

Subjective sleep quality in the elderly: relationship to anxiety, depressed mood, sleep beliefs, quality of life, and hypnotic use.

Melissa Galea

2008

**Doctor of Psychology (Clinical Neuropsychology)
School of Psychology, Victoria University**

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Doctor of Psychology Declaration

"I, Melissa Galea, declare that the Doctor of Psychology (Clinical Neuropsychology) thesis entitled 'Subjective sleep quality in the elderly: relationship to anxiety, depressed mood, sleep beliefs, quality of life, and hypnotic use' is no more than 40,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work".

Signature:

Date:28.02.08

Acknowledgements

My greatest appreciation is to God for all His love and guidance in all that I do.

I would like to thank my supervisor, Professor Dorothy Bruck for her assistance, advice, and dedication towards my thesis.

A special acknowledgement must be extended to Julio Slayo and my parents for their enduring love and support.

I would also like to thank Associate Professor Bernadette Hood and Brooke Davis for their contributions towards this research.

Finally, I would like to thank the participants for their much appreciated cooperation.

Abstract

The complaint of insomnia in older adults is associated with the use of hypnotics. This study was aimed at exploring possible differences between self-categorised good sleepers and poor sleepers on a range of variables, and the factors associated with the use of hypnotic medication amongst the elderly. Participants in the current study were aged 60 to 98 years old. A total of 74 (28 males, 46 females) older adult hostel residents participated in the current study. Twenty-two participants were self-reported good sleepers, and 52 participants were self-reported poor sleepers, with 21 using benzodiazepine hypnotic medication regularly and 31 not using hypnotics. The measures used included the Pittsburgh Sleep Quality Index (PSQI), Geriatric Depression Scale (GDS), The Beck Anxiety Inventory (BAI), Sleep Beliefs Questionnaire (SBQ), and World Health Organisation Quality of Life-Brief (WHOQoL). The findings demonstrated that self-categorised good sleepers had significantly better sleep quality and habitual sleep efficiency, and significantly shorter sleep latency, regardless of hypnotic use. It was also found that good sleepers and poor sleepers taking benzodiazepines had significantly longer sleep duration than poor sleepers not taking benzodiazepine hypnotics. The prediction that self-classified good sleepers would have lower depressed mood than their self-classified poor sleeping counterparts, whether they were taking benzodiazepine hypnotics or not, was supported. The findings suggested that while self-classified good sleepers had lower anxiety levels than poor sleepers using hypnotic agents, these poor sleepers had significantly lower anxiety levels when compared to their counterparts not using benzodiazepines. Further variables of difference between good and poor sleepers included the psychological and social relationships domains of quality of life. Interestingly, the results indicated that the main role benzodiazepine hypnotic agents have for poor sleepers appears to be longer sleep duration. Benzodiazepine use did not have a significant ameliorative effect on any of the other sleep variables measured in the current sample of hostel-dwelling, older adults. The findings indicated that sleep duration and habitual

sleep efficiency demonstrated good predictive validity for whether subjective poor sleepers take benzodiazepine hypnotics. It was concluded that an alternative to managing poor sleep needs to be education programs, such as education delivered by nurses or evidence based self-help programs (eg. Morawetz, 2003).

INTRODUCTION

A substantive literature base exists to describe and evaluate the diversity of variables associated with poor sleep in older adults. An overview of this literature identifies several key themes. Anxiety and depression have been linked to poor sleep, yet conflicting results still persist. Longitudinal research has indicated that a substantial percentage of incident cases of insomnia in older adults occur without depressed mood, and vice versa (Foley, Monjan, Simonsick, et al., 1999). Other longitudinal research found that self-reported good sleepers at a three year follow-up were least likely to have depressed mood, whilst those with newly reported poor sleep at the three year follow-up reported depressed mood (Cricco, Simonsick, & Foley, 2001). These conflicting results could be due to mediating factors such as sleep complaints simply being another indicator of depressed mood and/or anxiety, sleep related beliefs and negative thoughts (Alapin, Libman, Bailes, & Fichten, 2003). The impact of poor sleep on general quality of life issues has also emerged as a key variable (Lichstein, Durrence, Riedel, & Bayen, 2001; Hellstrom, Persson, & Hallberg, 2003). The current study seeks to explore possible differences between self categorised good sleepers and poor sleepers on a range of variables, and the factors associated with the use of hypnotic medication amongst the elderly. Specifically issues of sleep quality, mood, attitudes towards and beliefs about sleep, and general quality of life issues were explored as a function of self categorised sleep and hypnotic use. There has been a lack of attention in the literature to date, to self categorisation of good and poor sleep. The aim of this work was to make a contribution to the psychosocial knowledge of base factors associated with benzodiazepine hypnotic use, based on self categorisation of good and poor sleep. The literature shows that there is a wide range of factors associated with disturbed sleep and the associated use of hypnotics in the elderly population, and a broader understanding of these factors can potentially facilitate the reduction of such widespread use of benzodiazepines in the treatment of elderly insomnia.

Chapter 1. Sleep and sleep patterns in ageing.

Normal sleep architecture

Behaviourally defined, sleep is a reversible phase of perceptual detachment from, and inattention to, one's surroundings (Carskadon & Dement, 2000). Physiologically defined, sleep is a dynamic, elaborate, greatly organised neurological process, which entails various neuronal groups at numerous levels of the neuraxis (Carskadon & Dement, 2000). An aroused state is sustained by activity of the ascending reticular activating system (RAS) stemming from the mid-brain and pons. The RAS directs the thalamic gate to incoming sensory messages. While in a state of wakefulness, the RAS depolarises the thalamic reticular neurones allowing conveyance of sensory messages and hence, desynchronised cortical activation. However, during sleep the RAS is inhibited permitting thalamic repolarisation, sensory messages are inhibited, and cortical activity is synchronised (Douglas, 2002). It is believed that a fully aroused state is induced by prevalent cortical innervation supplied by noradrenergic neurons in the locus coeruleus and serotonergic neurons in the dorsal raphe nucleus. Along with the histaminergic neurons in the tuberomammillary nucleus (TMN), in the caudal hypothalamus, these neurons supply dispersed cortical innervation, fire most rapidly during wakefulness, become slower during slow-wave sleep, and cease firing completely throughout Rapid Eye Movement (REM) sleep (Carskadon & Dement, 2000). Inhibiting histaminergic neurons with GABA agonists, or injuring these neurons, leads to sleep (Saper & Sammel, 2003). Adenosine might promote slow wave sleep; it appears to accumulate gradually in brain extracellular fluid while awake and to decrease slowly while asleep, suggesting that it may propel sleep physiologically (Douglas, 2002). Serotonin may promote sleep onset and slow wave sleep (Douglas, 2002).

The normal human adult enters REM sleep after 80 minutes or more of non-Rapid Eye Movement (NREM) sleep. NREM and REM sleep interchange throughout the night, following an approximately 90 minute cycle. During stage 1 (which usually endures from around one to seven minutes at the onset of sleep), sleep can be effortlessly disrupted by, for instance touching the sleeper lightly or shutting a door quietly. Hence, stage 1 has a low arousal threshold. Stage 1 NREM sleep transpires as a transient stage during the night, additionally it plays a role in the initial shift from wakefulness to sleep. Following this short-lived stage 1 of sleep, stage 2 is delineated by K complexes and

sleep spindles in the EEG and persists for approximately 10 to 25 minutes (Carskadon & Dement, 2000). The same stimulus that leads to arousal in stage 1 often produces a stronger K complex but no arousal in stage 2 sleep; therefore a more extreme stimulus is necessary for awakening (Carskadon & Dement, 2000). With progression of stage 2, high-voltage slow wave activity begins to emerge. Stage 3 NREM emerges when slow wave activity reaches 2 or fewer cycles per second (cps) and encompasses more than 20% but less than 50% of the EEG activity. In younger adults Stage 3 sleep generally does not continue for more than a few minutes in the first cycle and is transitional to stage 4 with increasing high-voltage slow wave activity (Carskadon & Dement, 2000). Stage 4 sleep is distinguished when the high-voltage slow wave activity is over 50% of the record, and in the initial cycle of sleep typically endures for around 20 to 40 minutes. A considerably greater stimulus is often needed to generate arousal from stage 3 or 4 sleep or slow wave sleep (Carskadon & Dement, 2000).

Three main issues of the overall developmental changes in sleep will be discussed. Increased arousals and changes in sleep architecture are two changes which occur in older individuals. Furthermore, there is the whole issue of the circadian rhythm changes and whether these changes may be biological or environmental. It has been posited that alterations in the circadian sleep- wake rhythm in the aged lead to a shift from the normal adult dual phase sleep-wake cycle to a polyphasic rhythm like that seen in infants (Cistulli, Fiatarone-Singh, & Singh, 2001). These issues, which impact on the overall developmental changes in sleep, are not isolated factors, but rather factors that are interrelated in complex ways.

Aging and arousals from sleep

It appears that both the number and timing of arousals during sleep vary with ageing and this is linked to age-related changes in the central nervous systems underlying the circadian rhythm and sleep-wake homeostasis. Ohayon, Carskadon, Guilleminault, and Vitiello (2004) conducted a meta-analysis of international studies. They indicated that a decrease in sleep efficiency was evident from 40 years of age, and a 3 per cent decrease per decade was observed until very old age. Sleep disturbance measured by the number of awakenings after sleep onset were found to increase significantly with age. Waking after sleep onset consistently increased about 10 minutes per decade of age from the age of 30. It is indicated that there are pronounced age-related changes in

sleep quality in aging which consist principally of a distinct increase in the amount and timing of arousals interrupting sleep, along with a reduction in REM stages and stages 3 and 4 of NREM (Cauter, Plat, Leproult, & Copinschi, 1998; Lockley, Skene, & Arendt, 1999), as discussed in the following paragraphs. Mathur & Douglas (1995) conducted a study exploring the impact of ageing on arousals. They assessed the sleep of normal sleepers with polysomnography. The authors defined arousals according to the American Sleep Disorders Association (ASDA), requiring a quick shift in EEG frequency to >16 Hz or alpha or theta, of three seconds or more, followed by at least 10 seconds of sleep, and if arousal occurs in REM a rise in EMG tone must be present. The data indicated a distinct increase in arousals from middle aged adulthood to older age (Mathur & Douglas, 1995). Arousals may across all ages be momentary and probably unremembered, or may be longer awakenings which the individual remembers and can report (Carskadon & Dement, 2000).

A study undertaken by Murphy, Rogers and Campbell (2000) examined the effects of ageing on awakenings. Sleep episodes of younger (19-28 years) and older (60-82 years) individuals were compared using EEG, EOG and EMG. When allowed to sleep and spontaneously terminate sleep at any time of the day, young sleepers demonstrated a significant disposition to awaken from REM sleep while aged sleepers did not. This result suggested that age and/or quality of sleep might be crucial determinants of the stage in which termination of sleep takes place (Murphy, Rogers & Campbell, 2000). Even at circadian phases when REM inclination was low, young sleepers persisted in awakening from REM sleep. However, older individuals did not terminate sleep periods from REM even when circadian phases were at high REM propensity. The likelihood of awakening from REM was augmented when the sleep of older adults was less fragmented by bouts of arousals.

A wealth of objective and subjective studies of sleep in the elderly population has indicated an increase in both the quantity and duration of arousals that do and do not lead to awakening, during the night (Hume, Van & Watson, 1998; Mathur & Douglas, 1995; Yoon, Kripke, Youngstedt & Elliot, 2003). An implication of this age-related reduction in the propensity to maintain sleep is that the elderly may not need a 'gate' for the shift to wakefulness; instead this shift is readily made from any stage of sleep.

Sleep architecture and aging

The majority of slow wave sleep takes place in the initial third of the night, while REM persists for lengthier periods in the last third of the night. The young adult sleeper spends 75-80 per cent of the night's sleep in NREM, and the remaining 20-25 per cent in REM (Douglas, 2002; Marshall, Molle, Fehm, & Born, 1998). This however, is not the case for the elderly. Sleep patterns change markedly with age and the daily duration of total sleep declines. Sleep duration, or total sleep time has been found to be shorter in the elderly when measured by polysomnography (PSG), in comparison to young and middle-aged adults (Carrier, Land, Buysse, Kupfer, & Monk, 2001; Yoon, Kripke, Youngstedt, & Elliot, 2003). However, the meta-analysis indicated that when researchers compared people aged 60 or over, to people over 70 this trend was not observed, indicating that sleep duration does not continue to significantly decline among seniors (Ohayon, Carsden, Guilleminault, & Vitiello, 2004).

Sleep efficiency (the ratio of total sleep to time spent in bed) and REM sleep latency also decrease with ageing (Cistulli, Fiatarone-Singh, & Singh, 2001). The meta-analysis of international studies conducted by Ohayon and colleagues (2004) demonstrated a decrease in sleep efficiency evident from 40 years of age, and a 3 per cent decrease per decade observed until very old age, and also found that sleep onset latency and percentages of stage 1 and 2 increase significantly with age (Ohayon, Carsden, Guilleminault, & Vitiello, 2004). Sleep latency was observed to increase very progressively with age and became more apparent after 65 years of age. It has been suggested that the changes are linked to dendritic pruning and declining cortical metabolic rate. REM sleep is maintained with ageing but diminishes in dementia (Douglas, 2002). REM sleep has been associated with intellectual functioning, and REM decreases in instances of organic brain dysfunctions in older people. Slow wave sleep begins to abate in people over the age of 60. Women seem to retain slow wave sleep till a later age than men do. It has been asserted that the age-related decrease in nocturnal slow wave sleep may be associated with the loss of cortical synaptic density (Carskadon & Dement, 2000). The literature documenting age-related changes in sleep architecture would imply that insomnia increases in severity in the older old adults (over 75) in comparison to younger old adults (65-74). However, research has indicated that this is not necessarily the case (McCrae et al., 2003). Sleep patterns differ considerably between individual older people (Ohayon, Carsden, Guilleminault, & Vitiello, 2004).

Their large meta-analytic study indicated that after 60 years of age, only sleep efficiency continued to significantly decrease, with all the other sleep variables remaining unchanged.

Age-associated alterations in REM sleep may be due to a shift in the temporary curtailing of REM phase length to a more consistent length across the sleep phase, as opposed to a consecutive increasing (Murphy, Rogers & Campbell, 2000). Other explanations to age-linked changes in REM sleep include the decline in the REM solidity of the aged in comparison to younger people (Wauquier, 1993), and the change in autonomic arousal levels during periods of REM sleep (Zepelin & McDonald, 1987). The results of Murphy, Rogers and Campbell's investigation lend support to the premise that a process active in REM sleep opens the 'gating' to wakefulness, in that non-terminating REM phases were generally discontinued by a brief arousal (significantly more often than by a slip into another sleep stage), and these short-lived arousals took place at a similar temporal position within the ongoing REM phase as did sleep termination from the REM phase. Further studies conducted by Dijk, Duffy and Czeisler (2001) and Ficca and colleagues (2004) also supported the notion that REM sleep is the 'gate' to wakefulness, and that this process was impaired in the geriatric population.

In summary, age-related changes in sleep quality in aging consist mainly of a distinct increase in the amount and timing of arousals interrupting sleep, along with a reduction of stages 3 and 4 of NREM. Hence total sleep time and sleep efficiency decrease with age. When very young adults were compared to elderly people a significant increase in sleep latency emerged. Percentage of stage 1 and 2 sleep increased across all adulthood.

Circadian rhythms and aging

Core changes in the circadian rhythms of aging individuals include changes to both phase and amplitude. One of the factors modulating the sleep-wake cycle is the circadian rhythm originating from the suprachiasmatic nucleus, located in the anterior hypothalamus dorsal to the optic chiasm (Douglas, 2002). The circadian phase at which sleep occurs impacts the configuration of the sleep pattern (Carskadon & Dement, 2000). Light is the primary regulator of the main circadian pacemaker. Circadian rhythms may be shifted if individuals are exposed to light during a time when it is usually dark.

Exposure to light towards the end of darkness or early in the daylight period advances circadian rhythms while light directly prior to or soon after dark onset delays circadian control. Light exposure causes a swift reduction in circulating melatonin levels. Melatonin is usually discharged from the pineal gland during the hours of darkness; therefore levels are generally low by day and elevated at night. Melatonin secretion is an internal synchroniser. Evidence has indicated a significant age-associated reduction in N-acetyltransferase activity, which is a cardinal phase in melatonin formation (Selmaoui & Touitou, 1999). The reduction of melatonin amplitude and/or of the length of its nocturnal peak could be accountable for an endogenous transient desynchronization alongside insufficient adaptability to the external and internal environmental shifts (Magri et al., 2004).

Research has indicated circadian rhythm advances in the elderly population (Murphy, Rogers & Campbell, 2000; Yoon, Kripke, Youngstedt & Elliot, 2003). The circadian rhythm advance was made evident when even under an experimental condition where participants had no access to time cues, clock times of sleep periods were earlier in the older than in the younger adults (Murphy, Rogers & Campbell, 2000). When permitted to sleep at any time of day and to awaken spontaneously, young subjects (19 to 28 years of age) exhibited a significant tendency to awaken from REM sleep while older (60 to 82 years of age) subjects did not. The findings suggest that age and/or sleep quality may be important determinants of the stage from which awakenings occur. The lack of any preferential terminating stage in older subjects was consistent across time-of-day. The tendency to awaken from REM sleep was consistent in young subjects even at circadian phases when REM propensity was low, while older subjects did not awaken preferentially from REM even at circadian phases when REM propensity was high. When the sleep of older subjects was less fragmented by periods of wakefulness the tendency to terminate the sleep period from REM was increased (Murphy, Rogers & Campbell, 2000). Yoon and colleagues (2003) conducted an investigation comparing the napping of younger adults (18 to 32 years) and older adults (60 to 75 years) and its possible relation to circadian rhythm changes. Based on data from wrist-actigraphy, the study found that when young adults napped they were more likely to do so in the afternoon, whereas older adults napped more in the evening. The evening naps in the elderly were associated with earlier waking times from nocturnal sleep. However, older participants who refrained from evening napping still had earlier wake-up times than the

younger participants did. This reflected an advanced sleep-wake cycle in older adults. Hence, evening napping and earlier waking time in the elderly is a possible manifestation of the advanced circadian cycle phenomenon itself. The evening hours, around one to four hours preceding bedtime, have been described as a 'wake-maintenance' zone (Cajochen, Munch, Knoblauch, Blatter, Wirz-Justice, 2006). Taking naps in this period of expected minimal sleepiness could be a sign that the human circadian pacemaker, the suprachiasmatic nucleus, weakens as one gets older (Yoon et al., 2003). The declining amplitude of circadian rhythms, earlier bedtimes and earlier waking times in the geriatric population have been explained in terms of age-associated changes in the output of the suprachiasmatic nucleus (Czeisler et al., 1992; Van Someren, 2000a).

The circadian rhythm interacts with sleep-homeostatic mechanisms, which in turn play a role in the alterations in sleep timing and quality in older adults. Vasopressin is one of the chief peptides in the suprachiasmatic nucleus, and it has been shown that its quantity and synthesis is compromised in aged individuals (Hofman, 2000). These findings suggest a progressive disturbance with reduced amplitude in circadian functioning is embedded in the human biological clock itself. Core and skin temperature is also linked to the circadian rhythm. Body temperature could regulate the neuronal activation state in brain structures related to wakefulness and sleep. Age-related thermoregulatory changes may provide a reasonable argument for age-associated changes in sleep as it accounts for the shallow sleep experienced by many older adults and for the awakening closer to the nocturnal core temperature minimum (Greiffenstein, 2005; Van Someren, 2000b). Circadian rhythm advancement leading to sleep changes in older adults may also be linked to a variety of other factors. These factors may be physical, such as decreased sensitivity of sensory organs to time/light cues (e.g. Visual). Environmental factors, such as reduced environmental light, and decreased exposure to time cues due to lack of social interaction may also offer an explanation to circadian rhythm advancement in aging (Mishima, Okawa, Hozumi, & Hishikawa, 2000).

Summary

An examination of the key issues of sleep in ageing reveals a gamut of factors influencing the sleep of older people. It appears that there are a number of fundamental issues pertaining to sleep and sleep patterns in ageing. In summary, the three issues, of

increased arousals, changes in the sleep architecture, and the circadian rhythm changes (due to biological or environmental reasons) in older adults may all contribute to the experience of poor sleep in some older individuals. These issues which impact on sleep quality in aging are not discrete factors, but rather factors that are interrelated in complex ways. For instance, nocturnal arousals may be viewed as being linked to both changes in sleep architecture and changes to the strength of the circadian system. These age related endogenous changes to both the synchronisation of core body temperature and sleep/wake rhythms, may potentially lend an explanation for the high prevalence of sleep maintenance insomnia symptoms such as nocturnal arousals and early morning wakefulness, amongst older adults (Dijk and Duffy, 1999).

Chapter 2. Insomnia in ageing and the experience of insomnia.

