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The Why, When and Where of Selective Attention to Sleep in Psychophysiological Insomnia.

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Submitted for the higher degree of Ph.D to the Higher Committee of the College of Science and Engineering, University of Glasgow.

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Abstract

Psychophysiological Insomnia (PI) is characterized by morbid fear of insomnia, mental arousal and heightened somatic tension in bed (ICSD- 2). The most widely reported epidemiological study reports a prevalence of insomnia as between 9% and 15% (Ohayon 2002) and most studies conducted to date looking at prevalence report similar numbers. Espie and colleagues (2006) in their review paper outlined a pathway into PI with three main components; selective attention to sleep, explicit intention to sleep and sleep effort. This model moves forward from Espie's (2002) Psychobiological Inhibition Model of insomnia which considered both the psychological and physiological states of normal sleep and how these are affected in insomnia. The Attention-Intention-Effort (A-I-E) Model further addresses the loss of automaticity and flexibility in insomnia but moves towards specifically outlining the processes which are present in PI as compared to the good sleeper (GS).

The first step in the A-I-E is selective attention to sleep, more commonly reported as attention bias to sleep. The University of Glasgow have pioneered the work establishing this attention bias towards sleep as an indicator in insomnia by using several cognitive probe paradigms presenting neutral and sleep related words and images to PI and GS (Jones et al 2005, Marchetti et al 2006, MacMahon et al 2006, Woods et al 2009). The various paradigms applied to understanding attention bias in PI have confirmed that PI will selectively attend to sleep related stimuli compared to neutral and to GS. We now find ourselves at the juncture of wanting to further understand the underlying mechanism to this attention bias as the previous research has mainly attributed it to an anxiety provoked response. This has its basis in Harvey's (2002) cognitive model of insomnia which makes comparisons with insomnia and anxiety disorder as well as the absence of de-arousal, both physiological

and cognitive, in insomnia as outlined in Espie's (2002) Psychobiological Inhibition Model.

This thesis aims to further our understanding and answer our questions regarding the underlying mechanisms of attention bias in insomnia by addressing the time course, specificity and valence of attention bias in insomnia. Four experiments are used to address these three factors. Firstly, the specificity of AB is examined and compares the performance of GS and those going through a period of acute insomnia on a modified pictorial Posner paradigm in Experiment 1. Experiments 2 and 3 move on from Woods et al (2009) looking at AB to sleep and day times presented on an alarm clock using another modified Posner paradigm. By adding day times into the experiment and adjusting the presentation time of the salient stimuli we address the time course and valence questions. Finally, in experiment 4, an eye tracking experiment, which is new to insomnia research, has been developed where positive sleep, negative sleep and neutral words are presented to PI and GS.

. This definitive experiment addresses factors of time course and valence by experimentally manipulating the saliency of the stimuli presented as well as monitoring over a continuous period of presentation.

Overall, the findings of this thesis confirm that PI will selectively attend to salient stimuli at shorter presentation times but this attention bias changes into a performance deficit as presentation time increases. This prompts consideration on how the nature of the tasks are exposing elusive performance impairments in insomnia. Also, the saliency of stimuli representing the day presented to PI opens discussion into the 24 hour nature of PI.

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Declaration

This thesis embodies the results of original research carried out by the author.

References to existing work are made as appropriate.

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I would like to dedicate this thesis to my darling daughter Olivia. In everything I do, you have always been at the forefront of my thoughts and I look forward to sharing your adventures in life xx

Abbreviations

AASM American Academy of Sleep Medicine

AIE Attention-Intention-Effort pathway

ANOVA Analysis of Variance

APSS Associated Professional Sleep Societies

CBT Cognitive Behavioural Therapy

CBT-I Cognitive Behavioural Therapy for Insomnia

DBAS Dysfunctional Beliefs and Attitudes about Sleep scale

DFSAS Daytime Functioning and Sleep Attribution Scale

DSM-IV Diagnostic and Statistical Manual of Mental Disorders, 4th Edition

DSM-IV-TR Diagnostic and Statistical Manual of Mental Disorders, Text Revision

ES Effect Size

fMRI functional Magnetic Resonance Imaging

GS Good sleeper

ICSD-2 International Classification of Sleep Disorders, Second Edition

ISI Insomnia Severity Index

MRI Magnetic Resonance Imaging

MSLT Multiple Sleep Latency Test

NIH National Institutes of Health

PI Primary Insomnia/Psychophysiological insomnia

PIM Psychobiological Inhibition Model

PS Poor Sleepers

PSG Polysomnography

PSQI Pittsburgh Sleep Quality Index

RT Reaction Time

SD Standard Deviation

SE Sleep Efficiency

SOL Sleep Onset Latency

SRT Sleep Restriction Therapy

TST Total Sleep Time

UGSC University of Glasgow Sleep Centre

WASO Wake-time After Sleep Onset

Chapter 1

Insomnia Review

'Sleep is so close to home, nothing is closer- in fact home is sometimes defined as the place where we sleep. Sleep is woven into the fabric of our daily lives, into the most intimate regions of our beings, who we are, the way we see the world, the things we need to believe.' Gayle Green, *Insomniac*.

1.1 Insomnia definition

Office of National Statistics data shows that sleep disturbance is foremost of all primary mental health complaints (Singleton et al, 2001). Insomnia is the most widely reported psychological symptom in Britain (ONS, 2000: http://www.statistics.gov.uk/downloads/theme_health/psychmorb.pdf) and the main reason for benzodiazepine prescribing in primary care (NICE, 2004: <http://www.nice.org.uk/pdf/TA077fullguidance.pdf>). Insomnia is a heterogeneous complaint reflecting reduced quality, duration, or efficiency of sleep (Morin, Hauri, Espie, Spielman, Buysse, & Bootzin, 1999). It is defined as difficulty initiation or maintaining sleep or non-restorative sleep, associated with significant distress or daytime impairment and not due to other medical, psychiatric or sleep disorders (DSM-IV; APA, 1994). Riemann et al (2003) report pre-existing insomnia as the highest attributable, treatable, risk factor for first episode depression, and for recurrence of depressive episodes in younger and older adults. It is a distressing condition associated with reduced daytime alertness, productivity, and quality of life, impaired relationships and increased physical as well as psychological ill health (Dew, M.A., et al. 2003, Morgan, K. 1989).

Psychophysiological insomnia (PI) is the most common insomnia subtype found in 1-2% of the general population. According to diagnostic criteria, heightened arousal and learned sleep preventing associations form the foundations of this disorder, with patients exhibiting excessive focus upon and anxiety about sleep (ICSD, 1997 & 2005, DSM-IV; APA 1994). Numerous authors contend that PI is the result of a number of psychological factors, such as maladaptive beliefs about sleep or excessive pre-sleep intrusive thoughts (Harvey 2002, Espie 2002, Morin et al 1993). The most recent diagnostic criteria for insomnia can be found in the proposed criteria

for DSM V (<http://www.dsm5.org>) with the most recent proposed revisions dated as June 2010. The table below (Table 1.1) outlines how the diagnostic criteria have developed from DSM IV. What can be seen is that the specificity of symptoms and complaints has progressed; the symptoms can be multiple, age and stage of development is addressed, the daytime impact is acknowledged and detailed and the length of disorder has increased from one to three months.

At this point, the criteria for classification applied in this thesis should be considered. Previous research on attention bias to sleep in insomnia was conducted using the criteria for primary insomnia as found in DSM IV and this was the current criteria available when the research for this thesis was begun. Psychophysiological insomnia is a sub-category of primary insomnia as presented in ICSD 2 alongside idiopathic and paradoxical insomnia. The essential features of psychophysiological insomnia include learned or behavioural insomnia and heightened arousal. The primary components involved are intermittent periods of stress that result in poor sleep and maladaptive behaviours. These include a vicious cycle of trying harder to sleep, becoming tense and bedroom habits/routines that actually condition the patient to become frustrated and aroused. Patients often report "racing thoughts" and sensitivity to their environment. Bad sleep habits, such as those naturally acquired during periods of stress, are occasionally reinforced. These are therefore not resolved and become persistent. Insomnia continues for years after the stress has abated, and is labelled persistent psychophysiological insomnia. In the research presented here, the findings are applied to psychophysiological insomnia as this subtype considers the interaction of the individual with their environment including monitoring and arousal. These aspects would fit comfortably into attention bias and information processing.

DSM IV	DSM 5
<p>A. The predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month.</p> <p>B. The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.</p> <p>C. The sleep disturbance does not occur exclusively during the course of Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia.</p> <p>D. The disturbance does not occur exclusively during the course of another mental disorder (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, a delirium).</p> <p>E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.</p>	<p>A. The predominant complaint is dissatisfaction with sleep quantity or quality made by the patient (or by a caregiver or family in the case of children or elderly).</p> <p>B. Report of one or more of the following symptoms:</p> <ul style="list-style-type: none"> -Difficulty initiating sleep; in children this may be manifested as difficulty initiating sleep without caregiver intervention -Difficulty maintaining sleep characterized by frequent awakenings or problems returning to sleep after awakenings (in children this may be manifested as difficulty returning to sleep without caregiver intervention) -Early morning awakening with inability to return to sleep -Non restorative sleep -Prolonged resistance to going to bed and/or bedtime struggles (children) <p>C. The sleep complaint is accompanied by significant distress or impairment in daytime functioning as indicated by the report of at least one of the following:</p> <ul style="list-style-type: none"> -Fatigue or low energy -Daytime sleepiness -Cognitive impairments (e.g., attention, concentration, memory) -Mood disturbance (e.g., irritability, dysphoria) -Behavioral problems (e.g., hyperactivity, impulsivity, aggression) -Impaired occupational or academic function -Impaired interpersonal/social function -Negative impact on caregiver or family functioning (e.g., fatigue, sleepiness) <p>D. The sleep difficulty occurs at least three nights per week.</p> <p>E. The sleep difficulty is present for at least three months.</p> <p>F. The sleep difficulty occurs despite adequate age-appropriate circumstances and opportunity for sleep.</p> <p>Duration:</p> <ol style="list-style-type: none"> 1. Acute insomnia (<1 month) 2. Sub acute insomnia (1-3 months) 3. Persistent insomnia (> 3 months)

Table 1.1 DSM IV and DSM 5 diagnostic criteria for primary insomnia.

Benzodiazepine prescribing (ineffective for persistent insomnia) remains high in Scotland, and the newer 'Z' hypnotics are no more effective (NICE,2004). Antidepressants are increasingly prescribed (off label) for primary sleep disturbance, and the 'over the counter' market is booming. Figures for Scotland and the UK can only be extrapolated from abroad; the direct costs of assessing and treating insomnia were approximated as \$14 billion in the United States and FF10 billion in France in 1995 (Walsh & Engelhardt 1999, Leger et al. 1999). In the 12 months to March 2006, in England, 4.7 million items were dispensed for zaleplon, zolpidem and zopiclone (referred to as Z-drugs) at a cost of £13.5 million. For benzodiazepines used to treat insomnia (loprazolam, lormetazepam, nitrazepam, temazepam) 5.1 million items were dispensed at a cost of £8.8 million

<http://www.nice.org.uk/niceMedia/pdf/TA77NICEImplUptake.pdf>.

1.2 Insomnia classification

Insomnia can also be secondary to other diseases or disorders. In the case of neurological diseases e.g., Parkinson's, Huntington's and Alzheimer's disease, sleep disturbances are not uncommon. In such cases, the sleep disturbance can be caused by an underlying disorder involving the central nervous system, the motor system, or associated cognitive and psychological disorders. Insomnia can also be caused by the secondary effects of these diseases or as a side effect of the medication prescribed to treat them (Aldrich, 1993).

Insomnia is also widely accepted as a symptom of many psychological disorders e.g. anxiety, depression, post traumatic stress disorder. Here insomnia is generally viewed as not of major concern, a result of the 'primary' complaint and

dependent on the survival of the primary complaint. Until recently such thinking has been widely accepted throughout psychiatry.

However, contradictory evidence is now coming through. Johnson et al (2006) conducted a study looking at the association between insomnia and anxiety disorders and major depression among a community based sample of adolescents. They found that anxiety disorders preceded insomnia in 73% of cases while insomnia occurred first in 69% of comorbid insomnia and depression cases. Prior insomnia was associated with onset of depression but depression was not associated with onset of insomnia, compared to anxiety disorder which was associated with increased insomnia risk. The authors took these results to suggest distinct natural courses of development between insomnia, anxiety and depression during adolescents. This link between depression and insomnia is now a robust finding in the literature. The general consensus holds that if the 'primary' disorder is targeted in treatment, it is assumed that the secondary disorder will remit. However, experimental research has indicated that this is not the case for secondary insomnia. More specifically, Hauri et al 1974, demonstrated that patients who had been hospitalised for unipolar depression were still displaying residual sleep disturbances relative to normal controls six months after remission. Intriguingly, numerous researchers (e.g. Jacobs et al 1993, Vallieres et al 2000) have revealed preliminary data indicating that Cognitive Behavioural Therapy for insomnia can lead to reduced depression and anxiety symptoms. Irrefutably, these results indicate that a sleep-focused intervention is not only effective in reducing the sleep disturbance, but also reduces the accompanying pathology.

Insomnia is not merely a symptom or epiphenomenon of another disorder and this view may deprive patients of treatment, which might not only cure their insomnia, but also may reduce symptoms associated with the assumed 'primary'

disorder (Harvey 2001). Therefore it would subsequently appear important to discover and evaluate the genesis and maintenance of insomnia, as a primary disorder, in attempts to identify successful treatment routes.

According to DSM 5, making a reliable differential diagnosis between “Primary Insomnia” and “Insomnia related to another disorder” implies that a clinician can identify the cause and the consequence of the main condition, a determination that is often difficult, if not impossible to make and the recommendation is made to use “Insomnia Disorder” whenever diagnostic criteria are met, whether or not there is a co-existing psychiatric, medical, or other sleep disorders. This strategy moves on from the notion of insomnia as secondary to another disorder and prevents the clinician neglecting treating the sleep complaint which, as the research above highlights, can be a stand alone disorder as well as co-morbid with other sleep, psychological and physiological disorders.

1.3 Prevalence of insomnia

The most recent data on the epidemiology of insomnia comes from a study conducted in Norway by Sivertsen and colleagues (2010) looking at the prevalence of insomnia and associations with physical and mental health. With a final sample of over 45000, participants were administered questionnaires to complete as part of a general health screening program named HUNT-2. Within these questionnaires were two questions about the frequencies of sleep onset and terminal insomnia as well as collection of self reported diagnoses on somatic conditions, chronic pain conditions, pain conditions with uncertain organic etiology and mental conditions (anxiety and depression). Within this sample of the Norwegian population ranging from 20 to 89 years of age, 13.5% reported insomnia symptoms. 10% of the sample younger than 40

years reported insomnia symptoms with a significant increase with age which was most prominent in women. Strong associations were found between insomnia and mental conditions and pain with uncertain organic etiology as well as with chronic pain conditions and somatic conditions but to a lesser degree. These findings are in line with a previous review of the epidemiological studies conducted by Ohayon (2002) which found that between 8% and 18% of the population reported dissatisfaction with sleep quality or quantity and between 9% and 15% reported insomnia symptoms and daytime consequences.

With regard to UK prevalence of insomnia, the most recent study was conducted by Freeman and colleagues (2010) on findings from the second British National Survey of Psychiatric Morbidity (N=15 804), which was carried out between March and September 2000, reported sleep difficulties in 38% of the sample, insomnia of at least moderate severity in 11.9% and chronic insomnia in 6.6%. This study did not have a main aim of assessing prevalence of insomnia in the British population using strict diagnostic criteria but sleep problems were determined from the Clinical Interview Schedule Revised (CIS-R; Lewis et al 1992). The main aim of the Freeman et al (2010) study was to investigate the relationship between insomnia and paranoia to further understand the role insomnia contributes to the development and maintenance of these fears but it is also the most recent report of incidence of sleep problems in the UK.

Morphy et al (2007) published work reporting incidence rates of insomnia at 37%. This study determined insomnia by asking if the individual had trouble falling asleep, waking up several times a night, have trouble staying asleep and waking up after your usual amount of sleep feeling tired and worn out. Ratings were 'not at all', 'on some nights' or 'on most nights'. This study also followed up those participants

willing to be contacted again after 1 year where it was found that, of those without insomnia at baseline, the incidence of insomnia 12 months later was 15%, and this was significantly associated with baseline anxiety, depression and pain. Of those who did have insomnia at baseline, 69% had insomnia at 12-month follow-up; persistence of insomnia was significantly associated with older age. Insomnia at baseline was significantly associated with incidence of anxiety, depression, and widespread pain at 12-month follow-up. This study was, like Freeman et al (2010), reporting sleep outcomes which had been included within questionnaires administered as part of a wider study which in this case had the primary aim of investigating headache.

In summary, these studies highlight that insomnia is a common health problem in the UK and abroad which is linked to a number of other disorders both physiological and psychological. Irrespective of the international nature of this disorder, the features of insomnia remain consistent.

1.4 Impact of insomnia on everyday life

Poor sleep has a substantial impact on the life of the individual and society as a whole. The Harvard Work Hours and Safety Group (2005) showed that medical residents who drove home having been awake for at least 24hr have more than double the number of motor vehicle accidents and nearly six times more near misses when compared with those not working such extended shifts, with each commute home increasing their risk of crash by 16%. Drowsiness in sleep-deprived drivers is likely the cause of more than 100,000 crashes, 71,000 injuries and more than 1,500 deaths each year (Knipling and Wang 1995, US data). In the US, sleep disorders are estimated to cost over \$100 billion annually in lost productivity, medical expenses, sick leave and property and environmental damage (Stoller, 1994). Morin et al (2006)

reported the prevalence of insomnia symptoms and syndrome in the general population of Canada as 29.9% for insomnia symptoms and 9.5% for insomnia syndrome.

Despite the fact that at least 40 million Americans report having sleep problems, more than 60 percent of adults have never been asked about the quality of their sleep by a physician, and fewer than 20 percent ever initiated a discussion about it (National Sleep Foundation, 2000). In addition, over 40% of adults experience daytime sleepiness severe enough to interfere with their daily activities at least a few days each month, with up to 20% reporting problem sleepiness a few days a week or more (National Sleep Foundation, 2003). Morin et al (2006) found that 13% of all respondents had consulted a healthcare provider for sleep problems at least once in their lifetime. For the individuals with an insomnia syndrome the lifetime consultation rate increased to 42.3% and was significantly higher than lifetime consultations reported by the rest of the sample, 9.6%.

In their recent review paper, Leger and Bayon (2010) discuss the societal costs of insomnia. Concluding that the economic burden of insomnia is ‘enormous’ and public authorities need to be convinced on the long term collective gain of a good nights sleep, a sample of the findings these authors overview are that those with insomnia have double the absenteeism rate of good sleepers (Leger et al 2002), longer and more frequent sick leave (Sivertsen et al 2009), are eight times more likely to have a work related accident (Leger et al 2002) and have higher annual medical consultation rate (Weyerer and Dilling 1991). The general picture painted in this paper is that insomnia impacts every aspect of an individual’s life with the most recent (Canadian) estimated direct cost as Can\$547.5 million (approx. £343 million) and indirect cost as Can\$6.0 billion (approx. £3.8 billion).

Kyle et al (2010) conducted a study in response to the neglect of the existing literature to address the nature of impairment caused by insomnia. The Glasgow group used qualitative methodologies to characterize the daytime experience in individuals with insomnia using their own words. Interpretative analysis revealed three superordinate themes; 'just struggle through', 'isolated, feeling like an outsider' and 'insomnia as an obstruction to the desired self'. These themes certainly highlight that insomnia is a debilitating and wide ranging disorder with regards to impact on an individual's life. Interestingly, Kyle and colleagues also discuss the use of strategies employed by those with insomnia such as one participant in the study reporting that, although she had an unsatisfactory nights sleep, she would be at work earlier than colleagues which helped her cope with the social element of work. With regard to the higher levels of absenteeism reported elsewhere and discussed previously in this chapter, Kyle et al (2010) raise the question of absenteeism as being a measure of impact of insomnia as those individuals affected may be 'struggling through' and adopting strategies to avoid becoming overwhelmed.

