	**1000 Hz**	**2000 Hz**	**3000 Hz**	**4000 Hz**
60 – 69	25	25	45	65	75
70 – 79	35	40	55	70	80
80 - 92	50	55	70	80	85

Part 2: Comparison of mean auditory thresholds, when awake, for the participants in the current study with normative data from Cruickshanks et al. (1998).

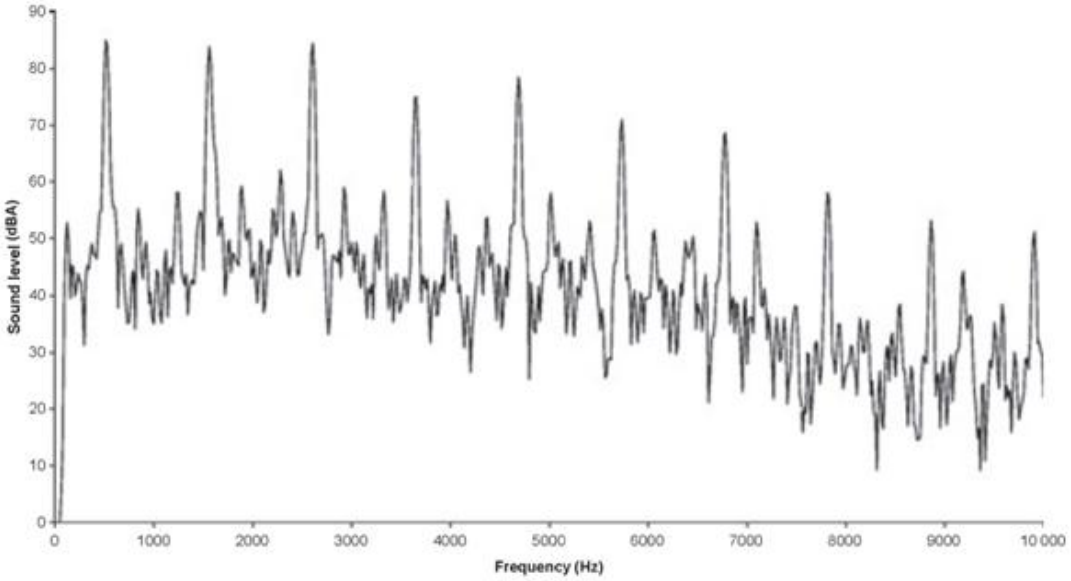
Age group - 60-69	Study *n*	500 Hz		1000 Hz		2000 Hz		3000 Hz		4000 Hz	
		Norms	Study	Norms	Study	Norms	Study	Norms	Study	Norms	Study
Females right ear	3	12.9 (14.1)	23.3 (2.9)	15.7 (14.4)	23.3 (2.9)	18.0 (16)	23.3 (7.6)	21.8 (16.7)	23.3 (11.5)	27.5 (17.6)	30 (13.2)
Males right ear	1	12.2 (13.6)	25.0	15.8 (14.6)	25.0	26.2 (20.9)	25.0	43.3 (23.6)	10.0	54.0 (23.7)	15.0
Females left ear	3	13.5 (13.1)	23.3 (2.9)	16.1 (14)	18.3 (7.6)	18.9 (15.3)	21.7 (7.6)	23.9 (17.2)	28.3 (7.6)	28.7 (17.6)	41.7 (7.6)
Males left ear	1	12.1 (11.6)	20.0	16.4 (12.8)	25.0	28.6 (19.3)	20.0	46.6 (21.6)	20.0	55.1 (21.7)	35.0

Age group - 70-79	Study *n*	500 Hz		1000 Hz		2000 Hz		3000 Hz		4000 Hz	
		Norms	Study	Norms	Study	Norms	Study	Norms	Study	Norms	Study
Females right ear	7	19.7 (16.6)	18.6 (7.5)	22.9 (17.4)	19.3 (5.3)	27.6 (18.6)	19.3 (7.3)	32.5 (19)	25.7 (6.1)	39.0 (19.8)	33.6 (12.8)
Males right ear	0	18.8 (14.5)	0	23.6 (17.5)	0	35.5 (21.1)	0	51.7 (20)	0	62.0 (19)	0
Females left ear	7	19.1 (16.5)	16.4 (6.9)	22.1 (17.6)	16.4 (5.6)	28.3 (18.7)	18.6 (6.9)	34.9 (19.1)	23.6 (6.3)	40.9 (19.8)	35 (14.1)
Males left ear	0	20.3 (17.1)	0	25.3 (19.4)	0	38.8 (21.5)	0	56.1 (19.1)	0	64.6 (19.3)	0

Age group - 80-89	Study *n*	500 Hz		1000 Hz		2000 Hz		3000 Hz		4000 Hz	
		Norms	Study	Norms	Study	Norms	Study	Norms	Study	Norms	Study
Females right ear	0	30.4 (16.8)	0	34.9 (17.8)	0	41.4 (18.3)	0	47.1 (18.1)	0	54.3 (17.5)	0
Males right ear	1	31.8 (22.9)	35	38.2 (22.7)	30	52.3 (19.9)	45	63.5 (17.1)	55	70.5 (17.3)	55
Females left ear	0	21.9 (16.4)	0	34.4 (18.0)	0	41.3 (17.7)	0	48.3 (16.8)	0	54.6 (17.0)	0
Males left ear	1	27.8 (18.1)	30	34.8 (19.5)	35	50.4 (17.7)	50	63.4 (16.4)	65	71.3 (16.8)	65

Appendix G

Spectral analysis of the 520 Hz square wave



Appendix H

Sound delivery technical details

Two Yamaha NS-P150PN speakers connected via a Yamaha HTR receiver.

Appendix I

INTRODUCTION AND PROJECT OVERVIEW

Hi, my name is Chris I am from Victoria University. You recently indicated that you might be interested in completing our important study on the impact sleeping tablets may have on the ability to wake up to different types of smoke alarms.

To see if you fit the criteria needed for the study I will need to ask you a few questions that will take around 10 minutes. Is this ok?

TERMINATE – Unfortunately you do not fit the specific criteria of the study. Is there someone else in the household who might like to complete the survey between the ages of 65-85 who takes sleeping pills or tablets?

SCREENERS

SC1. For classification purposes, could you tell me your age?

Under 65	1	Terminate
65-75	2	
76-85	3	
Over 85	4	Terminate

SC2. Do you suffer from any serious hearing problems?

Yes	1	Terminate
No	2	
Don’t know	3	

SC3. Have you ever been really concerned about your short-term memory, or do you get easily confused?

DISCUSS and terminate if major concerns that individual will not be able to follow study directions.

SC4. In the last four weeks have you taken the following types of medications?

	Yes	No	
Antidepressants (e.g., Prozac, Zoloft, Marplan)	1	2	
Antipsychotics (e.g., Thorazine, Haldol, Abilify)	1	2	
Mood stabilizers (e.g., Carbolith, Tegretol, Depakote)	1	2	
Stimulants (e.g., Ritalin, Focalin, Dexedrine)	1	2	
Anti-anxiety medication (e.g., Valium)	1	2	
Sleeping pills or sedatives (e.g., Temazepam, Stilnox)	1	2	IF NO TERMINATE

SC5. What types or brands of (ABOVE) have you taken in the last four weeks? (Cycle through each)

LIST OF DRUGS – TERMINATE AFTER ASSESSING FREQUENCY IF TAKING SEDATING DRUGS NOT ON THE APPROVED LIST OR NOT TAKING AN APPROVED DRUG ETC. (based on consultation with Sleep Physician)

SC6. And what condition is the medication prescribed for? (Cycle through each)

_____

SC7. What dosage do you usually take? (Cycle through each)

_____

SC8. How frequently would you take (ABOVE)? (Cycle through each)

Everyday	1	TERMINATE (IF HYPNOTICS)
More than once a week but not daily	2	
Only once a week	3	TERMINATE (IF HYPNOTICS)
Fortnightly	4	TERMINATE (IF HYPNOTICS)
Monthly	5	TERMINATE (IF HYPNOTICS)
Less frequently than once every month but still use	6	TERMINATE (IF HYPNOTICS)
Don’t use anymore	7	TERMINATE (IF HYPNOTICS)

DETAILS OF STUDY

Thanks, you are eligible to participate in the research.

The research involves an alarm being placed in your home that will sound between 3 to 5 times over an 11 night period. During this time you will be asked to take your sleeping pills in a pattern we prescribe, which will effectively mean taking the pills on and off for two nights at a time. It will be important to maintain the same dose throughout the 11 nights.

Should you wake up to an alarm you would only need to press a button setup next to your bed to stop the alarm. Each morning you will be required to fill in a simple sleep diary that would take a few minutes to complete.

All data collected will be de-identified and only analysed and presented in group form to ensure confidentiality.

You will also have to wear a watch like device at night that will allow us to monitor when you are sleeping.

For participating in the research we are offering a free 30 minute discussion about your sleep and good sleep habits with a qualified sleep psychologist over the phone.

Full details of the study will be sent to you via mail prior to the research commencing and you can withdraw from the project at any time.

Are you still interested in participating?

SLEEP HISTORY AND DRUG HABITS

We have some more questions we need to ask you now about your sleep history.

SH1. What time do you usually turn off the lights and attempt to fall sleep at night?

_____:_____ (use 24 hour clock)

SH2. After turning off the lights, how long does it usually take you to fall asleep on nights when you take your usual sleeping pill or sedative?

_____ minutes

SH3. After turning off the lights, how long does it usually take you to fall asleep on nights when you don't take your usual sleeping pill or sedative?

_____ minutes

SH4. How long before you usually fall sleep would you usually take your usual sleeping pill or sedative?

_____ minutes

SH5. How likely are you to fall asleep at roughly the same time on weekdays (vary less than 30 minutes)?

Very likely	1
Likely	2
Not likely	3
Not at all likely	4

DEMOGRAPHICS

Finally, we have to ask you a few more questions for classification purposes.

DG1. Sex

Male	1
Female	2

DG2. Which of the following would best describe your usual sleeping situation?

Sleep alone	1
Sleep together with another	2

DG3. Which of the following would best describe your current employment status?

Working (full-time)	1
Working (part-time)	2
Studying/ training	3
Retired	4
Unemployed	5
Not looking for work	6

It would be best if we can organise a tentative time for me to come over and setup the alarm equipment. I will call you a few days before this time to confirm you would still like to be in the study.

The study must begin on a Monday. Are you free....?

ARRANGE A TIME/ DATE

ADDRESS DETAILS?

That is all the information we need at this stage, thank you for your time.

Appendix J

INSOMNIA SEVERITY INDEX (ISI)

For each question, please *CIRCLE* the number that best describes your answer.

Please rate the *CURRENT (i.e. LAST 2 WEEKS) SEVERITY* of your insomnia problem(s).

Insomnia problem	None	Mild	Moderate	Severe	Very severe
1. Difficulty falling asleep	0	1	2	3	4
2. Difficulty staying asleep	0	1	2	3	4
3. Problem waking up too early	0	1	2	3	4

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

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

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

Not at all Noticeable	A Little	Somewhat	Much	Very Much Noticeable
0	1	2	3	4

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

Not at all Worried	A Little	Somewhat	Much	Very Much Worried
0	1	2	3	4

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

Not at all Interfering	A Little	Somewhat	Much	Very Much Interfering
0	1	2	3	4

CONSENT FORM
FOR PARTICIPANTS
INVOLVED IN RESEARCH

We would like to invite you to be a part of a study that aims to develop the best smoke alarm signal possible for waking different groups within the population, including older adults under the influence of sleeping pills or sedatives.

I**,** *(insert name)* _____

of *(insert address)* _____

certify that I am at least 18 years old and that I am voluntarily giving my consent to participate in the study entitled:

Psychotropic Drug Usage and Human Behaviour During Fire Emergencies

being conducted at Victoria University by:

Professor Dorothy Bruck, Dr. Michelle Ball, and Chris Lykiardopoulos.

I certify that the objectives of the study, together with any risks and safeguards associated with the procedures listed hereunder to be carried out in the research, have been fully explained to me by **Chris Lykiardopoulos**, and that I freely consent to participation involving the below mentioned procedures:

- Be interviewed about your sleep quality, medication intake, hearing capabilities and normal daily activities
- Complete a pencil and paper questionnaire on your sleep quality, medication intake, hearing capabilities and normal daily activities. Including a daily dairy of your wake and sleep times, detailing any activities that may have impacted your sleep and wake (e.g., drinking an unusual amount of coffee)
- Participate in a free hearing test
- Wear a device on your non-dominant wrist, similar to a watch in appearance, at night for 11 nights to measure your sleep patterns in your own home and usual bedroom
- Attempt to go to sleep at a similar time each night
- Be awoken on between three to five nights by an alarm signal via a speaker (connected to a laptop in another room) in your usual bedroom
- Take your usual sleeping pill or sedative for a total <u>five nights only</u> during the 11 night study in a pattern the research team prescribes. Sleeping pills will be taken for two nights then not taken for two nights, and then this process will be repeated

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

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

Signed: _____

Date: _____

Any queries about your participation in this project may be directed to the student researcher, Chris Lykiardopoulos via email in the first instance (chris.lykiardopoulos@live.vu.edu.au). Alternatively, the Principal Researcher, Professor Dorothy Bruck is available at 03 9919 2158. If you have any queries or complaints about the way you have been treated, you may contact the Ethics & Biosafety Coordinator, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4148.

INFORMATION TO PARTICIPANTS INVOLVED IN RESEARCH

You are invited to participate

You are invited to participate in a research project entitled: **Psychotropic Drug Usage and Human Behaviour During Fire Emergencies**

This project is being conducted by Professor Dorothy Bruck and Dr. Michelle Ball from the School of Social Sciences and Psychology at Victoria University, who are supervising a student researcher, Chris Lykiardopoulos, as part of a PhD study at Victoria University.

Project explanation

The study aims to develop the best smoke alarm signal possible for waking different groups within the population, including older adults under the influence of sleeping pills or sedatives.

What will I be asked to do?

You will be asked to do the following:

- Be interviewed about your sleep quality, medication intake, hearing capabilities and normal daily activities
- Complete a pencil and paper questionnaire on your sleep quality, medication intake, hearing capabilities and normal daily activities. Including a daily dairy of your wake and sleep times, detailing any activities that may have impacted your sleep and wake (e.g., drinking an unusual amount of coffee)
- Participate in a free hearing test
- Wear a device on your non-dominant wrist, similar to a watch in appearance, at night for 11 nights to measure your sleep patterns in your own home and usual bedroom
- Attempt to go to sleep at a similar time each night
- Be awoken on between three to five nights by an alarm signal via a speaker (connected to a laptop in another room) in your usual bedroom
- Take your usual sleeping pill or sedative for a total five nights only during the 11 night study in a pattern the research team prescribes. Sleeping pills will be taken for two nights then not taken for two nights, and then this process will be repeated

What will I gain from participating?

For participating, you will be offered the option of one 30 minute discussion about your sleep and sleep hygiene measures with a qualified sleep psychologist over the phone. Please note the discussion will be limited to one session only. However, a referral can be organised to an

appropriate sleep clinic at your own expense in consultation with the sleep psychologist (many sleep psychologists bulk bill the majority, if not all, of the service fee).

How will the information I give be used?

All data collected will be de-identified and only analysed and presented in group form to ensure confidentiality. The data will be used to understand what the best smoke alarm signal is for waking different groups within the population, including older adults under the influence of sleeping pills or sedatives.

What are the potential risks of participating in this project?