Definition and prevalence of insomnia

The Diagnostic and Statistical Manual of Mental Disorders, the DSM-IV-TR (American Psychiatric Association, 2000) defines primary insomnia as a complaint of trouble commencing or continuing sleep or of non-restful sleep persisting for at least one month, resulting in clinically significant disturbances or detriment in occupational, social, or other substantial aspects of functioning. Furthermore, the difficulty in sleep does not become apparent solely during the process of another sleep disorder or psychological disorder, and must not be due to the direct physiological impact of a drug or a medical condition. Insomnia is a subjective complaint of a lack of sleep or poor quality sleep, which can be broadly divided into two varied forms of complaints depending on the duration of the sleep disturbance (Martin & Ancoli-Israel, 2003). In describing specific age features of primary insomnia the DSM-IV-TR states that older adults are more likely to experience difficulty with sleep maintenance and early morning wakefulness, than younger adults. In the International Classification of Sleep Disorders- Revised (ICSD-R), primary insomnia is called psychophysiological insomnia. Psychophysiological insomnia is defined as an condition of somatised distress and trained sleep-impeding associations that lead to an experience of insomnia and related decreased functioning whilst awake (ICSD-R, 1997). Somatized distress refers to either the individual's subjective experience of, or objective measures of, somatic hyperarousal during sleep attempts. Somatic arousal is identified by peripheral nervous system activity, such as sweating, increased muscle tension, and faster heart rate (ICSD-R, 1997).

The estimates of the prevalence of insomnia across the general population vary widely (Holbrook, Crowther, Lotter, Cheng, & King, 2000a). In the 1991 National Survey of Sleep Complaints conducted in the United States by the National Sleep Foundation in conjunction with the Gallup Organization where one thousand individuals were interviewed, it was shown that 36 percent of the participants reported occurrences of insomnia during the course of the year. One in four of those experiencing insomnia stated that the complaint was chronic (The Gallup Organization, 1991). In Australia 18 per cent of women and 12.6 per cent of men aged 70 years and above suffer from insomnia virtually every night (Partinen & Hublin, 2000). Persisting insomnia is present in approximately a third of people over the age of 65 years old (Partinen & Hublin, 2000).

The DSM-IV-TR notes that the prevalence of primary insomnia in the geriatric population is around 25 per cent. A percentage of people older than 65 report problems falling asleep, i.e. sleep onset insomnia, however, a larger percentage of people in older age groups have middle night insomnia or early morning insomnia (Partinen & Hublin, 2000). The longitudinal study of over 6,000 elderly people undertaken by the National Institute on Aging, found 28 per cent of the participants claimed to have problems with sleep onset, while 42 per cent complained of difficulty with sleep onset and problems with remaining asleep (Foley et al., 1995). Three years later 15 per cent of the respondents who reported sleep complaints at the first interview did not experience any subjective insomnia, while five per cent of individuals without sleep difficulties at the first interview reported difficulties at the three-year follow up. The results showed that sleep difficulties are common in the geriatric population (Foley et al., 1995).

Trained sleep-impeding associations pertain to the type of presleep arousal that seems to be classically conditioned to the bedroom ambience, where unrelenting and intrusive cognitions and preoccupations are frequent signs of presleep arousal. Insomnia is defined as acute if enduring for less than one month, and is usually related to intense pain, stress, or substance abuse. Insomnia symptoms that persist for at least one month, and more commonly, for periods of six months or longer are considered chronic (Smith, Smith, Nowakow & Perlis, 2003). At present, there is no research that uses risk models to assess the natural process of insomnia. Hence, there exists no circumscribed definition of chronicity in terms related to when the disturbance becomes unrelenting, continuous, and self-perpetuating. Clinical hints for distinguishing between acute and chronic insomnia lie in the manner sufferers characterize their symptoms. Sufferers who cease to causally attribute their insomnia to its precipitant but imply that their sleep disorder appears 'to have a life of its own' may aid the characterization of the cut off point between the acute and chronic degrees of insomnia (Smith, Smith, Nowakow & Perlis, 2003).

Theoretical models of insomnia

The literature identifies at least three models of insomnia. The behavioural model of insomnia suggests that the sleep disorder presents itself acutely in terms of both predisposing (trait) and precipitating (state) elements and presents itself chronically in terms of enduring or perpetuating elements. Therefore, an elderly person may be subject

to insomnia because of certain genetic proneness, trait features, personality attributes, and physiological arousal (Smith, Smith, Nowakow & Perlis, 2003). Generally precipitating circumstances include acute pain, situational stress, grieving etc. Perpetuating determinants sustain chronic insomnia subsequent to the stabilization or resolve of the precipitating circumstances. Perpetuating factors are poorly adaptive, coping tactics used as an endeavour to compensate for insomnia symptoms, these may include spending too much time awake in bed, excessive diurnal napping, and using alcohol as a hypnotic (Smith, Smith, Nowakow & Perlis, 2003). The function of classical conditioning as the main sustaining element is pivotal to the behavioural theory of chronic insomnia. It asserts that over time, due to the recurring pairings of the bed with states of psychophysiological hyperarousal, insomnia becomes a conditioned response to the bed and bedroom surroundings.

On the other hand, the cognitive theory of primary insomnia posits that two linked groups of cognitions account for the disorder. One group relates to the individuals' beliefs surrounding insomnia; the other relates to cognitions like worry and ruminating thoughts (Smith, Smith, Nowakow & Perlis, 2003). The cognitive model of insomnia draws heavily from cognitive models of other psychological disorders (e.g., Beck, 1976; Clark, 1997), as well as from earlier theoretical studies outlining the importance of cognitive processes to insomnia (e.g., Espie, 2002; Lundh, 2000; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). According to the conceptualization put forward in the model, insomnia is maintained by a flow of cognitive processes that are activated at night and during the day. The cognitive model posits that worry activates the sympathetic nervous system (the 'fight or flight response') thereby triggering physiological arousal, and distress. This combination of worry, arousal, and distress plunges the individual into an anxiety state, a state that which results in difficulty falling asleep and maintaining sleep (Espie, 2002). Unhelpful beliefs regarding sleep may increase the potential for worry. For instance, if an individual believes one needs more than 8 hours of unbroken sleep each and every night to function adequately during the day, it is likely that the individual will worry about daytime functioning (because most people find that getting 8 hours of unbroken sleep is impossible to achieve). According to the theoretical framework proposed by Lundh (2000), insomnia results from the interaction between sleep-interfering processes (such as different types of arousal, and processes whereby various stimuli, behaviours, and cognitive activities lead to arousal) and sleep-interpreting processes, such as sleep

related beliefs, attitudes, and attributions. Lundh (2000) claimed that treatment of disturbed sleep should, in part, focus on modifying sleep-interpreting processes through behavioural experiments and psychoeducative interventions. Morin, Blais and Savard (2002) found that people with primary insomnia have a variety of negative sleep beliefs, such as disproportionate beliefs about the repercussions of insomnia and exaggerated perceptions of what constitutes satisfactory sleep. The researchers suggested that such tenets could possibly mediate insomnia through exacerbating sleep-related performance anxiety and inducing and encouraging maladaptive compensatory strategies (Morin, Blais & Savard, 2002). These findings lend support to the cognitive model of insomnia, however this model has been contrasted with the neurocognitive perspective of insomnia.

The neurocognitive perspective of insomnia posits that although cognitive factors might contribute to the occurrence and severity of insomnia when it is acute, when insomnia is chronic, cognition occurs secondary to arousal. Hence, according to the neurocognitive model, individuals suffering from chronic insomnia ruminate and worry due to being awake, rather than stay awake because of ruminating, as suggested by the cognitive perspective (Smith, Smith, Nowakow & Perlis, 2003). The neurocognitive theory of insomnia characterizes arousal as conditioned cortical arousal. This pattern of arousal may be detected in persons with primary insomnia as high-frequency EEG activity (14 to 45 Hz) at around sleep onset and throughout NREM sleep (Perlis, Smith, Orff, Andrews & Giles, 2001). This theory proposes that high-frequency EEG patterns permit abnormal levels of information and sensory processing and long-term memory creation. Increased information processing during PSG-determined sleep is believed to hinder the individual's capacity to perceive objective sleep as sleep. An increase in sensory processing has been postulated to interfere with the onset of sleep. While increased long-term memory formation (lengthening of the average mesograde amnesia of sleep) has been claimed to hinder the person's morning judgments on sleep quantity and quality (Smith, Smith, Nowakow & Perlis, 2003).

Insomnia in the elderly can be experienced through a psychophysiological malicious cycle. Maladaptive sleep habits are often exacerbated with excessive day time napping, irregular sleep wake rhythms, unnecessary lengthened time spent in bed, and other actions incompatible with sleep, such as excessive tea and coffee consumption

(Riemann, Fischer, Mayer & Peter, 2003). This in turn may lead to cognitions hindering sleep, like unrealistic expectations, anger because of insomnia, misattribution, and ruminating about the repercussions. Subsequent emotional, physiological, motor, and cognitive hyperarousal might follow, and this in turn may result in fatigue, impaired concentration and performance, depression, and a poorer quality of life. This might be followed by maladaptive sleep habits, and so the vicious cycle of the experience of insomnia continues (Riemann, Fischer, Mayer & Peter, 2003). Although day time napping has been suggested to contribute to poor night time sleep, other research has found that this might not necessarily be the case. Older adults who took brief afternoon naps reported better global sleep quality and sleep efficiency, according to the PSQI, than seniors who did not nap. They concluded that there might not be a need for health-care providers to restrict the day time napping of elderly insomniacs (Lai, 2005).

Subjective and objective sleep disturbance

The practice of insomnia treatment in geriatrics depends substantially on the self reports of older individuals experiencing sleep difficulty, as opposed to the objective measurement of their sleep parameters. Hence the subjective opinion of the aged person's symptoms must be taken into account (Bliwise, 2000). The elderly person's expectations of the amount and quality of sleep are considered important to most general practitioners when managing insomnia in the aged population (Dollman, LeBlanc, & Roughead, 2003). Characterizing insomnia as a subjective complaint without considering objective inspections of symptoms has benefits and drawbacks. The benefit of having subjective criteria is that it endorses the primacy of the sufferer's experience of the disorder and the distress it accompanies. That is, ultimately, the sufferer pursues, complies with and ends treatment grounded on one's own view of wellness. The drawback is that when used as sole measures, subjective accounts do not provide a holistic characterization of the individual's insomnia nor of the sleep disorder in general (Smith, Smith, Nowakow & Perlis, 2003). The older individual's point of view on the meaning of growing old may dictate whether he or she perceives 75 per cent sleep efficiency as insomnia or simply embraces this fact as an expected course in aging (Douglas, 2002). Davis, Hood, and Bruck, (2007) found that older adults categorising themselves as good sleepers utilised downward social comparison, while those classifying themselves as poor sleepers tended towards upward social comparisons. This occurrence might partially account for the discrepancy observed between subjective

and objective sleep quality evident in older adults. Research has explored the relationship between subjective sleep satisfaction and objective sleep measures and has sometimes found conflicting results. Davis, Hood, and Bruck (2007) indicated that the process of self-categorization was not essentially associated with sleep phenomena, because many of these experiences were similarly described by self-categorized 'good' and 'poor' sleepers. Instead, it appeared through the course of upward and downward social comparison, older adults conceptualize notions about 'normal' sleep. It was this 'normative characterization', as opposed to the sleep phenomena experienced, that the person used to establish a standard for their self-categorization of sleep quality.

Riedel and Lichstein (1998) examined the sleep of male and female adults over the age of 59 using polysomnography (PSG) and self-reported sleep satisfaction ratings. The 634 participants in this study were older adults residing within the community. Participants were classified as poor sleepers if they reported more than 30 minutes of sleep latency and arousal following sleep onset at least three nights a week for over 6 months, and were classified as good sleepers if they had no subjective complaint of insomnia (Fichten et al., 1995). Sleep latency and depth of sleep (decreased stage 1 sleep and increased slow wave sleep) were the clearest indicators of subjective sleep satisfaction. The authors found that arousals following sleep onset and sleep efficiency did not relate to sleep satisfaction on a particular night. Fichten and colleagues (1995) found significant differences on subjective ratings of total sleep time, total wake time, and sleep efficiency between elderly participants categorized as good sleepers and those categorized as poor sleepers on the basis of their reported sleep satisfaction. Hence, it appears that certain sleep parameters are more strongly related to subjective sleep satisfaction than others. An investigation by Vitiello, Larsen and Moe (2004) also involved community dwelling elderly; participants had a mean age of 67.5 years. Significant cognitive impairment was screened out by administering the Mini-Mental Status Examination and major depression was ruled out by administering the Geriatric Depression Scale. Participants rated their sleep subjectively using the Pittsburgh Sleep Quality Index (PSQI), their sleep was also monitored objectively using PSG. Analyses of the data revealed significant correlations between PSQI and PSG recorded sleep variables including total wake time, total sleep time, time in bed, sleep latency and sleep efficiency (Vitiello, Larsen & Moe, 2004). Other studies have also indicated significant correlations between subjectively reported and objectively measured (by PSG) sleep

parameters (Bliwise, 1992; Edell-Gustaffson, 2002). The converse has also been indicated, in that there was significant inconsistency between older adults' subjectively underestimated sleep latency and total sleep time in comparison to the results of polysomnography (Chong, Fujun, & Chunying, 2000). Individual self-perceptions might account for these disparate findings. For example, a study comparing self-report to PSG has demonstrated that some senior poor sleepers have a tendency to underestimate how much time they spend awake throughout the night (Libman, Creti, Levy, Brender, & Fichten, 1997). Furthermore, research had indicated that sleep quality, depth, and how rested participants felt upon awakening were not strongly correlated with objective sleep characteristics in those diagnosed with depression in comparison to healthy controls (Armitage, Trivedi, Hoffmann, & Rush, 1997). Hence, it is apparent that individuals with mood disorders may show some sleep-state misperceptions, and this may, at least in part account for some of the disparate findings between subjective and objective sleep characteristics between different study populations. It is difficult to account for the discrepancies in the studies where mood problems were screened out.

It is apparent that self categorized poor sleepers seem to demonstrate poorer sleep quality than self categorized good sleepers (McRae et al., 2003; Vitiello, et al., 2004). In an investigation using subjective and polysomnographic data, Bliwise (1992) found that good sleeping older women had significantly better sleep quality, shorter sleep latency, longer sleep duration, better habitual sleep efficiency, and less daytime dysfunction than poor sleeping women. Similar results were also indicated by investigations involving men and women of all ages and only older adults (Groeger, Zijlstra, & Dijk, 2004; Lichstein, Durrence, Riedel, & Bayen, 2001). These studies utilised a variety of measures to assess sleep quality, including the Pittsburgh Sleep Quality Inventory (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), sleep diaries, and structured interviews.

Bliwise (2000) cited a study undertaken by Middelkoop and colleagues which found that elderly people are increasingly likely to believe they are having sleep disturbances if they experience a problem initiating sleep rather than remaining asleep. This was an interesting finding considering that difficulty staying asleep is more customarily reported. With age, circadian rhythms are shifted earlier comparative to clock time. This often leads to the biologically driven sleep course shifting earlier than the desired sleep course (Martin & Ancoli-Israel, 2003). The experience of older adults often entails excessive

sleepiness prior to bedtime, then waking up before sunrise in the morning. This is potentially due to an advance in the sleep/wake cycle referred to as 'advanced sleep phase syndrome' (ASPS). If the elderly individual experiences problems sustaining sleep in the early morning, but has no trouble falling asleep, circadian rhythm advance is more likely to account for the insomnia (Martin & Ancoli-Israel, 2003). It is important to note that this pattern is also characteristic of depression, and this can pose a possible confound. Furthermore, seasonal changes may exacerbate or alleviate the experience of insomnia in aging. Research found that subjective complaints of insomnia occurred more often throughout the darker intervals of the year. The study suggested that insomnia was more prevalent in the elderly in autumn, winter, and spring than it was in summer (Partinen & Hublin, 2000).

Gender differences in sleep disturbance

Gender differences in insomnia prevalence are slight or nonexistent in children, adolescents and in adults under the age of 40 years. However, a distinct gender difference has been suggested in people over 60 years of age, with insomnia experienced about one and a half times more often in females than in males (Zorick & Walsh, 2000). This has been supported by Vitiello, Larsen and Moe (2004), who found that significantly more women than men experienced subjective sleep disturbance as rated on the PSQI. Interestingly, these participants had denied any substantial sleep disturbance during screening. When measured objectively it was revealed that women had greater total sleep time, sleep efficiency, stage 3 and stage 4 sleep, and REM, and less arousal following sleep onset when compared to men. Total wake time and sleep latency did not differ significantly between males and females (Vitiello, Larsen & Moe, 2004). McCrae and her colleagues (2003) found that older women only slept slightly worse than the older men. The results of their study also indicated that the proportion of non-complaining women was larger for the older old (75-98 years) than the younger old (60-74 years). Non-complainers were defined as poor sleepers that experienced at least 31 minutes of sleep latency and arousal following sleep onset at least three times a week but did not report a subjective insomnia complaint (McCrae et al., 2003).

Despite a greater subjective complaint in older women, one study using objective sleep measures illustrate that they appear to have 'better' sleep in comparison to men. Livingston, Blizard and Mann (1993) conducted a longitudinal study investigating sleep

disturbance among older adults. Measuring sleep disturbance subjectively, they found that it was more prevalent in females. However, Pallesen and colleagues (2002) found no gender differences between elderly people with insomnia and elderly good sleepers.

One explanation to these conflicting findings could potentially be that elderly women may evaluate their sleep quality using different criteria than men (Vitiello, Larsen & Moe, 2004). Results may also be confounding due to lack of methodological robustness in some studies, such as small sample sizes, poor screening for possible confounding and conflicting variables, and very lenient exclusion and inclusion criteria. A large meta-analysis indicated that the links between sleep variables and aging were roughly the same for both genders. However, gender-related differences were more pronounced on some sleep variables, with females experiencing a larger percentage of stage 1 sleep, longer REM latency, and a shorter total sleep time than their male counterparts. This suggests that increasing age in women has a stronger effect on these sleep variables than it does in men (Ohayon, Carskadson, Guilleminault, & Vitiello, 2004).

Summary

It was evident that the prevalence of sleep disturbance in older adults is high, particularly in contrast to its prevalence across younger adults. The literature identifies at least three theoretical models of insomnia, including the behavioural theory, the cognitive model, and the neurocognitive model. A wealth of research has found significant correlations between subjectively reported and objectively measured (by PSG) sleep parameters (Bliwise, 1992; Edell-Gustaffson, 2002; Vitiello, Larsen & Moe, 2004). However, other studies have found some conflicting results between subjectively and objectively measured sleep variables (Fichten et al., 1995; Riedel & Lichstein, 1998). It appeared that individuals with mood disorders may show some sleep-state misperceptions, and this may, at least in part account for some of the disparate findings between subjective and objective sleep characteristics between different study populations. However, it remains difficult to account for the discrepancies in the studies where mood problems were screened out.

Chapter 3. Sleep disturbance and associated factors.

Although insomnia increases with age, some researchers argue that this is not a result of the aging process per se. Insomnia is not a necessary condition of aging, and therefore physiological changes cannot provide a sufficient explanatory model. The sleep disturbances experienced with the increase of age can be linked to the significant increase of comorbidities like medical and mental health problems (Foley, Ancoli-Israel, Britz & Walsh, 2004). The researchers randomly recruited 1506 community residing adults aged 55 to 84 years old. The participants' perception of their sleep quality was very much related to their number of medical conditions. Around 40 per cent of those with major co-morbidity viewed their sleep quality to be only fair or poor, in comparison to those without medical conditions, where only 10 per cent perceived their sleep as fair or poor quality (Foley, Ancoli-Israel, Britz & Walsh, 2004). However, an investigation by Vitiello, Larsen and Moe (2004) found that in spite of the elderly participants being rigorously screened for health and medical problems, their objective sleep quality as measured by PSG was significantly less than that generally found in healthy younger adults.

A wide array of aetiological factors has been associated with the onset of insomnia in ageing. As described earlier in chapter 2, since insomnia is not a necessary condition of aging, physiological changes cannot provide a sufficient explanatory model. Sleep disturbance in the elderly can be secondary to a single or variety of underlying psychosocial influences, medical illness, and/or lifestyle factors and is generally not solely a consequence of the physiological changes associated with ageing. Anxiety and depression, although not directly linked to aging, may arise with changes in work and family status and finances, which sometimes lead to sleep disturbances (Schneider, 2002). A World Health Organization (Costa et al., 1996) study in 15 different countries found that over half of the people with an insomnia complaint were experiencing a mental disorder as defined in the International Classification of Diseases 10, such as anxiety and depression. Insomnia and depression are linked to each other in regards to epidemiology, neurobiology, clinical presentation, and implications for treatment.

Sleep disturbance in the elderly can also be related to factors such as bereavement and grief (Steeves, 2002). Environmental and lifestyle factors such as lack of physical activity

(Richards, Sullivan, Philips, Beck & Overton McCoy, 2001) and insufficient exposure to bright light (Haesler, 2004) have also been found to be associated with sleep disturbance in the elderly. Other lifestyle factors known to have a negative effect on sleep include alcohol and caffeine consumption (Martin & Ancoli-Israel, 2003). The following sections discuss these factors and how they may be related to sleep disturbance in the elderly population.

Psychological factors

The psychological problem most typically linked with insomnia is depression (Martin & Ancoli-Israel, 2003), and because of the pervasiveness of depression in the elderly, sleep difficulties associated with depression in this population are especially important. Symptoms of depression are related to an increase of complaints about sleep disturbances, however the sleep of aged individuals experiencing depression is more disturbed than younger individuals with depression (Martin & Ancoli-Israel, 2003). The older the depressed individual, the greater the sleep continuity difficulty is. Katz and McHorney (1998) found depression to be a risk factor related to mild and severe geriatric insomnia. The authors reported that depressive disorder was nearly three times more prominent as a risk factor of insomnia than chronic health conditions like hip problems and chronic obstructive pulmonary problems. There exists a complicated interaction between sleep complaints and depressed mood. While risk factors for insomnia are similar to those of depression, and depressed mood and insomnia commonly coexist, this is not always the case.