Chapter 2

Models of insomnia

The purpose of this chapter is to outline the theoretical perspectives of insomnia. To understand the models of insomnia, it is important to consider their foundations:

- Behavioural perspectives
- Physiological perspectives
- Cognitive perspectives.

However, it is important to note that these perspectives are not isolated but interact and this will be outlined in the more recent models such as Espie's Psychobiological Inhibition and Attention Intention Effort models. Therefore, this chapter begins by discussing the models and respective treatments of each of the perspectives mentioned above separately under the treatment headings as it is useful to approach these perspectives with the components of treatment i.e. understanding the solution helps in understanding the problem. This is followed by an integration of perspectives in the more recent models and how current research relates to these conceptualizations.

Cognitive Behavioural Therapy for Insomnia (CBTi) has been recommended as the first line treatment for insomnia (Stepanski, 2005) and the National Institutes of Health Consensus and State of the Science Statement (2005) concluded that CBT is "as effective as prescription medications are for short-term treatment of chronic insomnia. Moreover, there are indications that the beneficial effects of CBT, in contrast to those produced by medications, may last well beyond the termination of active treatment" (page 14). Numerous studies have been published providing evidence for its long term efficacy, an example of which is the American Academy of Sleep Medicine (AASM) taskforce reports (1999 and 2006) which included 85 clinical trials and reported a 70% improvement rate in patients which was sustained at 6 month

follow up. Therefore, the components of CBTi and the relevant models of insomnia will be presented chronologically.

2.1 Behavioural Perspectives

2.1.1 Sleep Hygiene and Stimulus Control

An early behavioural perspective was presented by Bootzin (1972) which proposed that insomnia results from the adoption of maladaptive sleep habits and resulted in the development of stimulus control. Good sleep is seen as coming under the stimulus control of the bedroom environment, which acts as a discriminate stimulus for sleep (Bootzin et al., 1991). Thus, difficulty in initiating sleep may result from faulty identification of discriminative stimuli for sleep or the presence of stimuli incompatible with sleep. This poor stimulus control therefore might interfere with a sleep drive and circadian timing by strengthening conditioned arousal.

This interest in the control that sleep-related environments might have over sleep behaviour is now long established. In simplistic terms, within a conditioning framework, bedroom environment objects might become discriminative stimuli for sleep (Bootzin et al., 1991), but when the bedroom-sleep contingencies are broken, they might become discriminative stimulus for wakefulness. Thus, perhaps as the individual processes the environment for sleep/sleeplessness cues, an Information Processing Bias (IPB) phenomenon, toward significant sleep-related stimuli within the sleep environment, may gradually evolve.

Other maladaptive sleep habits, commonly termed poor sleep hygiene i.e. sleeping in the armchair, napping during the day etc, strengthen the associations between sleep and non-sleeping environments.

Stimulus control therapy (SCT) was first introduced by Bootzin (1972) and addresses maladaptive sleep habits that exists in insomnia. An illustration which is given in the text mentioned above involves a dialogue between a therapist and patient highlighting that sleep and sex are not the only activities occurring in the bed and bedroom:

‘Sometimes I do things to keep my mind off the fact that I am not sleeping, maybe read, maybe watch some TV, sometimes I work on my laptop or surf the Internet. Sometimes I’ll lie in bed and meditate – I have heard this helps.’

This is very different to a GS who would perhaps have difficulty reporting other activities that occur in bed and the bedroom apart from sleep. In fact, I’m sure if a GS finds him/herself in the bedroom and has the opportunity, the temptation to have a quick lie down or at least look at the bed with longing may be difficult to overcome. Even if other activities do occur in the bedroom, such as reading or watching TV, these would not interfere with sleep in GS.

The rationale behind SCT includes the individual learning to use internal, physiological cues for knowing when to enter the bedroom for sleep rather than using time of day and calculation of hours of sleep required or other family members’ bedtime habits as cues for initiating sleep. With the aim of having time in bed asleep, this is an important skill to learn to achieve high sleep efficiency as many PI will spend a longer amount of time in bed compared to time asleep. This therefore leads to the opportunity to perform the other task e.g. TV watching during hours when they should actually be asleep.

Bootzin proposed that stimulus control treatment instructions, such as lying down to sleep only when sleepy, getting up if unable to sleep within 15 mins, avoiding napping etc., will significantly improve sleep efficiency (Bootzin 1972,

Bootzin & Epstein 2000). On reflection, these instructions may be inducing internal/external monitoring, by promoting awareness about successful sleeping environments and successful sleep promoting behaviour. Indeed, this may serve to further strengthen the awareness of positive and negative sleep-related cues that denote successful or unsuccessful sleep, respectively.

2.1.2 The 3P model and Sleep Restriction Therapy

Bootzin's work in the 1970s was followed by a framework for the development and maintenance of chronic insomnia which describes predisposing conditions, precipitating circumstances, and perpetuating factors (the 3-P model) as proposed by Spielman (Spielman 1986, Spielman & Glovinsky 1991) and further elaborated by Morin (1993).

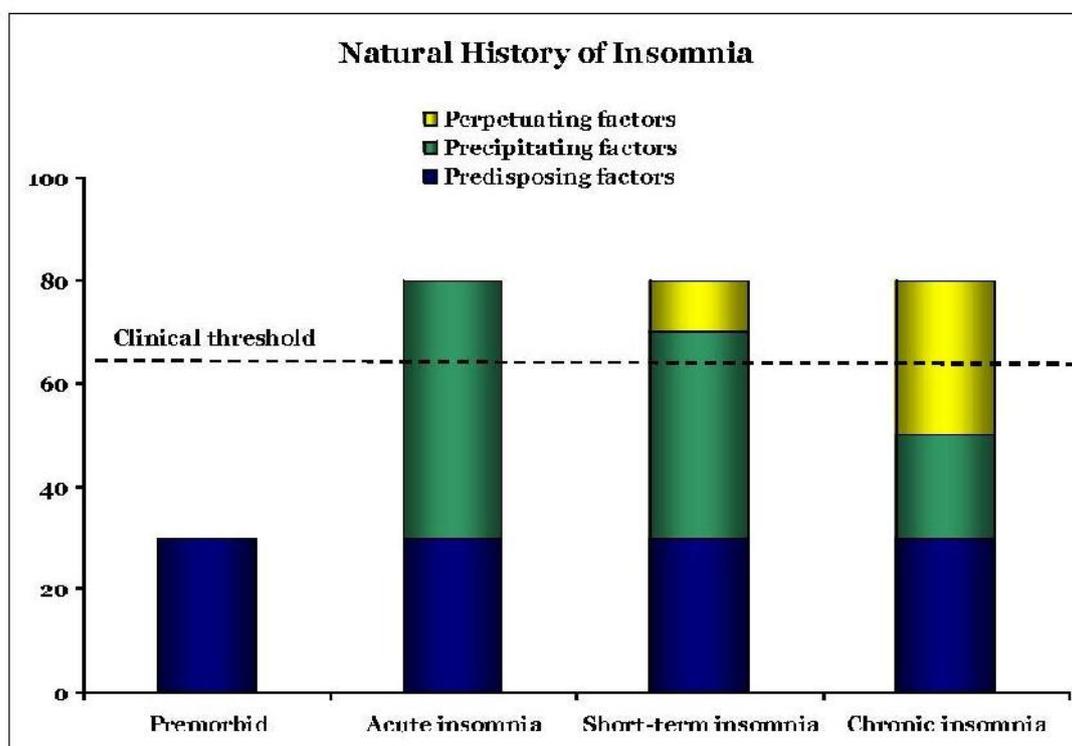


Figure 2.1 Adapted from Spielman, A.J. & Glovinsky, P. (1991). The varied nature of insomnia. In P.J. Hauri (Ed.). Case studies in insomnia. New York, Plenum Press.

Three types of factors are involved at different points during the course of insomnia.

- **Predisposing factors:** Some psychological or biological characteristics increase vulnerability, or predisposition, to sleep difficulties (e.g., female gender, anxiety, hyperarousal). These factors are not a direct cause of insomnia, but they increase the risk that an individual will develop sleep difficulties.
- **Precipitating factors:** These are the life events and the medical, environmental or psychological factors that trigger insomnia (e.g., divorce, death of a significant other, illness, medication, familial or occupational stress).
- **Perpetuating factors:** These elements maintain or exacerbate sleep difficulties. They are typically behaviors (e.g., extending time spent in bed to try to sleep more, naps) and/or beliefs and thoughts (fear of sleeplessness, excessive worries about daytime consequences) that people adopt in order to cope with sleeplessness. Although some of these behaviors (bed resting) can be useful in the short term, in the long run they have the opposite effect and tend to perpetuate insomnia.

The figure above (adapted from Spielman & Glovinsky, 1991) illustrates how transient insomnia can evolve into persistent insomnia. Everyone presents, to various degrees, some vulnerability to develop insomnia, which is more or less important depending on individual differences (predisposing factors). Different types of precipitating factor may trigger insomnia, even among those with little vulnerability. Once the initial precipitating event fades away, most people return to normal sleep. For others, perhaps those at greater risk for insomnia, sleep difficulties persist even

after the initial precipitating event has been removed or managed. For these people, insomnia develops a life of its own and several psychological and behavioural factors contribute to perpetuate the sleep difficulties over time. Effective management of chronic insomnia must directly target these perpetuating factors. According to the 3P model of insomnia, behavioural practices and cognitive tendencies that perpetuate sleep disturbance are often the most promising targets for intervention. Many of these perpetuating factors, such as spending too much time in bed, anticipatory anxiety about the prospects for sleep, and inordinate concern about daytime performance deficits, are addressed by sleep restriction therapy (SRT).

If you were to ask a good sleeper how they get to sleep they would probably have difficulty providing you with a definitive answer; it just happens. If you were to ask a PI how they get to sleep, they may provide you with a list of ‘strategies’ or things they do when they are not sleep. This is a key aspect of insomnia which we will return to later in the models of insomnia. One of the components of CBTi is to restrict the sleep opportunity. This strengthens the homeostatic sleep drive and increases the propensity for sleep on upcoming nights by limiting the sleep accumulated.

Sleep Restriction Therapy (SRT) addresses the unsatisfactory sleep efficiency of PI by providing a concentrated period for sleep. It removes the opportunity for PI to spend excessive time in bed awake by assigning a bedtime and wake time based on sleep diaries from the previous one to two weeks. The patient is instructed to follow this revised sleep schedule and retiring time may be altered only if greater than 85% sleep efficiency is achieved over a series of nights (Espie, 2001).

From personal experience of delivering CBTi at the University of Glasgow Sleep Centre, many of the individuals participating in the groups find this component of the treatment the most difficult to commit to but it has been found to be very

effective. It is interesting to get a reaction of how difficult it will be to stay up to 2am, for example, and rise at 7am from someone who is only sleeping 4 or 5 hours a night anyway but will usually spend a number of hours awake in bed awake, frustrated by their inability to sleep.

Spielman, Caruso and Glovinsky, (1987) listed excessive time in bed as one of a number of key factors that perpetuate insomnia. These authors suggested that sleep restriction therapy, stimulus control instructions, and sleep hygiene recommendations have all evolved from an appreciation that factors which perpetuated insomnia may operate long after precipitating factors have subsided. These authors administered SRT to 35 adults with psychophysiological insomnia and insomnia co-morbid with psychiatric disorders in eight weekly individual sessions. Treatment outcomes, as measured by subjective sleep onset latency (SOL), total sleep time (TST), sleep efficiency (SE), and ratings on insomnia symptoms were all improved after treatment as well as at a 36-week follow-up. Although initially restricted, TST eventually increased from 320 minutes to 343 minutes and SE improved from 67 percent to 87 percent.

2.1.3 Paradoxical Intention therapy

Paradoxical intention, as described by Frankl (1955) in the context of logotherapy, became of interest to behaviourists in the late 1970s in ameliorating problem behaviours. Ascher and Turner (1979) conducted a study comparing Paradoxical Intention therapy (PIT) against a control condition and found those with a sleep onset complaint had decreased SOL and WASO as well as improved restfulness. The rationale behind PIT is the automaticity of sleep onset and the lack of effort inherent in initiating sleep in GS. This is in contrast to insomnia in which sleep onset

becomes something effortful and conscious but elusive. The paradoxical nature of this therapy is that the individual is instructed to attempt to stay awake, lie in bed with the lights off but eyes open or, using a less concrete paradoxical behavioural method and moving more toward an acceptance and mindfulness notion, to simply give up trying and accept their insomnia.

Broomfield and Espie (2003) objectively tested the efficacy of PIT where those with sleep onset insomnia were instructed, at lights out, to stay awake for as long as possible by keeping their eyes open. The need to resist sleep-onset gently but persistently in an environment conducive to sleep was emphasized but the use of active methods to stay awake (e.g. reading, physical movement) was discouraged. The outcome measures of this study included subjective measures of sleep onset latency (SOL) and sleep efficiency (SE) using a sleep diary, measures of anxiety regarding sleep as measured by the Sleep Anxiety Scale (Fogle & Dyall, 1983) and Sleep Performance Anxiety Scale which was specifically designed for this study and, as an objective measure of SOL and SE, actigraphy. Results of this study showed that the group using PIT reported less sleep effort, sleep anxiety and a shorter subjective SOL than the control group not using PIT. With regard to the objective measures, no significant changes of SOL occurred over the treatment period. However the authors, when looking at the objective and subjective measures together, discuss the reduction in overestimation of SOL in subjective reports.

2.1.4 Relaxation Training

Morin et al (2006) describe relaxation training as clinical procedures aimed at reducing somatic tension (e.g. progressive muscle relaxation, autogenic training) or intrusive thoughts at bedtime (e.g., imagery training, meditation)

interfering with sleep. An American Academy of Sleep Medicine report (2006) recommended relaxation as an effective and recommended therapy in the treatment of chronic insomnia based on several studies evaluating the efficacy of either CBTi or relaxation therapy individually.

Means et al (2000) compared treatment outcomes of students with insomnia treated with progressive relaxation therapy against untreated students with insomnia and those with no sleep complaint. The outcomes measures were both sleep and daytime functioning. The sleep measures improved with the treatment resulting in improved sleep quality, decreased SOL and decreased WASO. The daytime functioning measure did not show such improvements which the authors attribute to the length of treatment being too short to produce improvements on these measures; treatment occurred over a 2 week period.

Edinger et al (2001) compared the efficacy of CBTi and relaxation therapy against placebo using both objective (PSG) and subjective measures. Although CBT produced the largest effects, which would be expected as a combination of all the therapies found to have a positive impact on improving insomnia symptoms, relaxation therapy still produced a 16% reduction on WASO and 12% of RT group achieved a 50% reduction in pre-treatment WASO.

2.2. Physiological Perspectives

Monroe (1967) reported that several autonomic indicators were significantly elevated among poor sleepers and proposed that there were distinct relationships between sleep variables and physiological arousal. Subsequent research has provided evidence for an association between insomnia and elevated electromyography EMG (Haynes et al., 1974), increased heart rate (Haynes et al., 1981), more beta and less

alpha frequencies in the electroencephalography EEG (Freedman et al., 1982), higher body temperature (Adam et al., 1986), and increased urinary cortisol and adrenaline excretion (Adams et al., 1986; Vgontzas et al., 1998). However results of these experiments have appeared inconsistent and fail to find an association, for example, with elevated body temperatures (Vgontzas et al., 1998; Mendelson et al., 1984) or an increased secretion of corticosteroids and adrenaline (Frankel et al., 1973).

Bonnet and Arand (1995) suggest these inconsistencies may be due to individual differences of the physiological systems and that a global measure, such as metabolic rate (whole body oxygen use), will show more consistent results. In a study of patients with primary insomnia, they found that metabolic rate (whole body VO₂) was consistently elevated in those with insomnia, as compared to normal controls, not only during the night but also at all measurement points during the day. The authors concluded that a general disorder of hyperarousal is responsible for both daytime symptoms and nocturnal poor sleep and this hyper arousal may be the result of biological conditions (e.g. genetic factors, caffeine, etc.) or of psychological (cognitive and emotional) processes.

Perlis et al (1997), in their neurocognitive model, discuss cortical arousal as a form of somatic arousal to the extent that it is a measure of brain, as opposed to mental, activity. Additionally, however, it is also the case that cortical arousal is an analogue of 'cognitive arousal' as it can be measured as a form of EEG that has been found to correlate with cognitive processes (cited in Perlis et al., 1997). This form of EEG activity, which occurs in beta and gamma ranges, has been found to be elevated in patients with insomnia (Freedman 1986; Mercia and Gaillard 1991). Perlis et al., propose that high frequent EEG activity at or around sleep onset is a primary feature of chronic insomnia and that this form of conditioned arousal allows for a variety of

sensory and cognitive phenomena that do not occur in good sleeper subjects (Perlis et al., 1997).

2.3 Cognitive Perspectives

Empirical evidence has been found in the past decade providing insight into the role of unhelpful or maladaptive cognitions in insomnia. Wicklow & Espie (2000) obtained voice-activated audio recordings of spontaneous thoughts and sleep actigraph data to investigate systematically the relationship between independently gathered objective data on mental activity and sleep pattern. Content analysis yielded 8 categories of pre-sleep intrusion, and the regression model indicated that thinking about sleep and the anticipated consequences of poor sleep, along with general problem solving were the strongest predictors of objective sleep onset latency. Intrusions were subsumed under one of 3 factors: active problem solving (e.g., rehearsing/planning events), present state monitoring (e.g., thinking about sleep/not sleeping) and environmental reactivity (e.g., attending to external noises).

Cognitive arousal is consistently associated with PI, and having an 'overactive mind' has been the attribution of poor sleep rated most highly, by PI and GS alike (Broman and Hetta, 1994; Espie, Brooks & Lindsay, 1989; Evans, 1977; Lichstein & Rosenthal, 1980; Nicassio, Mendlowitz, Fussell & Petras, 1985). Evidence for the role of cognition in sleep-onset problems can be drawn from three sources. First, in an investigation of self-reported attributions, those with insomnia were 10 times more likely to cite cognitive arousal as central to their sleep difficulties compared with somatic arousal (Lichstein & Rosenthal, 1980). Second, questionnaire measures of pre-sleep cognitive activity have a high correlation with length of sleep-onset latency (Nicassio, Mendlowitz, Fussell, & Petras, 1985; Van Egeren, Haynes, Franzen, &

Hamilton, 1983). Finally, sleep-onset latency in good sleepers was lengthened by telling participants that they would have to give a speech immediately following the nap (Gross & Borkovec, 1982) and by instructing participant to fall asleep as quickly as possible (Ansfield, Wegner, & Bowser, 1996).

Espie, Brooks & Lindsay (1989) reported cognitive items on the Sleep Disturbance Questionnaire (e.g. 'my mind keeps turning things over', 'I am unable to empty my mind') as the most highly rated by those with insomnia; findings recently replicated by Harvey (2000). The Sleep Disturbance Questionnaire has been found to have modest internal consistency ($\alpha = 0.67$) (Espie et al. 2000). Although there is no gold standard measure of cognitive activity (Espie 2000), the Pre-Sleep Arousal Scale (Nicasso et al. 1985) is widely used and has satisfactory internal consistency for its somatic and cognitive subscales ($\alpha = 0.81$ and $\alpha = 0.76$, respectively). This self-report instrument, in which subjects describe the intensity of cognitive and somatic symptoms of arousal at bedtime, revealed that, although both the cognitive and the somatic subscales were significantly associated with sleeping difficulty, the cognitive subscale showed the strongest association.

Coyle & Watts (1991) employed a questionnaire design and found two distinctive sleep-interfering cognitive factors: cognitions about sleep attitudes and mental activity of a non-specific kind. Watts Coyle & East (1994) extended these findings by defining two groups: 'non-worrying insomniacs' and 'worrying insomniacs'. Investigating the relationship between worry and insomnia, Watts et al. (1994) found that much of the pre-sleep mental activity of 'worried insomniacs' revolved around work and general mental activity. In contrast, thoughts of 'non worried' insomniacs focused on the sleep process itself. Those with insomnia may also feel less in control of their thinking (Watts et al. 1995).