The potential risks for participating in the study include:

- There is a small possibility that you may suffer withdrawal from your usual sleeping pills or sedative. Our prescription plan has been verified by a Sleep Physician who believes that altering the treatment plan presents minimal risks for someone taking sleeping pills or sedatives between two to six nights per week. In order to identify any difficulties you may experience, the researchers will telephone you on day four to monitor how you are coping with the changes to your normal medication schedule. Should you feel any adverse effects, you will be advised to contact your own doctor to ensure continuity of the best care. It will be your responsibility to contact your doctor. We will cover any fees incurred above the cost of the bulk billing fee. Furthermore, you are welcome to contact the researchers at any time, and may also withdraw from the research at any time with no penalty.
- There is a very small risk that the alarm may confuse your neighbours to a real danger. We will notify your neighbours prior to the research to avoid any confusion by putting a notice in their letterboxes.
- There is a small possibility that the results from the hearing test may uncover a hearing problem you did not know about, which may cause some psychological discomfort. However, if a hearing problem is detected you will be offered information on professional screening and assistance through H.E.A.R. Services.
- There is a small possibility that the change in routine may cause some discomfort. Dr. Gerard Kennedy (9919 2481) will be available to provide counselling services should you experience any discomfort.
- There is a small possibility that the people living together with you may experience possible nightly interruptions. We will offer ear plugs to any other people in the house to minimise this disruption.
- There is a small possibility that the interruptions to your sleep may cause daytime drowsiness the following day. We will brief you in more detail about these possible risks to prepare you for any next day drowsiness.

Note: you can withdraw from the project at any time.

How will this project be conducted?

We will come to your home at the beginning of the study and brief you on all aspects of the research, setup the equipment (including speakers and a bedside button) in your bedroom and laptop computer in an adjacent room, and give you a quick questionnaire to complete. After briefing you on the study we will leave until the conclusion of the project 11 nights later, but we will be available via phone or email should you have questions. On each of the 11 nights of the project you will be required to wear the watch-like device to measure your sleep patterns. On five of the 11 nights you will be asked to take your usual sleeping pills in a pattern we prescribe. During the 11 nights an alarm signal will be presented automatically via the speakers we setup in your bedroom, but you will not know which nights this will happen. An alarm will be presented between three to five times. The alarm will **not** be presented on a Friday or Saturday so you can enjoy your weekends. When an alarm signal is presented and you wake up you will be required to push the bedside button setup in your bedroom, then you can return to sleep. An alarm will only sound once on the allocated night. You should not be worried if you do not wake up to the alarm. Each day you will need to fill in a diary of your sleep and wake times, including details of any activities that may have impacted your sleep and wake (e.g., drinking an unusual amount of coffee). Finally, on the 12th day we will come and collect all the equipment and get your feedback.

Who is conducting the study?

The research team at Victoria University will include:

Principal Researcher, Professor Dorothy Bruck (03 9919 2158)
Principal Researcher, Dr. Michelle Ball (03 9919 2536)
Student Researcher, Chris Lykiardopoulos (0404 968 348)

Any queries about your participation in this project may be directed to the student researcher, Chris Lykiardopoulos, via email in the first instance (chris.lykiardopoulos@live.vu.edu.au). Alternatively, the research team can be contacted at the above numbers. If you have any queries or complaints about the way you have been treated, you may contact the Ethics and Biosafety Coordinator, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4148.

Appendix K

Project Pack

Psychotropic Drug Usage and Human Behaviour During Fire Emergencies

THE PROJECT PACK

Thank you for being involved in this important research.

This Project Pack includes all the information you will need in order to be part of this research. Read through the pack carefully and make sure you understand what needs to be done. Keep the Project Pack in a convenient place as you will need to look at it regularly.

The Project Pack includes the following items:

- A Sleep Diary that you must complete each day
- One Actiwatch
- A Prescription Plan
- Handy hints and reminders
- Contact details of the research team

THE ACTIWATCH

On each of the 11 nights of the project, you will be required to wear an Actiwatch. The Actiwatch is a watch-like device to measure when you are asleep and awake.

You **must** wear the Actiwatch on your wrist before you go to sleep **each night**. You may choose to wear it in the day as well – it's up to you.

The Actiwatch is worn on the wrist of your **non-dominant hand** (your non-dominant hand is the one you do not write with).

The Actiwatch also measures light, so please turn off the lights when you go to bed and ensure your shirt/ blouse does not block the light sensor as far as possible.

You can shower or bath while wearing the Actiwatches, **they are waterproof**. However, take the Actiwatch off if you go swimming or bath for longer than 25 minutes.

The Actiwatches are very expensive so please **ensure they are kept safe** and undamaged and returned to the researchers at the end of the study in the same condition they were delivered.

THE PRESCRIPTION PLAN

It is critical that the Prescription Plan that the research team has outlined is **followed exactly**. Please take your usual type and dose of sleeping pill and try to maintain your usual sleeping patterns as best as possible.

Our Prescription Plan has been verified by a Sleep Physician; however, should you experience any negative effects contact your own doctor immediately.

Night		Prescription Plan
N1	Monday	**Do not take** your usual sleeping pill
N2	Tuesday	**Do not take** your usual sleeping pill
N3	Wednesday	Take your usual sleeping pill
N4	Thursday	Take your usual sleeping pill
N5	Friday	**Do not take** your usual sleeping pill
N6	Saturday	**Do not take** your usual sleeping pill
N7	Sunday	Take your usual sleeping pill
N8	Monday	Take your usual sleeping pill
N9	Tuesday	**Do not take** your usual sleeping pill
N10	Wednesday	**Do not take** your usual sleeping pill
N11	Thursday	Take your usual sleeping pill
Day 12 - Pick up at agreed time on Friday		

REMINDERS AND TIPS

- **If you wake up** to an alarm then **press the bedside button THREE times** in quick succession.
- Once the bedside button is pushed three times the alarm currently being presented will stop and a set of acknowledgement beeps to confirm you have pressed the bedside button will sound.
- It's very important that you only take your usual hypnotic at your **usual dosage** on the **days we prescribe.**
- The Actiwatch **must be on** before you attempt to go to sleep each night. It is waterproof, so you can wear the Actiwatch in the shower or bath. You can wear it continuously (day and night) during the study if you prefer.
- As much as possible try and turn off the lights and attempt to fall asleep at your **usual bedtime** each night.
- The alarm will be presented between three to five times over the 11 nights.
- The alarm will **not** be presented on a Friday or Saturday night so you can enjoy your weekends.
- Look at the Sleep Diary each night to check any requirements and fill in the Sleep Diary **as soon as you wake up** in the morning so you don't forget.
- Look after the Actiwatch it is very expensive.
- If you sleep with someone else in the same room than please tell them not to touch any of the equipment or wake you up should they wake up to an alarm.
- You may be drowsy in the morning, so be careful when doing any activity where it's important that you are alert.
- You should not need to touch the laptop or any of the sound equipment. The laptop must be left on at all times.
- If you believe any of the equipment is not working properly, please do not touch the equipment, simply email the research team immediately.

EXAMPLE OF SLEEP DIARY

The Sleep Diary is an important part of the research. It is critical that you complete the Sleep Diary as best you can each **morning.**

Please look at the example below. If after studying the example you still have trouble completing the Sleep Diary, please contact the research team.

Each column and page in the table is designed to record the sleep and wake activities that occurred during the previous day and night. There are 11 blank columns and pages which will record 11 days worth of information.

If you have further information which you feel may be relevant to your sleep pattern, please include this in the Comments section below. If you take any medication that you have not previously told us about please write this in under Comments.

Remember, the Sleep Diary is just a useful guide. Do not be concerned if you are unsure of all the details. The completed Sleep Diary will be collected by the research team at the conclusion of the project.

SLEEP DIARY – Example		**Example Only**
	Today's calendar date	10 / 04 / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4.**	(Yes) / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	(Yes) / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	(Yes) / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	1pm to 2pm
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	15mg of Temazepam
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	10:25pm
7.	Last night, what time did you get in to your bed (AM or PM?).	10:30pm
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	10:45pm
9.	After turning off the lights, how many minutes did it take you to fall asleep?	20 minutes
10.	Today, what time did you get out of bed for the day (AM or PM?).	6:30am
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	6
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	Coffee at 5pm Loud noise at night

COMMENTS _____

MONDAY NIGHT (N1)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ Do **not** take any sleeping medication tonight

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on TUESDAY MORNING using information from MONDAY NIGHT		**Monday night N1** Complete on Tuesday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4**.	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7**.	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	

REMEMBER: BEFORE BED TONIGHT (TUESDAY NIGHT N2)
DO NOT TAKE YOUR USUAL SLEEPING PILL

COMMENTS _____

TUESDAY NIGHT (N2)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ Do **not** take any sleeping medication tonight

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on WEDNESDAY MORNING using information from TUESDAY NIGHT		**Tuesday night N2** Complete on Wednesday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4**.	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (WEDNESDAY NIGHT N3) **TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

WEDNESDAY NIGHT (N3)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ **Take** your sleeping medication tonight at _____

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on THURSDAY MORNING using information from WEDNESDAY NIGHT		**Wednesday night N3** Complete on Thursday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4.**	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (THURSDAY NIGHT N4) **TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

THURSDAY NIGHT (N4)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ **Take** your sleeping medication tonight at _____

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on FRIDAY MORNING using information from THURSDAY NIGHT	**Thursday night N4** Complete on Friday morning
Today's calendar date	_ _ / _ _ / 2011
1. Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4**.	Yes / No
2. Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3. Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4. What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5. What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6. What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7. Last night, what time did you get in to your bed (AM or PM?).	
8. Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9. After turning off the lights, how many minutes did it take you to fall asleep?	
10. Today, what time did you get out of bed for the day (AM or PM?).	
11. How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12. Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (FRIDAY NIGHT N5) **DO NOT TAKE** YOUR USUAL SLEEPING PILL	

COMMENTS _____

FRIDAY NIGHT (N5)

BEFORE BED

There will definitely be no alarm tonight so you do **not** need to wear your Actiwatch. However, it is important that you do **not** take a sleeping tablet.

☐ Do **not** take any sleeping medication tonight

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on SATURDAY MORNING using information from FRIDAY NIGHT		**Friday night N5** Complete on Saturday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
2.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, **go to Q4**.	
3.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
4.	Last night, what time did you get in to your bed (AM or PM?).	
5.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
6.	After turning off the lights, how many minutes did it take you to fall asleep?	
7.	Today, what time did you get out of bed for the day (AM or PM?).	
8.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
9.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (SATURDAY NIGHT N6) **DO NOT TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

SATURDAY NIGHT (N6)

BEFORE BED

There will definitely be no alarm tonight so you do **not** need to wear your Actiwatch. However, it is important that you do **not** take a sleeping tablet.

☐ Do **not** take any sleeping medication tonight

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on SUNDAY MORNING using information from SATURDAY NIGHT		**Saturday night N6** Complete on Sunday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
2.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, **go to Q4**.	
3.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
4.	Last night, what time did you get in to your bed (AM or PM?).	
5.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
6.	After turning off the lights, how many minutes did it take you to fall asleep?	
7.	Today, what time did you get out of bed for the day (AM or PM?).	
8.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
9.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (SUNDAY NIGHT N7) **TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

SUNDAY NIGHT (N7)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ **Take** your sleeping medication tonight at _____

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on MONDAY MORNING using information from SUNDAY NIGHT		**Sunday night N7** Complete on Monday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4.**	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (MONDAY NIGHT N8) **TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

MONDAY NIGHT (N8)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ **Take** your sleeping medication tonight at _____

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on TUESDAY MORNING using information from MONDAY NIGHT		**Monday night N8**
		Complete on Tuesday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4.**	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (TUESDAY NIGHT N9) **DO NOT TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

TUESDAY NIGHT (N9)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ Do **not** take any sleeping medication tonight

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on WEDNESDAY MORNING using information from TUESDAY NIGHT		**Tuesday night N9** Complete on Wednesday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4.**	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (WEDNESDAY NIGHT N10) **DO NOT TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

WEDNESDAY NIGHT (N10)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ Do **not** take any sleeping medication tonight

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on THURSDAY MORNING using information from WEDNESDAY NIGHT		**Wednesday night N10** Complete on Thursday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4**.	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: BEFORE BED TONIGHT (THURSDAY NIGHT N11) **TAKE** YOUR USUAL SLEEPING PILL		

COMMENTS _____

THURSDAY NIGHT (N11)

BEFORE BED

☐ Go to bed at _____ (plus or minus 30 minutes)

☐ **Take** your sleeping medication tonight at _____

☐ Ensure your Actiwatch is on your wrist (non-dominant hand)

☐ In the morning complete the Sleep Diary below

THE FOLLOWING MORNING

SLEEP DIARY – Complete this on FRIDAY MORNING using information from THURSDAY NIGHT		**Thursday night N11** Complete on Friday morning
	Today's calendar date	_ _ / _ _ / 2011
1.	Did you hear an alarm last night? If you did not hear an alarm last night, circle "NO", then **go to Q4.**	Yes / No
2.	Were you asleep when the alarm sounded (i.e., were you asleep before waking up to the alarm)?	Yes / No / Not Sure
3.	Did you press the bedside button three times in quick succession when you heard the alarm?	Yes / No
4.	What time did you take a nap yesterday (note time of all naps / AM or PM)? If you did not take a nap yesterday, simply write down "NA".	
5.	What item(s) did you take as a sleep aid(s) yesterday (include all prescription and over-the-counter sleep aids). If you did not take any items as sleep aids yesterday, simply write down "NA" and **go to Q7.**	
6.	What time(s) did you take the sleep aid(s) yesterday (AM or PM?).	
7.	Last night, what time did you get in to your bed (AM or PM?).	
8.	Last night, what time did you turn off the lights and attempt to fall asleep (AM or PM?).	
9.	After turning off the lights, how many minutes did it take you to fall asleep?	
10.	Today, what time did you get out of bed for the day (AM or PM?).	
11.	How would you rate the quality of last night's sleep: Very Poor Fair Excellent 1 2 3 4 5 6 7 8 9 10	
12.	Yesterday, were there any events that greatly impacted your sleep (e.g., caffeine, alcohol, worry, background noise, exercise)? If you did not experience any events that greatly impacted your sleep, simply write down "NA".	
REMEMBER: THE RESEARCH TEAM WILL ARRANGE A TIME TO COLLECT THE EQUIPMENT AND YOUR FEEDBACK TODAY		

COMMENTS _____

ON FRIDAY (DAY 12) THE RESEARCH TEAM WILL ARRANGE A TIME TO COLLECT THE EQUIPMENT AND GET YOUR FEEDBACK

CONTACT DETAILS

The preferred mode of contact is via email in the first instance. However, if you do not use email please ring.

EMAIL: chris.lykiardopoulos@live.vu.edu.au

RESEARCH HOTLINE: 0404 968 348

The research team at Victoria University includes:

Principal Researcher, Professor Dorothy Bruck
Principal Researcher, Dr. Michelle Ball
Student Researcher, Chris Lykiardopoulos

Should the change in routine cause discomfort, you can contact Associate Professor Gerard A. Kennedy who has kindly offered a counselling service on this project (03 9919 2481).