A longitudinal study involving over 6,500 people found that a substantial percentage of incident cases of insomnia in older adults occur without depressed mood, and vice versa (Foley, Monjan, Simonsick, et al., 1999). Another longitudinal investigation interviewed 6,444 community-dwelling men and women aged 65 and over. It was found that self-reported good sleepers at a three year follow-up were least likely to have depressed mood, whilst those with newly reported poor sleep at the three year follow-up reported depressed mood (Cricco, Simonsick, & Foley, 2001). The authors concluded that the effects of poor sleep did not seem to be additive with those of depressed mood since no substantial difference in risk was apparent between depressed older adults reporting poor or good sleep. Hence, in this group, sleep complaints might simply have been another indicator of depressed mood (Cricco, Simonsick, & Foley, 2001).

Bliwise (1992) conducted an investigation using polysomnography with older women and although no significant difference in depressive symptoms between good sleepers and poor sleepers were found, a trend was observed. One explanation to this finding not reaching significance could be due to the small sample size used in the study. Alapin, Libman, Bailes, and Fichten (2003) conducted an investigation using personality inventories with older adults and found no significant difference in depressive symptoms between good sleepers and poor sleepers. They demonstrated that when effects of sleep quality and cognitive arousal (continuous negative thoughts and relentless ruminating) were separated, poor sleep itself, appeared to be unrelated to higher levels of depression or other psychopathology. Rather, poor sleep quality was found to be linked with sleep related preoccupations, including sleep self-efficacy and sleep related distress. This indicates that depression or depressed mood does not necessarily dictate sleep quality in older adults. Hence, like other issues related to insomnia, mood should not be viewed as a discrete factor which impacts on sleep quality in ageing but rather, it is one of the factors that are interrelated in complex ways.

Some cross-sectional research has indicated that insomnia at one stage was a risk factor for the later development of depression (Buysse, 2004; Katz & McHorney, 1998). An earlier investigation had similar findings in a geriatric sample. The researchers in this longitudinal study (Livingston, Blizard & Mann, 1993) found that the highest predictor of future depression in aged people who were not depressed at the time, was current sleep disturbance. Another longitudinal study of a geriatric cohort aged 80 and above also found subjective sleep disturbances and insomnia to be significantly linked with depression and anxiety. Depression in this investigation was measured according to the ICD-10 (Jensen, Dehlin, Hagberg, Samuelsson & Svensson, 1998). These findings have also been supported by other methodologically strong cross-sectional studies when defining depression according to the DSM-IV (e.g. Xavier, Ferraza, Argimon, Trentini, Poyares, Bertollucci, Bisol & Moriguchi, 2002). Other research has indicated that a majority of insomnia sufferers who were depressed prior to non-drug treatment for insomnia were no longer depressed or significantly less depressed (according to Beck Depression Inventory results) after treatment for insomnia (Morawetz, 2003). The treatment for insomnia involved a self-help program which participants used at home to improve their sleep, it included learning strategies for sleep scheduling, sleep hygiene,

and keeping a sleep diary. It is often difficult to determine a cause and effect relationship between insomnia and depression or depression and insomnia, as they often have a circular association. Conflicting results have been found between longitudinal studies and between cross-sectional research as well.

Research involving 2,023 elderly people with an average age of 74.2 years was conducted to examine the relationship between sleep disturbances and depression. The Pittsburgh Sleep Quality Index (PSQI) and the Geriatric Depression Scale (GDS) were used to measure these two variables (Sukegawa et al., 2003). Based on the participants' GDS scores, they were divided into two groups, a depressive group (n=634) and a control group (n=1389). The participants with depression were found to have inferior sleep efficiency, problems falling asleep, and poor subjective sleep quality in comparison to their non-depressed counterparts. Pain associated with sleep disturbance was related to depression in the female elderly. Sukegawa and colleagues (2003) concluded that sleep disturbance and depression among Japanese older adults are significantly associated symptoms. It appears as though the association between depressive symptoms and sleep problems is a prevalent occurrence. However these findings, along with those of Bliwise (1992), and Alapin, Libman, Bailes, and Fichten (2003) support the suggestions in the literature that poor sleepers are not a homogenous group and that there exist subgroups with varying psychological profiles. "Depression affects both the subjective and objective measures of sleep" (Cistulli, Fiatrone Singh & Singh, 2001, p. 77). However when compared to control groups, the subjectively, but not objectively measured sleep of elderly individuals with minor depression was found to be affected (Xavier et al., 2002). This could be explained by negative thoughts and beliefs held by individuals experiencing depression. They might have an increased likelihood of pessimism towards their ability to sleep well. As indicated by the results of Alapin and colleagues' (2003) study other related factors, such as cognitive arousal (continuous negative thoughts and relentless ruminating) and other sleep related preoccupations might be confounding variables in the link between depressed mood and poor sleep. This suggests that depressed mood does not necessarily dictate sleep quality in older people and sleep related thought and beliefs are useful variables that warrant consideration.

People experiencing depression at any age display uniform changes in sleep physiology, and these alterations exacerbate with age (Brunello et al., 2000; Buysse, 2004). Laboratory sleep studies using PSG have ascertained four elementary kinds of sleep problems in patients with depression. The first was impaired sleep continuity; this includes lengthened sleep latency, frequent and prolonged arousals, and/or early morning wakefulness with inability to fall back to sleep. Since in aging the advance in the circadian rhythm itself is accompanied by earlier sleep timing and trouble maintaining sleep in the last half of the night, early morning wakefulness is generally the main characteristic of sleep disturbance in elderly adults experiencing depression (Buysse, 2004). This does not mean that early morning wakefulness is necessarily a depressive symptom, it is merely exacerbated in the presence of depressed mood (Brunello et al., 2000; Buysse, 2004). Lengthened sleep latency in depressed elderly individuals might be exacerbated by their experience of agitation or anxious thoughts about not being able to fall asleep (Cistulli, Fiarone Singh and Singh, 2001). The second sleep based indication of depression was alterations in REM sleep in the geriatric population (Buysse, 2004). Older adults with depression may present more physiological eye movements than non-depressed older adults during the REM sleep stages as implied by the quantity of actual eye movements or 'REM density' (Armitage, 1995; Buysse, 2004; Lauer, Riemann, Wiegand, & Berger, 1991). The third kind of sleep disturbance in older adults with depression is NREM sleep abnormalities. Stages 3 and 4 (slow wave sleep), is significantly reduced in depressed individuals when compared to non-depressed older adults. This is paralleled to a smaller degree in the ageing process, which is linked with a decline in slow wave sleep as well, but not to the extent of that which depressed older people experience (Brunello et al., 2000; Buysse, 2004). The fourth is alterations in timing of sleep stages. A decrease in deep sleep and increased REM 'pressure' in depressed individuals may lead to a reduced REM sleep latency (under 65 minutes), which is an earlier than usual appearance of REM sleep stage. No single sleep disturbance variable reliably discerns depressive individuals from healthy aged control subjects, suggesting that a series of sleep variables better account for the kind of sleep disturbances found in depression (Benca, Obermeyer, Thisted & Gillin, 1992).

Wrist actigraphy was utilized for objective recording of sleep/wake parameters in an investigation correlating depressed mood with sleep/wake patterns in a cohort of people aged 18 to 79 years (Mendlowicz, Jean-Louis, von Gizycki, Zizi & Nunes, 1999). A

qualified examiner assessed depressed mood using part of the Alzheimer's disease Assessment Scale. Those with major depression and depressed mood were characterised by significant rise in sleep latency, number of arousals, and total time awake following sleep onset in comparison to normal controls (Mendlowicz, Jean-Louis, von Gizycki, Zizi & Nunes, 1999). These findings of changes in sleep variables appear to be consistent with subjective reports of sleep disturbances being associated to depression. Ouellet and Morris (2006) reported that depression was negatively related to subjective overall sleep quality, as measured by the PSQI. This relationship remained significant even after controlling for age and number of illnesses.

Alongside alterations in sleep architecture, depressed elderly people have significantly changed patterns of nocturnal hormone secretion, potentially through mechanisms that connect sleep regulation with neuroendocrine activity (Steiger, Von Bardeleben, Guldner, Lauer, Roth, & Holsboer, 1993). An increase in the activity of the hypothalamo-pituitary-adrenocortical (HPA) system, which is part of sleep-endocrine activity, seems to appear with ageing, this possibly hinders the ability to deal with stress and heightens the risk for depression. Research has indicated a causal relationship between disturbed sleep, low levels of growth hormone (usually released amply across the initial half of the night in younger adults), and HPA overactivity in depression (Steiger, 2003). Sleep-endocrine alterations are manifested mostly in geriatric patients with depression. Hence, ageing and depression were found to exercise a synergistic impact on sleep-endocrine activity (Antonijevec, Murck, Frieboes & Steiger, 2000). The neurotransmitter serotonin is thought to be, in part, accountable for sleep patterns, as well as for mood. For instance, individuals with depression have a comparative decline of serotonin (Steeves, 2002).

Older adults experiencing untreated depression have an increased likelihood of disturbed sleep and developing insomnia. Although the pharmacological agents used to treat mood disorders may ameliorate sleep disturbance in the short-term, they may also cause sleep difficulty. Beta-blockers, calcium channel blockers, CNS stimulants, stimulating anti-depressants, and thyroid hormones can all be a cause of insomnia (Kryger, Monjan, Bliwise, Ancoli-Israel, 2004).

Another prominent psychological factor associated with sleep difficulties in the elderly is anxiety. A cohort of 60 older adults aged 60 to 84 who fulfilled the DSM-IV-TR (American

Psychiatric Association, 2000) criteria for primary insomnia were compared to adults aged between 63 to 83 assessed as proficient sleepers (Pallesen et al., 2002). Participants included in the study had no co-morbid DSM-IV Axis I Disorder (e.g. generalized anxiety disorder, major depression). In comparison to good sleepers, insomniacs scored significantly higher on dimensions of anxiety and phobic anxiety. Those with insomnia also had higher scores on dimensions of worry. The authors concluded that worry appeared to be a main characteristic of insomnia in the aged. Failure to tune out intrusive, deeply emotional thinking and images at night-time may lead to anxiety that exacerbates sleep disturbance and insomnia (Pallesen et al., 2002). Other researchers have also suggested that nocturnal negative, anxious, and worried self-talk about sleep may be linked to the complaint of insomnia in aged people (Alapin, Libman, Bailes, & Fichten, 2003). The researchers indicated that seniors experiencing high levels of cognitive arousal during nocturnal awakenings were more poorly adjusted than those with low levels of cognitive arousal. Older adults experiencing high levels of cognitive arousal during nocturnal awakenings had significantly more worried and anxious thoughts during nocturnal awakenings than those with low levels of cognitive arousal regardless of whether they classed themselves as a good or a poor sleeper, however those experiencing high levels of cognitive arousal were more likely to be poor sleepers. The authors suggested that poor sleeping older adults are not homogenous and that it appears that different subgroups may present varied psychosocial profiles (Alapin, Libman, Bailes, & Fichten, 2003).

Da Canhota and Piterman (2001) similarly found co-occurring feelings of anxiety and sleep disturbance in a group of Chinese elderly people over 65 years of age. A longitudinal study of a geriatric cohort aged 80 and above also found subjective sleep disturbances and insomnia to be significantly linked with anxiety (Jensen, Dehlin, Hagberg, Samuelsson, & Svensson, 1998). Anxiety and poor sleep have a similar circular association to that of depression and poor sleep. In an investigation into sleep disturbance in anxiety disorders Papadimitriou and Linkowski (2005) found that 60 to 70 percent of adults diagnosed with generalized anxiety disorder classify themselves as poor sleepers. The authors also posited that according to PSG measures they had an increased sleep onset latency and sleep disturbance (measured according to the number of awakenings), and a decreased sleep duration in comparison to adults without anxiety symptoms (Papadimitriou & Linkowski, 2005).

Generalised anxiety is quite often a symptom of depression particularly in older persons. In a large community sample of elderly people in Britain, Katona, Manela, and Livingstone (1997) found a high rate of comorbidity with 70 percent of those diagnosed with general anxiety being depressed, as opposed to 3 percent of those not experiencing generalised anxiety. They also found that older adults with sleep disturbance had a significantly higher likelihood of being depressed (Katona, Manela, & Livingstone, 1997). Research with a large population-based cohort of older adults living in the USA indicated anxiety symptoms in 43 percent of aged individuals with depression, while 15 percent of older people not depressed had anxiety symptoms (Mehta, et al., 2003). The researchers concluded that poor sleep is likely to occur concomitantly with anxiety symptoms, a milder version of the sleep-associated changes evident in anxiety disorders. Other research has also supported the notion of high comorbidity rates of anxiety and depressed mood in the aged population related to insomnia and sleep complaints (Voyer, Verreault, Cappeliez, Homes, & Nkogho Mengue, 2005).

Anxiety and depression are very much associated with sleep disturbances in the elderly (Bazargan, 1997; Brunello et al., 2000; Buysse, 2004; Pallesen et al., 2002). So much so, that they are sometimes misdiagnosed in place of each other. For instance, Olafsdottir, Marcusson, and Skoog (2001) recruited elderly patients above the age of 70, in primary care and conducted psychiatric examinations with a mean length of around 63 minutes, exploring anxiety and depressive symptoms, and contrasted the results with diagnoses contained in their medical records. The most frequent diagnoses in the psychiatric interview were anxiety disorders, depressive disorders, and dementia. However, the elderly patients' medical records did not concur, with the most common diagnoses being sleep disturbances and anxiety disorders (Olafsdottir, Marcusson & Skoog, 2001). Furthermore, among participants suffering from depression according to the research examination, 14 per cent of these participants' medical records had a diagnosis of an anxiety disorder, 24 per cent held diagnoses of a sleep disorder, while only 12 per cent of these people's medical records had a diagnosis of depression. This phenomenon may be explained by the possibility that older adults experiencing depression may only divulge sleep and anxiety issues to their general practitioners. It was also reported that sleep disorder was the most prevalent diagnosis in medical records of all kinds of mental disorders, implying that sleep difficulties should alert the

clinician to possible anxiety or depressive disorder (Olafsdottir, Marcusson & Skoog, 2001). Such findings have been paralleled by other studies, for instance Da Canhota and Piterman (2001) ascertained that when questioned directly, elderly patients reported a high to moderate percentage of psychological complaints such as sleep disturbances, feeling tense, sad, and anxious. This was contrary to these older patients' medical records which only had physical diagnoses registered and no such psychological problems. However, it has been found that subjectively and objectively classified poor sleepers do not necessarily have higher anxiety levels than their good sleeping counterparts. Bliwise (1992) ascertained that there was no significant difference in general anxiety between good sleepers and poor sleepers. The disparity in findings may be accounted for by the methodological differences between studies, such as sample size and genders included, and also by the notion suggested in the literature that poor sleeping older adults are not homogenous and that it appears that different subgroups may present varied psychosocial profiles.

Social influences

The controversy about specific variables contributing to poor sleep in the elderly has included links to work and household problems. Livingston, Blizard and Mann (1993) conducted a longitudinal study to investigate the pervasiveness of sleep disturbance among older adults and its associated factors. Sleep disturbance was measured subjectively, and found to be rather widespread, and was related to being unmarried, having solitary living arrangements, disability, and being female. However, Pallesen and colleagues (2002) found no gender differences between elderly people with insomnia and elderly good sleepers.

Many papers have noted an association between an increase in sleeping problems and residential care. Prominent psychosocial influences include a shift in residence to a hostel, nursing home, or child's home or possibly extended travel of a close family member. Cricco, Simonsick and Foley (2001) reported that one of the psychosocial risk factors associated with insomnia was nursing home placement. As many as 67 per cent of residents in residential aged care facilities have been reported to complain of disrupted sleep patterns (Haesler, 2004). This percentage is higher than that of elderly people with insomnia living independently (Beck-Little & Weinrich, 1998). Environmental factors unique to aged care institutions; such as light, noise and night-time nursing care

can impact the sleep of their elderly residents (Haesler, 2004). Another explanation as to why the prevalence of seniors in residential aged care facilities complain of poor sleep may be that sleep disturbance is a major trigger for the shift to alternate care, such as hostels or nursing homes (Pollack, Perlick, Lisner, Wenston, & Hsieh, 1990).

The hospitalization and/or death of a partner or loved ones are also a significant life event that may be linked to geriatric sleep problems (Cistulli, Fiatarone Singh & Singh, 2001; Schneider, 2002). With increasing age, people encounter losses that lead to sorrow, anxiety, depressed mood, loss of appetite, and insomnia. The subsequent conjugal mourning accompanies a heightened risk of depressive episodes, and significantly more anxiety symptoms. Byrne and Raphael (1997) indicated that widowers were more likely to experience sleep disturbances than married men; six weeks and six months following their wives' death, but not 13 months after. Sleep difficulties included trouble remaining asleep after sleep onset and losing sleep due to worry. Widowers had significantly higher levels of self-reported anxiety than matched married men at each of the three times interviewed post-bereavement. This study found that the grief over the death of a spouse in this cohort of community-dwelling elderly men was expressed more in sleep disturbance and anxiety than in symptoms of depression or loneliness (Byrne & Raphael, 1997). The researchers concluded that this grief-associated anxiety may portray an adult form of separation anxiety, or possibly a response to threatened loss of autonomy and authority, which in turn leads to disrupted sleep. A study conducted by Steeves (2002), similarly indicated that sleep disturbances were prevalent in elderly widowers, and many of the bereaved had longer sleep onset and more arousals during the night. Hence, early mornings or late evenings became problematic; these were periods when the grief was most powerful (Steeves, 2002).

Lifestyle factors

Sometimes insomnia in elderly individuals arises from lifestyle factors. Alcohol affects the quality of sleep, and older people are at greater risk of sleep difficulties because they metabolize alcohol slower than younger individuals (Martin & Ancoli-Israel, 2003). Stimulants such as nicotine and caffeine are also known to affect sleep quality. The sleep-disruptive effects of caffeine appear to be more elevated in middle-aged and older adults (Kryger, Monjan, Bliwise, Ancoli-Israel, 2004). Caffeine is usually consumed from coffee and tea, but alerting substances like caffeine or pseudoephedrine can also be

taken in the form of herbal preparations and over-the-counter medications, which are quite popular with some elderly people, and can promote sleep disturbance (Kryger, Monjan, Bliwise, Ancoli-Israel, 2004; Martin & Ancoli-Israel, 2003).

Lack of daytime activity or exercise might also have a negative impact on sleep quality in the elderly. These factors, that involve reduced environmental light and decreased exposure to time cues may also, in part, account for circadian rhythm advancement in aging (Mishima, Okawa, Hozumi, & Hishikawa, 2000). Lifestyle factors, such as exercise and walking and their relationship to sleep health were examined in a large cohort of individuals aged 60 to 93 years (Uezu et al., 2000). Such lifestyle factors were found to promote good sleep health. Individuals with lower rates of exercise had a longer sleep latency and more lengthy arousals after sleep onset when compared to those engaging in higher rates of exercise. The less active elderly individuals also had significantly lower subjective sleep efficiency than those participating in more exercise. Those who engaged in significantly lower rates of activity not only had poorer sleep health than their more active counterparts, but also had more episodes of fatigue and sleepiness (Bazargan, 1997; Uezu et al., 2000). A study by Richards, Sullivan, Philips, Beck and Overton-McCoy (2001) lent further support to the notion that lack of diurnal activity may be linked to sleep disturbances in the elderly. The researchers carried out an uncontrolled experiment involving five nursing home residents. The participants were involved in activity programs for around 2 hours a day. Sleep diaries and wrist actigraphy were utilized to assess sleep quality. All participants exhibited ameliorative nocturnal sleep, with an average increase of seven per cent in time spent asleep (Richards, et al., 2001). Morgan (2003) also suggested that lower physical activity levels, but not social activity levels, invariably emerged as one of the prominent risk factors for late-life insomnia. In contrast, Ohayon, Zulley, Guilleminault, Smirne and Priest (2001) asserted that both physical and social activity levels are protective factors against insomnia in older adults. These activities potentially act as zeitgebers strengthening the circadian control of sleep by establishing a routine in relation to the light-dark cycle. Consistent laboratory evidence relating exercise and psychosocial activity to enhanced sleep quality strengthens the possibility that the decline in proportion of physical activity typical of older age groups may contribute to geriatric insomnia (Morgan, 2003; Naylor et al., 2000; Ohayon et al., 2001). This could be linked to the physiological aspects of aging and to their sleep structure. Physical activity and psychosocial activity were found to be

the best predictors of circadian sleep-wake pattern maintenance in elderly people (Sullivan & Richards, 2004).

These lifestyle factors affect the sleep pattern of elderly individuals. Another factor favoring sleep disturbances was deficient exposure to bright light, which synchronizes circadian and sleep-wake rhythms. This is especially problematic in nursing home dwelling elderly (Douglas, 2002; Haesler, 2004). Circadian rhythm advancement in aging has been linked to reduced environmental light exposure (Mishima, Okawa, Hozumi, & Hishikawa, 2000). The cycle of light and dark, along with other factors such as physical activity synchronizes circadian rhythms to a 24-hour pattern. These external factors gain more importance with aging, due to the reduced activity of the suprachiasmatic nucleus (SCN), which is involved in the regulation of arousal level and sleep-wake rhythm (Staedt, 2005). Physical and psychosocial activity has also been found to increase slow-wave sleep in the elderly (Naylor et al., 2000), which may potentially strengthen the sleep-wake cycle. Many seniors have little or no daily commitments that condition their sleep-wake cycle (Naylor et al., 2000).