Formal analysis of sleep-interfering cognitions has been reported in several studies. An extended version of the Sleep Disturbance Questionnaire (Espie et al., 1989a) reported two distinct factors: "sleep attitudes," reflecting anxiety about the sleep process, and "mental activity," reflecting non-specific cognitive activity (Coyle & Watts (1991). In a study of young adults (Watts, Coyle & East, 1994) six factors of night-time intrusive thoughts, namely, thoughts about sleep; family and long term concerns; positive plans and concerns; somatic preoccupations; and work and recent concerns were identified. Extending these finding using a good sleeper comparison group, Harvey (2000) reported that the cognitions of individuals with insomnia were more focused upon worry about not getting to sleep, general worries, solving problems, the time and noises in the house, and less focused upon nothing in particular.

Type of monitoring	Examples
<i>During the night</i>	
Body sensations for signs consistent with falling asleep	Physical signs of "drifting off" such as slowing heart rate and loss of muscle tone
Body sensations for signs inconsistent with falling asleep	Heart pounding quickly, muscle tension
The environment for signs of not falling asleep	Noises outside and inside the house such as a dog barking or the neighbour arriving home
The clock to see how long it is taking to fall asleep	"It's 12:30am... I've been lying here for 2 hours and 25 minutes!"
The clock to calculate how much sleep will be obtained	"Oh no... it's already 2am... that means I'll only get 4 hours of sleep tonight!"
<i>On waking</i>	
Body sensations for signs of poor sleep	Heaving feeling in the head, heavy and tired eyes
The clock to calculate how many hours of sleep were obtained	"It's 7am... I finally got to sleep at 2am... and then woke up two more times... so that means I got about 4 and a half hours of sleep!"
<i>During the day</i>	
Body sensations for signs of fatigue	Heavy legs, sore shoulders, aching muscles, general feelings of fatigue, feeling "washed out"
Performance and functioning	No energy or motivation, memory problems, concentration problems
Mood	"I feel so miserable... I've really got to catch up on sleep tonight"

Table 2.1 Examples of Monitoring and Sleep Related Threats (taken from Harvey (2002) Behaviour Research and Therapy, 40 pp 876).

The three models below are presented separately to the behavioural, physiological and cognitive perspectives as integration of these is now starting to be seen. An important aspect to bear in mind here is that attention and monitoring are discussed within these models, which is relevant in the context of this thesis as a bias towards salient cues is central to the experiments discussed later. At the time of Harvey's Cognitive Model and Espie's Psychobiological Model the evidence for attention towards sleep salient environmental cues may have been secondary but objective evidence (MacMahon et al 2006, Marchetti et al 2006) is presented by Espie when formulating the Attention Intention Effort Model.

2.4 Harvey's Cognitive Model

Harvey published a cognitive model of insomnia in 2002 which made analogies with work carried out in the anxiety disorder field and focused on the cognitive processes which operate in insomnia leading to the individual becoming absorbed by their sleep problem. This model emphasises the excessive negatively toned cognitive activity when initiating sleep in insomnia. The key cognitive processes implicated in this model are attention, perception, counterproductive safety behaviours and erroneous beliefs which fuel the negative tone of the thoughts concerning sleep and lead to arousal and distress. As a consequence, sleep is disturbed as rumination over expected poor sleep and negative consequences of poor sleep alongside a lack of de-arousal does not facilitate sleep. Harvey proposes that the anxious state displayed by those with insomnia precipitates attentional narrowing and preferential allocation of attentional resources to sleep-related threat cues (Harvey 2001) and highlights that the sleep deficits seen in insomnia results from maladaptive

cognitive processes as opposed to a central deficit in the sleep/wake cycle and that the more repetitions around the cycle of negative cognitions leading to arousal and distress followed by selective attention and monitoring resulting in a distorted perception of sleep deficit will fuel further maladaptive cognitive processes, beliefs and behaviours..

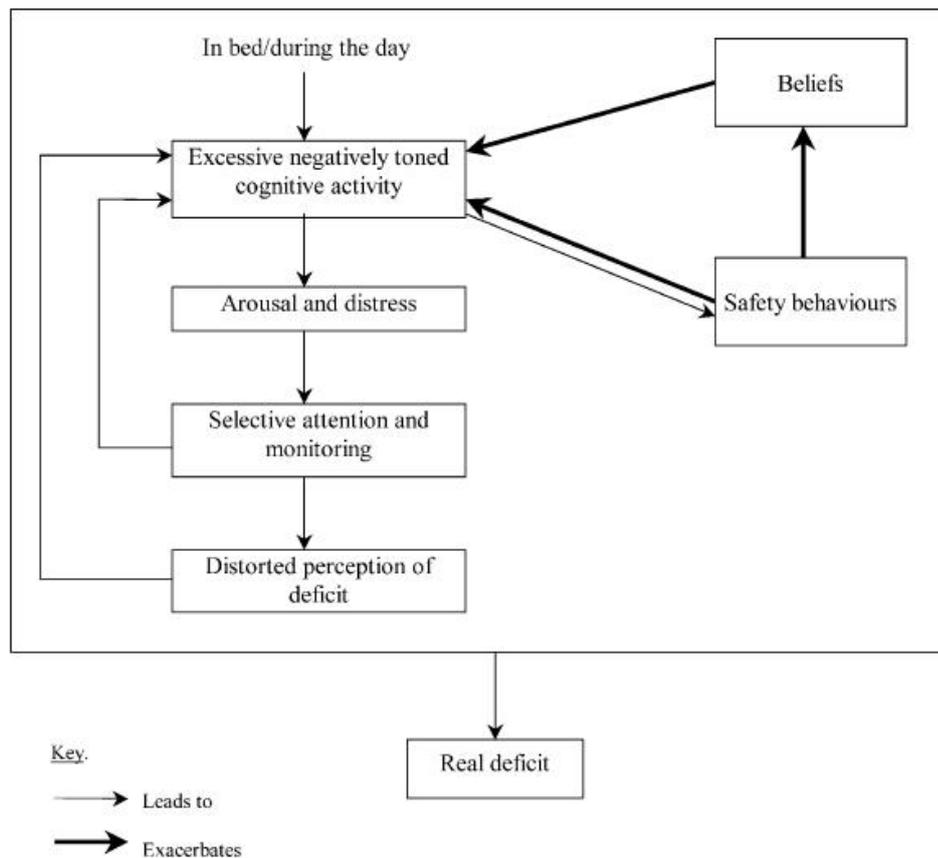


Figure 2.2 Harvey (2002) Cognitive model of insomnia.

Discussion of the cognitive factors and identification of monitoring as a major role in the maintenance of PI, relates to the previously discussed behavioural perspectives. There is a similar likelihood that PI will pick up on indicators compatible with sleep as indicators incompatible with sleep. Indeed, as the disturbance exists over time, faulty-conditioning becomes more likely and associations between sleep environments, objects and behaviours with poor sleep

become strong. Thus, over time these stimuli become concrete representations of sleep incompatible indicators generating maladaptive cognitive activity (worry, rumination).

2.5 Psychobiological Inhibition Model

With the aim of integrating the various factors which had been presented with empirical support into a conceptual framework, Espie (2002) proposed the Psychobiological Inhibition Model which at its core had good sleep and looked at the factors which disrupt normalcy rather than begin at the problem state. Automaticity and plasticity are important factors within this model as these are proposed to be compromised in insomnia with inhibitory insufficiency representing the 'critical mass of inhibition required to outweigh the stability of an individual's default sleep pattern'. Getting to sleep is not an action that a good sleeper undertakes with the aim of achieving sleep but sleep is flexible in that it does not immediately overwhelm us at a certain time every night, under normal conditions, but allows us to continue our personal and social lives into the night. It is these factors that Espie accounts as affected in insomnia through sleep-stimulus control, physiological de-arousal, cognitive de-arousal and daytime facilitation. Acute insomnia then is seen as logically arising at times of stress (cf. Healey, Kales, Monroe, Bixler, Chamberlin, et al, 1981; Kales & Vgontzas, 1992; Morgan & Clarke, 1997), but there should be a natural return to the 'default state', good sleep, after mental and emotional upset recedes. It is hypothesised then that in persistent insomnia there must be persisting inhibition. Perhaps to the extent to which sleep per se becomes a focus for concern is important in moderating whether or not there is a regression back to normal sleep, after an acute stressor is removed or the individual's response to stress habituates (Taylor, Espie &

White 2002). Espie has also highlighted the importance of paradoxical intention in insomnia. This model of insomnia proposes that anxiety responses may be conditioned not only to external, situational cues but also to the individual's behaviour, and thus the individual suffers from a performance anxiety. In paradoxical treatment counterproductive attempts to fall asleep are replaced by the intention to remaining passively awake or by giving up any direct effort to sleep. Indeed, this rationale is supported by the self reports of good sleepers who do not use any strategies to fall asleep (Espie, 1991).

The psychobiological inhibition model assumes such an interaction between haemostat and timer, but also describes how such an automated interaction may be inhibited by thoughts, emotions, and behavioural changes (Espie 2001) which builds on Spielman's (1991) model of insomnia acquisition.

In Morin's (1993) integrative conceptualization of insomnia hyper-arousal (emotional, cognitive, physiological) is the central mediating feature of insomnia, which interacts with dysfunctional cognitions, maladaptive habits and perceived consequences of insomnia. Although the psychobiological inhibition model makes no requirement of hyper-arousal, Morin's emphasis on cognitive factors parallels the cognitive/affective activation agent. The role of dysfunctional thoughts and beliefs in the psychological inhibition model is consistent with Edinger et al.'s (2000) interpretation that these influence self-perception of insomnia. Morin also stressed that bi-directional influence of the components, such that consequences often become causes and vice versa, similar to the proposed reciprocal interaction of the elements of the psychobiological inhibition model (Espie 2002).

2.6 Attention-Intention-Effort Model

Espie and colleagues (2006) proposed a route into PI along the attention-intention-effort (A-I-E) pathway which focuses on the inhibition of sleep-wake automaticity. In their conceptual paper, the authors propose that inhibition of sleep-wake automaticity can be attributed to 3 processes; selectively attending to sleep, explicitly intending to sleep and introducing effort into the sleep engagement process. This model has its roots in the Psychobiological Inhibition Model but develops these concepts further by outlining the actual processes which interfere with automaticity of sleep in insomnia. They acknowledge that the ICSD-2 criteria for PI which conveys both a sense of incrementing distress associated with sleeplessness and a preoccupying longing for sleep may serve as preconditions for attention bias. It is suggested that PI experiences sleep disruption, sleep loss and perceived sleep inadequacy that results in them becoming atypically motivated by sleep, which is increasingly incentivised in proportion to the preoccupation associated with it. Indeed under this framework, the desire for sleep of good quality may become a 'craving'.

However, Espie et al (2006) also propose that the perceived inability to sleep may also be conceptualised and experienced as a significant threat. Bedroom arousal may develop in PI as a result of the conditioning of non-verbal (environmental) and verbal signals (e.g. thoughts about sleeplessness) as threat cues which impact on selective attention. Taking the principle of automaticity into account, people who sleep well do not usually know how they do so. It is very possible that a normal sleeper would find it difficult to answer a question on how they sleep. Sleep is not in this sense an effortful process but rather passive and effortless. On the assumption that the individual with PI slept normally preceding the onset of insomnia, it is

understandable that the loss of such an automatic process may be considered as anxiety provoking or threatening.

What this model achieves is to bring together all the perspectives and models discussed so far in this chapter and factor them into a process leading to the development of the chronic insomnia disorder. With the initial attention component, we see the saliency of sleep/absence of sleep and how this has developed into something affective. This begins to develop the idea of sleep being an undemanding, natural transition for a GS but something which is very much attracting the attention of someone with insomnia. With attention to sleep as presented in this model, we see an attentive movement to sleep as presented in the research on attention bias to sleep but also a cognitive movement to sleep as presented in the Harvey model (2002). Monitoring as reported by Harvey (2001, 2002) is an appropriate way to highlight the integration of attention towards sleep and cognitive arousal, for example, the monitoring of the clock for time until rising.

With the intention part of the pathway, we now see behavioural aspects entering by sleep becoming a preoccupation and a task which needs to be resolved. Here we see the purposeful implementation of behaviours such as going to bed earlier or napping during the day to deal with the cognitive aspects of poor sleep. So not only are PI drawn to representations of sleep but behaviour is now modified with intent to alleviate the problem but which, in actuality, may increase impairment.

Inherent at this stage of the pathway is the notion of effort. Espie's psychobiological inhibition model really focuses on how automatic it is for GS to fall asleep i.e. very little effort is employed. Indeed, falling asleep may even happen when the GS is engaged in an activity such as reading. With insomnia, trying to get to sleep is a common feature which is obviously in opposition to the experience of GS. So

insomnia now presents itself as a disorder where we have sleep as a very conscious cognitive representation as well as behaviours carried out which can be counterintuitive to the initiation and maintenance of sleep.

The attention-intention-effort model therefore pulls together the various aspects which feed the insomnia disorder in an attempt to consolidate these concepts and present them rather than individual ideas but interacting to present unsatisfactory sleep.

2.7 Hyperarousal model

The most recent model proposed in the insomnia research field is the hyperarousal model (Riemann et al, 2010). This model incorporates higher level, for example cognitive, systems through to molecular and genetic processes in an attempt to consolidate the evidence particularly focusing on the neurobiological studies. Riemann and colleagues discuss how primary insomnia has mainly been conceptualized as a psychological disorder in recent times, initiated by a stressor and maintained by maladaptive behaviours alongside conditioned arousal. The notable absence of corroboration of the subjective experience of insomnia and objective measurement by PSG is highlighted with the authors proposing than an incorporation of the empirical evidence on several levels of neurobiological research with the psychological models, provides the concept of a psychobiological disorder which includes alterations on a psychological level which are associated with measurable transformations at physiological and neurological levels.

Empirical evidence related to insomnia and a hyperaroused state is presented on many levels by Riemann et al (2010) including, for example, genetic studies which show a familial link (Bastien and Morin, 2000), electrophysiology and MSLT tests

which show that the expected decreases in latencies to stages 1, 2 and REM are not seen but actually are increased in an insomnia sample (Bonnet and Arand 1995, 1998 and 2000), increased cortisol secretion pre-, during and post- sleep in insomnia (Vgontzas et al, 1998 and 2001) and hypoactivation of the medial and inferior prefrontal cortical areas in primary insomnia on a category and a letter fluency task conducted while awake in the MRI scanner alongside the absence of a behavioural deficit (Altena et al, 2008).

With the objective evidence for insomnia outlined in this hyperarousal model (Riemann et al, 2010) being integrated with the cognitive aspects of the disorder, evidence for which is provided for in the success and recommendation of CBTi as treatment for the disorder, the objective nature of attention bias in insomnia is highlighted. The contribution of the attention bias research to date (MacMahon et al 2006, Marchetti et al 2006, Spiegelhalder et al 2009 and Woods et al 2009) and the developing of that knowledge through the work contained in this thesis is important in providing that objective marker for the insomnia disorder.

Chapter 3

Attention bias

3.1 Attention bias

An attentional bias is said to have developed when disproportionate processing resources appear to be automatically allocated to exemplars, as compared with otherwise equivalent stimuli, producing a disproportionate impact on current cognitions. Selectively attending to salient stimuli has been investigated in a number of research fields in recent years including anxiety, alcohol, tobacco and other drug dependence, depression and cancer as well as insomnia research.

3.2 Alcohol and other drug attentional bias

Other studies have been carried out which indicate that regular use of certain drugs, such as tobacco and alcohol, is associated with biases in the processing of drug related cues, as they grab attention, elicit approach and are perceived as pleasant. Townshend and Duka (2001) used a dot probe paradigm where the alcohol and neutral stimulus pairs were presented for 500ms. The heavy social drinkers showed an attentional bias towards the alcohol related stimuli when compared to the occasional social drinkers.

Mogg et al (2003) investigated biases in overt orienting of attention to smoking related cues in cigarette smokers and to examine the relationship between measures of visual orienting and the affective and motivational valence of smoking cues. They measured direction and duration of gaze while participants completed a visual probe task (2000ms cue presentation time) as well as recording subjective and cognitive experimental measures of the motivational and affective valence of the stimuli. They found that smokers maintained their gaze for longer on smoking related pictures than control pictures compared to non-smokers. They were also faster to detect probes that replaced smoking related than control pictures, consistent with an

attentional bias for smoking related cues. Within smokers, longer initial fixations of gaze on smoking related pictures were associated with a bias to rate the smoking related pictures more positively, with greater approach tendencies for smoking pictures on the cognitive experimental task and with a greater urge to smoke. They concluded that this demonstrated that smokers show biased attentional orienting to smoking cues which is related to craving and the affective and motivational valence of the stimuli.

Bruce et al (2006) using an ICB flicker paradigm as a method of measuring attentional bias in problem drinkers in treatment where an artificially constructed visual scene comprising digitized photographs of real alcohol-related and neutral objects was presented. Problem drinkers detected a change made to an alcohol-related object more quickly than to a neutral object. Age- and gender-matched social drinkers showed no such difference. Second, problem drinkers given the alcohol-related change to detect showed a negative correlation between the speed with which the change was detected and the problem severity as measured by the number of times previously treated. Coupled with other data from heavy and light social drinkers, the data support a graded continuity of attentional bias underpinning the length of the consumption continuum.

Fadardi and Cox (2009), authors prolific in their research into motivation and attention to alcohol, carried out a study looking at alcohol related attention bias in social, hazardous and harmful drinkers and to determine whether it was related to their alcohol consumption level as well as if attention bias could be manipulated by alcohol attention control training in hazardous and harmful drinkers and if any change in attention bias effects actual alcohol consumption. The authors found that both hazardous and harmful drinkers showed significantly higher attention bias to alcohol

cues than social drinkers and that the outcome of a program of attention control training was positively correlated with the amount of alcohol they habitually consumed. Importantly, reductions in attention bias to alcohol were accompanied by reductions in alcohol consumption which maintained at 3 month follow up. This data would suggest that attention bias is strongly related to motivation to consume alcohol as a positive relationship exists between attention bias and consumption. In harmful and hazardous drinkers the attention bias to alcohol could be interpreted as a craving, physiologically and/or psychologically (Robinson and Berridge 1993, 2000, 2001).

3.3 Attention bias in depression

Gottlieb et al (2004) used a dot probe paradigm to understand the information processing biases of individuals diagnosed with clinical depression, general anxiety disorder and nonpsychiatric controls by presenting faces expressing anger, happiness or sadness for 1000ms. The authors found that those participants with clinical depression showed a significant processing bias towards sad faces only. This bias was specific to depression and emotional valence in that depressed patients showed the bias only towards sad faces and GAD patients did not show a bias towards any of the faces.

Kellough et al (2008) adapted a previously used paradigm by Eizenman et al (2003) which studies the visual scanning pattern of depressed and non-depressed individuals. Four complex emotional images (Figure 3.1) were presented simultaneously for 30 seconds which depicted dysphoric, positive and neutral themes. Depressed individuals spent more time attending to the dysphoric stimuli than their never depressed counterparts and this difference was affected by time i.e. it was consistent over the 30 second trial.