Appendix L

Participant	Psychotropic drug	Reported dose
1	Temaze	10mg
2	Zolpidem	10mg
3	Temaze	10mg
4	Temaze	10mg
5	Temaze	10mg
6	Temaze	20mg
7	Temaze	10mg
8	Nitrazepam	5mg
9	Temaze	15mg
10	Temaze	15mg
11	Temaze	10mg
12	Temaze	10mg

Appendix M

Drug/Group	Classified as:		
	Antidepressant	**Sedative**	**Illicit drug**
Amitriptyline	Yes	No	No
Citalopram	Yes	No	No
Dothiepin	Yes	No	No
Fluoxetine	Yes	No	No
Mirtazapine	Yes	No	No
Paroxetine	Yes	No	No
Sertraline	Yes	No	No
Tricyclics	Yes	No	No
Venlafaxine	Yes	No	No
Nefazodone	Yes	No	No
Alprazolam	No	Yes	No
Benzodiazepine	No	Yes	No
Diazepam	No	Yes	No
Nitrazepam	No	Yes	No
Oxazapam	No	Yes	No
Temazepam	No	Yes	No
Zolpidem	No	Yes	No
Amphetamine	No	No	Yes
Cannabinoids	No	No	Yes
Heroin	No	No	Yes
Methamphetamine	No	No	Yes
Carbamazepine	No	No	No
Celecoxib	No	No	No
Codeine	No	No	No
Diltiazem	No	No	No
Doxylamine	No	No	No
Frusemide	No	No	No
Gliclazide	No	No	No
Methadone	No	No	No
Morphine	No	No	No
Naproxen	No	No	No
Olanzapine	No	No	No

Oxycodone	No	No	No
Paracetamol	No	No	No
Phentermine	No	No	No
Promethazine	No	No	No
Thioridazine	No	No	No
Toluene	No	No	No
Tramadol	No	No	No
Verapamil	No	No	No

Appendix N

Australian Bureau of Statistics

43640DO009_20072008 National Health Survey: Summary of Results, 2007–2008 (Reissue)

Released at 11:30 am (Canberra time) 23 Nov 2010

Table 9 Mental health conditions: Medications and actions taken(a), Persons

								Total 18 years and over		
	35–44	45–54	55–64	Total 35-64	65–74	75 years and over	Total >65	Males	Females	Persons
Persons with a mental health condition										
Generic type of medication used(f)										
Amitriptyline	2.10	4.00	11.60	17.70	3.00	2.50	5.50	8.80	21.10	29.80
Other tricyclic antidepressants and mianserin	np	10.00	8.20	18.20	3.50	7.00	10.50	8.30	26.40	34.70
Citalopram	28.10	17.00	24.60	69.70	7.40	7.80	15.20	40.90	68.60	109.50
Paroxetine	3.20	4.30	5.70	13.20	np	np	0.00	7.50	16.00	23.50
Sertraline	25.00	21.30	15.20	61.50	14.30	6.50	20.80	40.20	62.90	103.20
Other serotonin reuptake inhibitors	20.10	16.90	10.10	47.10	3.60	1.50	5.10	34.50	42.90	77.40
Venlafaxine	41.00	26.10	28.20	95.30	3.40	1.70	5.10	44.90	75.90	120.80
Other antidepressants	3.40	10.00	8.80	22.20	5.40	4.80	10.20	19.60	21.40	41.00
Diazepam	9.20	17.90	8.20	35.30	8.60	2.30	10.90	23.90	40.50	64.40
Oxazepam	3.50	2.50	6.00	12.00	2.70	3.20	5.90	7.40	12.70	20.10
Temazepam	6.80	13.40	8.50	28.70	np	np	0.00	13.00	36.70	49.70
Other benzodiazepines	5.80	8.40	5.90	20.10	2.00	1.90	3.90	9.60	21.00	30.50
Other pharmaceutical medication	48.70	50.40	47.10	146.20	14.70	10.40	25.10	109.90	145.90	255.80
Total pharmaceutical medication	156.90	154.30	148.10	459.30	57.60	47.60	105.20	297.70	468.70	766.40
Vitamins, minerals and herbal treatments	42.30	45.30	35.60	123.20	5.10	3.70	8.80	70.00	120.10	190.10
Total(e)	159.30	154.30	149.50	463.10	57.60	47.60	105.20	307.20	470.20	777.50
Total persons with a mental health condition	*394.70*	*377.80*	*327.60*	*1100.10*	*132.40*	*116.60*	*249.00*	*1055.80*	*1253.90*	*2309.80*

np Not available for publication but included in totals where applicable, unless otherwise indicated

WORKINGS

(A) Total persons with a mental health condition using Benzodiazepines	*25.30*	*42.20*	*28.60*	*96.10*	*13.30*	*7.40*	*20.70*	*53.90*	*110.90*	*164.70*
(B) Total persons with a mental health condition using Antidepressants	*122.90*	*109.60*	*112.40*	*344.90*	*40.60*	*31.80*	*72.40*	*204.70*	*335.20*	*539.90*
(C) Population benchmark in '000s as at December 31(time of survey)	*3045.40*	*2884.40*	*2342.40*	*8272.20*	*1443.60*	*1157.50*	*2601.10*	*7770.90*	*7980.10*	*15751.00*
(A / C) % persons using Benzodiazepines rebased to general pop.	*0.83%*	*1.46%*	*1.22%*	*1.16%*	*0.92%*	*0.64%*	*0.80%*	*0.69%*	*1.39%*	*1.05%*
(B / C) % persons using Antidepressants rebased to general pop.	*4.04%*	*3.80%*	*4.80%*	*4.17%*	*2.81%*	*2.75%*	*2.78%*	*2.63%*	*4.20%*	*3.43%*

Appendix O

Variable	Definition	
	Unadjusted OR analysis	**BRT analysis**
Age	30-60/Other adult ages	Continuous (range 18-99)
Alarm operation	Not used	Not present/Present/Active
Alcohol intake	Positive/Negative	Continuous (range 0-0.37)
Sleep/wake	Awake/Asleep	Awake/Asleep
Conditions preventing escape	Present/Absent	Present/Absent
Location	In RFO/Elsewhere	In RFO/Elsewhere
Mental illness	Present/Absent	Present/Absent
People presence in home	Alone/Not alone	Alone/Not alone
Physical illness	Present/Absent	Present/Absent
Sex	Male/Female	Male/Female
Smoke alarm active	Active/All other possibilities	Merged-Alarm operation
Smoke alarm present	Present/Not present	Merged-Alarm operation
Smoking related materials	Involved/Not involved	Involved/Not involved
Time of fire	10:00-22:00/Other times	Continuous (range 00:00-23:59)

Appendix P

Drug	Classified as:	
	Psychotropic	**Sedative**
Temazepam	Yes	Yes
Diazepam	Yes	Yes
Alprazolam	Yes	Yes
Zolpidem	Yes	Yes
Nitrazepam	Yes	Yes
Benzodiazepine	Yes	Yes
Oxazapam	Yes	Yes
Promethazine	Yes	No
Paroxetine	Yes	No
Venlafaxine	Yes	No
Mirtazapine	Yes	No
Cannabinoids	Yes	No
Codeine	Yes	No
Morphine	Yes	No
Carbamazepine	Yes	No
Heroin	Yes	No
Methadone	Yes	No
Tricyclics	Yes	No
Fluoxetine	Yes	No
Sertraline	Yes	No
Amphetamine	Yes	No
Citalopram	Yes	No
Olanzapine	Yes	No
Toluene	Yes	No
Nefazodone	Yes	No
Amitriptyline	Yes	No
Oxycodone	Yes	No
Tramadol	Yes	No
Methamphetamine	Yes	No
Phentermine	Yes	No
Doxylamine	Yes	No
Thioridazine	Yes	No

Dothiepin	Yes	No
Paracetamol	No	No
Frusemide	No	No
Diltiazem	No	No
Verapamil	No	No
Celecoxib	No	No
Gliclazide	No	No
Naproxen	No	No

Appendix Q

STUDY 1

General Linear Model

Within-Subjects Factors

Measure: MEASURE_1

Alarm	Drug	Dependent Variable
1	1	lownodrug11
	2	lowdrug12
2	1	highnodrug21
	2	highdrug22

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df
Alarm	Pillai's Trace	.889	88.105[b]	1.000	11.000
	Wilks' Lambda	.111	88.105[b]	1.000	11.000
	Hotelling's Trace	8.010	88.105[b]	1.000	11.000
	Roy's Largest Root	8.010	88.105[b]	1.000	11.000
Drug	Pillai's Trace	.718	28.044[b]	1.000	11.000
	Wilks' Lambda	.282	28.044[b]	1.000	11.000
	Hotelling's Trace	2.549	28.044[b]	1.000	11.000
	Roy's Largest Root	2.549	28.044[b]	1.000	11.000
Alarm * Drug	Pillai's Trace	.003	.033[b]	1.000	11.000
	Wilks' Lambda	.997	.033[b]	1.000	11.000
	Hotelling's Trace	.003	.033[b]	1.000	11.000
	Roy's Largest Root	.003	.033[b]	1.000	11.000

Multivariate Testsa

Effect		Sig.	Partial Eta Squared
Alarm	Pillai's Trace	.000	.889

Drug	Wilks' Lambda	.000	.889
	Hotelling's Trace	.000	.889
	Roy's Largest Root	.000	.889
	Pillai's Trace	.000	.718
	Wilks' Lambda	.000	.718
	Hotelling's Trace	.000	.718
	Roy's Largest Root	.000	.718
Alarm * Drug	Pillai's Trace	.859	.003
	Wilks' Lambda	.859	.003
	Hotelling's Trace	.859	.003
	Roy's Largest Root	.859	.003

a. Design: Intercept

Within Subjects Design: Alarm + Drug + Alarm * Drug

b. Exact statistic

Mauchly's Test of Sphericity[a]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.
Alarm	1.000	.000	0	.
Drug	1.000	.000	0	.
Alarm * Drug	1.000	.000	0	.

Measure: MEASURE_1

Within Subjects Effect	Sig.	Epsilon[b]		
		Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Alarm	.	1.000	1.000	1.000
Drug	.	1.000	1.000	1.000
Alarm * Drug	.	1.000	1.000	1.000

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept

Within Subjects Design: Alarm + Drug + Alarm * Drug

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F
Alarm	Sphericity Assumed	1643.041	1	1643.041	88.105
	Greenhouse-Geisser	1643.041	1.000	1643.041	88.105
	Huynh-Feldt	1643.041	1.000	1643.041	88.105
	Lower-bound	1643.041	1.000	1643.041	88.105
Error(Alarm)	Sphericity Assumed	205.136	11	18.649	
	Greenhouse-Geisser	205.136	11.000	18.649	
	Huynh-Feldt	205.136	11.000	18.649	
	Lower-bound	205.136	11.000	18.649	
Drug	Sphericity Assumed	907.891	1	907.891	28.044
	Greenhouse-Geisser	907.891	1.000	907.891	28.044
	Huynh-Feldt	907.891	1.000	907.891	28.044
	Lower-bound	907.891	1.000	907.891	28.044
Error(Drug)	Sphericity Assumed	356.117	11	32.374	
	Greenhouse-Geisser	356.117	11.000	32.374	
	Huynh-Feldt	356.117	11.000	32.374	
	Lower-bound	356.117	11.000	32.374	
Alarm * Drug	Sphericity Assumed	1.048	1	1.048	.033
	Greenhouse-Geisser	1.048	1.000	1.048	.033
	Huynh-Feldt	1.048	1.000	1.048	.033
	Lower-bound	1.048	1.000	1.048	.033
Error(Alarm*Drug)	Sphericity Assumed	346.959	11	31.542	
	Greenhouse-Geisser	346.959	11.000	31.542	
	Huynh-Feldt	346.959	11.000	31.542	
	Lower-bound	346.959	11.000	31.542	

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Sig.	Partial Eta Squared
Alarm	Sphericity Assumed	.000	.889
	Greenhouse-Geisser	.000	.889
	Huynh-Feldt	.000	.889
	Lower-bound	.000	.889

Error(Alarm)	Sphericity Assumed		
	Greenhouse-Geisser		
	Huynh-Feldt		
	Lower-bound		
Drug	Sphericity Assumed	.000	.718
	Greenhouse-Geisser	.000	.718
	Huynh-Feldt	.000	.718
	Lower-bound	.000	.718
Error(Drug)	Sphericity Assumed		
	Greenhouse-Geisser		
	Huynh-Feldt		
	Lower-bound		
Alarm * Drug	Sphericity Assumed	.859	.003
	Greenhouse-Geisser	.859	.003
	Huynh-Feldt	.859	.003
	Lower-bound	.859	.003
Error(Alarm*Drug)	Sphericity Assumed		
	Greenhouse-Geisser		
	Huynh-Feldt		
	Lower-bound		

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	Alarm	Drug	Type III Sum of Squares	df	Mean Square	F
Alarm	Linear		1643.041	1	1643.041	88.105
Error(Alarm)	Linear		205.136	11	18.649	
Drug		Linear	907.891	1	907.891	28.044
Error(Drug)		Linear	356.117	11	32.374	
Alarm * Drug	Linear	Linear	1.048	1	1.048	.033
Error(Alarm*Drug)	Linear	Linear	346.959	11	31.542	

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	Alarm	Sig.	Partial Eta Squared
Alarm	Linear	.000	.889
Error(Alarm)	Linear		

Drug		.000	.718
Error(Drug)			
Alarm * Drug	Linear	.859	.003
Error(Alarm*Drug)	Linear		

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	149540.354	1	149540.354	428.117	.000	.975
Error	3842.278	11	349.298			

Estimated Marginal Means

1. Alarm

Estimates

Measure: MEASURE_1

Alarm	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	49.965	2.114	45.313	54.618
2	61.667	3.296	54.412	68.921

Pairwise Comparisons

Measure: MEASURE_1

(I) Alarm	(J) Alarm	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	-11.701*	1.247	.000	-14.445	-8.957
2	1	11.701*	1.247	.000	8.957	14.445

Based on estimated marginal means

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

b. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared

Pillai's trace	.889	88.105[a]	1.000	11.000	.000	.889
Wilks' lambda	.111	88.105[a]	1.000	11.000	.000	.889
Hotelling's trace	8.010	88.105[a]	1.000	11.000	.000	.889
Roy's largest root	8.010	88.105[a]	1.000	11.000	.000	.889

Each F tests the multivariate effect of Alarm. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

2. Drug

Estimates

Measure: MEASURE_1

Drug	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	51.467	2.385	46.218	56.716
2	60.165	3.196	53.131	67.200

Pairwise Comparisons

Measure: MEASURE_1

(I) Drug	(J) Drug	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	-8.698*	1.643	.000	-12.313	-5.083
2	1	8.698*	1.643	.000	5.083	12.313

Based on estimated marginal means

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

b. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared
Pillai's trace	.718	28.044[a]	1.000	11.000	.000	.718
Wilks' lambda	.282	28.044[a]	1.000	11.000	.000	.718
Hotelling's trace	2.549	28.044[a]	1.000	11.000	.000	.718
Roy's largest root	2.549	28.044[a]	1.000	11.000	.000	.718

Each F tests the multivariate effect of Drug. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

Correlations

Correlations

		ISI
ISI	Pearson Correlation	1
	Sig. (2-tailed)	
	N	12
lownodrug11	Pearson Correlation	-.218
	Sig. (2-tailed)	.496
	N	12
lowdrug12	Pearson Correlation	-.410
	Sig. (2-tailed)	.185
	N	12
highnodrug21	Pearson Correlation	-.330
	Sig. (2-tailed)	.296
	N	12
highdrug22	Pearson Correlation	-.533
	Sig. (2-tailed)	.075
	N	12

**. Correlation is significant at the 0.01 level (2-tailed).

Regression

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	highdrug22[b]	.	Enter

a. Dependent Variable: ISI

b. All requested variables entered.