The National Institute of Aging undertook a longitudinal investigation of over 6,000 aged people residing in the USA. Data from this study indicated that only 5.8 per cent of respondents with no risk factors for insomnia at the first interview reported new insomnia at the three-year follow up interview (Foley et al., 1995). The risk factors for insomnia are recognized as having psychosocial, psychiatric, or medical problems. The data indicated that developing new sleep disturbances was linked to psychological factors and/or poor health, and that alleviating disturbances of sleep is associated with a better quality of life (Foley et al., 1995). It is evident that life changes and co-morbidities accompanying old age augment the likelihood of sleep disturbance and more specifically, insomnia. Psychosocial factors including depression, anxiety, modality of residence, bereavement, and life style factors can adversely affect the sleep patterns and quality of life of older adults (Byrne & Raphael, 1997; Cricco, Simonsick, & Foley, 2001; Da Canhota & Piterman, 2001; Sukegawa et al., 2003).

Sleep beliefs and attitudes

As discussed previously, research and clinical observations have proposed that some poor sleepers have objectively marked sleep disturbances, while others apparently display only subjective insomnia or sleep-state misperception. There is literature (Ohayon, Caulet, & Guilleminault, 1997) that describes how self-characterised good sleepers may experience substantial sleeping difficulties without manifesting any sleep complaints. There is also evidence that even some people who acknowledge their sleep problems fail to convey marked sleep-related distress and dissatisfaction (Fichten et al., 1995; Ohayon, Caulet, & Guilleminault, 1997). Other subjective sleeper categories include self-reported good sleepers who experience objective good sleep, and self-reported poor sleepers who experience objective disturbed sleep. It is the differentiation of these categories that really underpins the understanding of insomnia in ageing, and it appears worthwhile identifying factors that predict actual sleep problems or self assessments.

The literature shows that individuals with sleep complaints report higher anxiety and depressed mood on psychometric measures than good sleepers. However, other aspects in addition to mood disturbances might contribute to sleep-related distress, such as the older adults' beliefs and attitudes about sleep (Edinger, et al., 2000). Some studies support the notion that negative sleep beliefs are linked to poor sleep. For example, through an inventory assessing sleep-related beliefs and attitudes, Morin, Stone, Trinkle, Mercer, and Remsberg (1993) demonstrated that aged poor sleepers had more negative beliefs about the impact of a poor night's sleep, and the extent of loss of control over their sleep than older adults who slept well. The authors concluded that dysfunctional sleep-related beliefs may have a significant part in the development of sleep disturbances. Similar results were found in younger adults (Smith and Trinder, 2001). The study illustrated that scores on the dysfunctional beliefs and attitudes measures discriminated well between DSM-IV diagnosed insomnia sufferers and good sleepers. Fichten and colleagues (1995) indicated that aged insomnia sufferers who experienced high levels of sleep difficulty related distress, expressed more dysfunctional self-statements throughout night waking periods than did other good sleeping seniors. In addition, a previous investigation evaluated the impact of cognitive-behavioural and pharmacological treatments for insomnia on sleep-related beliefs and attitudes and the relationship between those changes and sleep amelioration (Morin, Blais, & Savard,

2002). It was indicated that positive attitudes and beliefs about sleep at post-treatment were linked with better maintenance of sleep improvements at 3, 12, and 24 month follow up assessments. This implies that such beliefs need to be identified and targeted for change in the management of late-life sleep disturbance. It has also been suggested that individuals with certain attitudes may often ruminate over what they believe to be injustices, thereby, hindering their sleep. A WHO study (Costa et al., 1996) and a longitudinal study of older adults examined every year between the ages of 80 and 89 years conducted by Jenson, Dehlin, Hagberg, Samuelsson, and Svensson (1998) ascertained that an increasing severity of insomnia was linked with beliefs about a lack of justice in the world. The studies found that older adults with insomnia tended to think of themselves as being treated in an unjust way. These studies indicated that negative beliefs impacting sleep may not necessarily need to be sleep related. A study by Lichstein, Durrence, Riedel, and Bayen (2001) indicated no difference between poor sleep and negative beliefs and perceived negative consequences of insomnia and disturbed sleep. The inconsistencies in findings regarding sleep beliefs can, at least in part, be accounted for by utilisation of different tools to measure sleep beliefs and attitudes.

Quality of life

Sleep is important for quality of life at all ages. Diminished quality of life due to somatic and/or psychological conditions and lifestyle factors can increase the propensity to insomnia in older adults (Asplund, 1999). On the other hand, sleep disturbance can impact the individual's quality of life by encouraging a state of fatigue. These effects may be bi-directional and difficult to separate. Sleep disturbances in the elderly can lead to consequential deterioration in the quality of life including inability to execute day-to-day tasks, trouble with memory, and concentration, and may also interfere with the pleasure derived from interpersonal relationships (Brunello et al., 2000). A study comparing individuals suffering from insomnia with individuals not experiencing insomnia determined that insomnia sufferers report lower quality of life scores (Hatoum, Kong, Kania, Wong & Mendelson, 1998). A national German survey was conducted on the prevalence of insomnia according to DSM-IV criteria and its negative relationship with quality of life. Twenty two percent of insomniacs rated their overall quality of life as bad while 28 percent rated it as good. In comparison, 3 percent of those with no sleep complaints rated their overall quality of life as bad and 68 percent rated it as good

(Hajak, 2001). This indicated that sleep difficulties have a significant negative impact on an individual's quality of life, and this impact is often longstanding. A longitudinal study of a geriatric cohort aged 80 and above also found sleep disturbances to be related to a lower life satisfaction and quality of life (Jensen, Dehlin, Hagberg, Samuelsson & Svensson, 1998). Hellstrom and Hallberg (2001) found similar results after examining complaints linked to subjective quality of life in a large sample of people aged 75 and over. Between 25 to 30 percent of the sample stated that they had a poor quality of life. Elderly individuals who experienced nervousness, anxiety, and sleep disturbance had a significantly lower degree of quality of life than individuals who did not report these complaints (Hellstrom & Hallberg, 2001). There appears to be an inter-relation between factors contributing to lower life satisfaction and quality; worse self-reported opinions about sleep patterns, generalised anxiety, and minor depression symptoms are significantly associated with worse indexes of quality of life (Xavier, et al., 2002). Specifically, it has been found that the physical well-being domain of quality of life and the psychological/emotional well-being aspect of quality of life were lower in people who are classified as poor sleepers according to the global score of the PSQI (Hellstrom, Persson, & Hallberg, 2003; Manocchia, Keller, & Ware, 2001; Zeithofer et al., 2000). Reynolds and colleagues (2001) and Hajak (2001) ascertained that senior poor sleepers are more likely to have a poor quality of life within the social relationships domain, with an increase in social withdrawal and disengagement. The researchers suggested that poor sleep may lead to social withdrawal and poorer quality of life, which may lead to still poorer sleep (Reynolds et al., 2001).

Summary

Although some discrepancies have emerged within the literature, it appears that there are some consistent psychosocial factors that are risk factors for insomnia in older persons. The psychological problems most typically linked with insomnia are depressed mood and anxiety. Life-style factors such as lack of physical activity and reduced psychosocial activity were also found to be risk factors for poor sleep in elderly people, in particular to their hypothesised effect on the circadian sleep-wake pattern maintenance (Sullivan & Richards, 2004). Some conflicting results were evident regarding sleep beliefs and attitudes. Some studies indicated that dysfunctional sleep-related beliefs appear to play a significant part in the development of sleep disturbances while others did not indicate an association. Other research indicated that negative

beliefs impacting sleep may not necessarily need to be sleep related (Costa et al., 1996; Jenson, Dehlin, Hagberg, Samuelsson, & Svensson, 1998). The variety of tools used to measure this variable may partially account for the discrepant findings between studies. Furthermore, the literature made evident that poor physical, psychological, and social quality of life were associated with poor sleep in aged individuals (Manocchia, Keller, & Ware, 2001; Reynolds et al., 2001; Zeitlhofer et al., 2000).

Chapter 4: Hypnotic use in ageing

The complaint of insomnia in older adults is often associated with the use of hypnotics (Dollman, Le Blanc, & Roughead, 2003; Mant, de Burgh, Mattick, Donnelly, & Hall, 1996). Benzodiazepines are in the class of medications of preferred symptomatic treatment of primary insomnia (Hall, 1998; Holbrook, Crowther, Lotter, & Endeshaw, 2001). Even with the introduction of new non-benzodiazepine hypnotics, such as zolpidem, zaleplon, and zopiclone; benzodiazepines are still commonly used in the management of insomnia in the USA (Morin, Bastien, Brink, & Brown, 2003). Usage data in Australia also reflects widespread use of benzodiazepine agents in the management of insomnia. In 1998 over 3.4 million prescriptions for temazepam, a benzodiazepine hypnotic, were dispensed for the treatment of insomnia, costing the Commonwealth government more than 21 million Australian dollars through the Pharmaceutical Benefits Scheme (Dollman, Le Blanc, & Roughead, 2003). Statistics from the Australian Longitudinal Study of Aging demonstrated that in 1994, 19.3 percent of the cohort of 1664 people, aged over 70, were taking benzodiazepines for the management of insomnia, to the exclusion of any other health problem (Dollman, Le Blanc, & Roughead, 2003). Another Australian study found that 93.5 percent of sleep difficulties were being managed with benzodiazepines, with the majority as continuing treatment (Mant, de Burgh, Mattick, Donnelly, & Hall, 1996). These drugs produce immediate symptomatic relief for sleep disturbance symptoms in most individuals, provided they have not developed tolerance (Hall, 1998; Holbrook, Crowther, Lotter, & Endeshaw, 2001).

The use of benzodiazepines is of particular concern in relation to older people's health. This is especially pertinent, as they receive a disproportionate amount of the prescriptions relative to their numbers (Hall, 1998; Woodward, 1999). Benzodiazepine use in seniors has been linked to poor health, increased duration in time spent in hospital, and sleep disordered breathing (Hall, 1998; Woodward, 1999). Diminished abilities in day-to-day functioning has also been associated with usage of benzodiazepines, independent of coexisting medical conditions (Woodward, 1999), and these factors might contribute to impaired quality of life in elderly individuals. As will be demonstrated in this chapter, other risks of benzodiazepine use for aged people include day-time drowsiness, nocturnal confusion, sedation, light headedness, respiratory depression, and heightened risk of accident and injury due to lack of coordination, instability, ataxia, and difficulty walking (Hall, 1998; Mant, Mattick, de Burgh, Donnelly, &

Hall, 1995; Riemann, Fischer, Mayer, & Peter, 2003). Benzodiazepines have also been found to induce other consequences such as depressed mood (Patten, Williams, & Love, 1996), and cognitive performance, including short-term memory impairment (Curran et al., 2003; Mant, Mattick, de Burgh, Donnelly, & Hall, 1995). The safety of benzodiazepine use in older adults is unclear due to their increased sensitivity and increased slowness of metabolic functioning (Gleason, Schulz, Smith, Newsom, Kroboth, Kroboth, & Psaty, 1998).

Benzodiazepines have a rapid onset of action (Sand et al., 2000), and usually provide immediate symptomatic relief for symptoms of disturbed sleep (Hall, 1998). This class of drugs facilitate transmission of the inhibitory effects of gamma-amino butyric acid (GABA) throughout the central nervous system, leading to hypnotic, sedative, muscle relaxant, anxiolytic, and anti-epileptic effects (Dollman, LeBlanc, & Roughead, 2003). The GABA receptor is a chloride channel complex that mediates fast inhibitory transmission in the brain. By increasing the frequency of chloride channel opening, benzodiazepines augment the effects of GABA while hyperpolarizing the cell, subsequently, the responsiveness to incoming stimuli is reduced (Sand et al., 2003). Benzodiazepines act on the cerebellum, which is involved in coordination and balance; on the limbic system, which is implicated in emotion and memory; and on the cerebral cortex (movement, thought, sensation, decision making etc.; Gudex, 1991). The pharmacological properties that determine their appropriateness for different uses are their effective half-life and onset of action. Benzodiazepines favoured for insomnia, particularly in the elderly, usually have short or intermediate half-lives with intermediate onsets of action, such as temazepam, nitrazepam, and flunitrazepam; this is due to the potential daytime residual effects with long-acting benzodiazepines (Dollman, LeBlanc & Roughead, 2003; Morin, Bastien, Brink, & Brown, 2003). They are prescribed for the management of a wide variety of disorders, including parasomnias, insomnia, convulsions, epilepsy, panic disorder, anxiety, acute behavioural disturbance, muscle spasm, restless leg syndrome, and acute alcohol, barbiturate, and benzodiazepine withdrawal (Hall, 1998; Rivas- Vasquez, 2003; Woodward, 1999).

Older adults taking benzodiazepines for the management of insomnia often experience withdrawal symptoms when the medication is discontinued (Smith & Laundry, 1991). It appears likely that approximately half the people who have been receiving

benzodiazepines in therapeutic doses for more than six months will experience withdrawal symptoms on discontinuation of the medication (Cannard, 1996). Only a small percentage of people who have been taking benzodiazepines for less than six months are likely to have withdrawal symptoms (Cannard, 1996). Problems throughout withdrawal can be accounted for as symptoms of GABA deficiency. Due to benzodiazepines suppressing the REM phase of sleep, a compensatory excess occurs following withdrawal (Tyrer, 1993). The onset of withdrawal symptoms differs according to the length of action of the drug taken. The withdrawal syndrome typically manifests itself within two days following cessation of a short-acting benzodiazepine, and approximately a week for long-acting benzodiazepines (Smith & Laundry, 1991). The symptoms most typically linked with benzodiazepine withdrawal are increased depression, anxiety, gastrointestinal upsets, perceptual disturbances, severe headaches, and muscular aches and pains. However, variations of symptoms and their severity do occur between individuals (Cannard, 1996). Sudden withdrawal from benzodiazepine hypnotics may incite rebound insomnia, particularly with benzodiazepine hypnotics of a short or intermediate half-life. This consequence facilitates the chronic intake of the medication, hence resulting in dependence (Chouinard, 2004; Riemann, Fischer, Mayer, & Peter, 2003). In summary, the instant benefits of benzodiazepine hypnotics may often not outweigh the ensuing withdrawal symptoms and problems associated with longer term use.

Other concerns associated with long term use of benzodiazepine hypnotics include tolerance and dependency. Tolerance has been indicated with most commonly used benzodiazepine hypnotics (Woodward, 1999). With the occurrence of tolerance, increasing doses may be consumed to reach to same apparent clinical effects, and this may increase the risk of adverse effects. Tolerance to benzodiazepine hypnotics may occur very rapidly, typically within days or weeks. Benzodiazepines taken over a long period of time have little effect on sleep (Goldberg, 2005; Rosenberg, 2006). General practitioners have also been found to have significant concerns about disadvantages of benzodiazepines with regards to tolerance and hence, loss of efficacy after long-term use (Siriwardena, Qureshi, Gibson, Collier, & Latham, 2006). Epidemiological data on the prevalence and treatment of insomnia (Hohagen et al. 1993) support the notion that insomniacs' medication tolerance may be one of the principal reasons for chronic intake of hypnotics. In this study, older adults using prescribed hypnotics (almost exclusively

benzodiazepines) were asked about the duration of intake and the subjective effect of the hypnotics on their sleep. Whereas the duration of intake was longer than 6 months in 75% of all patients, a period far longer than advised, the overall efficacy was very small. Only 20% reported a distinct improvement of their sleep. The other patients taking a prescribed hypnotic either denied a significant amelioration of their sleep, or even noticed a deterioration, despite the intake of a hypnotic (Hohagen et al. 1993).

With regard to dependency, it has been found that around 35 per cent of older people taking benzodiazepine hypnotic agents become dependent after more than four weeks of use, and most regular benzodiazepine hypnotic users develop dependency after four months of use (Woodward, 1999).

The CNS depressant effects of benzodiazepines are the most problematic adverse events linked with their consumption. The most common side effects involve sedation, daytime drowsiness, and psychomotor slowing or un-coordination. Sedation is usually a passing issue initially experienced in treatment and tends to subside as tolerance develops within a few days. However, drowsiness has been found to interfere with daytime functioning and increases the dangers of driving, walking, and simple daily activities (Hall, 1998; Rickels, Lucki, Schweizer, Garcia-Espana, & Case, 1999; Rivas-Vasquez, 2003). The risks of falls due to ataxia and coordination problems increase the likelihood of head injuries or hip fractures. Benzodiazepine use has also been associated with reduced physical functional status in longitudinal studies (e.g. Gray et al., 2003). Benzodiazepine medications are known to have cognitive side effects and their use can not always be justified in older adults that may already be experiencing some degree of cognitive decline (Curran et al., 2003; Rivas- Vasquez, 2003). Rickels and colleagues (1999) found that cognitive functions, such as speed of information processing, were improved for many elderly long-term (mean, 8 years) benzodiazepine hypnotic users following discontinuation.

Benzodiazepines can be effective in the short-term treatment of insomnia, however, prescription of these agents in the elderly raises concerns regarding long-term use, dependence, safety and loss of efficacy, i.e. tolerance (Griffiths & Weerts, 1997; Roland, 1995). Even though some practitioners support short-term management with benzodiazepines, evidence suggests that older adults have an increased risk of physical instability during the initial few weeks of receiving these drugs (Neutel, Hirdes, Maxwell,

& Patten, 1996). It is apparent that the range of adverse effects of benzodiazepine hypnotics is wider in seniors than in younger adults (Woodward, 1999). Age-related physiological changes cause the benzodiazepines to be absorbed, distributed through the body, metabolised by the liver, and excreted in the urine at a slower rate than in younger adults. Generally, older adults encounter both the benefits and the side effects of benzodiazepines at lesser doses than do young and middle-aged adults (Juergens, 1993). In summary, the instant benefits of benzodiazepine hypnotics very rarely outweigh the ensuing withdrawal symptoms and problems associated with longer term use.

As the ageing population increases, there has been an increased emphasis within health policy on promoting healthy ageing (Holbrook, Crowther, Lotter, Cheng, & King, 2000a). However, relatively little empirical attention has been dedicated to factors that influence older adults experiencing disturbed sleep to take benzodiazepine hypnotics. Having an accurate illustration of the specific mood, beliefs, and quality of life of senior benzodiazepine hypnotic users would assist in identifying what should be developed in order to better respond to this population. Because the definition of 'normal' sleep is not well established, the estimates of the prevalence and severity of insomnia vary widely. Insomnia lacks diagnostic precision and is ill-defined by clinicians and seniors alike (Holbrook, Crowther, Lotter, Cheng, & King, 2000a). Lack of diagnostic specificity in turn leads to many elderly adults being prescribed hypnotic medication they do not necessarily require (Hall, 1998).

Benzodiazepines decrease REM sleep and usually, night-time awakenings. Furthermore, they suppress slow wave sleep. The increased time spent asleep during benzodiazepine use is thus linked to a modification of normal sleep architecture (Closser, 1991; Woodward, 1999). The reduction of REM sleep leads to decreased dreaming and nightmares, which may be particularly useful if these are interrupting sleep (Closser, 1991; Woodward, 1999). A meta-analysis of the findings contrasting sleep variables of poor sleepers using benzodiazepines with those not using benzodiazepines indicated that insomnia patients receiving benzodiazepines had increased total sleep duration, yet no significant difference in sleep latency was found (Holbrook, Crowther, Lotter, & Endeshaw, 2001). There is no association between long-term (more than four weeks of continuous use) benzodiazepine hypnotic use and self-reported quality of sleep

(Holbrook, Crowther, Lotter, Cheng, & King, 2000a; Petrovic, Pevernagie, Van Den Noortgate, Mariman, Michielsen, & Afschrift, 1999). This is an interesting finding that might warrant further investigation, given the frequent lack of association between subjective and objective measures of sleep quality in aging. A lack of association between benzodiazepine hypnotic use and self-reported quality of sleep may be, at least in part, accounted for by the effects of tolerance to the benzodiazepine hypnotics over time (Goldberg, 2005; Rosenberg, 2006). Petrovic and colleagues (1999) and Ramesh and Roberts (2002) compared the subjective sleep quality of aged individuals taking benzodiazepine hypnotics to their overall sleep quality after discontinuation of these hypnotics and found no significant difference in their self-reported overall sleep quality. It has been noted that in retirement homes, even the sleep quality of elderly insomniacs treated pharmacologically is worse than that of their non-treated counterparts (Berg & Dehlin, 1984).

A review by Estivill and colleagues (2003) outlined the body of literature that has indicated how short-term use of benzodiazepine hypnotics exert a variety of effects on sleep architecture, as measured by PSG. They indicated a decrease in slow wave sleep, REM sleep and sleep latency, and an increase in total sleep time and sleep quality. In a meta-analysis, Holbrook, Crowther, Lotter, and Endeshaw (2001) found that sleep latency for elderly patients receiving a benzodiazepine during a 4 week period was 4.2 to 14.3 minutes shorter than those receiving a placebo. The authors also reported that the total sleep duration (using sleep record results), for patients taking benzodiazepines was an average of 61.8 minutes longer than those taking a placebo. Patients' subjective estimates of sleep duration also indicated longer sleep duration than those in the placebo groups. Moreover, analyses of studies indicated that older adults receiving benzodiazepines were more likely than those receiving a placebo to complain of dizziness or light-headedness, and daytime drowsiness (Holbrook, Crowther, Lotter, & Endeshaw, 2001). Two other meta-analyses (Holbrook, Crowther, Lotter, Cheng, & King, 2000; Nowell et al., 1997) and a review (Smith et al., 2002) have posited a clear-cut superiority of short term benzodiazepine use in comparison to placebo in both subjective and objective parameters of sleep. However, the outcome had not been evaluated beyond four weeks.