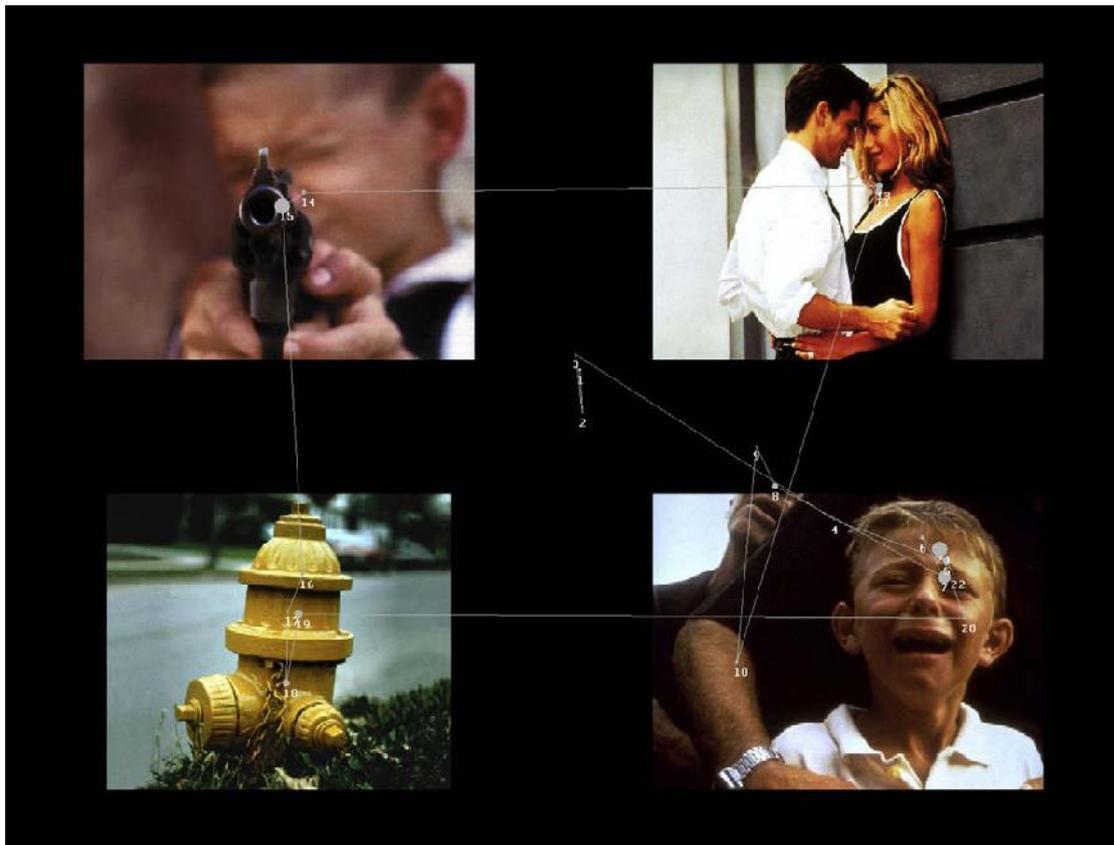


Figure 3.1 Example of stimulus presented with fixations superimposed, Kellough et al (2008).

Leung et al (2009) conducted a study to examine how inhibiting attention from and paying attention to preceding depression related information biased the attention to similar subsequent information in individuals with major depressive disorder. The ability to inhibit attention to depression related information was explored through the negative priming effect (inhibited reaction times) and the enhancement of attention to such information was explored through the positive priming effect (facilitated reaction times). The findings indicate that those with major depressive disorder as well as their healthy counterparts showed inhibition difficulties in attending to depression related information. However, only those with major depressive disorder showed

significantly facilitated reaction times on repeated related presentations relative to the initial presentation of the stimulus.

3.4 Attentional bias in anxiety

There is ample evidence that both generally and clinically anxious individuals pay particular attention to threatening stimuli (Mathews & MacLeod, 2005; Williams, Watts, MacLeod, & Mathews, 1997). According to Ohman's (1996) model of fear processing, fear-relevant stimuli capture the attention of individuals. This fast identification of fear-relevant stimuli activates an evolutionary-driven inborn defence system, which prepares the individual to take action to deal with the stimulus or situation. Such a natural and automatic response probably functions to reduce the presumed risk of being harmed by the fear-related stimulus. However, high trait and clinically anxious individuals probably display too much attention to aversive stimuli. As a result, they constantly explore the environment for potential threats and perceive the world as a dangerous place, which again makes them more anxious (Mogg & Bradley, 1998; Williams et al., 1997). This way the initially effective threat processing system eventually interferes with normal daily functioning, resulting in dysfunctional threat processing and even pathological fear.

Fox et al (2001) compared reaction time on a modified Posner paradigm using schematic face stimuli depicting neutral, happy or angry facial expressions which they presented to high and low trait anxious individuals for 100ms and 250ms. They found a cue validity effect, irrespective of anxiety group, when angry faces were presented compared to neutral or happy. However, this effect disappeared for the longer presentation time of 250ms i.e. the cue validity effect was moderated by anxiety group. The high state anxious individuals continued to show an inability to disengage

from the angry faces more than neutral or happy at this longer presentation time whereas the low state anxious individuals did not differ on performance on valid or invalid trials irrespective of facial expression. This would suggest that a 'normal' anxiety response to an angry face is seen at 100ms but what differentiates high anxiety from low is that we continue to see this delayed disengagement at 250ms in the high anxiety group.

Calvo and Avero (2005) monitored the eye fixations of high and low trait anxious individuals when presented with emotionally neutral, positive and negative (threat- or harm- related) scenes presented for 3 seconds. High trait anxiety was associated with attention towards all types of emotional stimuli in initial orienting i.e. probability of first fixation on emotional picture than on neutral of the picture pair, towards positive and harm stimuli in a subsequent stage of early engagement i.e. longer viewing times during the first 500ms following picture onset and attention away from harm stimuli in a later phase i.e. shorter viewing times and lower frequency of fixation during the last 1000ms of picture exposure. This study highlights the differential response to stimuli over presentation time.

Work has also been undertaken with phobic patients to determine the time course of attentional vigilance and avoidance. Rinck and Becker (2006) conducted an eye tracking study comparing highly spider fearful participants and nonanxious controls. They found that the spider fearful group's very first fixation was more frequently on a spider picture than the nonanxious controls but the spider fearful group quickly moved their eyes away from the spider they had first fixated resulting in shorter gaze durations. These continued for the remainder of the 1 minute presentation time. The authors concluded this early reflexive attentional bias towards

threat followed by avoidance may explain previous failures to find attentional biases in anxiety.

Roelofs et al (2010) carried out a study to investigate the approach and avoidance tendencies in socially anxious individuals to happy, angry and neutral faces by measuring reaction time of individuals to push a joystick forward (approach) or pull back (avoidance) in response to the colour of the facial images. The authors found that highly socially anxious individuals were faster in avoiding than approaching angry faces and this speeded avoidance was only seen when the gaze was directed towards the participant and not when averted. Also, the highly socially anxious group tended to avoid happy faces irrespective of gaze direction. This data was taken to reflect avoidance of subject directed anger and not of negative stimuli in general.

To summarize the attention bias research out with sleep, it would appear that attention allocation to salient stimuli varies with psychopathology. With alcohol and other drugs, attention is allocated to the salient stimulus quickly and gaze remains there. This could be said to be reflective of craving as when motivation to consume is present, salient stimuli are approached and engaged with for the duration of their presentation. There is also flexibility with their selective attention as alcohol consumption appears to decrease alongside attention bias to alcohol. With depressive stimuli, those with depressive disorder tend to initially avoid but return and engage with negative/dysphoric stimuli compared to anxiety disorders where threatening stimuli are approached and engaged with earlier in processing but then avoided reflecting an attention allocation of approach followed by avoidance.

Chapter 4

Attention bias in insomnia.

In this chapter, the research to date on attentional bias in insomnia will be reviewed and will lead into an outline of the terminology and constructs addressed within this area.

4.1 Attention bias to sleep in insomnia

Taylor et al (2003) investigated the role of attentional bias in the development of persistent insomnia by using an emotional Stroop task with two groups of people with cancer who developed sleep-onset difficulties. Both acute and persistent insomnia groups demonstrated attentional bias for cancer-related words but only the persistent insomnia group demonstrated attentional bias for sleep-related words. With the lack of a processing bias in the acute insomnia group, these findings are consistent with the reported association of sleep related mental preoccupation with development of persistent insomnia.

Marchetti et al (2006) demonstrated poor sleepers selectively attending towards sleep related stimuli using a computerized ICB flicker paradigm by comparing the change detection latencies for sleep and neutral stimuli in good sleepers (GS), individuals with delayed sleep phase syndrome (DSPS) and individuals with psychophysiological insomnia (PI). The DSPS group was employed as a further, clinical, control sample of people who, like PI participants, had sleep-onset problems, but who would not be expected to exhibit cognitive arousal as an explanatory mechanism for their continued wakefulness. PI were significantly faster to respond to sleep stimuli compared with GS and DSPS as well as significantly slower than GS and DSPS with neutral stimuli. The stimuli presented in this study were devised for Jones et al (2005) where individuals were asked to list five or more objects that they associated with sleep and going to bed. Evaluation of the lists yielded a 'top 12' most

commonly suggested items. These items were photographed and embedded in a collection of 12 neutral, individually photographed objects. A further 30 individuals were then asked to rate all 24 photographs on a 1–10 sleep-relatedness scale (1 highly sleep-related, 10 not sleep-related at all). In this study the item with the second highest rating (teddy bear) was used and paired with an entirely sleep-neutral item (a mug). In the previous study, Jones et al (2005) where the sleep ICB flicker paradigm was developed and first used, found a similar bias was demonstrated in PI but the authors also did a regression analysis which showed a significant relationship between sleep quality and bias for sleep stimuli with PSQI score accounting for 10.6% variance in number of flickers taken to identify the change.

MacMahon et al (2006), using the dot probe task, compared three experimental groups PS, GS and DSPS. Those in the PI group showing a significantly greater processing bias toward sleep-related words (in comparison to neutral words) when compared to the GS and DSPS groups. Notably, the DSPS groups did differ from the other 2 groups but not in that they displayed vigilance towards sleep but were slower to respond than PI and GS, suggesting that the underpinning mechanism maintaining DSPS is not an attention bias but a more general deficit in performance.

Spiegelhalder et al (2008) also used the emotional Stroop paradigm to investigate sleep related attentional bias in a novel way in that they included sleep experts as a comparison group along with PI and GS to address the question of frequency of concept usage (FOCU). The relevance of FOCU is demonstrated in a study conducted in alcohol attention bias research by Ryan (2002) where an attention bias to alcohol was seen in problem drinkers as well as the staff from the substance use clinics. This work suggests that experts may show a similar attention bias due to an emotional connection to the concept in question but not necessarily in the same

way as the clinical group being studied. Spiegelhalder and colleagues found that PI showed a significant bias towards sleep compared to the sleep expert group and not compared to the GS. This result is unexpected as it may be less surprising not to see any difference in performance between experts and PI due to emotional investment, familiarity and/or cognitive relevance but it would be expected that differences in performance would be seen between PI, sleep experts and GS as GS would be the group to which sleep was the least relevant. This finding brings into question relevance, saliency and motivation behind stimuli presented to participants in relation to their sleep status.

In a subsequent paper, Spiegelhalder et al (2009) raised the question of whether sleep related attentional bias is due to sleepiness or sleeplessness. Again, using the emotional Stroop presenting sleep and neutral words, the authors found that sleepiness had an impact on bias for sleep words as well as sleep quality. They found a bias towards sleep as sleep quality worsened but only when sleepiness was low. If sleep quality deteriorated alongside an increase in sleepiness, the attention bias was attenuated. This would confirm that the attention bias to sleep that has been established by research to date is not attributable to sleepiness due to sleep loss but cognitive factors underpinning the insomnia disorder. Spiegelhalder et al (2009) draw similarities with the substance dependence literature where an attentional bias has been attributed to craving and so attention bias to sleep may be due to a greater need for sleep being associated with an attentional preference for sleep related stimuli. This finding is relevant in the context of further understanding of what fuels attentional bias in PI.

From clinical practice, it is proposed that PI selectively attend to and monitor for sleep-related cues such as body sensations which are consistent or inconsistent

with falling asleep and the environment for signs of not falling asleep (Harvey, 2002). One of the sleep related cues which features heavily with stress of not falling asleep is monitoring the clock, for example, to see how long the individual has been in bed without sleep and how many hours are left before they have to start their day. In relation to this, Tang, Schmidt and Harvey (2001) compared NS and PI who were either encouraged or discouraged to monitor the clock. The researchers found that those individuals encouraged to monitor the clock increased their sleep onset latency, irrespective of their sleep efficiency. Hence, the clock displaying times normally associated with sleep is an ecologically valid stimulus which is relevant in both the perspective of the patient and clinical research but also within everyday life. Most people would be able to recall at least one occasion where sleep eluded them while being aware the time to rise was approaching. Woods et al (2009) presented images of an alarm clock showing sleep times using a modified Posner paradigm to PI and GS. PI were slower on invalid trials compared to GS demonstrating delayed disengagement from the clock showing sleep times and a trend towards enhanced engagement by performing faster on valid trials which is taken as further support of selective attention to sleep as well as monitoring of external cues such as the clock in PI (Appendix N). Interestingly, GS did not show any variability on performance over valid and invalid trials when the expected Posner effect would be faster on validly cued trials and slower on invalid trials but this is not seen here. This would suggest that sleep times do not influence attentional processing in the same way in GS than PI who hold sleep times as much more relevant.

The studies above demonstrate that PI show an attention bias towards sleep. This research has involved a variety of paradigms (ICB Flicker, dot-probe, emotional Stroop and modified Posner), stimuli types (words and images), presentation times

(100ms, 500ms and until response) and comparison groups (GS, DSPPS and sleep experts). Throughout all this, PI have shown a differential response and it is reasonable to conclude that an attention bias to sleep is part of the psychophysiological insomnia psychopathology.

At this stage in the research, it would be valuable to further understand what mechanism is underpinning the attention bias in PI. Espie et al (2006) proposed a route into PI along the attention-intention-effort (A-I-E) pathway which focuses on the inhibition of sleep-wake automaticity. In their conceptual paper, the authors propose that inhibition of sleep-wake automaticity can be attributed to 3 processes; selectively attending to sleep, explicitly intending to sleep and introducing effort into the sleep engagement process. They acknowledge that the ICSD-2 criteria for PI which conveys both a sense of incrementing distress associated with sleeplessness and a preoccupying longing for sleep may serve as preconditions for attention bias. It is suggested that PI experiences sleep disruption, sleep loss and perceived sleep inadequacy that results in them becoming atypically motivated by sleep, which is increasingly incentivised in proportion to the preoccupation associated with it. Indeed under this framework, the desire for sleep of good quality may become a ‘craving’.

However, Espie et al (2006) also propose that the perceived inability to sleep may also be conceptualised and experienced as a significant threat. Bedroom arousal may develop in PI as a result of the conditioning of non-verbal (environmental) and verbal signals (e.g. thoughts about sleeplessness) as threat cues which impact on selective attention. Taking the principle of automaticity into account, people who sleep well do not usually know how they do so. It is very possible that a normal sleeper would find it difficult to answer a question on how they sleep. Sleep is not in this sense an effortful process but rather passive and effortless. On the assumption that

the individual with PI slept normally preceding the onset of insomnia, it is understandable that the loss of such an automatic process may be considered as anxiety provoking or threatening.

Research out with the insomnia field has helped form the hypotheses of Espie and colleagues with regard to the role of attention in development and maintenance of insomnia disorder and this research has been reviewed in Chapter 3.

4.2 Constructs and terminology

At this point, it may be wise to address the terminology being used to discuss concepts to which we are attributing our findings. Within the A-I-E model, Espie and colleagues introduce the terms ‘threat’ and ‘craving’ when discussing the possible underlying mechanisms to the attention component of the pathway. The concept of ‘sleep cue as a threat’ is a reasonable application as the information processing literature on anxiety disorders emphasises attention bias toward emotionally threatening stimuli. However, the authors do address the possibilities that salience may be due to other reasons, such as craving.

It is worth taking a moment to consider the most appropriate terminology to be used in answering these questions. Is the jump from data points to ‘threat’, ‘anxiety’ and ‘craving’ one which can reasonably be made? How do we define these constructs in relation to PI? With these concerns in mind, I propose that we adopt the terms currently used in the literature on attentional bias in other research fields and when discussing the attention system.

4.3 Components of attention system- engage and disengage

Posner suggested that the attention system comprises measurable cognitive components (shift, engage, disengage; Posner, 1980), which are subserved by specific, neural sub-systems (Posner and Peterson, 1990) and which are open to modulation by negative emotional stimuli (Stormark et al., 1995). In the original Posner cue-target paradigm, participants responded to a target appearing in the same (valid) or opposite (invalid) location as a previously presented cue. Results indicated faster detection of targets on cued trials, particularly at short (<200ms) cue-target intervals. This facilitation effect was taken as evidence of the time-cost of disengaging attention from the cue to the target on invalid trials (Posner and Peterson, 1990; Posner, 1988). Over recent years, researchers have begun to apply Posner's attention model to develop a paradigm which can determine whether threatening stimuli can attract attention i.e. modulate the engagement component of covert attention, and/or hold attention, i.e. modulate the disengage component (Broomfield et al., 2004).

4.4 Overview of terminology

From all the above work, the main terms utilized are 'approach', 'engage', 'avoid' and 'disengage'. By using these terms we are able to compile a sequence of events with regard to an individual's attention in relation to a stimulus which we can then draw inferences from with a more complete picture, rather than drawing conclusions within constructs which require further definition. Therefore, 'approach', 'engage' and 'hold' will be used to refer to a positive, contact response and 'avoid' and 'disengage' to a negative, aversion response. It may be the case that these events

do not occur independently and so these terms may be used to build a sequence of events indicating attention allocation.

Chapter 5

Research questions and hypotheses

5.1 Research plan

We therefore find ourselves at the research juncture of having established selective attention to sleep in PI but being able only to hypothesize and draw parallels with other literature about what is fuelling this phenomenon. The aim of this thesis is to attempt to further our understanding of the underlying mechanisms of selective attention to sleep in PI. The way I have attempted to do this is by:

- Establishing if selective attention to sleep is specific to psychophysiological insomnia or if it is seen in other populations who have a sleep complaint.
- Manipulating the presentation time of the stimuli presented within the same cognitive probe paradigm.
- Employing methodologies which are new to the sleep research field which provide data over a longer time period.

This has resulted in four experimental chapters within this thesis. Three of these experiments, experiments 1-3, use modified Posner paradigms to measure attention bias in PI compared to GS. It would therefore be informative to explain some of the terms relevant to the Posner paradigm before outlining the hypotheses. The Posner paradigm provides a measure of reaction time on valid and invalid trials. What these terms mean is that a target is either validly or invalidly cued. Figures 5.1 and 5.2 below illustrate the differences between these trials. The information gained from the different trial types is a measure of attentional engagement on valid trials and disengagement on invalid trials. By presenting cues in a different position from the target, the participant has to disengage their attention and move it toward the target.

The longer the time taken to do this implies that attention is more difficult to disengage.

5.2 Experiment 1 Attention Bias to sleep and acute insomnia

Firstly, individuals with acute insomnia and GS are compared on their performance on a modified Posner paradigm presenting sleep and non-sleep stimuli for 100ms which provides information on the specificity of attention bias within insomnia and those showing dissatisfaction with their sleep.

Research Question: Is there evidence of a general attention bias in those expressing dissatisfaction with their sleep?

Hypothesis: Attentional bias to sleep will be not be evident in those with acute insomnia or GS.

Two chapters then cover the continuing work from Woods et al (2009) looking at engagement and disengagement of attention to sleep and day times presented on an alarm clock within a modified Posner paradigm in PI and GS. This is done over two experiments presenting the clock stimuli for 100ms and 250ms moving forward to build a picture of attention allocation over time from which we can then draw parallels with other research fields to understand what underlies attention bias to sleep in insomnia.

5.3 Experiment 2 100ms clock experiment

In the following experimental chapters, PI will be used in reference to the insomnia group and good sleepers as GS. This is for ease of reading and criteria for recruitment into these experimental groups can be found in chapter 6.

Research questions: Is it the clock to which PI selectively attend or the times displayed? Do PI only show selective attention to sleep times or are day times also salient?

Hypotheses:

- PI will have longer reaction times on invalid sleep time trials compared to invalid day time trials.
- PI will have longer reaction times on invalid sleep time trials compared to GS.
- PI will have shorter reaction times on valid sleep time trials compared to valid day time trials.
- PI will have shorter reaction times on valid sleep time trials compared to GS.

5.4 Experiment 3 250ms clock experiment

Research question: Do PI maintain their gaze on sleep times and therefore continue to show delayed disengagement at a longer presentation time?

Hypotheses:

- PI will have longer reaction times on invalid sleep time trials compared to invalid day time trials.
- PI will have longer reaction times on invalid sleep time trials compared to GS.

- PI will have shorter reaction times on valid sleep time trials compared to valid day time trials.
- PI will have shorter reaction times on valid sleep time trials compared to GS.

5.5 Experiment 4 Semantic eye tracking

The final experimental chapter involves PI and GS viewing sleep positive, sleep negative and neutral words alongside pseudo words while being fitted with an eye tracker so a continuous picture of where their attention is can be painted over time, again, allowing us to understand approach and avoidance to sleep.