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.533[a]	.284	.212	3.50661

a. Predictors: (Constant), highdrug22

Mann-Whitney Test

Ranks

	Filter	N	Mean Rank	Sum of Ranks
lownodrug11	.00	43	27.93	1201.00
	1.00	12	28.25	339.00
	Total	55		
lowdrug12	.00	43	25.77	1108.00
	1.00	12	36.00	432.00
	Total	55		
highnodrug21	.00	44	30.32	1334.00
	1.00	12	21.83	262.00
	Total	56		
highdrug22	.00	44	27.69	1218.50
	1.00	12	31.46	377.50
	Total	56		

Test Statistics[a]

	lownodrug11	lowdrug12	highnodrug21	highdrug22
Mann-Whitney U	255.000	162.000	184.000	228.500
Wilcoxon W	1201.000	1108.000	262.000	1218.500
Z	-.062	-1.984	-1.608	-.715
Asymp. Sig. (2-tailed)	.950	.047	.108	.475

a. Grouping Variable: Filter

T-Test

Paired Samples Test

		Paired Differences				
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference	
					Lower	Upper
Pair 1	First_night - Fourth_night	1.23284	14.40438	4.15819	-7.91927	10.38494
Pair 2	Second_night - Third_night	1.08916	13.74292	3.96724	-7.64268	9.82099

Paired Samples Test

	t	df	Sig. (2-tailed)

Pair 1	First_night - Fourth_night	.296	11	.772
Pair 2	Second_night - Third_night	.275	11	.789

STUDY 2

Odds Ratios

Sex * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
Sex	Female	24	15	39
	Male	43	26	69
Total		67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.006[a]	1	.936		
Continuity Correction[b]	.000	1	1.000		
Likelihood Ratio	.006	1	.936		
Fisher's Exact Test				1.000	.548
Linear-by-Linear Association	.006	1	.936		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 14.81.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for Sex (Female / Male)	.967	.431	2.171
For cohort drugusers = No drugs detected	.987	.725	1.344
For cohort drugusers = Drugs detected	1.021	.619	1.682
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	.006	1	.936
Mantel-Haenszel	.016	1	.900

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate	.967
ln(Estimate)	-.033
Std. Error of ln(Estimate)	.412

Asymp. Sig. (2-sided)			.936
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.431
		Upper Bound	2.171
	ln(Common Odds Ratio)	Lower Bound	-.841
		Upper Bound	.775

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

age1 * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
age1	Other ages	45	18	63
	30-60 years	22	23	45
Total		67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.663[a]	1	.017		
Continuity Correction[b]	4.746	1	.029		
Likelihood Ratio	5.656	1	.017		
Fisher's Exact Test				.026	.015
Linear-by-Linear Association	5.610	1	.018		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 17.08.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for age1 (Other ages / 30-60 years)	2.614	1.174	5.818
For cohort drugusers = No drugs detected	1.461	1.043	2.047
For cohort drugusers = Drugs detected	.559	.345	.907
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	5.663	1	.017
Mantel-Haenszel	4.702	1	.030

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.614
ln(Estimate)			.961
Std. Error of ln(Estimate)			.408
Asymp. Sig. (2-sided)			.019
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.174
		Upper Bound	5.818
	ln(Common Odds Ratio)	Lower Bound	.160
		Upper Bound	1.761

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

magesex * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
magesex	Male other ages	24	9	33
	Male 30-60 years	19	17	36
Total		43	26	69

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.918[a]	1	.088		
Continuity Correction[b]	2.130	1	.144		
Likelihood Ratio	2.954	1	.086		
Fisher's Exact Test				.135	.072

Linear-by-Linear Association	2.876	1	.090		
N of Valid Cases	69				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 12.43.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for magesex (Male other ages / Male 30-60 years)	2.386	.871	6.534
For cohort drugusers = No drugs detected	1.378	.949	2.001
For cohort drugusers = Drugs detected	.578	.300	1.112
N of Valid Cases	69		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	2.918	1	.088
Mantel-Haenszel	2.099	1	.147

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.386
ln(Estimate)			.870
Std. Error of ln(Estimate)			.514
Asymp. Sig. (2-sided)			.091
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.871
		Upper Bound	6.534
	ln(Common Odds Ratio)	Lower Bound	-.138
		Upper Bound	1.877

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

fagesex * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
fagesex	Female other ages	21	9	30
	Female 30-60 years	3	6	9
Total		24	15	39

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.933[a]	1	.047		
Continuity Correction[b]	2.536	1	.111		
Likelihood Ratio	3.861	1	.049		
Fisher's Exact Test				.063	.057
Linear-by-Linear Association	3.832	1	.050		
N of Valid Cases	39				

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.46.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for fagesex (Female other ages / Female 30-60 years)	4.667	.951	22.901
For cohort drugusers = No drugs detected	2.100	.810	5.447
For cohort drugusers = Drugs detected	.450	.220	.921
N of Valid Cases	39		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.932	1	.047
Mantel-Haenszel	2.471	1	.116

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			4.667
ln(Estimate)			1.540
Std. Error of ln(Estimate)			.812
Asymp. Sig. (2-sided)			.058
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.951
		Upper Bound	22.901
	ln(Common Odds Ratio)	Lower Bound	-.050
		Upper Bound	3.131

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

location * drugusers

Crosstab

Count

	drugusers		Total
	No drugs detected	Drugs detected	

location	Elsewhere in dwelling	23	6	29
	In RFO at fire start	37	26	63
Total		60	32	92

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.708[a]	1	.054		
Continuity Correction[b]	2.856	1	.091		
Likelihood Ratio	3.905	1	.048		
Fisher's Exact Test				.063	.043
Linear-by-Linear Association	3.668	1	.055		
N of Valid Cases	92				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 10.09.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for location (Elsewhere in dwelling / In RFO at fire start)	2.694	.963	7.538
For cohort drugusers = No drugs detected	1.350	1.022	1.784
For cohort drugusers = Drugs detected	.501	.232	1.084
N of Valid Cases	92		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.

Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.708	1	.054
Mantel-Haenszel	2.825	1	.093

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.694
ln(Estimate)			.991
Std. Error of ln(Estimate)			.525
Asymp. Sig. (2-sided)			.059
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.963
		Upper Bound	7.538
	ln(Common Odds Ratio)	Lower Bound	-.038
		Upper Bound	2.020

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

sleepwakefinal * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
sleepwakefinal	Asleep	29	14	43
	Awake	38	27	65
Total		67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.886[a]	1	.347		
Continuity Correction[b]	.546	1	.460		
Likelihood Ratio	.894	1	.345		
Fisher's Exact Test				.420	.231
Linear-by-Linear Association	.878	1	.349		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 16.32.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for sleepwakefinal (Asleep / Awake)	1.472	.657	3.296
For cohort drugusers = No drugs detected	1.154	.862	1.544
For cohort drugusers = Drugs detected	.784	.467	1.316
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	.886	1	.347
Mantel-Haenszel	.541	1	.462

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.472
ln(Estimate)			.386
Std. Error of ln(Estimate)			.411
Asymp. Sig. (2-sided)			.348
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.657
		Upper Bound	3.296
	ln(Common Odds Ratio)	Lower Bound	-.420
		Upper Bound	1.193

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

npeople * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
npeople	Not alone	20	7	27
	Alone	47	34	81
Total		67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.215[a]	1	.137		
Continuity Correction[b]	1.586	1	.208		
Likelihood Ratio	2.301	1	.129		
Fisher's Exact Test				.172	.103
Linear-by-Linear Association	2.194	1	.139		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 10.25.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for npeople (Not alone / Alone)	2.067	.786	5.437
For cohort drugusers = No drugs detected	1.277	.955	1.706

For cohort drugusers = Drugs detected	.618	.311	1.228
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	2.215	1	.137
Mantel-Haenszel	1.571	1	.210

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.067
ln(Estimate)			.726
Std. Error of ln(Estimate)			.494
Asymp. Sig. (2-sided)			.141
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.786
		Upper Bound	5.437
	ln(Common Odds Ratio)	Lower Bound	-.241
		Upper Bound	1.693

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

Smoking * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
Smoking	No smoking materials	38	15	53
	Smoking materials	29	26	55
Total		67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.125[a]	1	.042		
Continuity Correction[b]	3.358	1	.067		
Likelihood Ratio	4.163	1	.041		
Fisher's Exact Test				.049	.033
Linear-by-Linear Association	4.086	1	.043		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 20.12.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper

Odds Ratio for Smoking (No smoking materials / Smoking materials)	2.271	1.022	5.047
For cohort drugusers = No drugs detected	1.360	1.005	1.839
For cohort drugusers = Drugs detected	.599	.359	.998
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	4.125	1	.042
Mantel-Haenszel	3.327	1	.068

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.271
ln(Estimate)			.820
Std. Error of ln(Estimate)			.407
Asymp. Sig. (2-sided)			.044
Asymp. 95% Confidence	Common Odds Ratio	Lower Bound	1.022

Interval	ln(Common Odds Ratio)	Upper Bound	5.047
		Lower Bound	.022
		Upper Bound	1.619

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

apresence * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
apresence	No smoke alarm	25	11	36
	Smoke alarm present	25	20	45
Total		50	31	81

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.633[a]	1	.201		
Continuity Correction[b]	1.098	1	.295		
Likelihood Ratio	1.649	1	.199		
Fisher's Exact Test				.253	.147
Linear-by-Linear Association	1.613	1	.204		
N of Valid Cases	81				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 13.78.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for apresence (No smoke alarm / Smoke alarm present)	1.818	.724	4.568
For cohort drugusers = No drugs detected	1.250	.890	1.755
For cohort drugusers = Drugs detected	.688	.381	1.241
N of Valid Cases	81		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	1.633	1	.201
Mantel-Haenszel	1.085	1	.298

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.818
ln(Estimate)			.598
Std. Error of ln(Estimate)			.470
Asymp. Sig. (2-sided)			.203
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.724
		Upper Bound	4.568
	ln(Common Odds Ratio)	Lower Bound	-.323
		Upper Bound	1.519

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

aoperation * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
aoperation	All other possibilities	43	23	66
	Smoke alarm active	7	8	15
Total		50	31	81

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.768[a]	1	.184		
Continuity Correction[b]	1.072	1	.301		
Likelihood Ratio	1.725	1	.189		
Fisher's Exact Test				.242	.150
Linear-by-Linear Association	1.746	1	.186		

N of Valid Cases	81				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 5.74.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for aoperation (All other possibilities / Smoke alarm active)	2.137	.688	6.639
For cohort drugusers = No drugs detected	1.396	.790	2.466
For cohort drugusers = Drugs detected	.653	.367	1.163
N of Valid Cases	81		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	1.768	1	.184
Mantel-Haenszel	1.059	1	.304

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.137
ln(Estimate)			.759
Std. Error of ln(Estimate)			.578
Asymp. Sig. (2-sided)			.189
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.688
		Upper Bound	6.639
	ln(Common Odds Ratio)	Lower Bound	-.374
		Upper Bound	1.893

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

escape1 * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
escape1	No conditions preventing escape	49	23	72
	Conditions preventing escape	18	18	36

Total	67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.322[a]	1	.068		
Continuity Correction[b]	2.600	1	.107		
Likelihood Ratio	3.283	1	.070		
Fisher's Exact Test				.092	.054
Linear-by-Linear Association	3.291	1	.070		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 13.67.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for escape1 (No conditions preventing escape / Conditions preventing escape)	2.130	.938	4.837
For cohort drugusers = No drugs detected	1.361	.947	1.957
For cohort drugusers = Drugs detected	.639	.400	1.022
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.322	1	.068
Mantel-Haenszel	2.576	1	.109

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.130
ln(Estimate)			.756
Std. Error of ln(Estimate)			.418
Asymp. Sig. (2-sided)			.071
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.938
		Upper Bound	4.837
	ln(Common Odds Ratio)	Lower Bound	-.064
		Upper Bound	1.576

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

alc * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
alc	No alcohol intake	30	14	44
	Positive alcohol intake	36	26	62
Total		66	40	106

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.121[a]	1	.290		
Continuity Correction[b]	.732	1	.392		
Likelihood Ratio	1.131	1	.288		
Fisher's Exact Test				.316	.196
Linear-by-Linear Association	1.111	1	.292		
N of Valid Cases	106				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 16.60.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for alc (No alcohol intake / Positive alcohol intake)	1.548	.688	3.481
For cohort drugusers = No drugs detected	1.174	.877	1.573
For cohort drugusers = Drugs detected	.759	.450	1.279
N of Valid Cases	106		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	1.121	1	.290
Mantel-Haenszel	.725	1	.395

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.548
ln(Estimate)			.437
Std. Error of ln(Estimate)			.414
Asymp. Sig. (2-sided)			.291
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.688
		Upper Bound	3.481
	ln(Common Odds Ratio)	Lower Bound	-.374
		Upper Bound	1.247

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

mental.illness * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
mental.illness	No	31	9	40
	Yes	20	30	50
Total		51	39	90

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	12.726[a]	1	.000		
Continuity Correction[b]	11.245	1	.001		
Likelihood Ratio	13.207	1	.000		
Fisher's Exact Test				.001	.000
Linear-by-Linear Association	12.585	1	.000		
N of Valid Cases	90				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 17.33.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for mental.illness (No / Yes)	5.167	2.032	13.137
For cohort drugusers = No drugs detected	1.938	1.327	2.828
For cohort drugusers = Drugs detected	.375	.202	.696

N of Valid Cases	90		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	12.726	1	.000
Mantel-Haenszel	11.120	1	.001

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			5.167
ln(Estimate)			1.642
Std. Error of ln(Estimate)			.476
Asymp. Sig. (2-sided)			.001
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	2.032
		Upper Bound	13.137
	ln(Common Odds Ratio)	Lower Bound	.709
		Upper Bound	2.575

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

physical.illness * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
physical.illness	No	28	8	36
	Yes	38	33	71
Total		66	41	107

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.947[a]	1	.015		
Continuity Correction[b]	4.965	1	.026		
Likelihood Ratio	6.225	1	.013		
Fisher's Exact Test				.020	.012
Linear-by-Linear Association	5.891	1	.015		
N of Valid Cases	107				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 13.79.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for physical.illness (No / Yes)	3.039	1.219	7.579

For cohort drugusers = No drugs detected	1.453	1.100	1.920
For cohort drugusers = Drugs detected	.478	.247	.925
N of Valid Cases	107		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	5.947	1	.015
Mantel-Haenszel	4.918	1	.027

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			3.039
ln(Estimate)			1.112
Std. Error of ln(Estimate)			.466
Asymp. Sig. (2-sided)			.017
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.219
		Upper Bound	7.579
	ln(Common Odds Ratio)	Lower Bound	.198

	Upper Bound	2.025

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

tody * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
tody	Resting hours (Other times)	44	23	67
	Waking hours (10:00 am - 10:00 pm)	18	15	33
Total		62	38	100

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.162[a]	1	.281		
Continuity Correction[b]	.737	1	.390		
Likelihood Ratio	1.151	1	.283		
Fisher's Exact Test				.381	.195
Linear-by-Linear Association	1.150	1	.284		
N of Valid Cases	100				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 12.54.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for tody (Resting hours (Other times) / Waking hours (10:00 am - 10:00 pm))	1.594	.681	3.733
For cohort drugusers = No drugs detected	1.204	.843	1.719
For cohort drugusers = Drugs detected	.755	.458	1.244
N of Valid Cases	100		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	1.162	1	.281
Mantel-Haenszel	.730	1	.393