Literature that evaluates the effectiveness of benzodiazepine hypnotics beyond this time frame is scarce (Riemann, Fischer, Mayer, & Peter, 2003). Nonetheless, benefits in the use of benzodiazepines for over 30 days are rare (McLeod, Hung, Tamblyn, & Gayton, 1997). Other research has also suggested that the overall benefit of benzodiazepines compared with placebo appears to be minor and short-term, and linked to a variety of adverse effects (Rivas-Vasquez, 2003). Despite this, a majority of elderly people are prescribed benzodiazepines for months, and at times years (Ohayon, Caulet, Priest, & Guilleminault, 1998; Snowden, 1999). Withdrawal from these hypnotic drugs can also have an unfavourable impact on insomnia patients.

There appears to be a wide range of factors associated with insomnia and the associated use of hypnotics in the elderly population, and a broader understanding of these factors can potentially facilitate the reduction of such widespread use of benzodiazepines in the treatment of elderly insomnia. Physicians' prescribing behaviour, age, attitudes towards hypnotics, and psychosocial state are such factors, these are discussed in the next section.

In general practice settings, the management of insomnia typically involves benzodiazepines (Dollman, LeBlanc & Roughead, 2003). It has been suggested that there are problems with physicians' attitudes and skill concerning the methods of administration and intermission of hypnotic medication. A survey indicated that more than a third of physicians initiated administration of benzodiazepine hypnotics without considering a time period of administration and only 5 per cent stated that intermission of benzodiazepine medication was necessary (Uchimura et al., 2002). Moreover, it has been posited that clinicians' perspectives on medication efficacy are closely linked to their financial involvement with pharmaceutical companies (Stelfox, Chua, O'Rourke, & Detsky, 1998). Physicians are generally the first to be blamed for the long-term and excessive use of hypnotics in society; however the patients' beliefs and attitudes play a crucial part in the matter (Ohayon et al., 1999). Research suggests that the greatest barriers to general practitioners suggesting non-drug alternatives to elderly patients are perceived patient non-compliance and wanting/expecting medication. However, aged patients felt the brief time of a consultation was a barrier to receiving enough time to discuss non-drug alternatives with their general practitioners (Dollman, LeBlanc & Roughead, 2003). Yet, a survey of patient preferences suggested that seniors prefer

counselling from their physician to medication as a first line of treatment (Brody, Khaliq, & Thompson, 1997). In relation to elderly people residing in nursing homes, nurses are in a unique position to lessen the probability that inappropriate psychotropic drugs will be introduced to manage sleep. When residents complain about changes in sleeping patterns, nurses can provide education regarding the influence of the normal ageing process on sleep patterns. This would diminish misinterpretation and, subsequently, anxiety about the alterations in the quality and quantity of sleep that older individuals experience (Voyer & Schindel Martin, 2003). However, if it becomes evident that the elder's quality of sleep is substantially poor, prior to seeking medical treatment, the application of behavioural techniques should be tried (Grossberg & Grossberg, 1998).

Cognitive behavioural therapy encompasses adjusting poor sleep habits, correcting misinterpretations concerning sleep, and endorsing proper sleep hygiene. A meta-analysis conducted by Montgomery and Dennis (2002) on cognitive behavioural interventions for insomnia in adults aged over 60 demonstrated the efficacy of these non-pharmacological techniques. The application of cognitive and behavioural techniques has also been shown to effectively ameliorate the quality of sleep in older adults in other research (Cassel et al., 1997; Gatz et al., 1998). Furthermore, enhancement in sleep patterns is maintained when the techniques continue to be employed, in contrast to sustained use of hypnotic medications (Holbrook, Crowther, Lotter, & Endeshaw, 2001; Morin, Colecchi, Stone, Sood, & Brink, 1999). Pharmacological treatment alone always results in a relapse into insomnia symptoms after the medication has been ceased (Riemann, Fischer, Mayer, & Peter, 2003).

Specific benzodiazepine hypnotic user-related variables such as age have previously been investigated with varying results. Gleason and colleagues (1998) recruited a representative sample of 5,181 elderly in the USA. The sociodemographic characteristics among benzodiazepine hypnotic users indicated that out of the 115 users, the highest prevalence of users were individuals aged 70 to 79, followed by those aged 65 to 69. Only a small proportion of benzodiazepine hypnotic users were over 80 years of age. Miyamoto, Hirata, Miyamoto, Iwase, and Koshikawa (2002) used a computer-ordering system database in Japan to investigate benzodiazepine hypnotic prescriptions issued to outpatients. The researchers found that these hypnotics were

prescribed most often to patients in the sixties, followed by those aged in their seventies, fifties, forties, thirties, twenties, eighties, teens, and nineties.

It has also been ascertained that benzodiazepine hypnotic users are more likely to be white and have a tertiary education (Gleason, et al., 1998). Certain patient-related variables such as personality traits, and presence of psychiatric and somatic comorbidity can often determine whether a chronic benzodiazepine user will become dependent or not (Oldham et al., 1995; Wittchen et al., 1996, Martinez-Cano et al., 1999). It has been suggested that beliefs in the efficacy of benzodiazepine hypnotics, attitudes about continuing or stopping hypnotics, and self-report of insomnia despite their use, appear to play a role in older adults' decision to take benzodiazepine hypnotics (Iliffe et al., 2004).

Depression and/or anxiety are often major variables of difference between hypnotic users and non-hypnotic users, however the association of anxiety and depressed mood with benzodiazepine hypnotic use in poor sleepers remains unclear. One study revealed that nearly 40 per cent of insomniacs using hypnotics were also receiving mental health care for depression or anxiety (Kageyama et al., 1998). Furthermore, results found in a longitudinal study indicated high level of benzodiazepine use (43%) in older adults with anxiety (Schuurmans, Comijs, Beekman, de Beurs, Deeg, Emmelkamp, & van Dyck, 2005). While Voyer, Landerville, Moisan, Tousgnant, and Preville (2005) found that although anxiety and depression, as assessed by DSM-IV interviews, was not linked with benzodiazepine use, the self-report measures of anxiety and depressed mood did indicate a significant association with benzodiazepine medication use in older adults experiencing disturbed sleep. It has also been indicated that concomitant anxiety was significantly associated with higher consumption and higher daily dosage of hypnotics in older adults (Huang & Lai, 2005). It is difficult to ascertain whether older adults on benzodiazepines have higher anxiety since taking these medications or prior to taking benzodiazepine hypnotics. Personality pathology, particularly a predisposition to borderline personality disorder has been found to be more pronounced in benzodiazepine hypnotic users when compared to non-users (Petrovic, 2002). The findings also indicated that anxiety and dysthymic disorders are predominant clinical factors observed in patients on chronic benzodiazepine hypnotic treatment (Petrovic, 2002).

The Current Study

The issues examined in the following study of the elderly are informed most by the cognitive theory of primary insomnia. This theory states that that two linked set of cognitions account for the disorder. One set relates to the individuals' beliefs surrounding insomnia; the other relates to cognitions like worry and ruminating thoughts (Smith, Smith, Nowakow & Perlis, 2003). Thus variables of anxiety, sleep beliefs and attitudes, and quality of life issues were assessed across self categorised good and poor sleepers, with poor sleepers including those both taking and not taking hypnotics. Results that show major differences between good and poor sleepers on such variables would lend support to the cognitive model of insomnia. Understanding the variables of difference between self categorised good sleepers and poor sleepers, and the variables associated with the use of hypnotic medication amongst this group of older adults is of particular relevance as (i) sleep disturbance appears to be a likely outcome following admission to institutions, such as hostels and nursing homes, and a risk factor for older adults to require institutionalisation (Pollack & Perlick, 1991), (ii) literature exploring variables of difference between poor sleepers taking benzodiazepines and poor sleepers not using hypnotic medication is scarce but potentially important to inform clinical decision making, and (iii) there has been a lack of attention to variables of difference using self categorised good and poor sleepers in the literature to date.

Five hypotheses were tested, with all the dependent variables being assessed by questionnaires. The questionnaires and dependent variables are summarised in Table 1 and will later be described in detail.

Table 1

Details of the Questionnaires Administered

| Questionnaire | Abbreviation | Role within the study | Variables assessed |
|--|--------------|-----------------------|--|
| Mini Mental State Examination | MMSE | Screening tool | |
| The Sleep Apnea Screen | | Screening tool | |
| Pittsburgh Sleep Quality Index | PSQI | Dependent variables | Global sleep quality, sleep quality, subjective sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, and daytime dysfunction |
| Geriatric Depression Scale | GDS | Dependent variable | Depressed mood |
| The Beck Anxiety Inventory | BAI | Dependent variable | Anxiety |
| Sleep Beliefs Questionnaire | SBQ | Dependent variables | Beliefs about the impact sleep has on quality of life, loss of control, and social interactions |
| World Health Organisation Quality of Life-Bref | WHOQoL | Dependent variables | Quality of life in the physical domain, psychological domain, social relationships domain, and environment domain |

The first hypothesis was that overall sleep quality (global sleep quality index) in self categorised good sleepers would be better than that of self classed poor sleepers, but there would be no difference in overall sleep quality between poor sleepers taking benzodiazepines and poor sleepers not using benzodiazepines.

The second hypothesis was that there would be significant differences in specific sleep variables (poorer subjective quality, longer latency, shorter duration, poorer sleep

efficiency, and increased sleep disturbance and daytime dysfunction) in poor sleepers, as compared to good sleepers, and poor sleepers using benzodiazepines would have significantly longer sleep duration in comparison to poor sleepers not taking benzodiazepine hypnotics. No difference was expected in sleep quality, sleep latency, habitual sleep efficiency, sleep disturbance, and daytime dysfunction between poor sleepers not taking benzodiazepine hypnotics and poor sleepers using benzodiazepines.

The third hypothesis was that self categorised good sleepers would have better affective functioning (lower anxiety and depressed mood) than poor sleeping benzodiazepine users, and poor sleepers not taking benzodiazepine hypnotics. Poor sleeping benzodiazepine users would have lower anxiety than their counterparts not using benzodiazepines, but no difference in depressed mood.

A further hypothesis was that there would be a sleep beliefs (about quality of life, loss of control, and social impact) would be more negative in poor sleepers on benzodiazepines, and poor sleepers not using benzodiazepines when compared to those of good sleepers.

The fifth hypothesis stated that self-reported good sleepers would have higher quality of life (across the four domains of physical and health; psychological; social relationships and environment) in comparison to poor sleepers, but no significant difference between poor sleepers not using benzodiazepines and those taking benzodiazepine hypnotics.

The current study aimed to explore a selection of the affective functioning, sleep beliefs, and quality of life variables that might significantly predict global sleep quality as measured by the PSQI Global. Thus a multiple regression test was performed to determine which variables, if any, would best predict global sleep quality for all participants.

It also aimed to explore a selection of the dependent variables in Table 1 that might demonstrate significant differences between the two poor sleeper groups. Thus a discriminant analysis was performed to determine which variables, if any, best discriminated these two groups.

Finally, the study aimed to explore a selection of the dependent variables in Table 1 that might demonstrate significant differences between the good and poor sleeper groups (including both those taking and no taking benzodiazepines. Hence a discriminant analysis was performed to determine which variables, if any, best discriminated these two groups.

METHOD

Participants

Baptist Community Care and Catholic Homes managers were approached and asked permission to include a selection of their residents in the current study. The participants were from a variety of Baptist Community Care hostels including Karana, Strathalan, Westhaven, Hedley Sutton, and two Catholic Homes hostels including St Catherine's Hostel and St Bernadette's Hostel. A total of 74 (28 males, 46 females) older adult hostel residents participated in the current study. The participants completed a series of questionnaires. Their ages ranged from 60 to 98 years old ($M = 84.8$, $SD = 8.5$). Participation was voluntary.

Inclusion criteria for all participants were 1) age 60 or older, 2) do not have a severe physical illness known to disturb sleep, 3) do not have a serious disease that is associated with chronic pain, 4) are not depressed based on a score lower than 19 on the Geriatric Depression Scale, 5) have no major symptoms of cognitive dysfunction as suggested by a score of less than 23 on the Mini-Mental State Examination.

Within the sample, one group comprised twenty-one participants, 11 males and 10 females (28.4%), who were self-reported poor sleepers and chronic users of benzodiazepine medication for insomnia. The co-ordinator/nurse ensured that these participants' benzodiazepine use was for the treatment of poor sleep. Additional inclusion criteria for these participants were 1) history of using benzodiazepine medication for sleep at least 3 days per week for at least 6 months, 2) do not have a diagnosed sleep disorder other than insomnia, and 3) are not taking medications other than sedative hypnotics known to disrupt sleep.

Thirty-one participants, 17 male and 14 female (41.9%) were self-reported poor sleepers, but not using benzodiazepine hypnotics. Additional inclusion criteria for these participants were 1) no hypnotic use for at least 6 months, 2) do not have a diagnosed sleep disorder, although they complain of poor sleep, and 3) are not taking medications known to disrupt sleep.

Twenty-two female (29.7%) participants were self-reported good sleepers. Additional inclusion criteria were 1) do not have a diagnosed sleep disorder, and 2) are not taking medications known to disrupt sleep.

Design

This quantitative study had an independent samples design since differences were explored across the three sample groups. The dependent variables comprising the sleep variables were sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, and daytime dysfunction. The affective dependent variables were anxiety levels and depressed mood levels. Dependent variables exploring the individuals' sleep beliefs include beliefs about quality of sleep, beliefs about loss of control, beliefs about social impact. Moreover, the Quality of Life dependent variables include quality of life in the physical domain, psychological domain, social relationships domain, and environment domain. Satisfactory sleep, poor sleep without hypnotic use, and poor sleep assisted with benzodiazepine usage, were the independent variables. Refer to Table 1.

Materials

The following section describes each questionnaire (as set out in Table 1) and then describes the scoring procedure for each questionnaire.

Mini Mental State Examination (MMSE)

The Mini Mental State Examination (MMSE) (Folstein, Folstein, and McHugh, 1975) was initially designed to test the cognitive facet of the mental status of psychiatric patients. It is quick and easy to administer, and as a result has become widely utilised as a dementia screen in clinical and research populations.

The MMSE is an 11 item questionnaire with six sub-scales. The MMSE was administered by the researcher to screen for suspected cognitive dysfunction. The scale briefly assesses a restricted set of cognitive functions. Five discrete yet related domains are assessed, these include orientation, memory (delayed recall of three items), attention span (immediate recall of three items), concentration or working memory (serial

7s or spelling 'world' backwards), language and praxis (naming, following commands, and reproduction of a geometric design). Administration takes about five to 10 minutes.

The MMSE is a psychometrically sound screening instrument. In regards to the instrument's validity, when the conventional cut-off of 24 was applied to samples of patients referred for dementia evaluations, the MMSE had high specificity yet limited sensitivity: .90 and .69 in one investigation (Feher and Martin, 1992) and .96 and .63 in another (Kukell et al., 1994) respectively. The ideal screening inventory must emphasize sensitivity over specificity. However, a cut-off score of <25 yielded excellent sensitivity of .92 and specificity of .96, and a very high test effectiveness score (balance between sensitivity and specificity) of .99 (Heun, Papassotiropoulos, & Jennssen, 1998). Hence the <25 cut-off was identified as the optimal threshold for discerning demented and non-demented individuals. In studies of the convergent validity of the MMSE, it has been found that it concurs with verbally based neuropsychological tests, compared to those that are non-verbally based (Heun et al., 1998; Mitrushina & Satz, 1991). Furthermore, MMSE scores were most strongly correlated with scores on the Rey Auditory Verbal Learning Test (RAVLT) ($r = .31-.39$) (Mitrushina & Satz, 1991).

Test-retest reliability over a 24-hour period was high (.89) (Folstein, Folstein, and McHugh, 1975). This indicates that the MMSE renders an accurate assessment of cognitive mental state at a particular point in time, which makes it useful as a screening tool. This is further supported by acceptable convergent validity of the MMSE with tools that test similar constructs, and the superior sensitivity of the MMSE to the dementing process.

Survey Screen for the Prediction of Apnea

A Survey Screen for the Prediction of Apnea (Maislin et al., 1995) was used to investigate the presence of the sleep disorder. The survey contains 13 questions regarding symptom frequency across four domains as follows: sleep-disordered breathing; difficulty sleeping; excessive daytime sleepiness; narcolepsy-like factor. The index for domain 1 (I1), sleep-disordered breathing, is used as a screening tool for sleep apnea. I1 has a reported (Maislin et al., 1995) internal consistency of .85-.93, and a test-retest reliability of .92. Assessment of the predictive ability of I1 has shown that it has a test efficiency score of .7, and that the presence of sleep apnea ranges from 20% in

respondents with $I1 < 1$, to 74% of those with $I1 = 4$. The predicative ability of $I1$ is significantly less than that of the MAP index (Maislin et al., 1995), although that does not present a problem for the current study, given that the purpose of investigating apnea status was for information gathering, rather than for indication of participation eligibility.

Geriatric Depression Scale five/fifteen item version (GDS5/15)

The Geriatric Depression Scale was used in the current study as the questionnaire was specifically developed to measure depression in the elderly (Yesavage et al., 1983). The Geriatric Depression Scale five/fifteen item version (GDS5/15) (Weeks, McGann, Michaels, & Penninx, 2003) is a short form of the 30-item Geriatric Depression Scale (GDS30) designed by Yesavage and colleagues (1983). The GDS30 comprises of 30 yes/no questions designed for self-administration, however the questions can be read out to the respondent by the examiner. It is fast and simple to administer, and is suitable for use with healthy, medically ill, and elderly with mild to moderate cognitive impairment within the community, acute care, or long-term care settings.

The directionality of answers scored for depression changes randomly. An example of the questions is 'Do you prefer to stay home rather than go out and doing new things?' The aim of the instrument is partially disguised by the title "Mood Assessment Scale" at the top of the questionnaire. The scale required around 10 minutes for administration.

The item-total correlations range from .32 to .83, with a mean of .56. Internal consistency (alpha) was .94, and split-half reliability was .94 (Brink et al., 1982). Abraham (1991) reported an internal consistency between .69 and .88 over 18 occasions during a 30-week period in frail, multiply impaired nursing home patients, aged from 71 to 97 years. The GDS30 has a high reliability, with reports of internal consistency ranging from .69 to .94, and test-retest reliability ranging from .85 to .92 (Spren & Strauss, 1991). It was further reported to have convergent validity over .73 and satisfactorily discriminated between depression and dementia (Spren & Strauss, 1991).

The shorter version, the 5 item version of the GDS has been validated (Hoyl et al., 1999). A study by Weeks, McGann, Michaels, and Penninx, (2003) compared a variety of short forms of the GDS in a sample that was mostly female, elderly, and residing independently prior to a short stay in acute care. It was concluded that the GDS5 was

the most valid form, with sensitivity of 97.9 and specificity of 72.7% at a cut-off score of 2/3 for not depressed/depressed. However, a false positive rate of 22.3% for the GDS5, lead Weeks and colleagues (2003) to develop a two-tier approach (GDS5/15) to depression diagnosis.

The Beck Anxiety Inventory (BAI)

The Beck Anxiety Inventory was utilised in the present investigation due to its strong psychometric characteristics in its use with older adult populations. The BAI (Beck, Epstein, Brown, & Steer, 1988) contains questions relating to common symptoms of anxiety. The BAI is comprised of 21 items designed to measure the severity of anxiety symptoms such as nervousness, numbness, and fear of losing control. The questionnaire took approximately 5-10 minutes to complete. The BAI has been used by adults of up to 92 years of age (Wetherell & Arean, 1997).

Sound psychometric properties have been established for the measure among older community, medical, and psychiatric outpatient samples (Morin et al., 1999; Steer, Willman, Kay, & Beck, 1994; Wetherall & Arean, 1997). Specifically, the measure has strong internal consistency, .85 to .92, when assessed with elderly people, mixed psychiatric samples, and patients with anxiety disorders. Good test-retest reliability has also been found for anxiety patients, .83 (de Beurs, Wilson, Chambless, Goldstein, & Feske, 1997).

Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was originally designed to provide a standardised and psychometrically sound measure of sleep quality. The intention was for the PSQI to discriminate between good sleepers and poor sleepers, to provide an index that is simple for clinicians to interpret and for subjects to utilise, and also to yield a quick, clinically useful assessment of a range of sleep disturbances that may impact the quality of sleep (Buysse, 1989). Test items were derived from a review of previous sleep quality measures, field testing, and clinical experience (Buysse, 1989). The PSQI is a self-rated questionnaire, which assesses sleep quality and disturbances over a 1-month time interval.