Research question: On presenting sleep stimuli, do poor sleepers approach and then avoid or approach and maintain their gaze? These different responses reflect an anxious response or craving response, respectively.

Hypotheses:

- PI will show faster engagement to sleep related words compared to GS.
- PI will remain fixated on negative sleep words for longer than positive sleep and neutral words.

Chapter 6

Core Generic Methodologies

This chapter will describe factors which are common to all experiments discussed in this thesis. It will cover how participants were recruited, included in the studies, screened and the data analysed.

6.1 Participant Selection

Participants were recruited through advertising online within the University of Glasgow and through advertising on the psychology department's undergraduate student portal for students to obtain course credits. The title of the study as displayed on the departmental website was 'People with insomnia wanted' and the study description can be found in Appendix A.

When an individual selected the study an email was sent to myself as principal investigator notifying me of their interest and providing a contact email address. An email was then sent to the potential participant asking for a telephone number on which to contact them to ask some questions about their sleep. During this subsequent telephone conversation, the University of Glasgow Sleep Centre Preliminary Screening Interview (Appendix B) was completed. This tool allows participants to be prospectively assigned to a sleep quality group (PI or GS), screened for other sleep disorders as well as physical and/or mental health issues which could possibly effect their sleep. On successfully completing the phone interview and confirming that the individual met the criteria for inclusion in the study, an appointment was made to complete the experiment in the Psychology department.

6.1.1 Experiment 1

The data presented in this chapter is part of a larger scale ESRC funded study 'The role of psychological adjustment in the evolution of chronic insomnia'.

Participants were recruited into this study through emails sent through faculties of the University of Glasgow, poster and media advertisement. A phone number was provided which potential participants could call and request a call back. When each individual's call was returned, information was provided about the study and a standardised screening interview was conducted which included questions relating to insomnia as defined using Research Diagnostic Criteria for Insomnia and the International Classification of Sleep Disorders 2nd Edition (ICSD-2): a sleep-onset duration of at least 30 min, a wake time after sleep onset of 30 min or more, a sleep efficiency of less than 85%, calculated as the ratio between total sleep time and time spent in bed and that the sleep disturbance was causing distress and affecting normal psychosocial or occupational functioning with the period of insomnia lasting less than 3 months. Additional demographic information was also taken at this point.

During the initial telephone interview, those under 18 years old or in an assisted living environment were excluded. Additionally, individuals currently seeing a sleep specialist, taking hypnotics or reporting a history of neurological trauma, psychiatric illness, or other sleep disorder, according to the ICSD-2, were also excluded.

Following screening, all participants arranged to spend 2 consecutive nights at the University of Glasgow Sleep Centre. The 2 weeks prior to their overnight stay, participants were asked to complete a daily sleep diary and actigraphic assessment. On the designated nights for their stay at the Sleep Centre, participants arrived at approximately 6.30pm, were shown to their room and oriented with the facilities before completing the modified pictorial Posner paradigm followed by the questionnaire measures. This procedure provided the data reported here.

6.1.2 Experiments 2, 3 and 4.

Participants were recruited through advertising online within the University of Glasgow and through advertising on the psychology department's undergraduate student portal for students to obtain course credits. The title of the study as displayed on the departmental website was 'People with insomnia wanted' and the study description can be found in Appendix A. These different recruitment methods allowed access to both those experiencing difficulties with their sleep and good sleepers.

When an individual selected the study online an email was sent to myself as principal investigator notifying me of their interest and providing a contact email address. An email was then sent to the potential participant asking for a telephone number on which to contact them to ask some questions about their sleep. During this subsequent telephone conversation, the University of Glasgow Sleep Centre Preliminary Screening Interview (Appendix B) was completed. This tool allows participants to be prospectively assigned to a sleep quality group (PI or GS), screened for other sleep disorders as well as physical and/or mental health issues which could possibly effect their sleep. On successfully completing the phone interview and confirming that the individual met the criteria for inclusion in the study, an appointment was made to complete the experiment in the Psychology department.

6.2 Sleep Quality Assessment

The sleep quality assessment begins with the initial recruitment phone call and completion of the University of Glasgow Sleep Centre Preliminary Screening Interview which is structured around the ICSD-2 statement of criteria for psychophysiological insomnia and the DSM IV statement of criteria for primary

insomnia as well as self report measures i.e. Pittsburgh Sleep Quality Index and the Insomnia Severity Index. On completion of the experimental task, the assessment of sleep quality continued, and the participants completed the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989, Appendix C) as well as the Insomnia Severity Index (ISI; Bastien C.H., Vallières A. & Morin C.M., 2001, Appendix D). The PSQI provides a reliable, valid, and standardized measure of sleep quality i.e. to discriminate between "good" and "poor" sleepers. A PSQI global score > 5 indicates that a subject is having severe difficulty in at least two areas, or moderate difficulty in more than three areas of sleep quality. This global score conveys information about the severity of the subject's problem, and the number of problems present, through a single measure which has validated this cut off and confirmed reliability (Cronbach's $\alpha = 0.85$, test re-test $r = 0.84$; Backhaus et al., 2002). The ISI is a reliable and valid method of quantifying perceived insomnia severity showing adequate internal consistency and is a valid and sensitive measure to detect changes in perceived sleep difficulties with treatment. In addition, there is a close convergence between scores obtained from the ISI patient's version and those from the clinician's and significant other's versions (Bastien et al, 2001).

6.3 Assessment of Psychopathology

The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983, Appendix E) is a self-assessment scale found to be a reliable instrument for detecting states of depression and anxiety in the setting of a hospital medical outpatient clinic. The anxiety and depressive subscales are also valid measures of severity of the emotional disorder. A review of the literature on the validity of the HADS (Bjelland et al, 2002) found a mean correlation between the subscales

measuring anxiety (HADS-A) and depression (HADS-D) of 0.56 with a mean Cronbachs alpha for HADS-A of 0.83 and for HADS-D of 0.82. In previous studies (Marchetti et al, 2006) on attention bias insomnia, the Beck Depression Inventory (BDI) and Spielberger Trait Anxiety Inventory (STAI) had been used to provide measures of anxiety and depression. The HADS provides a clinical, diagnostic element as opposed to a predispositional measurement and allows for cut-offs of within normal range, mild, moderate and severe disordered state.

6.4 Insomnia Disorder Assessment

The Daytime Functioning and Sleep Attribution Scale (DFSAS; Kyle, Morgan and Espie 2010, Appendix F) is a new insomnia-specific measure to probe daytime impairment and poor sleep attributions. It is a two part measure designed to assess impairment in daytime domains commonly reported by individuals with insomnia (part 1), and, importantly, sleep-related attributions in accounting for such reported daytime impairment (part 2). Parts 1 and 2 successfully discriminated PI individuals and normal sleepers (both $p < .001$). Both parts 1 & 2 had high sensitivity and specificity (>87%). Cronbach's alpha was 0.81 for part 1 and 0.89 for part 2. DFSAS scores (part 1) were positively associated with insomnia severity (ISI; $\rho = 0.49$) and occupational impairment (OISQ; $\rho = 0.76$), and negatively associated with several SF-36 dimensions, including vitality ($\rho = -0.51$), general health ($\rho = -0.54$) and emotional role limitations ($\rho = -.043$).

The Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS; Morin et al 1993, Espie et al 2000, Appendix G) helps to identify particular salient, irrational and affect laden thoughts intruding prior to sleep onset. This 10 item scale is reported as having a satisfactory internal consistency of 0.69 and is sensitive to treatment

related improvements in sleep related cognitions as well as a means of descriptive quantification of maladaptive cognitions in insomnia (Espie et al, 2000).

The Sleep Preoccupation Scale (SPS; Ellis et al 2004, 2005) is a 20 item scale which assesses the frequency of sleep-related thoughts, feelings or behaviours during the day with higher scores indicating more sleep preoccupation. Items include 'I cannot stop thinking about the sleep during the day' and 'I keep checking to see if I look tired'. The SPS has been shown to have a high internal reliability in both student and older adult samples ($\alpha = 0.79$ and 0.82 , respectively) and to discriminate late-life insomniacs from normal sleepers (Ellis et al, 2007).

Both the DBAS and SPS provide measures of how much participant's thoughts are influencing their sleep or inability to sleep and how much of their cognitive space is being taken up by thinking about their sleep. This is relevant within the context of this thesis as the concern here is provide an objective measure of saliency and attraction to/avoidance of attention to sleep.

6.5 Inclusion/Exclusion Criteria

PI participants met combined DSM-IV and ICSD-2 criteria for primary insomnia as well as scoring >8 on the PSQI. DSM-IV criteria for primary insomnia is a difficulty initiating or maintaining sleep or non-restorative sleep associated with significant distress or daytime impairment, not due to other medical, psychiatric or sleep disorders (DSM-IV; APA, 1994). The ICSD-2 subdivides the disorder further into sleep disturbances that are either initial, maintenance or terminal insomnia with cognitive and/or physiological components. With regard to length of disorder, the DSM-IV criteria of at least 3 months is applied for the chronic condition.

Buysse et al (1989) reported the global PSQI score of > 5 yielded a diagnostic sensitivity of 89.6% and specificity of 86.5% (kappa= 0.75, $p > 0.001$) in distinguishing good and poor sleepers. The decision was made to create clinical poles using the PSQI by increasing the score cut off for PI. Exclusion criteria for PI included active psychological or drug interventions for sleep problems or when a sleep disorder other than insomnia was suspected. GS were required to score <5 on the PSQI, report no problems with their sleep and have no history of sleep problems. For both groups, the Hospital Anxiety and Depression Scale was used to provide a comparative measurement of anxiety between GS and PI but was not used for exclusion purposes.

6.6 Test Location

Participants were tested in an assessment room in the Department of Psychology at the University of Glasgow for the two modified clock Posner experiments, a specially equipped eye tracking room in the Department of Psychology for the eye tracking experiment and in an assessment room at the University of Glasgow Sleep Centre for the acute insomnia study.

6.7 Data analysis

All data obtained was entered into and analysis was carried out in the statistical program SPSS for Windows, version 15 (Release 15.0.0, 6th Sept. 2006). Each participant was assigned a unique number at recruitment so therefore no identifying data was stored. Questionnaire data was scored manually by the primary investigator and entered under appropriate headings into the SPSS database.

For each experiment conducted, mean reaction times were calculated and Analysis of Variance (ANOVA) carried out to determine whether manipulating the independent variables (IV) statistically significantly affected the variance seen within and between the PI and GS groups. Effect sizes were also calculated which enabled any effects of sleep group or the IVs to be quantified. A third set of analyses were carried on for Experiments 1 to 3 with regression analyses establishing whether a relationship exists between variables and whether prediction on one variable is possible on the basis of other variables of interest. Regression analyses was not carried out for the eye tracking data from Experiment 4 as other parameters were used to map out the performance over time. These parameters are outlined in detail in Chapter 10.

6.8 Experimental power

A power calculation carried out prior to the study suggested that 21 participants would be required in each of the groups to detect statistically significant differences at a power of 0.8 with an alpha level set at 0.05. This number had been shown to provide statistically significant differences in previous attention bias studies (Woods et al, 2009) and was therefore set as the ideal minimum number for recruitment.

Chapter 7

Experiment 1

Attention bias to sleep and acute insomnia.

The research on attention bias in insomnia outlined elsewhere in this thesis (Macmahon et al 2006, Marchetti et al 2006) has concentrated on the chronic primary insomnia condition when looking at attention bias to sleep. At this point, it would be reasonable to confirm that selective attention to sleep is specific to PI and not found in other populations where sleep is disrupted.

- **Research Question:** Is there evidence of a general attention bias in those expressing dissatisfaction with their sleep?
- **Hypothesis:** Attentional bias to sleep is a specific marker of the psychophysiological insomnia condition and will not be evident alongside a sleep complaint such as acute insomnia.

This research question and hypothesis are based on a body of work carried out by the sleep research group at the University of Glasgow which contributes to Espie et al's (2006) theoretical review and proposal of the Attention-Intention-Effort (A-I-E) pathway. These experiments have been presented previously in Chapter 4 (Taylor et al 2003, MacMahon et al 2006, Marchetti et al 2006) and have all shown, using various experimental paradigms and stimulus types, those with insomnia selectively attend to sleep. The graph below is taken from an experiment by Marchetti (2006) comparing reaction times of those with insomnia (PI), good sleepers (GS) and those with delayed sleep phase syndrome (DSPS) on a modified pictorial Posner paradigm. The same paradigm is used in the experiment presented in this chapter and therefore adds acute insomnia data alongside the previously collected data with a chronic, psychophysiological insomnia group.

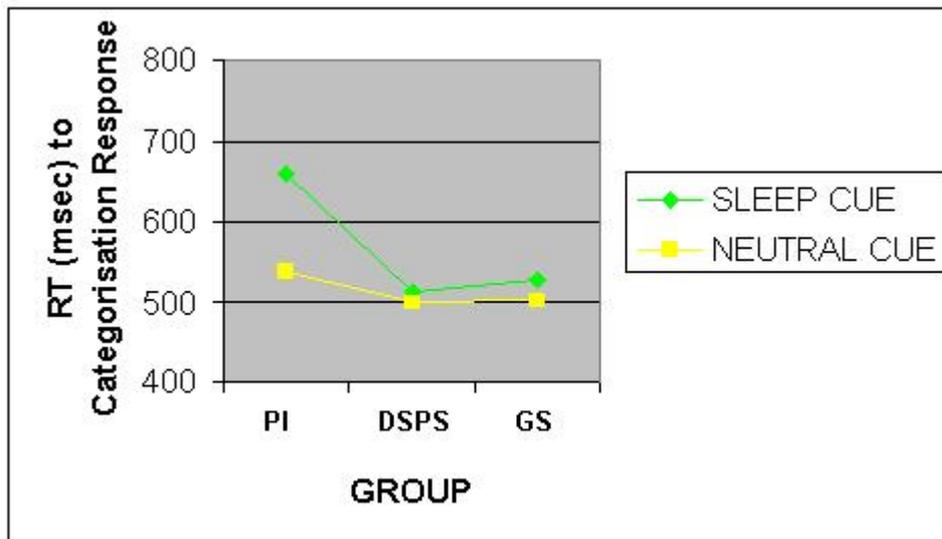


Figure 7.1 Reaction times on invalid trials for an insomnia group (PI), a delayed sleep phase group (DSPS) and a good sleeper group (GS). Taken from Marchetti (2006).

As the graph shows, those with insomnia take longer to react on an invalid trial presenting a sleep cue. This has been taken as evidence of delayed attentional disengagement to sleep compared to neutral cues by Marchetti (2006) which is in line with the other work done in this area of selective attention to sleep in chronic insomnia.

In this experimental chapter, the premise that selective attention to sleep is specific to the chronic condition and not an indicator of poor sleep is investigated by establishing the absence of selective attention to sleep in an acute insomnia group. To clarify, the research to date has compared insomnia defined as persistent or chronic in that the complaint had been present for at least 3 months. This is in line with ICSD-2 criteria for persistent insomnia. The experiment

discussed in this chapter compares an acute insomnia group where the complaint has been present for less than 3 months.

7.1 Methods

7.1.1 Apparatus and Stimuli

Forty-eight digitised single stimuli pictures represented the entire experimental stimulus set. There were 24 sleep-related picture stimuli and 24 non-sleep related picture stimuli. Presentation of each picture was repeated randomly 3 times generating a total of 144 critical trials. In addition, there were four catch trials (no target) to prevent participants developing an automated response (see Stormark et al., 1997). Target stimuli consisted of either a horizontal or vertical colon (: or ..). Cue and target stimuli were all presented inside two boxes (5.3 cm high and 3.0 cm wide) and positioned 2.0 cm to the left and the right of the central fixation point (cross shape).

Altogether, two thirds of the trials were valid (target replaces cue) and one third invalid (target in opposite location to cue) as illustrated in Appendix I. This unbalanced ratio of trials is routine in emotional cue-target paradigm studies (Fox et al. 2002) and leads to the cue-validity effect. The typical paradigm effect reveals that valid trials are detected quicker than invalid trials as the exogenous cue induces a covert orienting of attention to the cued location leading to faster reaction times on valid trials and slower on invalid trials. In the Posner et al. (1978) original study, the ratio of valid to invalid trials was 80% valid to 20% invalid. The authors proposed this unequal ratio allowed them to examine both the benefits of knowing where the target stimulus would appear as well as the costs of it appearing in an unexpected position. In Posner (1980), the author reports ‘highly significant benefits from valid information and highly significant costs when the trial is invalid...’.

Following more recent discussion in the literature on selective attention tasks, Fox et al. (2001) used a modified Posner task to investigate the pattern i.e. engagement and disengagement of selective attention found in individuals with high and low state anxiety levels. They modified the Posner task by cueing the target with emotionally salient words or faces. Fox and colleagues found that such salient cues did not facilitate response on valid trials but did influence response times on invalid trials by delaying disengagement from the salient cue. These results were found using a two thirds valid and one third invalid trial ratio, the unequal proportion of trials following on from the original Posner (1978) paper. This one third to two thirds ratio overcomes the notion of predictability and ‘inhibition of a prepared response’ (Fox et al. 2001). This ratio was used by Marchetti (2006) from which this experiment taken. Participants were given 4 practice trials to ensure they were comfortable with completing the task.

7.1.2 Procedure

The data presented in this chapter is part of a larger scale ESRC funded study ‘The role of psychological adjustment in the evolution of chronic insomnia’. Participants were recruited into this study through emails sent through faculties of the University of Glasgow, poster and media advertisement. A phone number was provided which potential participants could call and request a call back. When each individual’s call was returned, information was provided about the study and a standardised screening interview was conducted which included questions relating insomnia as defined using Research Diagnostic Criteria for Insomnia and the International Classification of Sleep Disorders 2nd Edition (ICSD-2): a sleep-onset duration of at least 30 min, a wake time after sleep onset of 30 min or more, a sleep

efficiency of less than 85%, calculated as the ratio between total sleep time and time spent in bed and that the sleep disturbance was causing distress and affecting normal psychosocial or occupational functioning with the period of insomnia lasting less than 3 months. Additional demographic information was also taken at this point.

During the initial telephone interview, those under 18 years old or in an assisted living environment were excluded. Additionally, individuals currently seeing a sleep specialist, taking hypnotics or reporting a history of neurological trauma, psychiatric illness, or other sleep disorder, according to the ICSD-2, were also excluded.

Following screening, all participants arranged to spend 2 consecutive nights at the University of Glasgow Sleep Centre. The 2 weeks prior to their overnight stay, participants were asked to complete a daily sleep diary and actigraphic assessment. On the designated nights for their stay at the Sleep Centre, participants arrived at approximately 6.30pm, were shown to their room and oriented with the facilities before completing the modified pictorial Posner paradigm followed by the questionnaire measures. This procedure provided the data reported here.

At the start of the experiment, each participant was presented with the instructions screen (Appendix J). Once the experimenter was certain the instructions were understood, the participant, on pressing the keyboard, began the experiment.

Participants continued on to have 2 nights polysomnography (PSG) carried out alongside a number of measures which form the larger ESRC study. The PSG provides continuous recording of specific physiological variables during sleep and typically records brain wave changes (electroencephalogram), eye movements (electrooculogram), muscle tone (electromyogram), respiration, electrocardiogram (ECG) and leg movements. Actigraphy provides a measure of body activity and not

sleep but allows inference on the basis of the more movement, less sleep. The acute insomnia group recruited were measured on attention bias as well as stress and coping and therefore actigraphy would be expected to show a higher level of movement alongside higher stress levels. PSG data is always a valuable objective measure of sleep quality and every effort was made to obtain that data but, due to circumstances surrounding the movement of principal investigator of the ESRC grant to another institution, this data became unavailable for inclusion in this thesis.

Reaction time data from the modified Posner paradigm was gathered and uploaded onto an online database which allowed mean values for each participant to be selected for each trial type i.e. invalid trials presenting sleep related stimuli, valid trials presenting sleep unrelated stimuli etc. This data was then entered into the SPSS database for analysis.