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.594
ln(Estimate)			.466
Std. Error of ln(Estimate)			.434
Asymp. Sig. (2-sided)			.283
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.681
		Upper Bound	3.733
	ln(Common Odds Ratio)	Lower Bound	-.384
		Upper Bound	1.317

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

age8 * drugusers

Crosstab

Count

		drugusers		Total
		No drugs detected	Drugs detected	
age8	Other ages	43	22	65
	35-64 years	24	19	43
Total		67	41	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.175[a]	1	.278		
Continuity Correction[b]	.777	1	.378		
Likelihood Ratio	1.169	1	.280		
Fisher's Exact Test				.315	.189
Linear-by-Linear Association	1.164	1	.281		

N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 16.32.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for age8 (Other ages / 35-64 years)	1.547	.701	3.414
For cohort drugusers = No drugs detected	1.185	.863	1.629
For cohort drugusers = Drugs detected	.766	.475	1.235
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	1.175	1	.278
Mantel-Haenszel	.770	1	.380

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.547
ln(Estimate)			.437
Std. Error of ln(Estimate)			.404
Asymp. Sig. (2-sided)			.280
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.701
		Upper Bound	3.414
	ln(Common Odds Ratio)	Lower Bound	-.355
		Upper Bound	1.228

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

```
CROSSTABS
  /TABLES=Sex age1 magesex fagesex location sleepwakefinal npeople Smoking apresence aoperation escape1 alc
mental.illness physical.illness tody age8 BY sedateusers
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ RISK CMH(1)
  /CELLS=COUNT
  /COUNT ROUND CELL.
```

Crosstabs

Sex * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
Sex	Female	26	13	39
	Male	51	18	69
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.639[a]	1	.424		
Continuity Correction[b]	.334	1	.563		
Likelihood Ratio	.632	1	.427		
Fisher's Exact Test				.508	.280
Linear-by-Linear Association	.633	1	.426		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 11.19.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for Sex (Female / Male)	.706	.300	1.661
For cohort sedateusers = Not taking sedatives	.902	.694	1.173
For cohort sedateusers = Taking sedatives	1.278	.704	2.318
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	.639	1	.424
Mantel-Haenszel	.331	1	.565

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			.706
ln(Estimate)			-.348
Std. Error of ln(Estimate)			.437
Asymp. Sig. (2-sided)			.425
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.300
		Upper Bound	1.661
	ln(Common Odds Ratio)	Lower Bound	-1.204
		Upper Bound	.507

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

age1 * sedateusers

Crosstab

Count

	sedateusers		Total
	Not taking sedatives	Taking sedatives	

age1	Other ages	50	13	63
	30-60 years	27	18	45
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.810[a]	1	.028		
Continuity Correction[b]	3.910	1	.048		
Likelihood Ratio	4.772	1	.029		
Fisher's Exact Test				.033	.024
Linear-by-Linear Association	4.766	1	.029		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 12.92.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for age1 (Other ages / 30-60 years)	2.564	1.092	6.019
For cohort sedateusers = Not taking sedatives	1.323	1.010	1.732
For cohort sedateusers = Taking sedatives	.516	.283	.942
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	4.810	1	.028
Mantel-Haenszel	3.874	1	.049

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.564
ln(Estimate)			.942
Std. Error of ln(Estimate)			.435
Asymp. Sig. (2-sided)			.031
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.092
		Upper Bound	6.019
	ln(Common Odds Ratio)	Lower Bound	.088
		Upper Bound	1.795

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

magesex * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
magesex	Male other ages	27	6	33
	Male 30-60 years	24	12	36
Total		51	18	69

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.050[a]	1	.152		
Continuity Correction[b]	1.339	1	.247		
Likelihood Ratio	2.085	1	.149		
Fisher's Exact Test				.179	.123
Linear-by-Linear Association	2.020	1	.155		
N of Valid Cases	69				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 8.61.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for magesex (Male other ages / Male 30-60 years)	2.250	.731	6.921
For cohort sedateusers = Not taking sedatives	1.227	.926	1.626
For cohort sedateusers = Taking sedatives	.545	.231	1.287
N of Valid Cases	69		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	2.050	1	.152
Mantel-Haenszel	1.320	1	.251

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.250
ln(Estimate)			.811
Std. Error of ln(Estimate)			.573
Asymp. Sig. (2-sided)			.157
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.731
		Upper Bound	6.921
	ln(Common Odds Ratio)	Lower Bound	-.313
		Upper Bound	1.935

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

fagesex * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
fagesex	Female other ages	23	7	30
	Female 30-60 years	3	6	9
Total		26	13	39

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.850[a]	1	.016		
Continuity Correction[b]	4.063	1	.044		
Likelihood Ratio	5.594	1	.018		
Fisher's Exact Test				.039	.024
Linear-by-Linear Association	5.700	1	.017		
N of Valid Cases	39				

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.00.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for fagesex (Female other ages / Female 30-60 years)	6.571	1.296	33.330
For cohort sedateusers = Not taking sedatives	2.300	.894	5.916

For cohort sedateusers = Taking sedatives	.350	.158	.776
N of Valid Cases	39		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	5.850	1	.016
Mantel-Haenszel	3.958	1	.047

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			6.571
ln(Estimate)			1.883
Std. Error of ln(Estimate)			.828
Asymp. Sig. (2-sided)			.023
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.296
		Upper Bound	33.330
	ln(Common Odds Ratio)	Lower Bound	.259
		Upper Bound	3.506

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

location * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
location	Elsewhere in dwelling	25	4	29
	In RFO at fire start	43	20	63
Total		68	24	92

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.320[a]	1	.068		
Continuity Correction[b]	2.454	1	.117		
Likelihood Ratio	3.598	1	.058		
Fisher's Exact Test				.079	.055
Linear-by-Linear Association	3.283	1	.070		
N of Valid Cases	92				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 7.57.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper

Odds Ratio for location (Elsewhere in dwelling / In RFO at fire start)	2.907	.892	9.473
For cohort sedateusers = Not taking sedatives	1.263	1.011	1.578
For cohort sedateusers = Taking sedatives	.434	.163	1.157
N of Valid Cases	92		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.320	1	.068
Mantel-Haenszel	2.427	1	.119

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.907
ln(Estimate)			1.067
Std. Error of ln(Estimate)			.603
Asymp. Sig. (2-sided)			.077
Asymp. 95% Confidence	Common Odds Ratio	Lower Bound	.892

Interval	ln(Common Odds Ratio)	Upper Bound	9.473
		Lower Bound	-.114
		Upper Bound	2.248

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

sleepwakefinal * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
sleepwakefinal	Asleep	32	11	43
	Awake	45	20	65
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.340[a]	1	.560		
Continuity Correction[b]	.134	1	.714		
Likelihood Ratio	.343	1	.558		
Fisher's Exact Test				.665	.359
Linear-by-Linear Association	.337	1	.561		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 12.34.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for sleepwakefinal (Asleep / Awake)	1.293	.545	3.068
For cohort sedateusers = Not taking sedatives	1.075	.847	1.365
For cohort sedateusers = Taking sedatives	.831	.444	1.556
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	.340	1	.560
Mantel-Haenszel	.133	1	.716

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.293
ln(Estimate)			.257
Std. Error of ln(Estimate)			.441
Asymp. Sig. (2-sided)			.560
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.545
		Upper Bound	3.068
	ln(Common Odds Ratio)	Lower Bound	-.607
		Upper Bound	1.121

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

npeople * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
npeople	Not alone	23	4	27
	Alone	54	27	81
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.393[a]	1	.065		
Continuity Correction[b]	2.549	1	.110		
Likelihood Ratio	3.720	1	.054		
Fisher's Exact Test				.086	.051
Linear-by-Linear Association	3.362	1	.067		

N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 7.75.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for npeople (Not alone / Alone)	2.875	.903	9.152
For cohort sedateusers = Not taking sedatives	1.278	1.025	1.592
For cohort sedateusers = Taking sedatives	.444	.171	1.156
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.393	1	.065
Mantel-Haenszel	2.525	1	.112

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.875
ln(Estimate)			1.056
Std. Error of ln(Estimate)			.591
Asymp. Sig. (2-sided)			.074
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.903
		Upper Bound	9.152
	ln(Common Odds Ratio)	Lower Bound	-.102
		Upper Bound	2.214

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

Smoking * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
Smoking	No smoking materials	42	11	53
	Smoking materials	35	20	55
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.213[a]	1	.073		
Continuity Correction[b]	2.496	1	.114		
Likelihood Ratio	3.251	1	.071		

Fisher's Exact Test				.090	.057
Linear-by-Linear Association	3.184	1	.074		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 15.21.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for Smoking (No smoking materials / Smoking materials)	2.182	.922	5.165
For cohort sedateusers = Not taking sedatives	1.245	.977	1.587
For cohort sedateusers = Taking sedatives	.571	.303	1.073
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.213	1	.073
Mantel-Haenszel	2.473	1	.116

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.182
ln(Estimate)			.780
Std. Error of ln(Estimate)			.440
Asymp. Sig. (2-sided)			.076
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.922
		Upper Bound	5.165
	ln(Common Odds Ratio)	Lower Bound	-.082
		Upper Bound	1.642

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

apresence * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
apresence	No smoke alarm	29	7	36
	Smoke alarm present	30	15	45
Total		59	22	81

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.950[a]	1	.163		
Continuity Correction[b]	1.311	1	.252		
Likelihood Ratio	1.992	1	.158		
Fisher's Exact Test				.211	.126
Linear-by-Linear Association	1.926	1	.165		
N of Valid Cases	81				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 9.78.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for apresence (No smoke alarm / Smoke alarm present)	2.071	.738	5.815
For cohort sedateusers = Not taking sedatives	1.208	.930	1.570
For cohort sedateusers = Taking sedatives	.583	.267	1.276
N of Valid Cases	81		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	1.950	1	.163
Mantel-Haenszel	1.295	1	.255

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.071
ln(Estimate)			.728
Std. Error of ln(Estimate)			.527
Asymp. Sig. (2-sided)			.167
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.738
		Upper Bound	5.815
	ln(Common Odds Ratio)	Lower Bound	-.304
		Upper Bound	1.760

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

aoperation * sedateusers

Crosstab

Count

	sedateusers		Total
	Not taking sedatives	Taking sedatives	

aoperation	All other possibilities	51	15	66
	Smoke alarm active	8	7	15
Total		59	22	81

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.541[a]	1	.060		
Continuity Correction[b]	2.434	1	.119		
Likelihood Ratio	3.271	1	.071		
Fisher's Exact Test				.104	.063
Linear-by-Linear Association	3.497	1	.061		
N of Valid Cases	81				

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 4.07.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for aoperation (All other possibilities / Smoke alarm active)	2.975	.927	9.551
For cohort sedateusers = Not taking sedatives	1.449	.887	2.368
For cohort sedateusers = Taking sedatives	.487	.242	.981
N of Valid Cases	81		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.

Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	3.541	1	.060
Mantel-Haenszel	2.404	1	.121

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.975
ln(Estimate)			1.090
Std. Error of ln(Estimate)			.595
Asymp. Sig. (2-sided)			.067
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.927
		Upper Bound	9.551
	ln(Common Odds Ratio)	Lower Bound	-.076
		Upper Bound	2.257

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

escape1 * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
escape1	No conditions preventing escape	53	19	72
	Conditions preventing escape	24	12	36
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.566[a]	1	.452		
Continuity Correction[b]	.277	1	.599		
Likelihood Ratio	.558	1	.455		
Fisher's Exact Test				.502	.297
Linear-by-Linear Association	.560	1	.454		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 10.33.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for escape1 (No conditions preventing escape / Conditions preventing escape)	1.395	.585	3.325
For cohort sedateusers = Not taking sedatives	1.104	.844	1.445
For cohort sedateusers = Taking sedatives	.792	.434	1.445

N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	.566	1	.452
Mantel-Haenszel	.275	1	.600

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.395
ln(Estimate)			.333
Std. Error of ln(Estimate)			.443
Asymp. Sig. (2-sided)			.453
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.585
		Upper Bound	3.325
	ln(Common Odds Ratio)	Lower Bound	-.536
		Upper Bound	1.202

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

alc * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
alc	No alcohol intake	35	9	44
	Positive alcohol intake	41	21	62
Total		76	30	106

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.283[a]	1	.131		
Continuity Correction[b]	1.670	1	.196		
Likelihood Ratio	2.340	1	.126		
Fisher's Exact Test				.189	.097
Linear-by-Linear Association	2.261	1	.133		
N of Valid Cases	106				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 12.45.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for alc (No alcohol intake / Positive alcohol intake)	1.992	.808	4.908

For cohort sedateusers = Not taking sedatives	1.203	.953	1.518
For cohort sedateusers = Taking sedatives	.604	.306	1.190
N of Valid Cases	106		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	2.283	1	.131
Mantel-Haenszel	1.654	1	.198

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			1.992
ln(Estimate)			.689
Std. Error of ln(Estimate)			.460
Asymp. Sig. (2-sided)			.134
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	.808
		Upper Bound	4.908
	ln(Common Odds Ratio)	Lower Bound	-.213

Upper Bound	1.591

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

mental.illness * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
mental.illness	No	35	5	40
	Yes	25	25	50
Total		60	30	90

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	14.063[a]	1	.000		
Continuity Correction[b]	12.426	1	.000		
Likelihood Ratio	15.116	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	13.906	1	.000		
N of Valid Cases	90				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 13.33.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for mental.illness (No / Yes)	7.000	2.356	20.794
For cohort sedateusers = Not taking sedatives	1.750	1.295	2.364
For cohort sedateusers = Taking sedatives	.250	.105	.594
N of Valid Cases	90		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	14.063	1	.000
Mantel-Haenszel	12.288	1	.000

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate	7.000
ln(Estimate)	1.946
Std. Error of ln(Estimate)	.555

Asymp. Sig. (2-sided)			.000
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	2.356
		Upper Bound	20.794
	ln(Common Odds Ratio)	Lower Bound	.857
		Upper Bound	3.035

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

physical.illness * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
physical.illness	No	31	5	36
	Yes	45	26	71
Total		76	31	107

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.998[a]	1	.014		
Continuity Correction[b]	4.944	1	.026		
Likelihood Ratio	6.515	1	.011		
Fisher's Exact Test				.015	.011
Linear-by-Linear Association	5.942	1	.015		
N of Valid Cases	107				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 10.43.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for physical.illness (No / Yes)	3.582	1.240	10.348
For cohort sedateusers = Not taking sedatives	1.359	1.090	1.693
For cohort sedateusers = Taking sedatives	.379	.159	.904
N of Valid Cases	107		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	5.998	1	.014
Mantel-Haenszel	4.898	1	.027

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			3.582
ln(Estimate)			1.276
Std. Error of ln(Estimate)			.541
Asymp. Sig. (2-sided)			.018
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.240
		Upper Bound	10.348
	ln(Common Odds Ratio)	Lower Bound	.215
		Upper Bound	2.337

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

tody * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
tody	Resting hours (Other times)	53	14	67
	Waking hours (10:00 am - 10:00 pm)	19	14	33
Total		72	28	100

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.083[a]	1	.024		
Continuity Correction[b]	4.071	1	.044		
Likelihood Ratio	4.919	1	.027		
Fisher's Exact Test				.033	.023

Linear-by-Linear Association	5.032	1	.025		
N of Valid Cases	100				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 9.24.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for tody (Resting hours (Other times) / Waking hours (10:00 am - 10:00 pm))	2.789	1.126	6.912
For cohort sedateusers = Not taking sedatives	1.374	1.000	1.888
For cohort sedateusers = Taking sedatives	.493	.267	.909
N of Valid Cases	100		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	5.083	1	.024
Mantel-Haenszel	4.031	1	.045

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.789
ln(Estimate)			1.026
Std. Error of ln(Estimate)			.463
Asymp. Sig. (2-sided)			.027
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.126
		Upper Bound	6.912
	ln(Common Odds Ratio)	Lower Bound	.118
		Upper Bound	1.933

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

age8 * sedateusers

Crosstab

Count

		sedateusers		Total
		Not taking sedatives	Taking sedatives	
age8	Other ages	51	14	65
	35-64 years	26	17	43
Total		77	31	108

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.096[a]	1	.043		
Continuity Correction[b]	3.263	1	.071		
Likelihood Ratio	4.044	1	.044		
Fisher's Exact Test				.052	.036
Linear-by-Linear Association	4.058	1	.044		
N of Valid Cases	108				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 12.34.

b. Computed only for a 2x2 table

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for age8 (Other ages / 35-64 years)	2.382	1.017	5.576
For cohort sedateusers = Not taking sedatives	1.298	.987	1.705
For cohort sedateusers = Taking sedatives	.545	.301	.986
N of Valid Cases	108		

Tests of Homogeneity of the Odds Ratio

	Chi-Squared	df	Asymp. Sig. (2-sided)
Breslow-Day	.000	0	.
Tarone's	.000	0	.