Clinical and clinimetric properties of the PSQI were assessed over an 18-month period with "good" sleepers (healthy subjects, $n = 52$) and "poor" sleepers (depressed patients, $n = 54$; sleep-disorder patients, $n = 62$). Satisfactory measures of internal homogeneity, consistency (test-retest reliability) and validity were obtained. It has a test-retest reliability of .85 for the global PSQI score, and internal consistency of .83 for both component and individual item scores (Buysse et al., 1989). These high values for internal consistency imply that each component measures a specific facet of sleep quality, while each item measures a specific aspect of the component. The PSQI also has high validity. A global PSQI score greater than 5 yielded a diagnostic sensitivity of 89.6% and specificity of 86.5% ($\text{kappa} = 0.75$, p less than 0.001) in distinguishing good and poor sleepers in clinical assessments. The clinimetric and clinical properties of the PSQI suggested its utility both in psychiatric clinical practice and research activities (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI also has good convergent validity, with reported correlations between the PSQI and related constructs all exceeding $r = .69$, and discriminant validity, with none of the reported correlations between the PSQI and unrelated constructs exceeding $r = .37$ (Carpenter & Andrykowski, 1998).

Sleep Beliefs Questionnaire (SBQ)

The Sleep Beliefs Questionnaire (SBQ) (Ware, Hood, Perlstrom, & Bond, 1996) provides a global estimate of attitudes towards sleep and potential effects of disturbed sleep. It is a 40-item self-report questionnaire, which consists of 40 statements concerning a variety of potential effects of disturbed sleep on life across the following four categories: quality of life, loss of control, expected or standard effects, and social impact. An example of the questions asked include: 'People are less likely to function well at work if they get less than seven hours sleep'. Although no literature on the psychometric properties of this scale was available, this scale was utilised as it was unique in the way it asked participants about their beliefs regardless of their central complaint. Instead of asking about personal effects of poor sleep, questions in the instrument were phrased about people's sleep in general.

World Health Organisation Quality of Life-Bref (WHOQoL)

The World Health Organization Quality of Life—Bref Version (WHOQOL—Bref) (Murphy, Herrman, Hawthorne, Pinzone, & Evert, 2000) was developed as a multi-dimensional

tool to produce a comprehensive and subjective quality of life (QoL) profile. The instrument, which has been developed from the longer 100 item version, has been adapted to the Australian environment. It was derived from data collected by using the WHOQOL–100 and even though this original instrument is highly reliable and valid, the shorter version was provided as an alternative form, suitable for use with those for whom a faster administration is preferable, such as with older adults.

The Australian WHOQOL-Bref (Murphy et al., 2000) is a 26 item instrument developed by the World Health Organisation to be used as an abbreviated standard measure of quality of life issues. It produces scores for four domains of QoL (physical health, psychological, social relationships, and environment) and a single item rating of perceived satisfaction of general health status and overall quality of life. A number of questions is dedicated to each domain; (a) physical health, for instance, “To what extent do you feel that physical pain prevents you from doing what you need to do?” (b) psychological, for instance, “How often do you have negative feelings such as blue mood, despair, anxiety, or depression?” (c) social relationships, for instance, “How satisfied are you with the support you get from your friends?” and (d) environment, for instance, “How healthy is your physical environment?”.

The questionnaire is identified as a cross culturally valid and sensitive measure of quality of life. The scale has been widely used in cross-sectional research with particular relevance to health sector issues. Validity evidence is strong and has been shown to be comparable to the WHOQOL–100, which has been shown to have an excellent ability to discriminate between ill and well respondents. WHOQOL–BREF scores correlate highly (.89 or above) with the WHOQOL–100. High values were found for construct validity of each domain (Physical health $r = .58$ to $.80$; Psychological $r = .20$ to $.70$; Social $r = .10^*$ to $.48$; and Environment $r = .17$ to $.45$) when correlated with widely used measures of health-related quality of life. * This non-significant result represents the correlation between the social WHOQOL-Bref and the physical function scale of the SF-36. That scale only measures the physical function facet of health-related QoL, and hence would not be expected to correlate highly with the social domain of the WHOQoL-Bref (Hawthorne, Richardson, et al., 2000). Coefficient alpha estimates have ranged from .66

to .94 in each domain (Chronister & Chan, 2006; WHOQOL Group, 1998), and test–retest reliability estimates have ranged from .56 to .87 with intervals from 2 to 8 weeks (WHOQOL Group, 1998).

Participant Information Sheet

The Participant Information Sheet described the nature and aims of the study. This was used to assist in explaining the research to potential participants (see Appendix 1).

Consent Form

A Victoria University Consent form was utilised to record the individuals' consent to participate in the current investigation (see Appendix 2).

Test Scoring

Mini Mental State Examination (MMSE)

Correct answers receive a score of '1' and wrong answers receive a score of '0'. The scale was scored by summing the number of correct answers. The highest possible total score for the MMSE was 30; high scores denote sound cognitive functioning. Generally, a total MMSE score of <24 is the recommended threshold for distinguishing demented and non-demented individuals (Folstein et al., 1975).

The Sleep Apnea Screen

Participants were asked to indicate the extent to which they had experienced each of the 13 symptoms during the past month. This was measured using a 6-point Likert-type scale (0 = 'never', and 5 = 5-7 times per week'). An average score was obtained for questions 1 to 3 to make up the disordered breathing factor, the only one of the 4 factors relevant to the current study. A cut-off score of 1 was used to exclude potential participants who were determined to have sleep apnea symptoms. An index is calculated for each domain by taking the average of non-missing responses for that domain. The index for domain 1 (I1), sleep-disordered breathing, is used as a screening tool for sleep apnea, where an I1 of ≥ 1 indicates the likely presence of sleep apnea (Maislin et al., 1995). The remaining domain indices can be used in conjunction with BMI, age, and gender, to calculate a multivariable apnea risk index (MAP index), although the MAP index was not calculated for the participants in this study.

Geriatric Depression Scale five/fifteen item version (GDS5/15)

The scale was scored by giving one point for each of the negatively answered 'yes or no' responses. The GDS5 was initially administered and if a score of 0 or 1 was obtained, the individual was classified as 'not depressed' and the assessment was complete. A score of 2 or more, however, indicated the administration of the GDS15 was required to render a more reliable assessment of the suggested depression. The recommended cut-off scores for mild depression was less than 4 and severe depression was more than 9.

The Beck Anxiety Inventory (BAI)

The respondents was required to rate how much he or she had been bothered by each of the 21 symptoms over the past week on a 4-point scale ranging from 0 = 'not at all' to 3 = 'severely'. The inventory was scored by summing the scores of the items. The highest possible total score was 63, whilst the lowest was 0. Scores under 7 indicate minimal anxiety or possibly denial, scores between 8 and 15 indicate mild anxiety, scores between 16 to 25 suggest moderate levels of anxiety, while scores ranging from 26 to 63 indicate severe anxiety.

Pittsburgh Sleep Quality Index (PSQI)

Nineteen individual items generate seven "component" scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The inventory was scored by summing the scores to generate a global PSQI score (range 0 to 21) from these seven component scores (range 0 to 3), with the higher scores reflecting more severe sleep complaints. Global scores at or above 5 indicated sleep disturbance.

Sleep Beliefs Questionnaire (SBQ)

The participants rated their agreement with each of the 40 statements on a 5 point Likert scale ranging from 1 = 'Strongly Agree' to 5 = 'Strongly Disagree'. All items were reversed scored, then summed into 4 different factors: the SBQ quality of life, SBQ loss of control, SBQ expected and standard effects, SBQ social impact. A higher score reflects a higher perceived impact of disturbed sleep.

World Health Organisation Quality of Life-Bref (WHOQoL)

Participants were asked to indicate their experience related to each of the items in the last 2 weeks by using a 5-point Likert-type rating scale. Domain scores have a range of 0 to 100, where a higher score indicates a better quality of life. Questions 3, 4, and 26 were reverse scored. Raw scores for each of the 4 domains were transformed using a formula where the lowest possible raw domain score was subtracted from the actual raw domain score and divided by the possible raw domain score range, this was then multiplied by 100.

Procedure

Ethical clearance to undertake the research was sought and obtained from the Victoria University Human Research Ethics Committee (HREC). The co-ordinator of each hostel was approached by the researcher. The inclusion and exclusion criteria were discussed and the co-ordinator provided a list of potential participants, who were then approached by the researcher. The participants were asked whether they classed themselves as a good sleeper or a poor sleeper. Potential participants that were suspected to suffer from a serious cognitive decline were administered the Mini Mental State, and excluded if they obtained a score below 23. The potential participants were screened by administering the GDS; anyone scoring above 19 was excluded from the study. Potential participants also took a Sleep Apnea test to determine whether they suffered from Sleep Apnea, and excluded from the study if potentially suffering from this condition. The participants that satisfied the inclusion and exclusion criteria and categorised themselves as either good sleepers or poor sleepers, were interviewed, meaning all measures were read out to the participants by the researcher. The researcher administered the PSQI to obtain data about the participants' range of sleep characteristics. This was followed by administration of the SBQ to assess the participants' attitudes towards sleep. The researcher subsequently administered the WHOQoL-Bref to measure the participants' quality of life. Finally, the researcher gathered data on the participants' anxiety by administering the BAI. The participants were informed that they could withdraw at anytime. Responses were scored and the overall scores entered into a data file, after which statistical analyses were performed.

Data Analysis

Data were entered into a spreadsheet and analysed using the Statistical Package for the Social Sciences (SPSS Version 14.0), and means and standard deviations were calculated for all the variables. Alpha level was set at .05 unless stated otherwise.

The first hypothesis were that overall sleep quality in self-categorised good sleepers (GS), would be better in self-categorised poor sleepers taking benzodiazepines (PSB), and self-classed poor sleepers not using benzodiazepines (PSNB). All three group means for global PSQI score were subjected to an Analysis of Variance (ANOVA) with between-subjects group factors. Where the univariate results were significant, the Least Significant Difference (LSD) post hoc analyses were performed.

The next hypotheses were that there would be significant differences in sleep variables between GS, PSB, and PSNB, these group means were analysed using a multivariate analysis of variance (MANOVA) with between-subjects group factors for the PSQI sleep variables, i.e. sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, and daytime dysfunction.

The hypotheses that GS would have better affective functioning (lower anxiety and depressed mood) than PSB, and PSNB, was tested using a MANOVA (between-subjects group factors). The dependent variables were depressed mood and anxiety.

The hypotheses which indicated that there would be significant mean differences in sleep beliefs between GS, PSB, and PSNB was tested using a between-subjects MANOVA. The dependent variables include general beliefs of quality of life, beliefs of loss of control, and beliefs of sleep's social impact.

Further hypotheses predicted higher quality of life in self-reported GS, when compared to self classified PSB and PSNB. The group means were analysed using a between-subjects MANOVA with dependent variables of global quality of life, overall perception of health, physical domain, psychological domain, social relationships domain, and environment domain.

The PSQI global score was then submitted to stepwise linear regression analyses as a dependent variable to determine which of the independent variables measured in the study provided the best predictors of global sleep quality. The independent variables in this analysis were all the questionnaire dependent variables (as shown in Table 1), except those from the PSQI.

Finally, two separate stepwise discriminant function analysis were computed to determine which, if any, of the dependent variables measured in the study could best differentiate the following groups from each other:

- PS on/off benzodiazepines
- GS and PS (both on/off benzodiazepines)

The stepwise method of discriminant analyses was preferred over the standard method in order to eliminate discriminating variables that do not add to the power of the discriminant functions.

For the above analysis the default settings for SPSS (Version 14) were applied.

RESULTS

General Sleep Quality (PSQI)

General sleep quality was derived from the global score of the PSQI of good sleepers (GS), poor sleepers using benzodiazepines (PSB), and poor sleepers not taking benzodiazepines (PSNB). Lower mean global scores depicted better sleep quality (Figure 1). Tests of assumptions (e.g. Homogeneity of the variance/covariance matrix) were conducted to establish that the parameters of the data were within acceptable limits allowing the use of analysis of variance with the present data.

An Analysis of Variance (ANOVA) was performed to examine differences in overall sleep quality between GS, PSB, and PSNB. Figure 1 demonstrates the extent to which self-categorised poor sleepers rated their quality of sleep negatively compared to good sleepers. The results showed that the mean global sleep quality for GS was 6.36 (SD = 3.24), for PSNB it was 11.16 (SD = 2.79), and for PSB it was 12.66 (SD = 4.06). The global sleep quality scores differed significantly between groups ($F(2,71) = 21.65, p = .00$). LSD post hoc contrasts showed that the global sleep quality was significantly better (i.e. lower) in self-categorised GS than in PSB and PSNB. PSB did not differ significantly from PSNB on global sleep quality.

Sleep Variables (Specific PSQI variables)

Sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, and daytime dysfunction were derived from responses of GS, PSB, and PSNB on the PSQI. Table 2 reports the means and standard deviations of responses to the PSQI on these variables between GS, PSB, and PSNB. Lower mean values denoted better subjective sleep (Table 2). These six sleep quality independent variables were analysed using a multivariate analysis of variance (MANOVA) and the results were reported in terms of Wilks' Lambda. The MANOVA demonstrated a significant difference in the sleep variables across the three groups ($F(12,132) = 7.02, p = .000$).

Univariate results indicated a significant difference between the three groups in sleep quality ($F(2,71) = 28.68, p = .000$) and a significant difference between groups in sleep

latency ($F(2,71) = 4.45, p = .015$). Furthermore, there was a significant difference across all the three groups in sleep duration ($F(2,71) = 11.86, p = .000$) and a significant difference was also found between groups in their habitual sleep efficiency ($F(2,71) = 11.55, p = .000$). Where the univariate results were significant, LSD post hoc tests were carried out. LSD post-hoc contrasts (summarised in Table 2) indicated significantly ($<.01$) lower sleep quality in PSB than in GS; and significantly lower sleep quality in PSNB in comparison to GS. LSD post-hoc contrasts demonstrated significantly longer sleep latency in PSB than in GS; it also indicated significantly longer sleep latency in PSNB in comparison to GS. LSD post-hoc contrasts demonstrated significantly longer sleep duration in PSB than in PSNB. LSD post-hoc contrasts observed significantly shorter sleep duration in PSNB when compared to GS. LSD post-hoc contrasts indicated significantly poorer habitual sleep efficiency in PSB than in GS. It also observed worse habitual sleep efficiency in PSNB when compared to GS.

Table 2

Means (Standard Deviations) of Sleep Variables (PSQI) Between Groups of Sleepers (Lower Means Depict Better Sleep)

| | Good sleepers (GS) | Poor sleepers not using benzodiazepines (PSNB) | Poor sleepers using benzodiazepines (PSB) | Significant Post hoc comparisons |
|---------------------------|--------------------|--|---|----------------------------------|
| Sleep quality | .68 (.65) | 2.16 (.58) | 1.95 (.97) | GS < PSB GS < PSNB |
| Sleep latency | 1.14 (1.08) | 1.94 (1.15) | 2.00 (1.00) | GS < PSB GS < PSNB |
| Sleep duration | .95 (.95) | 2.23 (.88) | 1.52 (1.03) | GS < PSNB PSB < PSNB |
| Habitual sleep efficiency | .91 (1.19) | 2.29 (1.04) | 2.24 (1.14) | GS < PSB GS < PSNB |
| Sleep disturbance | 1.32 (.57) | 1.26 (.44) | 1.14 (.57) | |
| Daytime dysfunction | .64 (.79) | 1.10 (.65) | .86 (.85) | |

Affective Functioning (Anxiety and Depressed Mood)

Tests of assumptions (e.g. Homogeneity of the variance/covariance matrix) were conducted to establish that the parameters of the data were within acceptable limits allowing the use of a multivariate analysis of variance with the present data.

Anxiety levels were derived from responses on the BAI and depressed mood was derived from responses on the GDS of GS, PSB, and PSNB. Higher mean scores indicate higher anxiety and depressed mood levels (Table 3). These two affective functioning dependent variables were analysed in a multivariate analysis of variance (MANOVA) across the three groups of sleepers and the results were reported in terms of Wilks' Lambda. The MANOVA demonstrated a significant difference in the anxiety and depressed mood levels across the three groups ($F(4,140) = 5.06, p = .001$). There was a significant difference between sleepers in anxiety ($F(2,71) = 6.83, p = .002$), and a significant difference between sleepers in depressed mood ($F(2,71) = 4.55, p = .014$). Descriptive statistics and post hoc results for anxiety and depressed mood between GS, PSB, and PSNB were reported in Table 3. LSD post-hoc contrasts demonstrated significantly lower anxiety levels in PSB than PSNB and significantly higher anxiety levels in PSNB when compared to GS. LSD post-hoc contrasts indicated significantly higher depressed mood in PSB than in GS and significantly higher depressed mood in PSNB in comparison to self-reported GS. All other affective functioning group differences were non-significant.

Table 3

Means (Standard Deviations) of Affective Disturbance Between Sleepers (Higher Means Depict Higher Levels of Anxiety and Depressed Mood Levels)

| | Good sleepers (GS) | Poor sleepers not using benzodiazepines (PSNB) | Poor sleepers using benzodiazepines (PSB) | Post hoc comparisons |
|----------------|--------------------|--|---|-------------------------|
| Anxiety | 4.50 (4.03) | 11.74 (9.31) | 6.52 (6.88) | GS < PSNB PSB < PSNB |
| Depressed mood | 0.64 (2.13) | 2.84 (3.48) | 3.43 (3.83) | GS < PSB GS < PSNB |

Sleep Beliefs (SBQ)

Sleep beliefs about quality of life, beliefs about loss of control, and beliefs about the impact sleep has on social interactions were derived from responses of GS, PSB, and PSNB on the SBQ. Higher mean scores depicted more negative sleep beliefs (Table 4). The three factors of sleep beliefs were analysed using a MANOVA and the results were reported in terms of Wilks' Lambda. The MANOVA yielded a non-significant difference in the sleep beliefs of GS, PSB, and PSNB ($F(8,136) = 1.53, p = .154$). There was no significant difference between the three groups in sleep beliefs related to quality of life, in sleep beliefs related to loss of control, or in sleep beliefs associated with social impact. Table 4 depicts the means and standard deviations of sleep beliefs related to their life quality, loss of control, and beliefs about the social impact of sleep between the three groups.

Table 4

Means (Standard Deviations) of Sleep Beliefs Between Sleepers

| | Good sleepers | Poor sleepers not using benzodiazepines | Poor sleepers using benzodiazepines |
|-------------------------------|---------------|---|-------------------------------------|
| Beliefs about quality of life | 67.32 (2.57) | 63.77 (2.16) | 59.38 (2.63) |
| Beliefs about loss of control | 23.36 (3.25) | 22.22 (2.74) | 22.42 (5.39) |
| Beliefs about social impact | 27.40 (4.73) | 25.67 (5.50) | 25.38 (7.47) |

Quality of life (WHOQOL-BREF)

Quality of life variables included quality of life in the physical domain, psychological domain, social relationships domain, and environment domain derived from responses to the WHOQOL-BREF. Higher mean scores denoted higher quality of life within the respective domain (Table 5). These four domains variables were subjected to a

MANOVA and the results were reported in terms of Wilks' Lambda. The MANOVA demonstrated a significant difference in the quality of life facets of the three groups ($F(12,132) = 1.86, p = .045$). On a univariate ANOVA there was no significant difference between sleepers in the physical and health domain of quality of life. There was a significant difference between the three groups in the psychological domain of quality of life ($F(2,71) = 4.75, p = .012$) and significant differences between groups in the social relationships domain of quality of life ($F(1,71) = 4.23, p = .018$). However, there were no significant differences across the three groups in the environment domain of quality of life. Descriptive statistics and post hoc results for quality of life in the physical, psychological, social relationships, and environment domains between GS, PSB, and PSNB were reported in Table 5. LSD post-hoc contrast indicated a significantly higher psychological domain quality of life difference in GS, in comparison to PSNB. It also showed a higher quality of life in the psychological domain of GS when compared to PSB. Furthermore, LSD post-hoc contrasts demonstrated significantly higher quality of life in the domain of social relationships in GS individuals than in PSB.

Table 5

Means (Standard Deviations) of Quality of Life Domains Between Sleepers (Higher Means Denoted Higher Quality of Life)

| | Good sleepers (GS) | Poor sleepers not using benzodiazepines (PSNB) | Poor sleepers using benzodiazepines (PSB) | Post hoc comparisons |
|-----------------------------------|--------------------|--|---|-----------------------|
| QoL – Physical and health domain | 67.05 (3.36) | 57.81 (2.83) | 61.91 (3.44) | |
| QoL – Psychological domain | 76.96 (3.29) | 64.07 (2.77) | 66.52 (3.37) | GS > PSNB GS > PSB |
| QoL – Social relationships domain | 73.41 (3.10) | 68.48 (2.61) | 60.62 (3.17) | GS > PSB |
| QoL – Environment domain | 86.91 (2.35) | 84.84 (1.98) | 86.43 (2.40) | |

PSQI Predictors

A stepwise multiple regression test was performed to determine whether any of the affective functioning, sleep belief, and quality of life variables significantly predicted global sleep quality as measured by the PSQI Global score using all the participants in the study (n=74). The results are presented in Table 6. The following variables did not enter the regression results: depressed mood, beliefs about loss of control, beliefs about social impact, QoL – Physical and health domain, and QoL – Social relationships domain.

Table 6

Stepwise Multiple Regression Results for the PSQI Global Score.

| PSQI Global score | |
|-------------------|--------|
| Adjusted R2 | 0.47 |
| <i>df</i> | 5, 73 |
| <i>F</i> | 14.085 |
| Significance | 0.000 |

Note. Independent variables: Anxiety, day-time dysfunction, sleep beliefs concerning quality of life, QoL- psychological domain, and QoL – environment domain.
Dependent variable: PSQI global score.