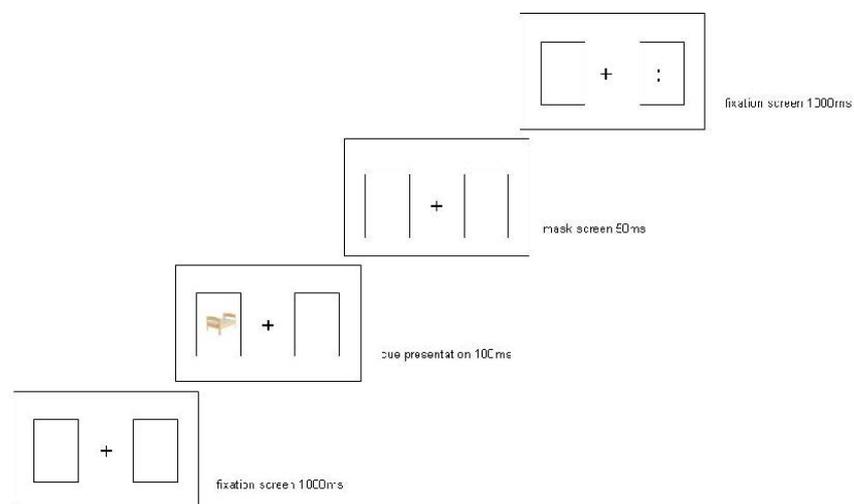


Figure 7.2 Illustration of an invalid trial in the modified pictorial Posner paradigm (Marchetti 2006).

7.2 Results

7.2.1 Subjective measures

The acute and GS groups did not differ in age or sex profile; the acute group had a mean age of 32.2 years (range 19-64 years) with 13 males and 22 females and the GS group's mean age was 32.9 years (range 20-64) with 12 males and 14 females.

The mean length of insomnia period was 4.2 weeks (range 2-12 weeks). The following tables outline the sleep characteristics of the acute and GS groups using both questionnaires and sleep diaries.

	Acute N=35		GS N=26		MEAN DIFF.	SIG.
	MEAN	SD	MEAN	SD		
ISI	12.4	4.4	2.3	3.0	10.1	.000*
PSQI Global	8.7	2.7	3.8	2.2	4.9	.000*
PSQI sleep quality	1.4	0.8	0.5	0.5	0.9	.000*
PSQI sleep latency	1.6	1.1	0.8	0.9	0.8	.002*
PSQI sleep duration	1.1	0.8	0.4	0.5	0.7	.000*
PSQI sleep efficiency	1.6	1.0	0.5	0.9	1.1	.000*
PSQI sleep disturbance	1.2	0.4	1.0	0.4	0.2	.054*
PSQI use of sleep meds.	0.5	0.8	0.0	0.2	0.5	.007*
PSQI daytime dysfunction	1.3	0.8	0.6	0.7	0.7	.000*

Table 7.1 Subjective sleep measures for the acute insomnia and GS groups. ISI= Insomnia Severity Index, PSQI= Pittsburgh Sleep Quality Index Global and 7 component factors.

* denotes a statistically significant difference between the acute and GS groups.

	Acute N=35 MEAN	SD	GS N=26 MEAN	SD	MEAN DIF.	SIG.
SOL	27.3	15.4	15.1	10.3	13.2	.002*
WAKE	1.4	1.0	2.6	5.1	1.2	.188
WASO	32.4	35.6	10.0	13.3	22.4	.007*
TST	404.9	60.1	452.9	50.3	48.0	.004*
TIB	503.9	47.4	520.2	48.9	16.3	.240
SE	79.5	10.9	86.8	5.6	7.3	.006*

Table 7.2 Sleep diary measures for the acute insomnia and GS groups. SOL= Sleep Onset Latency, WAKE= No. of awakenings after sleep onset, WASO= Time awake after sleep onset, TST= Total Sleep Time, TIB= Time in Bed and SE= Sleep Efficiency. * denotes a statistically significant difference between the acute and GS groups.

As can be seen from the tables above, those with acute insomnia and GS differed significantly on all sleep parameters apart from the number of awakenings after sleep onset and time in bed. The PSQI sleep disturbance factor is only marginally significant which is interesting with regard to the groups we are comparing. The contributory questions to this factor cover a number of complaints which disturb sleep, ranging from an inability to get to sleep within 30 minutes to having to use the bathroom. The marginal difference between GS and the acute insomnia group on this factor may be suggestive of a difficulty identifying an exact sleep difficulty in the acute group.

7.2.2 Actigraphy

Lichstein et al (2006) validated actigraphy with insomnia using SE, WASO and TST. Using these measures, as well as SOL, GS and acute insomnia groups were

compared as a more objective measure of complaint compared to the sleep diary. The actigraphy measures did not highlight any significant differences between PI and GS. As an objective measure to confirm subjective sleep diary report, no significant differences were found on SE $t=0.3$, $df=26$, $p=0.78$, SOL $t=1.1$, $df=26$, $p=0.31$, WASO $t=0.5$, $df=26$, $p=0.6$ or TST $t=0.1$, $df=26$, $p=0.9$.

	Acute MEAN	SD	GS MEAN	SD
SE	82.0	11.9	80.0	23.3
WASO	50	19.6	46.5	16.9
TST	361	124.1	357	41.3

Table 7.3 Mean and SD (mins) for sleep efficiency (SE), wake after sleep onset (WASO) and total sleep time (TST).

7.2.3 Posner task

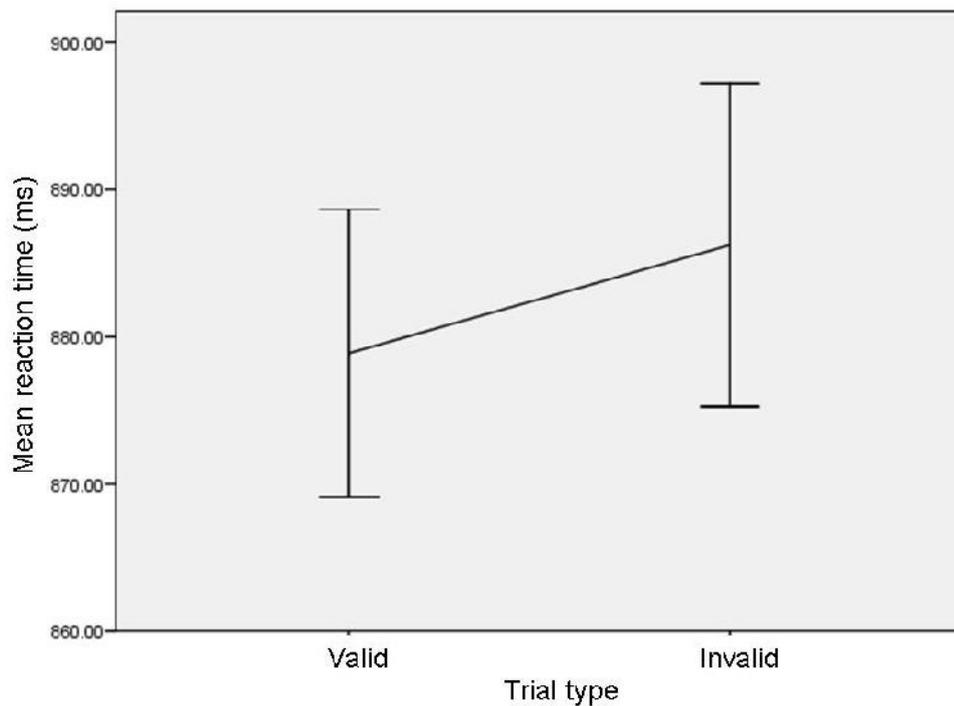


Figure 7.3 Reaction times irrespective of cue presented on valid and invalid trials.

Mean reaction time data can be found in Appendix K.

The graph above illustrates the typical response pattern for a Posner paradigm i.e. responses are faster when the target is validly cued and slower when invalidly cued. It is now of interest to analyse the performance of the GS and acute insomnia groups on trials presenting sleep and non-sleep stimuli. For descriptive purposes, Marchetti's (2006) data is presented again in the graph below to demonstrate an expected attention bias response using this paradigm.

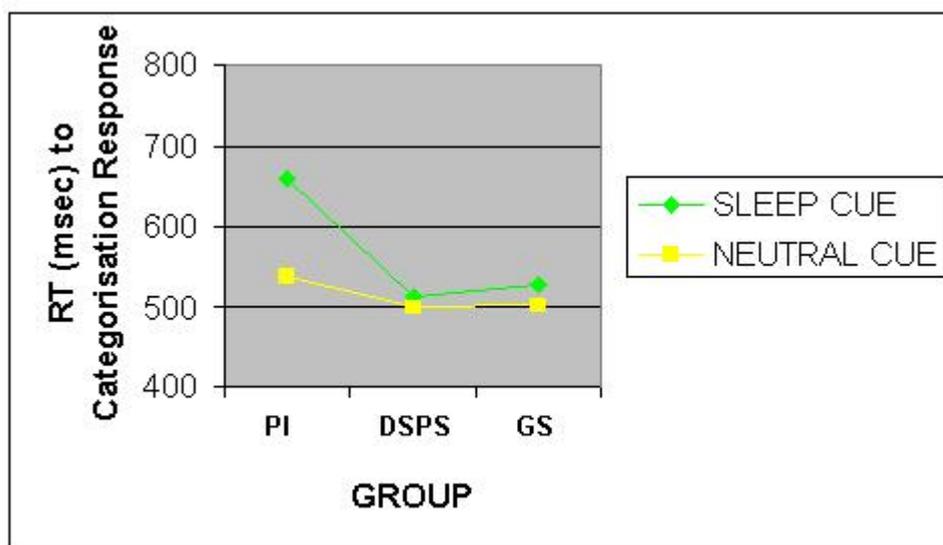
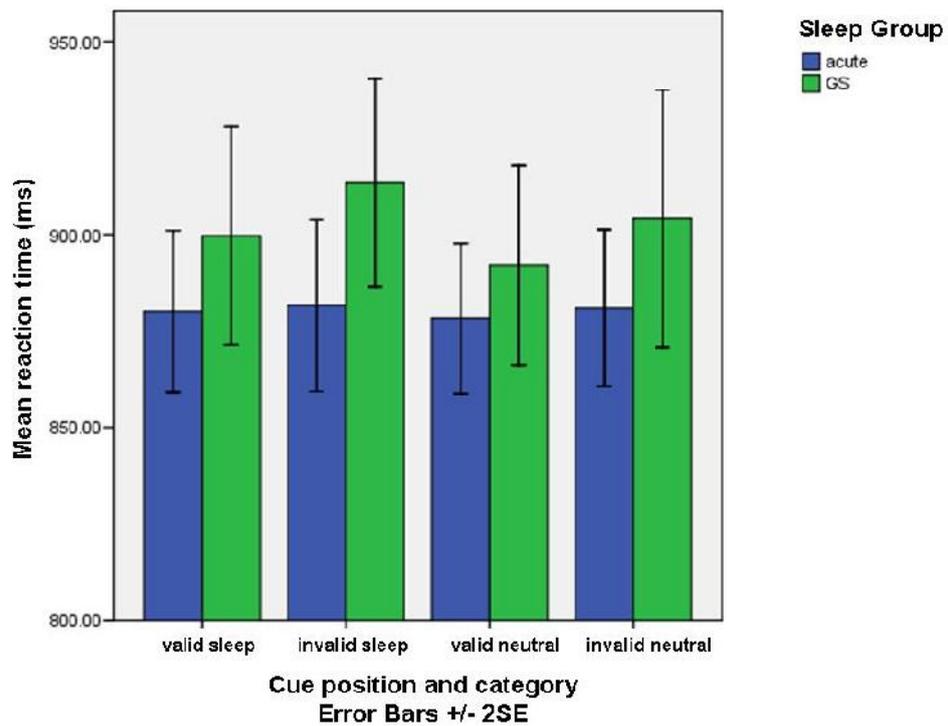


Figure 7.4 Reaction times on invalid trials for an insomnia group (PI), a delayed sleep phase group (DSPS) and a good sleeper group (GS). Taken from Marchetti (2006).

The graph below shows reaction time performance of both the acute insomnia group and GS on the modified Posner task.



Graph 7.5 Mean reaction times for GS and those with acute insomnia on a modified Posner task, by cue position and category.

Descriptively, both the GS and acute insomnia groups show the expected Posner pattern of responses; reaction times are faster on valid trials compared to invalid. Those with acute insomnia appear to react faster on trials or all cue positions and stimuli categories compared to GS but this difference is not confirmed in the analysis [$F(1,220)=2.7, p=0.1$]. No within-groups significant differences were found for validity [$F(1,220)=0.98, p=0.3$] or cue [$F(1,220)=0.1, p=0.7$]. No significant sleep x validity [$F(1,220)=0.002, p=0.9$] sleep x cue [$F(1,220)=0.2, p=0.6$] and validity x cue [$F(1,220)=0.18, p=0.89$] interactions were found as well as the 3-way interaction [$F(1,220)=0.2, p=0.89$].

7.2.4 Effect sizes

Although no significant difference was found between the GS and acute insomnia group, descriptively, it appears that the acute insomnia group react faster and with less variability over the different trial types. Effect sizes were therefore calculated to gain some further perspective on this subtle between group difference and lack of variability within the acute insomnia group. These are presented in the table below.

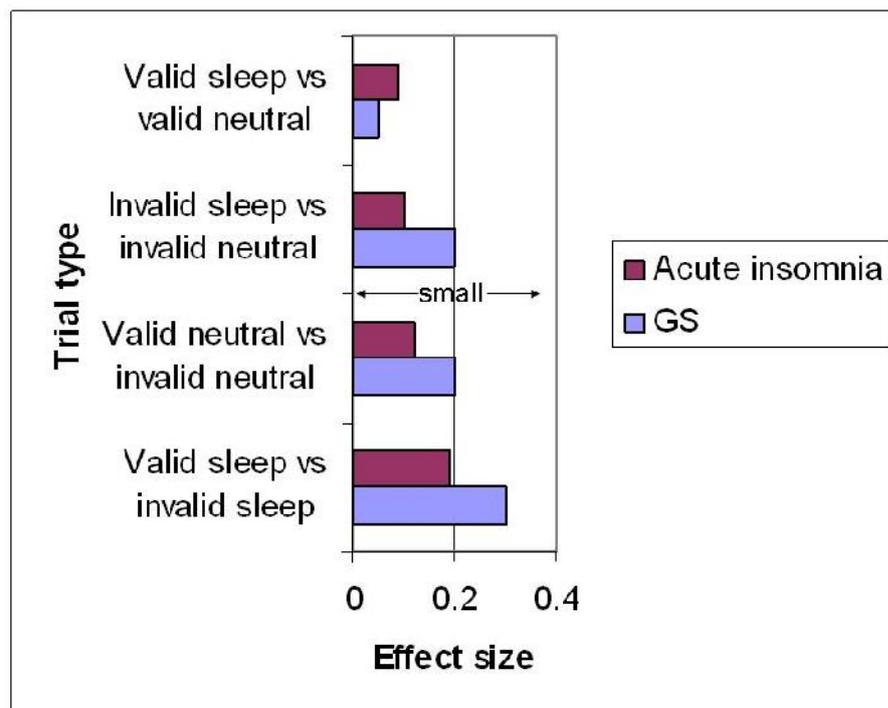


Figure 7.6 Within group effect sizes comparing trial types with relative sizes highlighted (Cohen's d effect sizes taken from Cohen, *J Psychological Bulletin*, Vol 112(1)). Effect size data can be found in Appendix K.

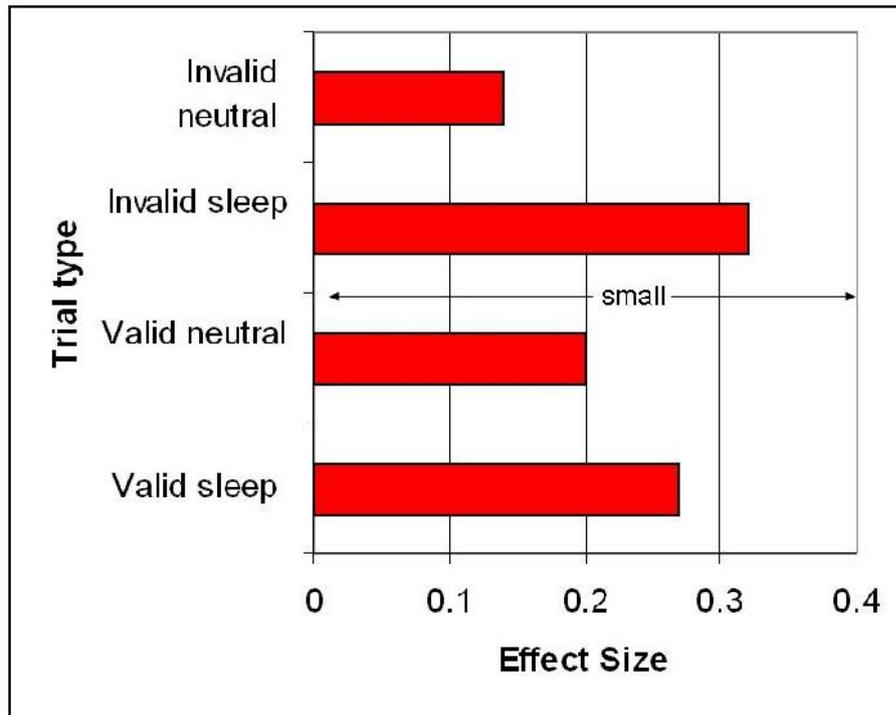


Figure 7.7 Between group effect sizes comparing trial types with relative sizes highlighted (Cohen's d effect sizes taken from Cohen, J Psychological Bulletin, Vol 112(1)).

The effect sizes highlight that the acute insomnia group show less variability in their reaction times across trials compared to GS who show more distinguishable differences on their performances across trials, especially on invalid trials.

7.2.5 Error rates

The number of errors made by each sleep group were compared. No significant differences were found $F(1,55)=0.02$, $p=0.88$.

7.2.6 Insomnia duration

Insomnia duration (weeks).	%
2	12.1
3	6.1
4	9.1
6	6.1
8	33.3
10	3.0
12	30.3

Table 7.4 Percentage split of acute insomnia duration in weeks.

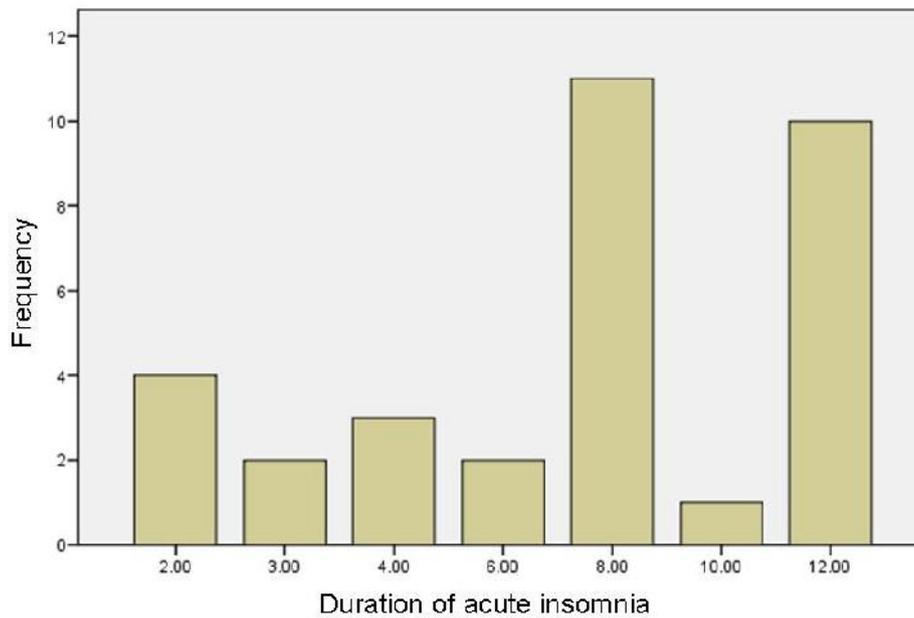


Figure 7.8 Distribution of duration of acute insomnia in weeks.

By finding a trend towards those with acute insomnia reacting faster than GS, it is of interest to address possible reasons for this speeded response. One of the

relevant factors within the acute insomnia group is the duration of their sleep disruption/dissatisfaction. The table and graph above show that the majority of those with acute insomnia reported having an issue with their sleep for 8 and 12 weeks which accounted for two thirds of the acute insomnia group; 33.3% and 30.3% respectively. This means that the majority of participants in this experiment had been experiencing acute insomnia for over the period required for diagnosis as outlined in DSM-IV (1 month) and towards the end of duration criteria according to ICSD-2.