Tests of Conditional Independence

	Chi-Squared	df	Asymp. Sig. (2-sided)
Cochran's	4.096	1	.043
Mantel-Haenszel	3.233	1	.072

Under the conditional independence assumption, Cochran's statistic is asymptotically distributed as a 1 df chi-squared distribution, only if the number of strata is fixed, while the Mantel-Haenszel statistic is always asymptotically distributed as a 1 df chi-squared distribution. Note that the continuity correction is removed from the Mantel-Haenszel statistic when the sum of the differences between the observed and the expected is 0.

Mantel-Haenszel Common Odds Ratio Estimate

Estimate			2.382
ln(Estimate)			.868
Std. Error of ln(Estimate)			.434
Asymp. Sig. (2-sided)			.046
Asymp. 95% Confidence Interval	Common Odds Ratio	Lower Bound	1.017
		Upper Bound	5.576
	ln(Common Odds Ratio)	Lower Bound	.017
		Upper Bound	1.718

The Mantel-Haenszel common odds ratio estimate is asymptotically normally distributed under the common odds ratio of 1.000 assumption. So is the natural log of the estimate.

Appendix R

Machine Learning Glossary

Cross validation

Cross validation involves testing a subset of the data against another independent subset multiple times (Hastie, et al., 2009). Many types of cross validation exist. The most prominent type and the method used in this analysis is 10 folds cross validation, which involves partitioning the data into 10 equal proportions, fitting the data to nine datasets and testing on the holdout or remaining dataset, then repeating this process 10 times and taking the average deviance from the 10 tests as an indication of model performance (Hastie, et al., 2009).

Regression Trees

Regression trees are a form of binary recursive partitioning (Breiman, Friedman, Olshen, & Stone, 1984). This means that each step in a decision tree is split into two groups, and this process is repeated, forming separate groups of data until some performance criteria is satisfied. This subdivision gives rise to a tree data structure.

Boosting

Boosting is a method for improving model accuracy that combines an ensemble or set of weak models in order to create a strong model (Elith, et al., 2008). There a many types of boosting methods. Friedman's (2001) stochastic gradient boosting is used for regression problems, usually in combination with regression trees. Each individual model in the set consists of a simple regression tree. The gradient boosting algorithm builds the final model in a stage-wise manner where the residuals or error from previous trees are fitted. This places emphasis on difficult to fit observations. The final model is formed by adding the weighted contribution of each tree (Friedman, 2001).

Parameter Selection in BRT

The key parameters for optimising BRTs are the tree complexity, learning rate, and bag fraction (Elith, et al., 2008). The tree complexity refers to the interaction depth for each tree (Elith, et al., 2008). An interaction depth of one would be a main effects model. The learning rate, which is also known as the shrinkage parameter, regulates the amount of learning possible in each tree (Elith, et al., 2008). While the bag fraction specifies the amount of stochasticity or randomness in the modelling procedure (Elith, et al., 2008). For example, a bag fraction of 0.5 indicates at each iteration the models were built from a random sample of 50% of the raw data.

AUC

The AUC is a measure of discrimination and can be interpreted as the probability that a classifier will rank a randomly chosen positive instance (sedative detected) higher than a negative instance (sedative not detected) that is randomly chosen (Hanley & McNeil, 1982). AUC ranges from 0 to 1, with values below 0.6 indicating a performance no better than random, values between 0.7 to 0.9 considered as useful, and values greater than 0.9 as excellent (Hanley & McNeil, 1982).

Deviance

Deviance is a goodness of fit measure. The residual deviance is equivalent to the residual sum of squares in traditional regression and indicates how much of the variation in sedative usage the model did not explain (Zuur, et al., 2009). A smaller residual deviance will result in a better model. The null deviance is analogous to the total sum of squares and signifies the total variation in sedative usage in the model (Zuur, et al., 2009). The proportion of deviance explained (D = null deviance – residual deviance / null deviance) is a measure of overall model accuracy, analogous to the R^2 in linear regression (Yee & Mitchell, 1991).

An increase in deviance (y axis in Figure 11) will result in a decrease in predictive performance. The simplification method was run within a 10 fold cross validation procedure. This process yielded four variables for removal (Figure 11).

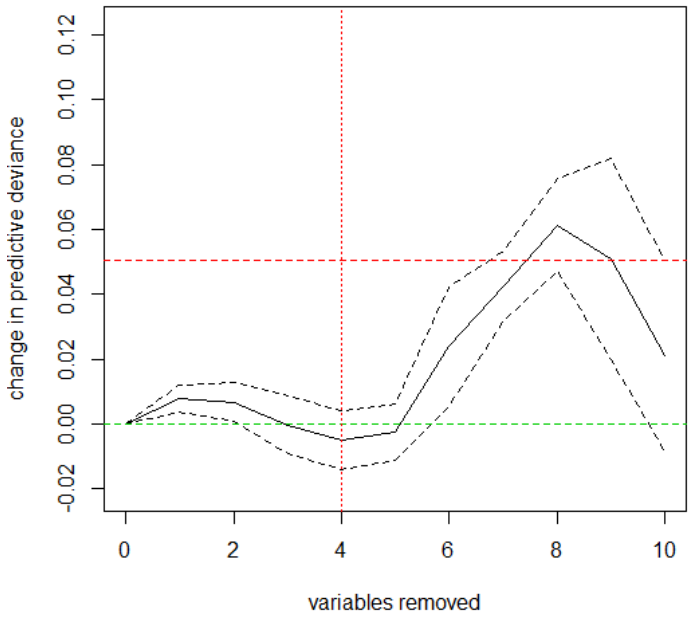


Figure 11. Change in deviance as variables are removed from the model. The solid line indicates the mean change in predictive deviance, and the dotted line one standard error, calculated over the 10 folds of the cross-validation. The optimal number of variables to drop is indicated by the vertical dotted line.

Appendix S

```
################ DATA ENTERED ##############################################
data <- read.table("c:/final.csv", header=TRUE, sep=",")

################ PACKAGES LOADED ###########################################
require(gbm)
require(dismo)

################ BRT MODEL FITTED – Figure 8 ###############################
gbmstep <- gbm.step(data=data, gbm.x = 3:14, gbm.y = 2,family = "bernoulli", tree.complexity
= 4, learning.rate = 0.001, bag.fraction = 0.50)

################ BRT MODEL SIMPLIFIED – Figure 11 ##########################
gbm.simp <- gbm.simplify(gbmstep, n.drops = 4)

################ INTERACTIONS OF SIMPLIFIED MODEL TESTED – Table 11 #####
find.int <- gbm.interactions(BRTsimp)
find.int$interactions

################ SIMPLIFIED BRT MODEL FITTED – Figure 8 ##################
BRTsimp <- gbm.step(data=data,gbm.x=gbm.simp$pred.list[[4]], gbm.y=2,tree.complexity=4,
learning.rate=0.001)

################ TWO WAY PARTIAL PLOTS – Figure 9 ######################
gbm.plot(BRTsimp, variable.no=0, n.plots=15, write.title = FALSE, rug=TRUE,
plot.layout=c(3,4))

################ THREE WAY PARTIAL PLOT – Figure 10 ####################
gbm.perspec(BRTsimp, 2, 3, z.range=c(0,.6), theta=310, phi=20,col="light blue")
```

Appendix T

Illicit drug
Pain-killers/analgesics*
Tranquillisers/sleeping pills*
Steroids*
Meth/amphetamines*
Cannabis
Heroin
Methadone or buprenorphine**
Other opiates (opioids)*
Cocaine
Hallucinogens
Ecstasy
Ketamine
GHB

Note: *used for non-medical purposes; ** non-maintenance program.

Appendix U

VICTORIA UNIVERSITY
HUMAN RESEARCH ETHICS COMMITTEE

Application for Approval of Project Involving Human Participants in Victoria University

REGISTER NUMBER (*office use only*) : **HRETH** _____

INFORMATION FOR APPLICANTS

1. Applicants are advised to follow the *Guidelines for Applications* prior to submitting *Application for Approval*. Applicants are to forward **a)** Twelve (12) hard copy applications (including one original copy)* with any accompanying documentation to your Faculty Ethics Officer and **b)** an electronic application to your Faculty Ethics Officer. **Note**: *Non Minimum Risk applications* may be forwarded directly to the Secretary, Victoria University Human Research Ethics Committee (researchethics@vu.edu.au).
2. A *Consent Form for Participants Involved in Research* template and *Information for Participants* template is also available on-line.
3. The above documents are located at: http://research.vu.edu.au/hrec.php

* *Applications to be considered at the Faculty of Business & Law need to submit one original hard copy application.*

YOU ARE REMINDED THAT THIS PROJECT MUST NOT COMMENCE WITHOUT PRIOR WRITTEN APPROVAL

FROM THE APPROPRIATE HUMAN RESEARCH ETHICS COMMITTEE.

Please Note:

- Ethics approval will not be finalised until electronic & hard copy applications and copies of all necessary materials have been received by the Secretary of the relevant Human Research Ethics Committee.
- This application form is included in the Human Research Register. If your project includes information of a commercial or patentable nature, this information should be sent separately and marked as confidential.
- If an institution other than Victoria University is to be involved in the project, please provide this information and evidence of ethics approval from the other institution with this application.
- Research projects undertaken by individuals who are not staff members of VU that require access to a cohort of VU staff or students for research must be 'sponsored' by a member of VU staff who will take responsibility for all interactions with the University and the HREC. A copy of the approved project and approval letter must be forwarded to the Ethics & Biosafety Administration Group.
- If sufficient space is not available on the form for your answer/s, please attach additional page/s.
- Ensure **all questions** are appropriately answered and the hardcopy application is **authorised** by appropriate staff (Applications will not be processed without the appropriate authorisation).
- To avoid unnecessary delays, please ensure your full application (both hard copies and soft copy application) has been received by the relevant Human Research Ethics Committee submission date. Refer to University/Faculty *Committee Meeting Dates* at http://research.vu.edu.au/hrec.php

University & Faculty Forwarding Details:

Victoria University Human Research Ethics	Faculty Human Research Ethics Contacts
Send electronic applications to: E-mail address: researchethics@vu.edu.au Hard copy applications to be delivered to: Ethics Secretary, Victoria University Human Research Ethics Committee Office for Research Victoria University PO Box 14428, Melbourne VIC 8001 **Or** deliver in person to the Ethics & Biosafety Administration Group located within the Office for Research at Building C, Room 302, Footscray Park campus.	Send electronic applications to: **Faculty of Arts Education & Human Development:** AEHDEthics@vu.edu.au **Faculty of Business & Law:** BLEthics@vu.edu.au **Faculty of Health Engineering & Science:** HESEthics@vu.edu.au Hard copy applications to be delivered: Faculty Ethics Officer *Nominated* Faculty Human Research Ethics Committee Office for Research Victoria University PO Box 14428, Melbourne VIC 8001 **Or** deliver in person to the Ethics & Biosafety Administration Group located within the Office for Research at Building C, Room 302, Footscray Park campus.
For Further Information: Web: http://research.vu.edu.au/hrec.php	**Telephone: 9919 4148 or your Faculty Ethics Officer**

I attach a proposal for a project involving human participants for the purposes specified on the attached sheets.

Note: The Human Research Ethics Committee normally grants approval for periods of up to two years, subject to annual review. Consideration will be given to granting approval for a longer period in certain circumstances. Applications for extension of approval should be lodged prior to expiry of existing approval.

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1. Project Title:

Drug use and human behaviour during fire emergencies

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2. Principal Investigator/s:

(Projects to be undertaken by students should list the Supervisor as the Principal Investigator)

Title	*First Name*	*Surname*	*School/Centre*	*Phone Number*	*Mobile Number*	*VU E-Mail Address*
Prof.	Dorothy	Bruck	Psychology/ CESAR	9919 2158	0428 139 88	Dorothy.Bruck@vu.edu.
Dr.	Michelle	Ball	Psychology/ CESAR	9919 2536		Michelle.Ball@vu.edu.a

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***3. (a)* Associate Investigator/s and/or Co-Investigator/s:**

(Please insert additional lines & information if there is more than one)

Title	*First Name*	*Surname*	*School/Centre*	*Phone Number*	*Mobile Number*	*E-Mail Address*
Dr.	Simon	Frenkel	Sleep Clinic			SimonFrenkel@hotmail.co

3. (b) VU Sponsor:
*(For a*pplications for research involving participants from individuals who are not staff members of VU. *Please refer to declaration page for further details and signature)*

Title	First Name	Surname	School/Centre	Phone Numb	Mobile Numb	VU E-Mail Addres

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***4.* Student Project**
(Please insert additional lines & information if required)

4.1. Is the application part of a student project? Yes ✓ No ☐

4.2. If YES, select the appropriate tick box:

PhD ✓ Masters by Research ☐ Honours ☐ Postgraduate Coursework ☐ Undergraduate (not honours) ☐

- Has this research project been approved by the Postgraduate Research Committee? Yes ☐ No ✓

Student details

Title	First Name	Surname	School/Centre	Student Number	Phone Number	VU E-Mail Address
Mr.	Chris	Lykiardopoulos	CESARE	61 3 9919 8036	0404 968 348	Chris.Lykiardopoulos@live.vu.edu.