The results of the regression analysis demonstrated that anxiety, day-time dysfunction, sleep beliefs concerning quality of life, QoL- psychological domain, and QoL – environment domain were significant predictors of global sleep quality. Using the Global PSQI as the dependent variable, these five variables entered the model with Adjusted R2 values as shown below.

- QoL- psychological domain .261
- Sleep beliefs concerning quality of life .338
- QoL – environment domain .390
- Anxiety .435
- Day-time dysfunction .473

Anxiety, day-time dysfunction, sleep beliefs concerning quality of life, QoL- psychological domain, and QoL – environment domain were significant predictors of global sleep quality, and together explained 47.3 percent of the variation (using the adjusted R²) in PSQI global sleep quality. Individuals' beliefs concerning sleep's impact on quality of life was the best single predictor of PSQI global sleep quality ($t = 3.07$, $p = .003$, Beta = .298).

Differences Between Poor Sleepers Not Taking Benzodiazepines and Poor Sleepers Taking Benzodiazepines.

Tests of assumptions (e.g. Equality of the variance/covariance) were conducted to establish the validity of using a stepwise discriminant analysis with the present data of poor sleepers not using benzodiazepines and those on benzodiazepines. These tests demonstrated that none of the stepwise discriminant analysis assumptions were violated.

All the variables listed as dependent variables in Table 1 (except Global PSQI) were subjected to a stepwise discriminant analysis and the results were reported in terms of Wilks' Lambda. The stepwise discriminant analysis demonstrated a significant difference in the sleep duration, habitual sleep efficiency, and beliefs concerning the impact of sleep on quality of life, of the two poor sleeper groups (Wilks = .731, Chi-square(3) = 15.21, $p = .002$). All the other variables listed as dependent variables in Table 1 (except Global PSQI) that were subjected to a stepwise discriminant analysis were not significant, hence not included. Table 7 indicates the variables that were entered into the stepwise discriminant analysis.

Table 7

Stepwise Discriminant Function Analysis Between Poor Sleepers on Benzodiazepines and Not on Benzodiazepines.

| | Wilks Lambda | F | P |
|---|-----------------|-------|------|
| Sleep duration | .879 | 6.908 | .011 |
| Habitual sleep efficiency | .796 | 6.274 | .004 |
| Beliefs about impact on quality of life | .731 | 5.895 | .002 |

The results indicated that 67 percent of the self classed poor sleepers on benzodiazepines and 77 percent of the poor sleepers not taking benzodiazepines were correctly classified using the three variables of sleep duration, habitual sleep efficiency, and beliefs concerning the impact of sleep on quality of life.

Differences Between Poor Sleepers and Good Sleepers.

In this analysis the two groups of poor sleepers were combined into one group. Tests of assumptions were conducted to establish the validity of using a stepwise discriminant analysis with the present data. These tests demonstrated that none of the stepwise discriminant analysis assumptions were violated.

All the dependent variables in Table 1 (except Global PSQI) were subjected to a stepwise discriminant analysis and the results were reported in terms of Wilks' Lambda. The stepwise discriminant analysis demonstrated a significant difference in the sleep quality, habitual sleep efficiency, and sleep disturbance, of the good and poor sleeper groups (Wilks =.490, Chi-square(3) = 50.22, p = .000). All the other variables listed as dependent variables in Table 1 (except Global PSQI) that were subjected to a stepwise discriminant analysis were not significant, hence not included. Table 8 indicates the variables that were entered into the stepwise discriminant analysis.

Table 8

Stepwise Discriminant Function Analysis Between Good and Poor Sleepers

| | Wilks Lambda | F | p |
|---------------------------|--------------|--------|------|
| Sleep quality | .562 | 56.322 | .000 |
| Habitual sleep efficiency | .522 | 32.472 | .000 |
| Sleep disturbance | .490 | 24.239 | .000 |

The results indicated that 90.9 percent of the self categorised good sleepers and 84.6 percent of the self categorised poor sleepers cases were correctly identified using the variables of sleep quality, habitual sleep efficiency, and sleep disturbance.

Discussion

Sleep variables

The first hypothesis was that self categorised good sleepers would have better overall sleep quality than self categorised poor sleepers, but no significant difference in overall sleep quality in poor sleepers taking and not benzodiazepines. This was supported by the data of the present study. The mean values for the Global PSQI scores indicated that older adults who categorised themselves as good sleepers had better global sleep quality than those who categorised themselves as poor sleepers. However, as expected, due to the possibility of tolerance effects, there was no difference in global sleep quality between the poor sleepers that were taking benzodiazepine hypnotics and older adults not using these medications to improve their sleep. Previous research with older adults in residential care has proposed that self-classified good and poor sleepers categorise themselves in that manner due to the way that they thought about their own sleep quality, in the wider context of their construction of normal sleep, which was based on their cognitive comparison strategies, rather than their classification being grounded on their actual experience of sleep (Davis, Hood, & Bruck, 2007). The process of downward social comparison, whereby people search for comparison with others worse off than themselves, is suggested to improve personal satisfaction, while upward social comparison may contribute to the individual feeling more disappointed with their experiences. For older people downward social comparison is believed to lend a degree of protection against the loss of control that is often an adjunct to aging (Heckhausen & Brim, 1997; Frieswijk et al., 2004). Davis, Hood, and Bruck (2007) found that those categorising themselves as good sleepers utilised downward social comparison, while those classifying themselves as poor sleepers tended towards upward social comparisons. This occurrence might also, at least in part, account for mixed results as to whether subjective and objective sleep quality and sleep parameters concur in older people (Chong, Fujun, & Chunying, 2000; Riedel & Lichstein, 1998; Vitiello, Larsen & Moe, 2004).

The findings of the current study for the Global PSQI scores in older people who classed themselves as good sleepers had better global sleep quality than the self-categorised poor sleepers. This was consistent with past research which has shown that self categorized poor sleepers appear to demonstrate poorer overall sleep quality than self

categorized good sleepers (Lichstein, Durrence, Riedel, & Bayen, 2001; McRae et al., 2003; Vitiello, Larsen and Moe, 2004). It appears that this result is parallel to other studies regardless of the type of instrument used to measure global sleep quality. Vitiello, Larsen and Moe (2004) highlighted that both subjectively and objectively categorised good sleepers reflected better global sleep quality on the PSQI than subjectively and objectively categorised poor sleepers.

The finding that there was no difference in global sleep quality between the poor sleepers that were taking benzodiazepine hypnotics and older adults not using these medications to improve their sleep was supported by that of Holbrook and colleagues (2000a) who suggested there was no association between benzodiazepine hypnotic use and self-reported overall quality of sleep. Previous research using a within groups design provided further support for the findings of the current investigation. Petrovic and colleagues (1999) and Ramesh and Roberts (2002) compared the subjective overall sleep quality of aged individuals taking benzodiazepine hypnotics to their sleep quality after discontinuation of these hypnotics and found no significant difference in reported overall sleep quality. While the mean and median values show important and significant differences between good sleepers and poor sleepers, both on and not on benzodiazepine hypnotics, there is a large overlap and a large range. Furthermore, this is demonstrated by the high standard deviation, as a measure of variability. Clearly some good sleepers are reporting sleep that is equivalent to that reported by some poor sleepers.

Overall or global sleep quality, as measured by the PSQI was the sum of all the self-report variables, while sleep quality was the individual score to the rating of one question regarding the individuals' perception of their sleep. The prediction that self-reported good sleepers would have significantly better sleep quality than poor sleepers, but no significant difference in sleep quality between poor sleepers not taking benzodiazepine hypnotics and poor sleepers using benzodiazepines was supported by the current data set. The results indicated that older adults who were self-categorised good sleepers had better sleep quality than those who were self-categorised as poor sleepers. However, as predicted there was no difference found in the sleep quality of poor sleepers on benzodiazepine hypnotics and those not using benzodiazepines. Such findings replicate those already reported in the literature (Lichstein, Durrence, Riedel, & Bayen, 2001).

Lichstein and colleagues highlighted that the seniors diagnosed with insomnia, whether primary or secondary, had poorer ratings of the one question describing quality of sleep from a subjective perspective than those not diagnosed with insomnia. The current result of hypnotic and non-hypnotic poor sleepers was inconsistent with the studies reviewed by Estivill and colleagues (2003) who outlined that benzodiazepine hypnotics increase sleep quality, as measured by PSG, when compared to non-hypnotic users with insomnia. This inconsistency can be accounted for by the short-term duration the participants were taking benzodiazepine hypnotics for. As outlined in the literature review, older adults taking benzodiazepine hypnotics in the short term reported better sleep quality objectively and subjectively, however, when considering older adults taking these pharmacological agents for longer than four weeks the association between benzodiazepine hypnotic use and self-reported better sleep quality is no longer apparent objectively or subjectively (Holbrook, Crowther, Lotter, Cheng, & King, 2000a; Petrovic, Pevernagie, Van Den Noortgate, Mariman, Michielsen, & Afschrift, 1999).

A significant difference in parameters of self-rated sleep onset latency between good sleepers and poor sleepers taking and not taking benzodiazepines, was found, with the good sleepers taking less time to fall asleep. The results of the current study were consistent with previous literature which has shown that good sleepers had subjectively and objectively shorter sleep latency when compared to poor sleepers (Bliwise, 1992; Lichstein, Durrence, Riedel, & Bayen, 2001; Riedel & Lichstein, 1998). Bliwise (1992) found that subjectively, poor sleepers reported significantly longer sleep latency, and that their sleep latency was significantly longer when measured objectively with polysomnography. Subjective sleep latency has been found to be the best measure in predicting sleep-satisfaction differences in older people with insomnia (Riedel & Lichstein, 1998). The authors highlighted the importance of subjective short sleep latencies in predicting greater satisfaction with sleep.

The current finding of a lack of significant difference in sleep latency between poor sleepers taking and not taking benzodiazepine hypnotics, was concordant with a paper by Holbrook, Crowther, Lotter, and Endeshaw (2001). Their meta-analysis indicated no significant difference in subjective and objective sleep latency when comparing insomnia patients using benzodiazepine hypnotics with those not using benzodiazepines. The current result was inconsistent with the studies reviewed by Estivill and colleagues

(2003) who found that benzodiazepine hypnotics decrease sleep latency, as measured by PSG. The differences in findings may be accounted for by the inconsistencies between study cohorts' length of time of hypnotic agent consumption, the dosage, and also the severity of poor sleep or insomnia.

A significant difference in self-rated sleep duration between good sleepers, poor sleepers not taking benzodiazepine hypnotics, and poor sleepers using benzodiazepines, was found. It was ascertained that self-categorised good sleepers had longer sleep duration than poor sleepers not taking benzodiazepine hypnotics. Furthermore, the results showed that poor sleeping older adults using benzodiazepines had longer sleep duration when compared to self-categorised poor sleepers not on hypnotics. However, there was no difference in sleep duration between good sleepers and poor sleepers on benzodiazepines. This was consistent with past research which has demonstrated that good sleepers had longer total sleep time in comparison to poor sleepers (Bliwise, 1992; Groeger, Zijlstra, & Dijk, 2004; Lichstein, Durrence, Riedel, & Bayen, 2001; Ouellet and Morris, 2006). Bliwise (1992) found that subjectively, good sleepers reported significantly longer sleep duration than poor sleepers not on benzodiazepines, and this was supported by the polysomnographic data. Moreover, sleep duration as measured by the PSQI was found to be one of the best descriptors of subjective sleep satisfaction (Ouellet and Morris, 2006). The findings of the current study are aligned with those of Holbrook, Crowther, Lotter, and Endshaw's (2001) meta-analysis contrasting sleep variables of poor sleepers using benzodiazepines with those not taking benzodiazepines. The researchers found that poor sleepers receiving benzodiazepines had significantly increased total sleep duration, but the shortened length of sleep latency was not significant.

The results of the current study were consistent with the meta-analysis and review findings that indicated that insomnia patients receiving benzodiazepines had increased total sleep duration in comparison to those not using benzodiazepines (Estivill et al., 2003; Holbrook, Crowther, Lotter, & Endshaw, 2001). Estivill and colleagues (2003) reviewed a series of investigations that compared objectively measured total sleep time or sleep duration between benzodiazepine users and non-users.

The present study found a significant difference in self-reported habitual sleep efficiency between good sleepers and poor sleepers, regardless of whether or not they were taking benzodiazepine hypnotics, with good sleepers having better habitual sleep efficiency. However, no significant difference in habitual sleep efficiency was indicated between poor sleepers taking benzodiazepines and those not using sleep medications. The result that good sleepers had better habitual sleep efficiency when compared to poor sleepers supported past research (Bliwise, 1992; Lichstein, Durrence, Riedel, & Bayen, 2001). Bliwise (1992) found that good sleepers reported significantly better habitual sleep efficiency, and this was consistent with their polysomnographic data. Sleep efficiency has been found to correlate highly with the sleep satisfaction reported by good and poor sleepers (Riedel & Lichstein, 1998). The researchers concluded that satisfied sleepers reported significantly better sleep efficiency than poor sleepers.

No significant difference in self-reported sleep disturbance between good sleepers and poor sleepers was found. Moreover, as predicted, no significant difference in sleep disturbance was found between poor sleepers taking and not taking benzodiazepine hypnotics. These results were consistent with the existing literature. Sleep disturbance measured by the self-reported number of awakenings during the night was not found to correlate with whether aged persons categorised themselves as good sleepers or poor sleepers (Bliwise, 1992). One explanation for this could be that when self-report of time spent awake was compared to PSG recordings it was found that senior poor sleepers have a tendency to underestimate how much time they spend awake throughout the night (Libman, Creti, Levy, Brender, & Fichten, 1997). Furthermore, sleep disturbance as evaluated by the PSQI was found to be one of the best descriptors of subjective sleep satisfaction (Ouellet and Morris, 2006).

The result of no difference in daytime dysfunction between poor sleepers not taking benzodiazepines and those using benzodiazepine hypnotics failed to parallel the meta-analysis findings that older adults receiving benzodiazepines were more likely than those receiving a placebo to complain of daytime dysfunction (Holbrook, Crowther, Lotter, & Endeshaw, 2001). The differences in results could possibly be explained by the differences in design, including the measurement of daytime drowsiness or dysfunction, and the administration of a placebo.

Anxiety and depressed mood

Significant differences in anxiety between poor sleeping benzodiazepine users, poor sleepers not taking benzodiazepine hypnotics and good sleepers were found in the present data set. The results showed that good sleeping older adults had lower levels of anxiety in comparison to poor sleepers not on hypnotics. As expected, it was also indicated that elderly individuals taking benzodiazepine hypnotic medication were less anxious than poor sleepers not using benzodiazepines. Conversely, the results demonstrated no difference in anxiety levels between good sleepers and poor sleepers on benzodiazepine hypnotics. This can be explained by the pharmacological properties of benzodiazepines. This finding is similar to the findings in a study undertaken by Voyer, Landerville, Moisan, Tousgnant, and Preville (2005). Voyer and colleagues (2005) highlighted that when anxiety was assessed by DSM-IV interviews, it was not linked with benzodiazepine use, however, their responses on the self-reported measure (State Trait Anxiety Inventory) did indicate a significant association between anxiety and benzodiazepine medication use in older adults experiencing disturbed sleep. The authors demonstrated that according to DSM-IV structured interviews anxiety disorders in older adults with insomnia were not associated with benzodiazepine consumption. This appears to contrast with results found in a longitudinal study that indicated high level of benzodiazepine use (43%) in older adults with anxiety (Schuurmans, Comijs, Beekman, de Beurs, Deeg, Emmelkamp, & van Dyck, 2005), and findings that showed anxiety to be a predominant clinical factor observed in patients on chronic benzodiazepine hypnotic treatment (Petrovic, 2002). They interpreted their findings in terms of the idea that mood status may be influenced according to how it is measured. It is probable that a lower prevalence of anxiety symptoms according to self-report measures might stem from denial or social desirability, which may incline older adults to deny the presence of symptoms in questionnaires such as the STAI or BAI (Voyer, et al., 2005).

The result of the current investigation is parallel with the finding of Lichstein, Durrence, Riedel, and Bayen (2001) and Jensen, Dehlin, Hagberg, Samuelsson, and Svensson (1998) that indicated poor sleepers had higher anxiety levels than those that had no sleep problems, as assessed by the American Sleep Disorder Association (1990) criteria. Analyses of severity of daytime impairment found that the effects of anxiety were linked to disturbed sleep (Lichstein, Durrence, Riedel, & Bayen, 2001). The result of the

present investigation also lends support to the cognitive theory of insomnia, which posits that a combination of worry, arousal, and distress plunges the individual into an anxiety state, a state that which results in difficulty falling asleep and maintaining sleep (Espie, 2002). Furthermore, the current result is consistent with the finding in a previous investigation of at least a trend of difference in general anxiety between good sleepers and poor sleepers (Bliwise, 1992). Poor sleepers consistently reported more psychological symptoms, such as phobic anxiety and paranoid ideation, and a trend of differences were observed for general anxiety (Bliwise, 1992). As discussed earlier, the Bliwise (1992) study had a relatively small sample size that might have accounted for this result proposing a trend but not reaching statistical significance. Elderly individuals taking benzodiazepine hypnotic medication having lower levels of anxiety than poor sleepers not using benzodiazepines could be accounted for by the anti-anxiety or sedative properties of benzodiazepines. However, as discussed further on, poor sleepers not on benzodiazepine hypnotics did not have clinically high anxiety.

A significant difference in depressed mood between both poor sleeping older adults taking and not taking benzodiazepine hypnotics and good sleepers was found. The result of the present study did not indicate depression in poor sleeping older adults, in fact they were excluded from the study. The results showed that self-categorised poor sleepers not on benzodiazepines and those on benzodiazepines have higher levels of depressed mood in comparison to elderly individuals who reported they slept well. However, no difference in depressed mood was observed between poor sleepers not taking benzodiazepines and those on benzodiazepine hypnotics. The finding that poor sleepers have higher levels of depressed mood than good sleepers was supported by previous research (Jensen, Dehlin, Hagberg, Samuelsson & Svensson, 1998; Lichstein, Durrence, Riedel, & Bayen, 2001). Some of these studies included participants with depressed mood, while some involved clinically depressed older adults. Lichstein, Durrence, Riedel, and Bayen (2001) found significantly higher scores on the GDS in individuals who met sleep disturbance criteria according to the American Sleep Disorder Association (1990) than those who failed to meet sleep disturbance criteria.

Jensen and colleagues (1998) assessed depression according to the ICD-10 (World Health Organisation, 1992) and included older individuals with depression ranging from mild to severe. They found that depression was associated with increasing severity of

poor sleep (Jensen, Dehlin, Hagberg, Samuelsson, & Svensson, 1998). The result supported the finding of a previous investigation using polysomnography that found a trend of difference in depressive symptoms between good sleepers and poor sleepers. The result may have not reached significance in the Bliwise investigation due to the small sample size and the exclusion of males (Bliwise, 1992). The current investigation did not use polysomnographic data, and hence cannot rule out or evaluate the specific contribution of specific sleep pathologies to either the sleep complaint or to the reported worse mood. The finding of the current study that indicated no difference in depressed mood between poor sleepers not taking benzodiazepines and those on benzodiazepine hypnotics supported the findings of an investigation conducted by Voyer, Landerville, Moisan, Tousgnant, and Preville (2005). The authors suggested that according to DSM-IV structured interviews depression in older adults with insomnia was not associated with benzodiazepine consumption, however, it was associated when using self-report measures. Voyer and colleagues (2005) outlined that although depression, as assessed by DSM-IV interviews, was not linked with benzodiazepine use, the self-reported measure (GDS) did indicate a significant association with benzodiazepine medication use. They argued that aged individuals may opt for expressing their psychological difficulties as a physical symptom such as sleeping problems (Voyer et al., 2005). The discrepancies in associations and prevalence between sleep disturbance and psychosocial and mental health problems such as anxiety and depression can also be accounted for by the method of report selected by researchers. The number of anxiety and depressed mood symptoms identified can potentially be influenced by the type of instrument and manner of administration, such as an interview on the telephone, questionnaire sent by mail, self-report, or face-to-face questioning (Voyer et al., 2005).

It was also evident that poor sleepers not taking benzodiazepines were not at a higher risk for psychosocial/mental health problems such as depressed mood. The literature indicates that dysthymic disorders and depression are predominant clinical factors observed in patients on chronic benzodiazepine hypnotic treatment (e.g. Petrovic, 2002; Voyer, et al., 2005). However, it was apparent from the current results that since poor sleepers with depression were excluded, there was no difference in depressed mood between seniors taking benzodiazepines and those not. The literature indicates mixed results about the association of anxiety with benzodiazepine hypnotic use in older poor sleepers. It was evident from the current investigation that elderly individuals taking

benzodiazepine hypnotic medication were less anxious than poor sleepers not using benzodiazepines. This may be accounted for by the bio-physiological effects of benzodiazepines on reducing anxiety levels. This raises the question as to whether poor sleepers not on benzodiazepine hypnotics are more at risk for psychosocial and mental health problems such as anxiety than those taking benzodiazepines. Although the subjective poor sleepers not taking benzodiazepine hypnotics had higher anxiety levels, these levels did not reach the cut-off scores to indicate clinical anxiety. Perhaps the current sample was not large enough to demonstrate these differences of benzodiazepine users having anxiety scores more consistent with good sleepers and lower than poor sleepers not taking benzodiazepines hypnotics, or perhaps they were not recommended as participants for the study by the nurses. This indicates a trend that needs to be further explored. Concomitant anxiety has been found to be significantly associated with higher consumption and higher daily dosage of hypnotics in older adults (Huang & Lai, 2005). Unfortunately, daily dosage was not explored in the current investigation. Older adults on benzodiazepine may have lower anxiety levels similar to self-reported good sleepers but due to the finding that poor sleepers not on benzodiazepines do not have clinically high anxiety, and the risk of increased anxiety following benzodiazepine hypnotic withdrawal (Cannard, 1996), not prescribing and not taking benzodiazepine hypnotics certainly has benefits worth considering.