As a consequence of the majority of the acute group having a duration of insomnia longer than 8 weeks, analysis was carried out comparing the reaction times of short (< 8 weeks) and long (> 8 weeks) duration acute insomnia periods. The analysis did not provide any significant differences on reaction time between the durations.

This is a possible explanation of our reaction time findings compared to GS as it is perhaps taking the participants this long to get to the point of addressing their sleep difficulty and investigating relevant action to alleviate the problem. Therefore, it may be the case that we have documented the reactions of individuals who are moving from holding another factor as most salient i.e. the stressor that has led to their sleep disruption towards their attention being turned to their sleep.

7.3 Discussion

The graphical representation of the data suggested that, descriptively, the acute insomnia group showed less variability over trial types than GS as well as faster responses which led to between group effect sizes being calculated to further understand any differences in performance. Over the 4 different trial types, the effect sizes were all small. This is worthy of further discussion with regard to power and the

presence of a small effect between acute insomnia and good sleepers being found. Within the context of previous research on attention bias in insomnia, and in other disorders such as anxiety, effects have been found with 25 participants per group. A power calculation carried out previously suggests that 21 participants per group would detect statistically significant differences at a power of 0.8 with an alpha of 0.05 (Macmahon et al 2006). This has been taken as the effective standard in attention bias studies. However, these previous studies compared a chronic insomnia group to good sleepers whereas the current study compares acute insomnia and good sleeper groups. The present research question was concerned with establishing if the same attention bias found with chronic insomnia was found in an acute insomnia group. If an attention bias was present in acute insomnia, a similar reaction time pattern as shown by Marchetti (2006) would be expected. The reaction time pattern in this acute insomnia experiment is not similar to Marchetti et al (2006) in any way. If there had been a trend in the acute insomnia group showing delayed disengagement then power may have been an issue in detecting a similar bias as established in the chronic condition. A post-hoc power calculation based on a 0.25 between group effect size with an alpha level set at 0.05, as achieved in this experiment, produced a power of 0.5. This calculation suggests that the experiment is adequately powered.

Differences in the number of errors made in the acute insomnia group and GS cannot account for the effects of sleep group on reaction time. Another factor relevant in understanding this data is duration of insomnia in the acute insomnia group. Previous research has shown those with the chronic condition will selectively attend to sleep related stimuli and therefore by looking at the length of time sleep has been a problem may be predictive of a difficulty disengaging attention. However, when

duration is considered, there are no significant differences between short and long durations.

7.3.1 Results in context of A-I-E model

Espie and colleagues (2006), when discussing the use of computerized attention bias tasks to establish selective attention to sleep in psychophysiological (chronic) insomnia, address length of disorder and the identification of a initiating stressor. The first study carried out in this area by the Glasgow group was conducted by Taylor et al (2003) with a cancer population using a modified Stroop paradigm. The use of the cancer population in this study is interesting as all those recruited had been good sleepers prior to their cancer diagnosis so development of insomnia is attributable to a particular event. Two groups of people with cancer and insomnia, 0-3 months and 12-18 months after cancer diagnosis, completed the computerized emotional Stroop task comprising cancer-related, sleep-related and neutral word cues. The terms adjustment insomnia and persistent insomnia were used by Taylor and colleagues (2003) as descriptors for the 0-3 and 12-18 month groups respectively.¹

Both groups demonstrated attention bias for cancer related words but only the persistent insomnia group showed an attention bias for sleep-related words. As interference effects are seen with the persistent group and not with the adjustment² group, Espie and colleagues (2006) suggest that selective attention to sleep may play a role in the transition from adjustment insomnia to psychophysiological insomnia.

From this study by Taylor and colleagues (2003) as well as the other attention bias studies carried out comparing chronic insomnia and good sleepers by the Glasgow group (MacMahon et al 2006, Marchetti et al 2006, Woods et al 2009), it is

¹ According to ICSD-2, the essential feature of Adjustment Insomnia is: “the presence of insomnia in association with an identifiable stressor. The sleep disturbance of Adjustment Insomnia has a relatively short duration, typically a few days to a few weeks” (p. 1–3).

suggested that selective attention to sleep is specific to the chronic insomnia condition, rather than a more acute period of poor sleep. Espie's A-I-E model proposes that selective attention toward sleep feeds into the inhibition of de-arousal leading to insomnia. Due to the most salient stressor not being sleep in adjustment or acute insomnia, selective attention to sleep is unlikely.

This experiment set out to further examine the nature of selective attention to sleep as either a contributory factor in the development of the chronic disorder or as a perpetuating factor in the chronic disorder. The reaction time results obtained suggest that it is a perpetuating factor in the chronic disorder and this is supported by comparing those with short and long duration acute insomnia complaints and finding length of the acute episode does not cause differential reactions to sleep cues within the acute insomnia period. These findings further suggest that selective attention to sleep is a marker of the chronic insomnia condition as shown in the studies by Marchetti et al (2006, Fig. 7.1), Taylor et al (2006) and others (MacMahon et al 2006, Woods et al 2009).

7.3.2 Other considerations

Predispositional and precipitating factors are still relevant within the acute insomnia population and perhaps this data descriptively highlights a precipitating factor within this group in their, non-significantly, faster reaction times. It would appear from this data that those with acute insomnia react faster on this task, independent of cue type or location which could be suggestive of those with acute insomnia having an increased readiness to react on the task compared to GS. Hyperarousal has been implicated as a contributing factor to insomnia within the literature, however, to date this has been with reference to chronic insomnia. Bonnet

and Arand (1995) evaluated on sleep, performance, mood, personality and metabolic measures over a 36-hour sleep laboratory stay. Within the context of arousal, the authors found the individuals with insomnia (chronic) had increased MSLT scores, although they reported higher levels of sleepiness, as well as increased whole body VO₂ throughout the day and one night of sleep, demonstrating an increased metabolic rate which led the authors to propose that those with insomnia suffered from a more general hyperarousal disorder.

Drake et al. (2004) demonstrated that individuals scoring high on a measure of stress-related sleep disturbance, the Ford Insomnia Response to Stress Test (FIRST), have greater sleep disruption on the first night in the laboratory, significantly elevated latency on the MSLT and a significant correlation between FIRST scores and MSLT score. These differences in sleep as a function of FIRST scores support the hypothesis that there are individual differences in vulnerability to transient insomnia. These results are consistent with previous studies of transient insomnia and show that particular individuals may have a vulnerability to sleep disturbance induced by stress. Moreover, the results demonstrate that this vulnerability can be reliably assessed, is associated with physiological hyperarousal, and is present independent of exogenous influences such as excessive daytime sleepiness. Unfortunately, this study is unable to report measures of hyperarousal and stress although it can be reliably assumed that stress is a relevant factor within this acute insomnia group. As far back as 1987, Spielman, Caruso and Glovinsky proposed a stress-diathesis model which suggests existing predisposing factors make some more vulnerable to insomnia than others. These predisposing factors are compounded by precipitating events, i.e. stress, resulting in an acute form of insomnia.

One factor which should be considered here is the absence of any differences on the actigraphy measures between the acute insomnia group and GS. Although the groups differed on subjective, questionnaire and sleep diary measures and the issue of complaint regarding their sleep was present in the acute insomnia group, there does not appear to be any objective differences on the actigraphy measure. The groups were compared on factors which had been validated by Lichstein et al (2006) to reliably differentiate insomnia from GS. Therefore, it may be that we do not see any significant differences on the attention bias task due to the absence of a quantifiable sleep disturbance. Polysomnography data would help in further establishing the presence of sleep disturbance as actigraphy is purely a measure of movement. It may be the case that the acute insomnia are experiencing more sleep disturbance but are perhaps lying in bed, attempting sleep and not moving significantly more than the GS group.

With regard to the objective sleep parameters, no statistically significant differences were seen with regard to WAKE and TIB. WAKE is higher in the GS group compared to the acute group which is unexpected as those with acute insomnia are the group least satisfied with their sleep and would perhaps be expected to have the most disrupted sleep by waking. However, the SOL and WASO in the acute insomnia group is higher than GS which may suggest that the complaint in the acute group is mostly difficulty getting to sleep and are having longer periods awake after fewer awakenings. Interestingly, the acute insomnia group and GS only marginally differ on the sleep disturbance component of the PSQI with both groups having a mean score of around 1.0 which would suggest that any disturbance only occurs once per week.

It is acknowledged that a possible limitation to this study is the absence of a chronic insomnia group. Previous published research by this lab on attention bias in a chronic insomnia population forms a suitable comparison group as a bias towards sleep in the chronic population has been well established and the aim of this experiment was to confirm its exclusivity to the chronic condition.

In summary, no significant differences were seen between the acute insomnia and GS groups on performance on this modified Posner task, although descriptively the acute insomnia group performed faster over all trials types with between group effect sizes showing the effect of group was small. This lack of significant difference between the groups would confirm our hypothesis that an attention bias towards sleep is unique to a chronic insomnia group suggestive of a maintaining, perpetuating factor and not a precipitating factor in acute insomnia. The small effect seen between groups would be suggestive of the hyperarousal attributed to the acute insomnia disorder as marginally faster reaction times are seen.

Chapter 8

Experiment 2

*Modified Posner Paradigm presenting
a clock cue showing sleep and day
times for 100ms.*

This experiment follows on from Woods et al (2009) which presented the clock cue in a modified Posner paradigm for 100ms but only sleep times i.e. 2am to 6.45am in 15 minute increments. To briefly review, this paper showed that PI were delayed in disengaging from sleep times compared to GS and also there was a trend for PI to show enhanced engagement (Graph 8.1). This suggests that sleep times on a clock cue attract and hold attention of PI compared to GS. The natural progression for this work is to experimentally test whether it is the sleep times that are grabbing the attention of PI or the clock cue itself.

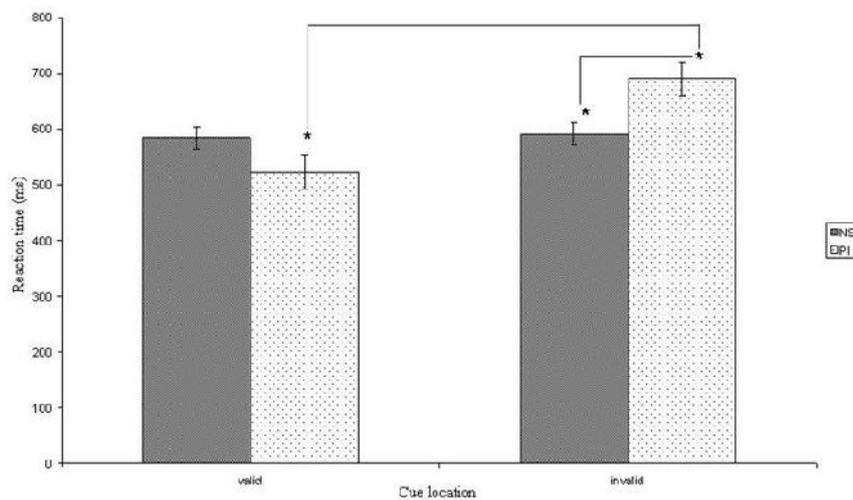


Figure 8.1 Significant differences were found between valid and invalid trials for PI and also between GS and PI on invalid trials. There was a non-significant trend towards a faster response by PI than GS on valid trials.

Day times (9am to 5pm) are now added into the paradigm as well as sleep times (11pm to 7am) enabling the influence of sleep quality, validity and time presented on the clock to be investigated. The sleep time window was extended to ensure the same number of trials as day times.

8.1 Methods

- Research questions: Is it the clock to which PI selectively attend or the times displayed? Do PI only show selective attention to sleep times or are day times also salient?

Hypotheses:

- PI will have longer reaction times on invalid sleep time trials compared to invalid day time trials.
- PI will have longer reaction times on invalid sleep time trials compared to GS.
- PI will have shorter reaction times on valid sleep time trials compared to valid day time trials.
- PI will have shorter reaction times on valid sleep time trials compared to GS.

8.1.1 Apparatus and Stimuli

Figure 7.2 illustrates the modified Posner paradigm used in this experiment. The cues presented were photographs of a digital clock showing a time either associated with sleeping in a normal sleep pattern, 11pm through till 7am, or with daytime, 9am through till 5pm. These times were chosen as they reflect sleep and daytime functioning but also so there was an equal number of trials for both sleep and day times. The stimulus set consisted of digitised pictures of single stimuli which were presented on the clock in the left hand box and in the right hand box. Target stimuli and instructions were the same as described above in the acute insomnia experiment, with participants being asked to react to either 2 horizontal dots (. .) or 2 vertical dots (:). These boxes were continually presented on the screen. Participants

were given 4 practice trials to ensure they were comfortable with completing the task just as in the modified pictorial Posner used in the acute insomnia study.

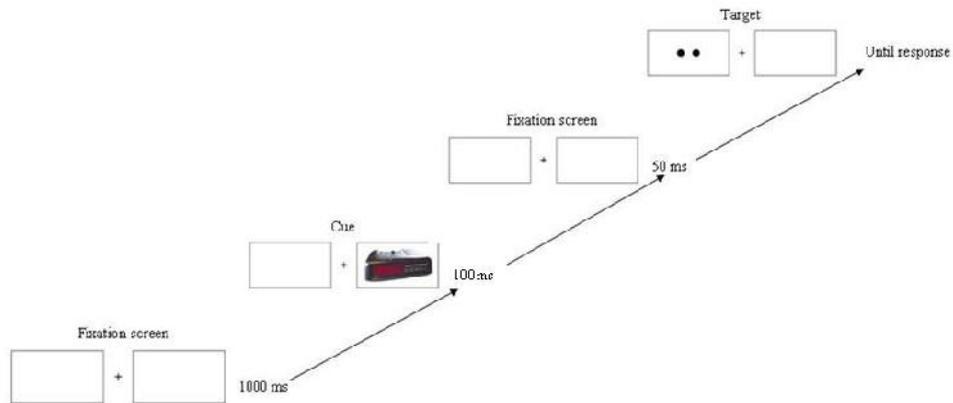


Figure 8.2 The modified Posner paradigm presenting the clock cue for 100ms. As the clock cue and target are presented on opposite sides, this illustrates an invalid trial.

8.1.2 Participant Selection

Participants were recruited through advertising online within the University of Glasgow and through advertising on the psychology department's undergraduate student portal for students to obtain course credits. The title of the study as displayed on the departmental website was 'People with insomnia wanted' and the study description can be found in Appendix A. These different recruitment methods allowed access to both those experiencing difficulties with their sleep and good sleepers.

When an individual selected the study online an email was sent to myself as principal investigator notifying me of their interest and providing a contact email address. An email was then sent to the potential participant asking for a telephone number on which to contact them to ask some questions about their sleep. During this

subsequent telephone conversation, the University of Glasgow Sleep Centre Preliminary Screening Interview (Appendix B) was completed. This tool allows participants to be prospectively assigned to a sleep quality group (PI or GS), screened for other sleep disorders as well as physical and/or mental health issues which could possibly effect their sleep. On successfully completing the phone interview and confirming that the individual met the criteria for inclusion in the study, an appointment was made to complete the experiment in the Psychology department.

8.1.3 Procedure

Individual trials consisted of a fixation cross, presented for 1s, followed by the clock picture displayed for the time relevant to the experiment being undertaken. Targets were then presented in the same or a different box (central to where one of the pictures was positioned) and remained on the screen until response. If the participant saw a target which was vertical, they were instructed to press C on the computer keyboard, if the target was horizontal, then they were instructed to press M. When the target is presented in the box on the same side as the stimuli, this is considered a valid trial. However, if the target is presented in the other box on the other side of the computer screen, this is considered an invalid trial as illustrated in Appendix M. Latencies to detect these targets were used to index the extent to which the groups show an attentional bias. The same instructions outlined in the acute insomnia experiment were given to each participant.

The analysis undertaken in this experiment permitted comparison of reaction times of PI and GS to a validly or invalidly presented clock cue showing sleep times and day times. With regard to our hypotheses, we would expect valid trials to be reacted to faster than invalid for both PI and GS but for PI to react slower on invalid

sleep time trials compared to invalid day time trials and GS as this would be in agreement with the findings of Woods et al (2009).

8.2 Results

8.2.1 Subjective measures

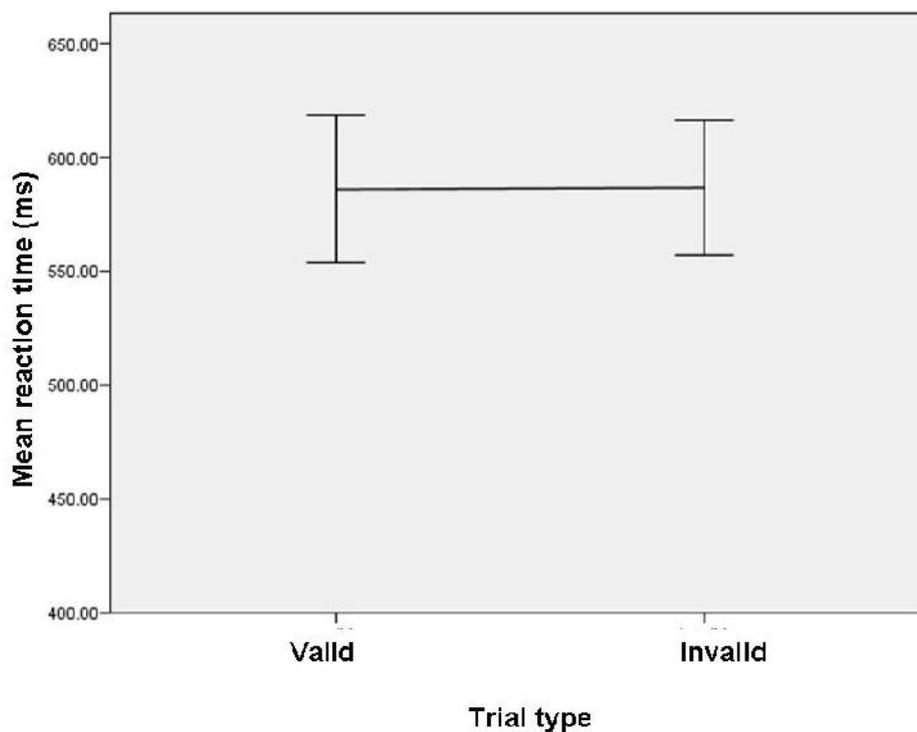
The PI and GS groups did not differ in age or sex profile; the PI group had a mean age of 23.5 years (range 18-37) with 16 females and the GS group's mean age was 22.8 years (range 19-44) with 10 females. PI and GS classification of sleep quality is confirmed, as can be seen in the table below. PI scored higher on both the PSQI and ISI indicative of poorer sleep and a more severe insomnia complaint. They also scored higher on the DBAS and SPS as well as appearing significantly more anxious than GS. PI and GS did not significantly differ on the depression measure although PI scored slightly higher. The measure of daytime functioning did not differ between our sleep quality groups which would perhaps be suggestive of no particular salience of day times in one group compared to another.

	PI N=18		GS N=21		MEAN DIFF.		SIG.
	MEAN	SD	MEAN	SD			
PSQI	11.9	2.7	3.9	2.0	8.0		.000*
ISI	17.3	4.3	5.9	4.8	11.4		.000*
DFSAS PART 1	21.1	5.9	17.2	9.3	3.9		.133
DFSAS PART 2	23.2	7.5	18.2	11.4	4.9		.124
HADS Anxiety	10.9	3.5	6.4	3.4	4.5		.000*
HADS Depression	5.0	3.2	3.2	2.6	1.8		.06
DBAS	79.7	15.8	63.3	15.4	16.4		.002*
SPS	108.7	24.5	85.3	26.9	23.3		.008*

Table 8.1 Mean and standard deviation scores of subjective measures for PI and GS.

As the subjective measures above confirm classification of the PI and GS groups by sleep quality and complaint, the performance measures of reaction time can now be analysed. Our first aim is to investigate if the reaction times on invalid trials were slower than valid, irrespective of the time displayed on the clock. This would tell us if it was the clock that was causing the attention bias in Woods et al (2009) as opposed to the sleep time which it showed.