- Is the student currently enrolled at Victoria University? Yes ✓ No ☐

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5. Type of Project:
(please select Yes or No to the following questions)

5.1. Type of Program

(a) Is application for a higher degree program? Yes ✓ No ☐

(b) Is this application for a pilot program of a higher degree? Yes ☐ No ✓

[If yes, please note that a second application will be required for the full program]

(c) Is application for an honours program of an undergraduate degree? Yes ☐ No ✓

*If yes, please indicate semester dates:*_____

(d) Other student project? Please specify _____

5.2. Funded Program

(a) Is application for a funded research program? Yes ☐ No ✓

*If yes, please indicate source of funding:*_____

_____

(b) Do you require ethical approval prior to funding being granted? Yes ☐ No ✓

If yes, ***attach*** *any necessary forms to be completed by the Ethics Committee and indicate* ***grant closing date****.*

*D*ate:_____

5.3. Intrusiveness of Project
(please select Yes or No to the following questions)

a) Uses physically intrusive techniques Yes ✓
No ☐

b) Causes discomfort in participants beyond normal levels of inconvenience Yes ✓
No ☐

c) Examines potentially sensitive or contentious areas Yes ☐
No ✓

d) Uses therapeutic techniques Yes ☐ No ✓

e) Seeks disclosure of information which may be prejudicial to participants Yes ☐
No ✓

f) Uses ionising radiation Yes ☐ No ✓

g) Uses of personal information obtained from a Commonwealth department or agency
Yes ☐ No ✓

I. If YES, and the project is <u>not</u> medical research, does the research meet the Guidelines under Section 95 of the Privacy Act 1988? Yes ☐ No ☐

II. If YES, and the project is medical research (including epidemiological research) does the research meet the Guidelines under Section 95A of the Privacy Act 1988? Yes ☐ No ☐

h)Clinical trial Yes ☐ No ✓
(A clinical trial is a study involving humans to find out whether an intervention, including treatments or diagnostic procedures, which it is believed may improve a person's health, actually does so. A clinical trial can involve testing a drug, a surgical or other therapeutic or preventive procedure, or a *therapeutic, preventive or diagnostic device or service. Any intervention, including so-called "natural" therapies and other forms of complementary medicine, can be tested in this way).*

i) Research focuses on Aboriginal and/or Torres Strait Islander Peoples
Yes ☐ No ✓

- If YES, does the project involve health research? Yes ☐ No ☐

j) Involves potentially vulnerable groups (eg children, people in dependent/unequal relationships, highly dependent on medical care, cognitive impairment or intellectual disability, may be involved in illegal activities)
Yes ☐ No ✓
If YES, please provide additional detail:
_____

k)Involves deception or covert observation Yes ☐ No ✓
If YES, please provide additional rationale: _____

Note: If you have ticked "YES" to any of the items g to k, please forward your ethics application to the Secretary, Victoria University Human Research Ethics Committee (VUHREC). Note that Faculty HREC submission deadlines differ to that of the VUHREC, and this may impact on your project's timelines.

6. Aim of project:

(In brief terms, state the aims and the expected benefits of the project in no more than 250 words)

The overall aim of the project will be to investigate the response of older adults who intermittently use hypnotics to various alarm signals. This will involve answering the following key questions. First, are users of hypnotics, particularly older adults, overrepresented in fire fatalities? Second, what is the relationship between hypnotic usage and other risk factors? Third, will an average smoke alarm (3100 Hz pure tone) wake an older adult user of hypnotics? Finally, will the new low frequency (520 Hz square wave) alarm be more effective at waking older adult users of hypnotics?

7. Plain language statement of project:

(Provide a brief summary of the project [not more than 2 pages] outlining the broad aims, background, key questions, research design/approach and the participants in the project. Include a theoretical background or context of the research. If there are multiple participant groups or interventions/phases, please specify relevant information for each. Please make sure implications associated with multiple groups/phases is addressed throughout the application. It is recognised that in some areas of research, it may be appropriate that this statement is repeated elsewhere in this application form, and that it may comprise part of your response to questions 6, 8, 15, 16 and 17. This section is to be stated in simple language and any terms or jargon must be accompanied by explanation).

Sleeping is a risk factor for death in a residential fire, even if a smoke alarm sounds (Ahrens, 2008). Research has shown that there are many groups within the population who are at a heightened risk of not waking to the standard high pitched (3100 Hz pure tone) smoke alarm signal at a pillow volume of 75 dBA [which is widely recommended, including in Australia (Australian Standard 1670.4, 2004)]. These groups include children (Bruck, 1999; Bruck & Bliss, 2000; Bruck, et al., 2004), those aged over 65 years (Bruck & Thomas, 2007a), people with hearing loss (Bruck & Thomas, 2007c), and the alcohol impaired (Ball & Bruck, 2004b; Bruck, et al., 2007).

Approximately 1 in 20 Australians have taken hypnotics (i.e., sleeping pills or sedatives) in the last two weeks and these were most likely to be benzodiazepines (Bz), such as temazepam (Australian Bureau of Statistics, 2010a). The purpose of hypnotics is to make sleepers less responsive to both external (e.g., alarms) and internal stimuli (e.g., worrying thoughts). Thus it is to be expected that people who use hypnotics will also be at higher risk of sleeping through a fire alarm in the event of an emergency; however, this has only been indirectly confirmed. There exists only one previous study that has investigated the effect of hypnotics on the ability to wake to a smoke alarm; however, this study was limited because participants did not have insomnia and did not normally take Bz, which may have resulted in higher auditory arousal thresholds. An auditory arousal threshold is the necessary sound level (in dBA) required to awaken an individual from sleep.

Users of hypnotics are significantly more likely to be aged over 65 years (Smith & Tett, 2009). Coupled with a higher risk of dying in a fire and other associated disadvantages with advanced age, older adults appears to be a particularly vulnerable demographic in a fire

emergency. The likelihood of dying in a fire increases with each decade over 60 compared to younger people (Hall, 2005).

Older adults who take hypnotics are also more likely to be suffering from insomnia (i.e., an inability to sleep). Prior research has often found no significant differences in arousal thresholds between people suffering from insomnia and normal sleepers (Johnson, et al., 1979; Mendelson, et al., 1986); however, this research has typically recruited younger adults experiencing sleep-onset (i.e., problems in falling asleep) difficulties. People suffering from sleep-maintenance (i.e., difficulties in staying asleep) problems, which are significantly more common in older adults (Webb & Campbell, 1980), may be more vulnerable to evening arousal than people suffering primarily from sleep-onset issues because of a predisposition to arousal when asleep.

Previous research has raised the possibility that an alarm of a different frequency (520 Hz square wave) may be more effective for waking sleeping individuals than the current high pitched (3100 Hz pure tone) alarm (Bruck & Thomas, 2007b). A pure tone is a single frequency, like striking one note on a piano, while a square wave carries a number of frequencies and is like playing several notes on a piano at once, as in playing a chord. The new low frequency alarm has been successfully tested in older adults (Bruck & Thomas, 2007a), but no tests have been conducted on users of hypnotics at any age.

The overall aim of the study will be to investigate the response of older adults who intermittently use hypnotics to various alarm signals. Previous research recruited participants who had not used hypnotics, which may have resulted in higher arousal thresholds. Therefore, the first objective will be to determine the extent to which hypnotic ingestion increases auditory arousal thresholds to the current smoke alarm signal in intermittent users of hypnotics. The second objective will be to determine whether the current standard alarm intensity level of 75 dBA at the pillow is adequate for waking intermittent users of hypnotics under the influence of their usual hypnotic. It is hypothesised that hypnotic ingestion will increase arousal thresholds above the standard alarm intensity. The third objective will be to compare arousal thresholds under the current (3100 Hz pure tone) smoke alarm and the 520 Hz square wave signal in both a hypnotic and non-hypnotic condition to determine if there are lower arousal thresholds to the 520 Hz square wave alarm compared to the current (3100 Hz pure tone) smoke alarm when under the influence of hypnotics. This will involve comparing auditory arousal thresholds in intermittent hypnotic users on and off their medication to the two different alarms. It is hypothesised that there will be lower arousal thresholds to the 520 Hz square wave alarm in the hypnotic condition. Previous research is mixed on the difference in arousal thresholds of people suffering from insomnia and normal sleepers. Therefore, a fourth objective will be to investigate the relationship between arousal thresholds and the severity of insomnia symptoms in intermittent hypnotic users, and to determine whether such a relationship may exist when they are on or off their medication, or both. It is hypothesised that as insomnia symptoms increase arousal thresholds will decrease.

An additional objective will be to compare the auditory arousal thresholds (for both types of alarm signals) of sleeping intermittent hypnotic users when they are subject to the peak concentration of their hypnotic with the auditory arousal thresholds of existing age and sex matched healthy controls where sleep stage was controlled (data from Bruck and Thomas 2007a). The existing data comes from the project “Reducing fire deaths in the aged: Optimising the smoke alarm signal”. Ethics approval for this study was obtained from the Human Research Ethics Committee in 2005. Data in this existing study was tested with the same signals under

conditions of electroencephalography (EEG) determined deep sleep (stages 3/ 4). EEG is a device that records electrical activity in the brain. This comparison will add methodological rigour to the study. It is hypothesised that healthy controls will have significantly lower arousal thresholds compared with intermittent hypnotic users.

In all cases participants' bedtimes and sleep and wake will be measured via actigraphy, which is a device, similar to a watch in appearance, used to measure arm movement and the alarm will be presented at peak hypnotic concentration.

Demonstrating the improved effectiveness of the new signal in another at-risk group (people taking sleeping medication) will add significant weight to the argument for changing the smoke alarm signal in residential buildings as well. Furthermore, highlighting the role of hypnotics and psychoactive drugs more broadly in emergency fire situations may prevent death and injury.

This project will involve two related experiments in the area of human behaviour in fire and emergency settings when under the influence of psychoactive drugs. The study described above will examine the specific effect of hypnotics and alarm signals on arousal thresholds. A second study examining coronial data on the likelihood of dying in a fire if under the influence of psychoactive drugs will also be conducted. Approval for this second study is covered under a previous application and has already been obtained.

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8. Nature of research, including methodology and a list of all procedures to be used on human participants. Please include a statistical power analysis statement if applicable.

Participants

The experimental group will be aged between 65 to 85 years of age and be intermittent users of prescription hypnotic medication (to be defined with the assistance of a Sleep Physician, Dr. Simon Frenkel), defined as normally taking hypnotics on between two to six nights per week. The assistance of a Sleep Physician will be of particular importance in excluding participants who are taking sedating drugs that are not specifically designed as hypnotics (e.g., anti-depressants). This will ensure participants are not taking other drugs that might impact their ability to wake to an alarm. A Sleep Physician will also ensure we only include participants who are taking commonly prescribed hypnotics so that the findings of the study are representative of typical hypnotic users. This age group has been selected because: (a) they are overrepresented in fire fatalities and as users of hypnotics; and (b) there is matching historical data for this age group.

Forty-five experimental participants are proposed, which will provide approximately 99% power for the key main effect comparisons assuming a medium effect size and alpha at 0.05 (Lenth, 2006). Experimental participants will be exposed to repeated measures; therefore the same participants will be tested under both the hypnotic ingestion and control (no hypnotic ingestion) conditions.

Participants will be excluded from the study if they:

- Are not aged between 65 to 85 years.

- Do not have a sufficient understanding of English to understand the risks and procedures of the study and also complete the questionnaires as we can not fund translations.
- Advise us by self-report that they have been diagnosed with serious neurological or psychiatric disorders.
- Are not intermittent users (2-6 nights per week) of a hypnotic/ sedative on our medication list to be determined by a Sleep Physician.
- Are regularly taking any sedative drug (other than hypnotics listed in the inclusion criteria).
- Undertake a free auditory examination and are not in the top 50% of the population of their age for hearing in both ears at frequencies from 500Hz to 4000Hz.

A recruitment interview (attached as Appendix A) has been developed and will include detailed information on the study and questions regarding participants' treatment plan, standard demographics, and subjective sleep history. The aim of the interview is primarily to inform participants of the study and apply the exclusion criteria to willing participants. The interview is expected to be between 5 and 10 minutes in duration.

Materials

Awakenings will occur via a computer program specifically designed for the current project therefore the participant will not be disturbed by having a stranger in the house and their sleep will not be monitored. This allows for a more naturalistic assessment of auditory arousal. The computer program will run on a small laptop to be placed in an adjacent room, which will be connected to a button placed within reach of the participants' beds, to ensure participants are easily able to signal when they have awoken.

Signal presentation will use a method of limits procedure adapted from Bonnet (1982). This involves presentation of a signal at a low volume (e.g., 35 dBA), which will increase by 5 dBA every 30 seconds until the participant wakes up and presses the button. There will be a silence of approximately 10 seconds between each signal presentation, which has demonstrated positive methodological properties (Bruck, et al., 2009). Participants' arousal thresholds will be the sound level (dBA) being presented at the time each participant presses the bedside button. Sounds will be presented via speakers attached to the laptop, and the sound level at the pillow will be calibrated using a sound level meter.

Sleep and wake will be measured via actigraphy, which is a device, similar to a watch in appearance, used to measure arm movement. An algorithm interprets the arm movement recorded via the actigraph as a proxy for sleep and wake. Actigraphy will be used to confirm bedtime and to have some objective record of participants' activity levels at the time of signal presentation. Participants will be instructed to wear the actigraph continuously at night for the duration of the study on their non-dominant wrist. The actigraph includes an event button, which the participants will be asked to press when taking a hypnotic. Use of the actigraph will comply with the current American Sleep Association (ASA) standards. For example, only validated scoring algorithms will be utilised.

An in-home questionnaire pack (attached as Appendix B) has also been developed and will include a two-week sleep diary and the Insomnia Severity Index (ISI). The ISI is a 7-item instrument designed to briefly evaluate and screen for insomnia in the general population

following criteria from the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV; American Psychiatric Association, 1994) and the International Classification of Sleep Disorders (ICSD; ICSD; American Sleep Disorders Association, 1997). The ISI provides a global index of the severity of insomnia, which can then be categorised into one of four different severity profiles: (1) no clinically significant insomnia; (2) sub-threshold insomnia; (3) clinical insomnia (moderate severity); and (4) clinical insomnia (severe). The ISI has been found to be a reliable and valid instrument to assess primary insomnia. The ISI will be used to determine if insomnia severity moderates arousal thresholds. The in-home interview is expected to be between 10 and 15 minutes in duration.

Procedure

The researcher will begin by conducting a short recruitment interview of no longer than 10 minutes with all available participants. In-home appointments will be arranged with participants who meet the study criteria. Participants will also be sent information and consent forms at this time. Shortly after the recruitment call, the researcher will communicate with each participant confirming the in-home appointment and their willingness to participate having read through the information and consent forms.

Prior to attending the in-home appointment, the researcher will determine the appropriate time for alarm presentation. Alarm presentation time will be individualised across participants. It will be calculated based upon each participant's typical bed time (from the recruitment interview), their prescribed hypnotic medication, and usual ingestion time. The goal will be for the alarm signal to be presented when the medication is at peak concentration and the participants are asleep. Alarm presentation will be at this same time on the hypnotic and hypnotic-free night for each condition to ensure consistency. The responses will be evaluated and decision rules will be created around the movement profiles yielded by the actigraphy to ensure data is not included where participants appear to be awake before the alarm sounds. This will be determined both subjectively (self-report) and objectively (actigraph movement profile).

Upon arrival at the participants' homes the researcher will again inform the participants of the details of the study and secure the signed consent forms. The participants will be briefed on all materials used in the study and given a set of instructions, requiring the participants to maintain their usual sleep routines and ensure other family members do not interfere (e.g., wake up the participant if they awake to the alarm) with the tests. Participants will then be given the auditory examination and will listen to both sounds that will be played to them, to ensure there are no confounding effects due to differential familiarity with the alarm sounds. Finally, participants will then be given the in-home questionnaire to complete.

Experimental design: The experiment will involve monitoring the participants' sleep patterns and arousal thresholds over 11 nights, modulating hypnotic intake (their usual hypnotic) and alarm presentation (the current 3100 Hz pure tone cf. the 520 Hz square wave) to ensure all independent variable combinations occur. Table 1 presents the enumerated design.