As demonstrated by the current results and those of other researchers, insomnia, depressed mood, and anxiety have a strong relationship. Researchers have asserted that anxiety and depressed mood can cause insomnia and, among aged persons, the individual experiencing insomnia can develop an anxiety disorder or depression (Voyer, et al., 2005). Psychosocial and mental health problems such as anxiety and depressed mood appear to have a circular association with sleep for insomnia. On one hand, difficulty sleeping is a diagnostic indicator for anxiety and depression, while anxiety and depression represent the factors most often attributed as the cause of insomnia or poor sleep (Buysse, 2004; Pallesen et al., 2002; Olafsdottir, Marcusson & Skoog, 2001; Steiger, 2003). "Depressed adults who are not treated are more likely to develop insomnia, and adults with unresolved insomnia are more likely to develop depression" (Kryger, Monjan, Bliwise, Ancoli-Israel, 2004, p. 29). The issue of somatisation of psychosocial and mental health problems in the elderly population needs to be addressed in order to determine the extent of the cause and effect relationship with self-

reported poor sleep. This issue also has clinical implications for physicians prescribing benzodiazepine hypnotic agents to seniors.

Sleep beliefs

The results from the current data indicated a trend demonstrating that self-rated good sleepers had stronger beliefs about poor sleep having a negative impact on a person's quality of life. However, no significant difference in beliefs of loss of control over disrupted sleep, and in beliefs about the negative impact disruptive sleep has on the person's social life between good sleepers and poor sleepers using and not using benzodiazepines hypnotics was found. The current results indicated that self-rated good sleepers did not have stronger beliefs of loss of control over disrupted sleep, or in beliefs about the negative impact disruptive sleep has on the person's social life, when compared to the beliefs of poor sleepers taking or not taking benzodiazepines. There was also no difference in sleep beliefs between poor sleeping elderly taking benzodiazepines and those not. These results failed to parallel the findings of previous research that indicated differences between poor sleep and negative beliefs and perceived negative consequences of insomnia and disturbed sleep (Lichstein, Durrence, Riedel, & Bayen, 2001). The investigators found that time of sleep onset latency was significantly related to heightened beliefs and perceptions of the negative repercussions of insomnia and its impact on the sufferers' daytime dysfunction.

A WHO study (Costa et al., 1996) and a longitudinal study of older adults examined every year between the ages of 80 and 89 years conducted by Jenson, Dehlin, Hagberg, Samuelsson, and Svensson (1998) ascertained that an increasing severity of insomnia was linked with beliefs about a lack of justice in the world. They interpreted their findings as a reflection that individuals with this type of attitude may often ruminate over what they believe to be injustices, in so doing, hindering their sleep. Costa and colleagues, alluded to the possibility that although beliefs may be associated with poor sleep, these beliefs may not necessarily be about the negative consequences of disrupted sleep.

The results of the current study departed from the sleep-interfering and sleep-interpreting process theoretical model, as discussed earlier (Lundh, 2000). The model proposed that insomnia results from the interaction between sleep-interfering processes (such as different types of arousal, and processes whereby various stimuli, behaviours,

and cognitive activities lead to arousal) and sleep-interpreting processes, such as sleep related beliefs, attitudes, and attributions. The sleep-interpreting processes measured in the current study were not a significant factor of difference between those who reportedly slept well and those that complained of poor sleep. Hence, the result of the present study did not lend support to the findings of a body of literature that indicates that poor sleepers have more negative beliefs about the impact of a poor night's sleep (Edinger, et al., 2000; Fichten, et al., 1995; Morin et al., 1993). The current results departed from the findings that demonstrate that poor sleepers have more negative beliefs about the extent of loss of control over their sleep, in comparison to good sleepers (Edinger, et al., 2000; Fichten, et al., 1995; Morin et al., 1993). These investigations demonstrated that older adults with objective insomnia had significantly more dysfunctional beliefs about the negative consequences on daily life and their loss of control over sleep than did their objective good sleeper counterparts. It was also demonstrated that that positive attitudes and beliefs about sleep at post cognitive-behavioural treatment were linked with better maintenance of sleep improvements at 3, 12, and 24 month follow up assessments (Morin, Blais, & Savard, 2002). It was concluded that particular dysfunctional attitudes and beliefs pertaining to sleep may lend to actual sleep difficulty, however they agreed that more research is required to confirm a cause-effect relationship (Edinger, et al., 2000). The specific difference in populations may account for the present results' lack of support, results of the current study were based on older adults that self-reported poor sleep and did not necessarily have a diagnosis of insomnia. Age might also be a possible reason as to why these results differ from previous findings, as these previous studies have included younger and older adults in their sample population. It is possible that sleep related beliefs, attitudes, and attributions in older adults are not as strongly linked to poor sleep as they are in younger adults. A further possible reason that might explain the current results' lack of support may be due to the fact that the Sleep Beliefs Questionnaire used has not been validated for an older population.

Quality of life

The results of the present data showed a significant difference in the psychological domain of quality of life between self-reported good sleepers and poor sleepers using and not using benzodiazepine hypnotics. It was found that self-categorised good sleepers had a higher quality of life within the psychological domain in comparison to

poor sleepers whether they were taking benzodiazepines or not. On the other hand, as hypothesised, the results showed no difference in the psychological domain of quality of life between poor sleepers on benzodiazepines and those not using any hypnotic medications. This was consistent with prior research which has indicated that the psychological aspect of quality of life was one of the strongest aspects contributing to poor quality of life in older adults who experienced sleeping difficulties (Hellstrom, Persson, & Hallberg, 2003). The result within the current investigation also parallels findings by Lichstein and colleagues (2001) who observed that poor sleepers were below the 25th percentile in psychological health related quality of life, in comparison to older adults who slept well. Zeithofer and colleagues (2000) found that poor sleepers, as indicated by the global PSQI score, had significantly lower quality of life within the psychological and emotional well-being domain. The authors reflected that the major reasons for sleep disturbance were personal problems and events of the day, and that the effect of these psychological/emotional factors increased in later periods of life (Zeithofer et al., 2000).

Furthermore, a significant difference in the social relationships domain of quality of life between self-reported good sleepers and poor sleepers taking benzodiazepine hypnotics was found. The results demonstrated that good sleeping elderly had a better quality of life within the social relationships domain than those who reported poor sleep and took benzodiazepine hypnotics. Although the difference in quality of life within the social relationships domain between good sleepers and poor sleepers not using benzodiazepines did not reach statistical significance, a trend was evident, with good sleepers having better social relationships quality of life than poor sleepers on benzodiazepines. Furthermore, the results also indicated that poor sleepers taking benzodiazepines had no differences in the social relationships domain of quality of life in comparison to poor sleepers not using hypnotics. The result that good sleeping elderly had a better quality of life within the social relationships domain than those who reported poor sleep and took benzodiazepine hypnotics was concordant with previous findings (Hajak, 2001; Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001; Reynolds et al., 2001). In one paper the authors observed that those experiencing sleep disturbance in later life have a higher likelihood of poor sleep in the context of social withdrawal, disengagement, and poor quality of life within the social relationships domain. It was suggested that poor sleep may lead to social withdrawal and poorer quality of life, which

may lead to still poorer sleep. Poor sleep quality may be the antecedent of a decline in social activity level and life satisfaction, as well as being a consequence of them (Reynolds et al., 2001). Other research has demonstrated that in comparison to good sleepers, poor sleepers were functioning at the 25th percentile or lower within the social functioning domain of quality of life (Lichstein, Durrence, Riedel, & Bayen, 2001). Ohayen and colleagues (2001) suggested that being active and having a satisfying social life appeared to be protective factors against poor sleep at any age.

No significant differences in the environment domain of quality of life between self-reported good sleepers and poor sleepers using and not using benzodiazepines hypnotics was found. This could be explained by the high quality living conditions experienced by all the participants from the current sample, as they all lived in quality hostels, and also their consistency for all sleepers. Hence, regardless of their sleep quality, they all had sufficient leisure time and financial resources. They were also all provided with accessibility to required information and safety in their daily life and living conditions.

Poor sleep and its associations with poor quality of life in the psychological and social domains exposes a distinct pattern, such that dimensions of mental health, from anxiety and depressed mood to daytime functioning, may be impacted. Sleep disturbance has the potential to pervade the older adult, from disconcerting emotional feelings to affecting social relationships. This supports Manocchia, Keller, and Ware's (2001) study comparing sleep problems among the chronically ill, when they illustrated significantly worse quality of life within the physical, psychological, and social domains. These differences were evident in good and poor sleeping patients within most of the disease cohorts studied. Their research outlines that disease does not account for the lower quality of life domains observed, as they controlled for the occurrence of chronic conditions. It was concluded that psychological quality of life was the factor most associated with sleep disturbance severity (Manocchia, Keller, & Ware, 2001). The current study lends support to this conclusion.

Sleep predictors

The expectation that selective affective functioning, sleep beliefs, and quality of life variables would significantly predict global sleep quality as measured by the PSQI

Global score of self-reported GS, PSB, and PSNB was supported by the data of the present investigation. The results indicated that anxiety, day-time dysfunction, sleep beliefs concerning quality of life, quality of life within the psychological domain and environment domain were significant predictors of global sleep quality between GS, PSB, and PSNB. The results that indicated three variables (sleep beliefs concerning quality of life, daytime dysfunction, and QOL-environmental domain) as predictors of global sleep quality between GS, PSB, and PSNB appeared inconsistent with the findings observed in the MANOVAs, where these variables did not show group significant differences. The most obvious explanation, which is nevertheless not clear-cut, was that the regression analysis was performed on the basis of Global PSQI, while the MANOVAs were obtained on the basis of self classifications of sleep quality. What was being reflected in the disparate results may be the inconsistencies in the two classifications, and this interpretation is supported by the overlap in Global PSQI scores across the different groups. As alluded to earlier in the Discussion, this cohort may be presenting different evaluations of their sleep depending on how the questions are framed, possibly because they are using downward and upward social comparisons.

Group classification

Discriminant analysis of the factors derived from the present investigation (i.e. from sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, and daytime dysfunction, anxiety, depressed mood, beliefs about the impact sleep has on quality of life, loss of control, and social interactions, and quality of life in the physical domain, psychological domain, social relationships domain, and environment domain) indicated that sleep duration, habitual sleep efficiency, and beliefs concerning the impact of sleep on quality of life correctly classified 67 percent of the poor sleepers on benzodiazepines and 77 percent of the poor sleepers not taking benzodiazepines (N= 52). The findings demonstrated a significant difference in the sleep duration, habitual sleep efficiency, and beliefs concerning the impact of sleep on quality of life, of the two poor sleeper groups. The inclusion of the beliefs about quality of life variable in the derived factors of this discriminant analysis, appeared inconsistent with the findings observed in the MANOVA, which indicated no significant difference in sleep beliefs regardless of self classed quality of sleep. Perhaps the failure to find a significant difference in the overall MANOVA (with three dependant variables across three groups)

masked an otherwise important difference between two of the groups or this one variable.

Sleep duration and habitual sleep efficiency demonstrated good predictive validity for whether subjective poor sleepers take benzodiazepine hypnotics or not. The main role benzodiazepine hypnotic agents have for poor sleepers from the current and previous studies (e.g. Estivill et al., 2003; Holbrook, Crowther, Lotter, & Endeshaw, 2001) appears to be longer sleep duration. Benzodiazepine use did not have a significant ameliorative effect on any of the other sleep variables measured in the current sample of hostel-dwelling, older adults.

Discriminant analysis of the factors derived from the current study indicated that the sleep quality, habitual sleep efficiency, and sleep disturbance accurately predicted good or poor sleeper classification in 90.9 percent of the good sleepers and 84.6 percent of the poor sleepers cases (N = 74). This result informs about the basis on which older adults classify themselves in regards to their sleep. This was consistent with the body of literature which has demonstrated that subjectively classed poor sleepers appear to demonstrate poorer overall sleep quality than older adults who class themselves as good sleepers (Lichstein, Durrence, Riedel, & Bayen, 2001; McRae et al., 2003; Vitiello, Larsen and Moe, 2004). The literature has also indicated that good sleepers have better habitual sleep efficiency when compared to poor sleepers (Bliwise, 1992; Lichstein, Durrence, Riedel, & Bayen, 2001; Riedel & Lichstein, 1998). Moreover, sleep disturbance, as measured by the PSQI was found to be one of the best descriptors of subjective sleep satisfaction (Ouellet and Morris, 2006).

Conclusion

Overall, the current study found that sleep quality, habitual sleep efficiency, and sleep disturbance demonstrated good predictive validity for whether older adults classify themselves as good sleepers or poor sleepers. Seniors classifying themselves as good sleepers differed from those that categorised themselves as poor sleepers on all but one of the PSQI sleep variables.

Self-categorised good sleepers had significantly better sleep quality and habitual sleep efficiency, significantly shorter sleep latency, significantly longer sleep duration, and

significantly less daytime dysfunction. Self-classified good sleepers also had lower depressed mood and anxiety levels than their self-classified poor sleeping counterparts. The findings reinforce the importance of research to tease out the possible cause and effect relationship between poor sleep and feeling depressed. Interestingly, there were no significant differences in sleep related beliefs and attitudes between subjective good and poor sleepers, however the instrument used to measure beliefs and attitudes associated with sleep has not been validated within an aged population. Further variables of difference between good and poor sleepers included the psychological and social relationships domains of quality of life.

Assessment of depression and excluding depressed older adults from the sample was a major strength of current work, as it eliminated the depressive-type bias in their reporting of negative events. As older adults on anti-depressant medication were not excluded, this could have biased the findings regarding psychosocial and mental health problems faced by the sleepers. A limitation was that daily dosage of benzodiazepine hypnotic consumption was not taken into account in the current investigation; this has been identified as a significant factor in its association with psychosocial and mental health problems such as anxiety and depression in elderly individuals with insomnia (Huang & Lai, 2005). Results of the current investigation cannot be generalised to the general older independent living population, as participants in the current study were all hostel dwellers. This is important limitation to note as substantive differences exist in the literature pertaining to community dwelling versus hostel residents (Beck-Little & Weinrich, 1998; Cricco, Simonsick & Foley, 2001). Moreover, results of the present study should be interpreted with caution, as since it was cross-sectional in design, the findings cannot be regarded as providing cause-effect relationship. Although the current data are based on self-report measures, subjective data are of specific relevance to healthcare providers, who usually rely exclusively on patients' reports for clinical assessment and treatment of geriatric insomniacs. Furthermore, it is the subjective complaint of poor sleep that results in the frequent use of hypnotic agents, hence the current findings are clinically relevant.

The importance and need to describe issues faced by older adults increases as the population ages. An understanding of the factors of difference between older poor sleepers on benzodiazepines and those not, is of far more than theoretical interest. It is

evident that the subjective experience of poor sleep and its subsequent management by hypnotic medications is associated with substantial risk for older adults. The current investigation has contributed to this end with its focus on poor sleep and hypnotic use in this population. Several implications can be drawn from the results of the current study. An accurate and holistic approach to assessment of sleep satisfaction by nurses and other clinicians must include an appraisal of not merely the older adult's typical sleep patterns but also the person's unique perception about their daily functioning. Specifically, their views of the cause of their poor sleep and their affective reaction to the reported sleep disturbance should also be assessed. Anxiety and depressed mood evaluations should be included for a holistic evaluation of sleep related factors. Participants in the current study were all hostel dwellers, hence nurses have a major role in providing education around sleep medication to older adults in residential care.

There is a need for education to be provided to older adults about the modification of the sleeping pattern with age, to discourage them from seeking pharmacological prescriptions. This is an important key point with regards to potential benzodiazepine hypnotic use, this should be conveyed to the residents as part of an educational program because as suggested by the results of the current study, the way they view their sleep duration and sleep efficiency are key factors in predicting benzodiazepine hypnotic use in self-classified poor sleepers. A thorough explanation of 'normal' sleep duration and sleep efficiency for older adults should be provided. The findings of this investigation suggest that education around 'normal' sleep duration and sleep efficiency may be helpful in the reduction of benzodiazepine use and initial prescription. The knowledge that benzodiazepine hypnotics should be avoided due to their effectiveness being only short-term and because alternative aids are as effective for the long-term and without any side effects, needs to be forwarded on to older adults.

The current study has strengthened previous conclusions about the factors of difference between older adults that classify themselves as good sleepers and those classifying themselves as poor sleepers. It has also identified potential variables of difference between self-reported poor sleepers that take benzodiazepine hypnotics in an attempt to alleviate their sleep disturbances and those that choose not. These findings allow for further exploration into other types of variables of difference between poor sleepers taking and not taking benzodiazepine hypnotics, such as knowledge about alternate

treatments, and knowledge of 'normal' sleep patterns in older adults. The findings also support an alternative to the use of hypnotic agents in the management of insomnia amongst aged persons, as subjective sleep quality, sleep latency, habitual sleep efficiency, sleep disturbance, and daytime dysfunction were not significantly different among poor sleepers, regardless of whether they were taking benzodiazepine hypnotics or not. The alternative to managing poor sleep needs to be education programs, such as education delivered by nurses or evidence based self-help programs (e.g. Morawetz, 2003).

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Appendix A: Information to Participants Letter



227 Burwood Road
Hawthorn 3122
Victoria Australia
Telephone
(03) 9818 7344
Facsimile
(03) 9818 2128
Email: info@baptcare.com.au



Victoria University
PO Box 14428
MELBOURNE VIC 8001

Telephone:
(03) 9919 4000
Facsimile:
(03) 9919 4069

Victoria University of Technology

The sleep patterns and use of hypnotic medications amongst aged persons

Thank you for expressing interest in this study being conducted by Victoria University. It is designed to look at the sleep patterns of older people and the decisions people make around using sleeping tablets to help with sleeping at night.

- If you agree to participate, you will be interviewed in a session that will take between 30 and 45 minutes. During this time you will be asked about your**
 - **Personal details such as your age, health status, medications etc.**
 - **Your sleep patterns (when you go to bed/wake up)**
 - **Your use of sleeping tablets**

- You will also be asked to complete 5 questionnaires about**
 - **Your sleep habits**
 - **Your attitudes to sleep**
 - **Your general life satisfaction and emotional well-being**

- You will be asked to complete a sleep diary for the next week, which involves recording sleep details (such as the time you went to bed) from the night before.**

- **You will engage in a second interview next week for 60-90 minutes. During this time you will be asked to give detailed information about your**
- **experiences and thoughts on good and poor sleep**
 - **what you do to improve your own sleep**
 - **sleep medications**

Please tell the researcher if you feel tired during the interview and would like to take a break. Also note, that participation in this study is voluntary. This means that if you begin the interview but then change your mind, you are free to withdraw at any time.

All the information you provide will be collected along with information from other participants, and you will not put your name on any of the questionnaires. This means that you will not be identifiable from your responses, and the data will only be reported as group data.

Following the interview, if you wish to talk about the experience, feel uncomfortable about the interview, or would like further information, you can contact Dr Bernadette Hood on 9365 2334. This phone has an answering machine for you to leave your name and phone number, after which your call will be returned.

Appendix B: Consent Form



227 Burwood Road
Hawthorn 3122
Victoria Australia
Telephone
(03) 9818 7344
Facsimile
(03) 9818 2128
Email: info@baptcare.com.au



Victoria University
PO Box 14428
MELBOURNE VIC 8001

Telephone:
(03) 9919 4000
Facsimile:
(03) 9919 4069

Victoria University of Technology

**Consent Form for Participants Involved in Research
INFORMATION TO PARTICIPANTS**

We would like to invite you to be a part of a study into “The sleep patterns and use of hypnotic medications amongst aged persons”

CERTIFICATION BY PARTICIPANT

I,.....
of.....
certify that I am voluntarily giving my consent to participate in the
experiment entitled:

**The sleep patterns and use of hypnotic medications amongst aged
persons**

As conducted at Victoria University of Technology by:
Associate Professor Bernadette Hood
Professor Dorothy Bruck

I certify that the objectives of the study, together with any risks to me associated with the procedures listed here under to be carried out in the study have been fully explained to me by:

Associate Professor Hood, Professor Bruck, or their research officer

And that I freely consent to participation as outlined below:

- I understand that I will be interviewed for between 30 and 45 minutes today, and I will answer a range of questions about my personal details, my sleep habits and my use of hypnotic medications.
- I will also fill out 5 questionnaires, which will involve a series of questions relating to my sleep behaviours, my attitudes to sleep, and my satisfaction with my current life experiences.
- I will keep a sleep diary for 1 week.
- I will be interviewed for between 60 and 90 minutes next week, about the quality of my sleep, ways I try to improve my sleep, and hypnotic medications.
- I understand that my interview will be recorded on audio tape.

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 jeopardize me in any way.

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

Signed:

Witness (not researcher) Date

Any queries about your participation in this project may be directed to the researcher (Associate Professor Bernadette Hood ph. 9365 2334). If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University of Technology, PO Box 14428 MCMC, Melbourne, 8001 (ph. 03 9688 4710)