Table 1
Experimental design

Night	Day	Alarm	Hypnotic	Row
1	Mon	No alarm	No drug	A
2	Tue	Alarm 1	No drug	B
3	Wed	No alarm	Drug	C
4	Thu	Alarm 2	Drug	D
5	Fri	No alarm	No drug	E
6	Sat	No alarm	No drug	F
7	Sun	No alarm	Drug	G
8	Mon	Alarm 1	Drug	H
9	Tue	No alarm	No drug	I
10	Wed	Alarm 2	No drug	J
11	Thu	No alarm	Drug	K

Presentation of the two alarm signals (the current high pitched 3100 Hz pure tone signal and the 520 Hz square wave) will be counterbalanced (to avoid bias from presenting in only one order) and presented over four nights, one night for each treatment combination. Participants will be advised that the alarm will not be presented on Friday and Saturday to allow participants to enjoy irregular bedtimes on traditional leisure days. The alarm will also not be presented on Sunday to avoid any possible hang-over effects but participants will not be informed of this setting. To minimise priming effects (experience of the experiment that affects subsequent responses from the participant) participants will be told that the alarm may be presented between three to five times. In addition, the alarms will be presented in a pseudorandom arrangement, occurring on the second, fourth, eighth, and tenth night (rows B, D, H, and J respectively in Table 1).

Hypnotic intake will occur in blocks every third night, ensuring adequate wash-out time. There will be two blocks of two nights of hypnotic intake followed by a block of one night, totalling five nights. The hypnotic medication will be the participants' normal dose and type.

Participants will be required to alter their normal drug regimen to adhere to the above prescription plan. Participants will be made aware of this point prior to participation. This is less likely to be a cause for concern because we are only recruiting intermittent users of hypnotics who are less likely to be troubled by alterations to their treatment plans. Further efforts will be made to manage this risk and have been detailed later in the application.

9. Description of those techniques which are considered by the profession to be established and accepted. Please give details of support for their application.
(If, in the course of your research, procedures are significantly varied from those stated here, the Human Research Ethics Committee must be informed).

Signal presentation will use a method of limits procedure adapted from Bonnet (1982). This involves presentation of a signal at a low volume (e.g., 35 dBA), which will increase by 5 dBA every 30 seconds until the participant wakes up and presses the button. There will be a silence of approximately 10 seconds between each signal presentation, which has demonstrated positive methodological properties (Bruck, et al., 2009). Participants' arousal thresholds will be the sound level (dBA) being presented at the time each participant presses the bedside button. Sounds will be presented via speakers attached to the laptop, and the sound level at the pillow will be calibrated using a sound level meter.

Sleep and wake will be measured via actigraphy, which is a device, similar to a watch in appearance, used to measure arm movement. An algorithm interprets the arm movement recorded via the actigraph as a proxy for sleep and wake. Actigraphy will be used to confirm

bedtime and to have some objective record of participants' activity levels at the time of signal presentation. Participants will be instructed to wear the actigraph continuously at night for the duration of the study on their non-dominant wrist. The actigraph includes an event button, which the participants will be asked to press when taking a hypnotic. Use of the actigraph will comply with the current American Sleep Association (ASA) standards. For example, only validated scoring algorithms will be utilised.

10. Proposed start and end date of project:
(Note: new research projects may not commence prior to approval by the Human Research Ethics Committee).

Proposed start date:	(01/02/2011)	**Proposed end date:**	(01/02/2012)

11. Details of participants:

Name of Phase/Group	Group 1	Group 2 (Existing Data)	
Number of Participants	45	39	
Type	Hypnotic users	Healthy controls	
Age Range	65-85	65-85	

12. Source of participants
(specify for each group/phase if relevant), and means by which participants are to be recruited)

Group 1
Two possible recruitment methods have been canvassed. The first method will involve placing advertisements in local newspapers (an example of the advertisement is attached as Appendix F). The purpose of the study and incentive will be outlined in the advertisements. All participants will be offered the option of a one month free trial of a new, non-pharmaceutical insomnia treatment (normally valued at $99, supported by Philips Respironics) as compensation. Participants will be invited to contact a toll-free number, which will divert to an answering machine. A brief message will thank participants for their interest in the study and request that they leave their personal details in order for the researcher to contact them at a later date. The second method will involve recruiting through word of mouth. Appointments will be made with older adult groups and the researcher will visit and explain the research to be undertaken. Interested participants will be asked for their contact details or to contact the toll-free number and register their details at a later date if they require more information before agreeing to participate.

Group 2

Group 2 participants were involved in an electroencephalography (EEG) study also measuring arousal thresholds where sleep stage was controlled. This is existing data from the project "Reducing fire deaths in the aged: Optimising the smoke alarm signal". Ethics approval for this study was obtained from the Human Research Ethics Committee in 2005. We are seeking approval to use this data for the purpose described in this application.

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13. Is there any payment of participants proposed: Yes ☐ No ✓

There will be no monetary payment, but participants will be offered the option of a one month free trial of a new, non-pharmaceutical insomnia treatment device (normally valued at $99, supported by Philips Respironics) as compensation for the inconvenience incurred in participating in the study. Everyone who commences the project will receive the trial even if they fail to complete the project.

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14. Premises on which project is to be conducted:
If using an institution/s other than Victoria University, attach a copy of documents giving approval to use participants or premises in the relevant institution/s.

The study will be conducted in the private homes of the participants.

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15. Dealing with potential risks (for each phase/group where applicable):

*(a) Indicate any **physical risks** connected with the proposed procedures*

- There is a small possibility that any deviation from their routine drug treatment plan might create withdrawal symptoms. It is also possible that participants may wish to take their medication on a night when we are asking them to abstain.
- The interruptions to their sleep may cause daytime sleepiness the following day.

*(b) Indicate any **psychological risks** connected with the proposed procedures*

- There is a small possibility that participants will experience some psychological distress should the hearing test unveil a hearing problem they were not aware of.
- Potential respondents will be intermittent users of hypnotics, which are almost exclusively prescribed for sleep disorders. Sufferers of sleep disorders are more likely to be suffering psychological distress and suffer from borderline depression. There is a small possibility the change in routine has the potential to exacerbate any symptoms.

*(c) Indicate any **social risks** connected with the proposed procedures*

- There is a risk that people who live with the participants may have their sleep disturbed.

- There is a very small possibility, particularly in apartment dwellings, for the experimental alarm to confuse neighbours to a real danger either in their own home or their neighbours.

*(d) Indicate any **legal risks** connected with the proposed procedures*

*(e) Indicate if there are any **other risks** connected with the proposed procedures*

(f) Management of the potential risks identified above- indicate how each of these potential risks will be minimised and/or managed if they occur (if risks have <u>not</u> been identified in 15 a – e, go to item 16).

(i) how risks are to be minimised:

(ii) how adverse events would be managed if they were to occur:

Physical risks

- In order to manage the risk of withdrawal, we are only recruiting intermittent users of hypnotics (who have a very small risk of withdrawal), who are less likely to be troubled by alterations to their treatment plans. In addition, we have written approval from a Sleep Physician (attached as Appendix C) verifying the safety of our intended alteration of existing treatment plans. However, should problems arise, we will request that the participant seek advice from their regular doctor immediately.

- Overcoming the risk of daytime sleepiness or a mild hangover is basically about being aware that such a risk is possible and avoiding vulnerable tasks. Participants will be briefed of the dangers. However, participants are likely to have existing sleep difficulties and be more familiar and prepared to deal with the consequences of lack of sleep.

Psychological risks

- The psychological risk that an unknown hearing difficulty may emerge for some participants through the hearing test will be minimised by targeting people who believe their hearing is intact. In addition, most people recognise the benefits of becoming aware of a treatable problem. In the event that a person fails the hearing test they will be offered information on professional screening and assistance through H.E.A.R. Services.

- The risk that participants will experience more severe symptoms of depression or anxiety will be managed by referring participants at first to their treating physicians or alternatively to an experienced counsellor (letter of support from Gerard Kennedy attached as Appendix D).

Social risks

- The social risk will be managed by offering ear plugs to any other people present in the home.

- To manage the issue of an alarm confusing neighbours of the true threat of fire or an emergency in their own homes or their neighbours, we will notify neighbours of the participant in writing (attached as Appendix E) prior to the experiment of an alarm going off at a particular time at a particular location, and advise the participant of this information. Neighbours will be told to check their alarms first just in case a fire has taken place. The participant will not be told of the exact timing of the alarms, just that the neighbours have been alerted of the project for their safety. The neighbours will be instructed to keep the exact timing of the alarms confidential.

Other risks

(g) If you consider there to be no potential risks, explain fully why no potential risks have been identified.

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16. If you consider the participants to be 'at risk', give your assessment of how the potential benefits to the participants or contributions to the general body of knowledge would outweigh the risks.

The risks in this study are manageable and are outweighed by the potential of this research to result in an improved smoke alarm which may reduce fire fatalities for people who have taken hypnotics.

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17. Informed Consent (If materials are to be distributed in languages other than English, a copy of non-English version and a letter from an independent person verifying accuracy of content is required):

(a) *As part of the informed consent process, it is necessary to provide information to participants prior to obtaining consent.* ***Please attach a copy of your 'Information to Participants Involved in Research' Letter*** *[See http://research.vu.edu.au/hrec.php for a template] with information about your research that you intend to give to potential participants. This needs to:*

- *state briefly the aims, procedures involved and the nature of the project, including a clear indication of any potential risks associated with this project;*
- *if you consider participants to be 'at risk' (see Question 16), state exactly what the researcher will communicate to the participant (this must be stated in clear and concise language) in order to obtain informed consent. This must be in a written format that is given to the participant particularly for this purpose; and*
- *be written in language which may readily be understood by members of the general public, with explanation of any technical terms.*

(b) *Please **attach a copy of your Consent form** [See http://research.vu.edu.au/hrec.php for a template consent form.]*

(c) *State the process you will use to obtain documentation of informed consent hereunder… (It is essential to clearly detail the steps involved in obtaining informed consent. It is recommended that a procedure or flow chart be attached as an appendix commencing from the recruitment stage to consent taking into consideration issues such as communications and awareness of recruitment, provision for considering participation, etc.)*

Potential participants will be informed of the project in the local newspaper advertisement or via the presentation at the older adult group. After registering their interest, volunteers will also be given a full project briefing during the follow up recruitment telephone interview. If the volunteer still opts to participate they will be sent a consent form via mail and asked for the signed consent form prior to the experiment beginning at the in-home appointment. This process provides considerable time for participants to consider their participation.

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18. Confidentiality:

(a) Describe the procedures you will adopt to ensure confidentiality.

All volunteers will be identified by ID only in all documentation. The cross referencing of ID with name and address will be stored separately and securely.

(b) Indicate who will be responsible for the security of confidential data, including consent forms, collected in the course of the research. (Note: the Principal Investigator should be nominated as the responsible person in this section. An alternative person may be nominated with clear justification)

Professor Dorothy Bruck

(c) Indicate the period for which the data will be held. (Data must be held for at least 5 years post-publication. Please refer to section 3.2 of the University's **Code of Conduct for Research, 1995***).*

Five years

(d) Name all people who will be granted access to the data and the reason for the access. People identified are required to maintain all aspects of confidentiality.

In the course of collecting data only the members of the VU research team will know individual participant names and IDs, although all data collection will be identified by ID code as far as possible. The VU research team will consist of the Principal Investigators, and the student (Professor Dorothy Bruck, Dr. Michelle Ball, and postgraduate student,

Chris Lykiardopoulos). All data analysis will proceed using ID codes and no individuals will be identified in any report from this research.

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19. Privacy:

(a) Does this project involve the use of personal information obtained from a Commonwealth department or agency?

Yes ☐ No ✓

If YES you may need to comply with the requirements of the Privacy Act 1988.

Under the Commonwealth Privacy Act 1988 disclosure of personal information by Commonwealth agencies is not permitted except in a number of circumstances specified in Information Privacy Principle (IPP) II. These include consent by the individual concerned. Where consent has not been given, and where none of the other circumstances specified in IPP II apply, additional guidelines for consideration of the project application and for conduct of research apply. Note that the Act does not apply to publicly available material (such as electoral rolls).

If a Commonwealth agency (for instance, the Australian Bureau of Statistics, Commonwealth Government departments, Australian Electoral Commission, most Repatriation Hospitals) is involved in the collection, storage, security, access, amendment, use or disclosure of personal information for a research project investigators must ensure that the project complies with the requirements of the Act.

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20. Conflict of interest

Is there a conflict of interest between any of the researchers and potential participants in the research (i.e due to a relationship between researcher and participant population)?

Yes ☐ No ✓

No recruitment will take place via Simon Frenkel's place of practice.

If yes, provide details and ensure that the conflict is identified and addressed in Section 15.

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21. Research in other countries.
Is any part of the program to be conducted in another country?

Yes ☐ No ✓

If yes, please provide information about any relevant legal or regulatory requirements and any ethical review processes in that other country.

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22 Is approval required for data collection from other organisations? If so, please provide information of consent process (attach evidence of approval/s)

NA

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23. Collaborative program

Does the program involve collaboration with another institution?
Yes ☐ No ✓

If YES, please describe the arrangements with the other institution/s for managing the program including, if appropriate, confidentiality, intellectual property, ethics and safety clearances, reporting to appropriate agencies and the dissemination of research findings.

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24 Other relevant comments (including information that you deem necessary to inform the HREC that may impact on the project)

NA

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25. Application Review Check list

A completed and signed *Application Review Check List* must be submitted with all applications. A copy may be downloaded from the Victoria University Human research ethics webpage at: http://research.vu.edu.au/hrec.php

Important: Attach Application Review Form on cover

Has the Principal Investigator completed and signed the Application Review Form?

Yes ✓ No ☐

Is the Application Review Form attached with a hard copy of this application?

Yes ✓ No ☐

DECLARATION FORM

I, the undersigned, have read the current NH&MRC Statement on Human Experimentation and the relevant Supplementary Notes to this Statement, or Code of Ethics for the Australian Psychological Society, (or *) and accept responsibility for the conduct of the experimental and research procedures detailed above in accordance with the principles contained in the Statement and any other condition laid down by the Human Research Ethics Committee.

Principal Investigator (1) Print Name: Prof. Dorothy Bruck

Signature [redacted] Date 7/10/2010

Principal Investigator (2) Print Name: Dr. Michelle Ball

Signature [redacted] Date 7/10/2010

Associate Investigator ** Print Name: Dr. Simon Frenkel

Signature [redacted] Date 6/10/2010

VU Sponsor ***

Print Name: _____

Signature _____ Date _____

Student/s Details (*If the project is to be undertaken by a student, please provide details):*

Name: Chris Lykiardopoulos

Signature [redacted] Date 7/10/2010

Co-Investigator Print Name: _____

Signature _____ Date _____

I, the undersigned, understand that the above person/s have read the current NH&MRC Statement on Human Experimentation and the relevant Supplementary Notes to this Statement, or Code of Ethics for the Australian Psychological Society, (or *) and that responsibility is accepted by the above person(s) and by this Department for the conduct of the experimental and research procedures detailed above in accordance with the principles contained in the Statement and any other condition laid down by the University Human Research Ethics Committee and fully support the project undertaken within the Department and Faculty.

Head of Department

Print Name: Graham Thorpe

Signature [redacted] Date 27/9/2010

* If NHMRC Statement or APS Code are not appropriate to your project, please identify your professional code of ethics under which this project would operate.

** The Associate Investigator will assume responsibility for the project in the absence of the Principal Investigator.

*** Applications for research involving participants from individuals who are not staff members of VU and who require access to the cohort of VU staff or students to undertake their research. Such research proposals are to be 'sponsored' by a member of staff, who would be required to take responsibility for all interactions with the University and the HREC in relation to ethics issues and their management.