8.2.2 Posner task



Graph 8.3 Reaction times irrespective of time displayed on clock cue for valid and invalid trials. Mean reaction time data can be found in Appendix O.

The graph above suggests that there is no difference on invalid trials compared to valid trials and analysis confirms that there is no statistically significant difference $F(1,73)=0.001$, $p=0.98$. This finding is interesting as it would suggest that some other process is affecting performance as we would expect to see a delay on invalid trials i.e. when the individual has to reallocate their attention, compared to valid trials. Further analysis was carried out to investigate this taking into consideration the remaining factors of interest, sleep quality and time presented on the clock cue.

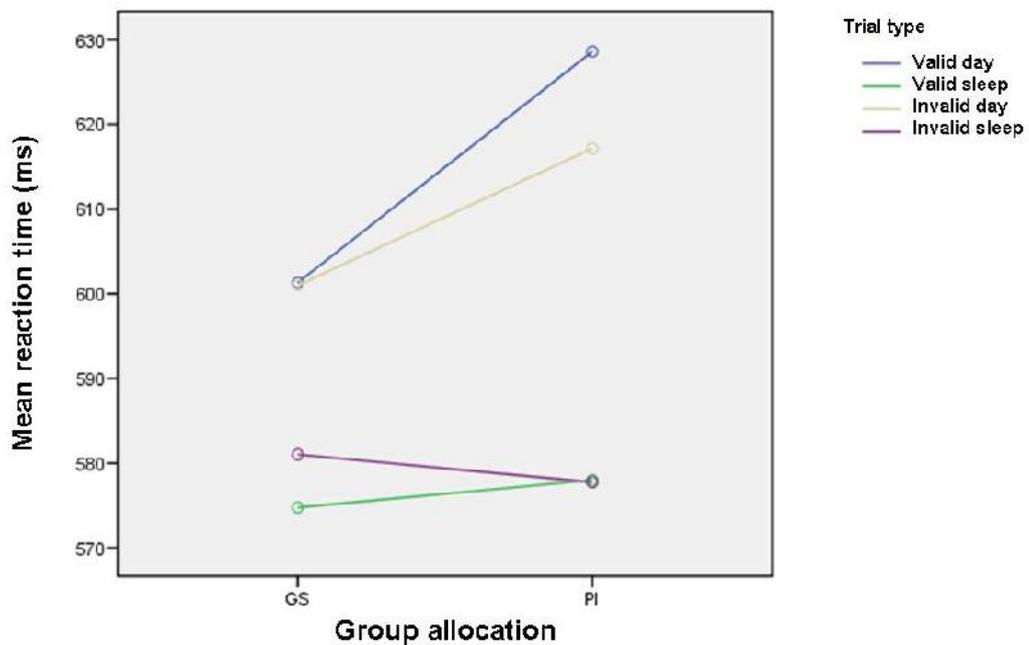


Figure 8.4 Reaction time (ms) of PI and GS on valid and invalid trials when day times and night times are presented on the clock cue.

The graph above suggests that reactions to sleep times are faster than day times and the pattern of reactions is different between PI and GS. These observations were confirmed by a 4 (trial type: valid day, valid sleep, invalid day, invalid sleep) x 2 (sleep quality: GS and PI) ANOVA which identified significant effects of trial type $F(1,31)=17.6, p<0.000$ and a significant interaction between trial type and sleep quality $F(1,31)=4.97, p<0.05$. Both GS and PI react faster to sleep times than day times but the pattern of reactions are different within these groups. GS show a typical Posner reaction pattern with sleep times in that they react faster to valid than invalid trials. GS show no difference in reaction time on valid or invalid sleep time trials. PI show no difference on sleep time trials and show an untypical reaction pattern to day times in that they react faster on invalid trials compared to valid.

8.2.3 Effect sizes

To substantiate these findings, both within and between group effect sizes were calculated. The graphs below highlight the significantly larger effect sizes between the trials when day and night times are presented on the clock cue as well as sleep quality having a substantial effect on reaction time to the different trial types.

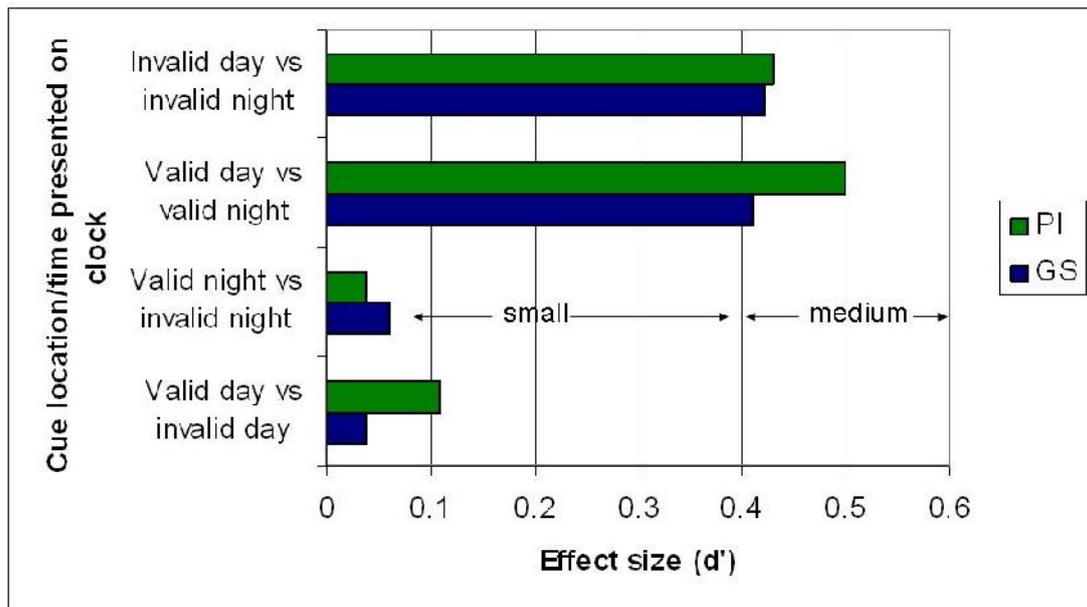


Figure 8.5 Within group effect sizes for PI and GS by trial type.

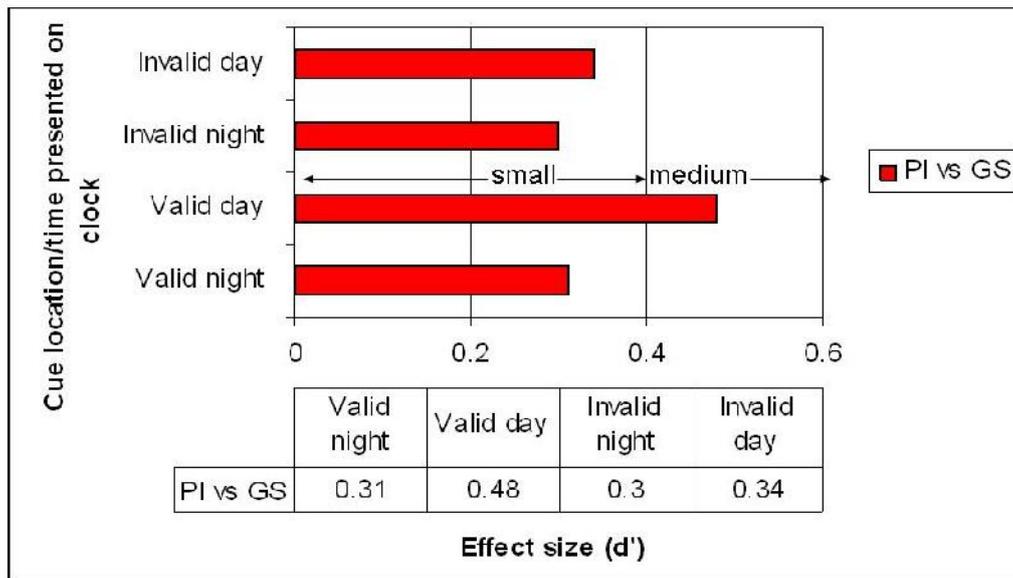


Figure 8.6 Between group effect sizes by trial type. Effect size data can be found in Appendix O.

At this point, with trial type and sleep quality having such a consequential effect on performance on this task, insight into the relationship between sleep complaint as quantified by the subjective measures of sleep quality, insomnia complaint etc recorded for each participant and task performance appeared to be a valuable next step to see if any of these measures of complaint could be a predictor for performance on this task.

8.2.4 Regression analyses

Four stepwise regression analyses were carried out, using the four levels of trial type (valid day, valid night, invalid day and invalid night) as separate dependent variables and the DFSAS parts 1 and 2, PSQI, ISI, HADS Anxiety and Depression measures and the Sleep Preoccupation Scale as predictor variables, our hypotheses being that sleep quality as measured by the PSQI and ISI as well as the effects of poor

sleep as measured by the remaining the subjective measures listed above would significantly predict performance on this task.

Two models came out as significant which had valid day trials as the criterion variable. Firstly, when PSQI was entered into the model, it deviated from the null, $F(1,35)=4.8$, $p < 0.05$ and the incremental variance explained on the basis of adjusted R^2 was 9.5% ($p < 0.05$) with a beta value of -0.347 ($p < 0.05$). Secondly, when PSQI and part 1 of the DFSAS were entered the model deviated from the null, $F(2,34)=4.99$, $p < 0.05$ and the incremental variance explained on the basis of adjusted R^2 was an additional 8.7% compared to PSQI only, at 18.2% ($p < 0.05$) with a beta of -0.473 ($p < 0.05$). No other significant models were found for the other 3 levels of trial (valid night, invalid day and invalid night).

8.3 Discussion

To summarize, time presented on the clock cue affects performance with sleep times facilitating response compared to day times. This confirms that time presented on the clock is salient, not only to PI but also to GS as time presented affects performance in both sleep groups. With sleep times, PI perform no differently on valid and invalid trials, however, GS show a more typical pattern of responses on this task with faster reactions on valid trials compared to invalid. With day times, no difference in performance is seen with GS on valid and invalid trials but PI show a more atypical pattern of responses by reacting slower on valid day trials than invalid day trials. This is where the largest effect size is seen and, due to its unexpected direction, it is worth addressing the power of the experiment. A post-hoc power calculation based on a 0.35 between group effect size with an alpha level set at 0.05,

as achieved in this experiment, produced a power of 0.6. This calculation suggests that the experiment is adequately powered.

8.3.1 Results in context of A-I-E model

The attention bias studies which formed the basis for the selective attention component of Espie et al's (2006) A-I-E model demonstrated a facilitated response to sleep stimuli in PI compared to GS and neutral stimuli. Marchetti's (2006) work demonstrated a delayed disengagement from sleep stimuli using a modified Posner paradigm with sleep or neutral pictorial stimuli as cues. These studies are suggestive of an attention bias towards and difficulty disengaging attention from sleep. Woods et al (2009) went on to demonstrate a delayed disengagement from sleep times, in line with the previous research. However, the results of the present study are interesting as they show a different response pattern than the research mentioned above and, therefore, the hypotheses formulated.

This experiment's first hypothesis, that PI will have longer reaction times on invalid sleep time trials compared to invalid day time trials, was formulated from Woods et al (2009). This is not supported by the current study as PI show no difference in reaction times on valid or invalid sleep time trials suggesting that sleep times are not influencing attention disengagement. The second hypothesis, that PI will have longer reaction times on invalid sleep time trials compared to GS, is also not supported as both sleep groups react faster on sleep time trials than day time trials. At this point, it is clear that a replication of Woods et al (2009) with delayed disengagement from sleep times is not the outcome and day times must be having an influence on attention allocation. This is confirmed by rejection of our third and fourth hypotheses as PI do not have shorter reaction times on valid sleep trials

compared to valid daytime trials nor do they react faster on valid sleep trials compared to GS.

To refer back to the A-I-E model (Espie et al, 2006), the attention bias studies which contributed to this model presented sleep and neutral stimuli. The current study introduces two novel categories of stimuli; sleep and daytimes. As this is the only difference from Woods et al (2009) and the acknowledgement of insomnia as a 24 hour disorder in the current literature, it was perhaps hasty to consider daytimes as the less salient cues compared to sleep times. Therefore the findings are interesting in two ways; that valid trials had the largest effect and that this effect was with trials presenting daytime cues.

To consider this finding in light of the A-I-E model and the contributing attention bias studies (MacMahon et al 2006, Marchetti 2006), it may be that an attention bias towards sleep is evident when sleep and neutral are the comparison stimuli types. By presenting two stimuli categories which represent different aspects of insomnia, sleep time and day time, attentional avoidance of stimuli representative of daytime is seen. In fact, not only are day times causing the most unexpected and substantial results within this experiment but also providing a predictive relationship with sleep quality and perceived daytime impairment accounting for nearly 20% of variance in performance on these trials. This confirms the salience and influence day times have over attention allocation, and therefore reaction time, in this experiment.

To now apply these results to the constructs outlined earlier in this thesis, with regard to engagement and disengagement, this experiment has further informed us with regard to attention allocation in PI. GS show an expected facilitated response on valid trials and a comparatively delayed response on invalid trials but only when sleep related times are presented on the clock cue. This would be due to attentional

engagement on valid trials with the delay on invalid providing a measure of attentional disengagement and movement. As we only see this with sleep times in GS we can theorize that this is due to sleep times being more salient to GS than day times.

PI do not show the same pattern of facilitation and inhibition of responses on valid and invalid trials as GS or for the sleep times. Instead of seeing facilitation on valid trials and inhibition or delay on invalid trials, we see inhibition on valid day trials and a comparative facilitation on invalid day trials. This is also where we see the biggest influence on performance in terms of effect size between presentation of sleep and day times.

Since the current study follows on from Woods et al. (2009) it is worth commenting on the movement from seeing delayed disengagement from sleep related times in PI in the previous study to now seeing no variation in performance on sleep related times but day times having the biggest influence on performance and in an unexpected manner.

In previous attention bias in PI studies, enhanced processing of sleep related items is suggested. Jones et al (2005) found that PI reacted to sleep related changes in an ICB flicker 'spot the difference' task faster than neutral changes and faster than GS. MacMahon et al (2006) showing a significantly greater processing bias toward sleep-related words (in comparison to neutral words) in a PI group when compared to the GS and Delayed Sleep Phase groups. Spiegelhalder et al (2008) also used the emotional Stroop paradigm to investigate sleep related attentional bias in a novel way by raising the question of whether sleep related attentional bias is due to sleepiness or sleeplessness. In line with the studies discussed above, Spiegelhalder and colleagues found a positive association between selective attention towards sleep and poor sleep quality, however, a novel finding was that sleepiness also has an impact. The authors

make an analogy with substance dependence attentional bias studies in that a greater need for sleep seems to be associated with an attentional preference for sleep related stimuli and that this possibly reflects craving. This finding is relevant in the context of further understanding what fuels attentional bias in PI. Woods et al (2009) found a trend towards enhanced reactivity to validly cued sleep times compared to GS and invalidly cued sleep times on a similar modified Posner paradigm as used in the current study. These studies suggest that sleep cues are particularly salient to PI which enhances their reaction times to these cues when, within the context of the Posner, reacting to a target spatially replacing a sleep cue. However, in the current study, the biggest effect on performance is seen on trials where a target is spatially replacing a non-sleep time. This would suggest that the previously seen delayed disengagement to sleep times is over-ridden by the relationship which exists between performance on trials presenting day times and perceived daytime impairment. This is an important consideration within the nosology of the insomnia disorder.

8.3.2 Other considerations

Two possible explanations will be proposed here for the effects seen in this study. Firstly, avoidance may result in a delay in reacting to the targets which replaced the day times. This would mean that attention is not held in the space where the clock showing day times was presented followed by the target. If this was the case, then perhaps facilitated responses to invalidly cued day times would be found. PI do react faster on invalid day time trials than validly cued day time trials which could be due to attentional movement away from a day time presented on the clock resulting in faster reaction times to targets presented elsewhere.

The second possible explanation for the reaction pattern seen on valid day trials is that day times are *least* salient to the PI group and are not grabbing their attention and holding it in place for reaction to the target which then replaces the clock. Since the sleep times provoke faster reaction times in both PI and GS, this would suggest that sleep times are the more universally salient stimuli. Perhaps valid day trials hold the least amount of interest to either group but particularly PI and so attention is not engaged in the task as much as with sleep times being presented or where a shift in attention is required. If this was the case, it would perhaps be more expected that we would see delayed disengagement from sleep times in line with Woods et al (2009) and a relationship between performance on the trials presenting sleep times and sleep quality. However, what we find here is that valid trials presenting day times produce the largest effects; there is no magnification of the delayed disengagement to sleep related times found in GS seen in PI and the significant relationships we find do not involve sleep times but are with sleep quality, perceived daytime impairment and performance on validly cued trials presenting day times.

Once again, research in the anxiety disorder field provides us with evidence that a salient stimulus will produce differential performance compared to non-salient. Eldar et al (2010) compared reaction times of high- and low-anxious participants on a dot-probe task which presented either angry-neutral, happy-neutral or neutral-neutral pairs of face stimuli. The authors found a small but significant difference between the anxiety levels on bias scores with the anxious group having a bias towards (reacting faster to) the target when it replaced the angry face which was interpreted as anxious individuals are biased towards threatening stimuli. In addition to their findings, Eldar and colleagues obtained electrophysiological data which revealed that anxious

participants had more pronounced C1 negativity than the nonanxious participants exclusively in the threat condition (angry–neutral pairs). C1 modulation by threat stimuli has been observed in previous ERP studies of nonselected populations (Pourtois et al., 2004; Stolarova et al., 2006). From this work carried with anxious individuals, it could be expected that an anxious response would lead to a bias towards and engagement with that relevant stimulus.

The relationship between sleep quality as measured on the PSQI and impairment in daytime domains as measured on the DFSAS (part 1) are significant in this study, in that for a 1SD increase in PSQI and DFSAS (part 1) score, a decrease of 0.5SD and an increase of 0.35SD respectively is predicted in reaction time on trials validly presenting a day time. This would suggest that sleep quality and daytime impairment produce different response patterns. As the sleep complaint increases, the reaction time to validly cued day times becomes faster whereas with the complaint of daytime impairment increasing, the impairment of online performance on the task with valid day times increases. This further intertwines sleep quality and daytime impairment and the view of insomnia as a 24 hour disorder.

Approximately one fifth of the variance on performance on this task is accounted for by these two measures, sleep quality and daytime impairment. These findings would make accepting the argument that validly cued day times are the least salient of the trials and therefore allowing attention to waver difficult to defend. They also suggest that moving on from Woods et al (2009) and including day times as well as sleep times within the experiment and measuring daytime impairment as well as sleep quality informs us that time on the clock is important as well as consideration of the 24 hour insomnia complaint.

In a recently published paper on attentional bias training in depression, Baert et al (2010) discuss how attentional bias in depression is distinct from the same phenomenon found in anxiety in that the anxious attentional response is found to be an early vigilance toward threat compared to the depressed response being characterized by a difficulty disengaging attention from depressogenic content once it has eventually been engaged with. The authors also report a growing body of evidence for mood congruent attentional bias when negative material is self-referent and when the cues are presented for long durations i.e. >1 second. This lack of early engagement with salient cues is consistent with the valid daytimes data for the current study in that there appears to be a difficulty with early engagement on these trials. Philips et al (2010), in a meta-analysis on implicit cognition and depression, also discuss the studies carried out to date which tend to find that those participants with depressive symptoms will show a bias towards dysphoric stimuli but at longer presentation times. PI and GS did differ on the HADS measure of anxiety but did not on the depression measure. If anything, this data would suggest that a more anxious i.e. faster shift and engagement response would be expected in PI, as they are the more anxious group, however this is not borne out in the data.

Chapter 9

Experiment 3

*Modified Posner Paradigm presenting
a clock cue showing sleep and day
times for 250ms.*

The previous experiment which presented the clock cue for 100ms within the modified Posner paradigm showed that day times provided the largest effect on reaction time in PI which is interesting within the context of insomnia as a 24 hour disorder. As has been discussed within the review of attention bias literature earlier in this thesis, the psychopathology being investigated influences the pattern of engagement and disengagement alongside the time the salient cue is presented for. This next experiment aims to investigate the pattern of engagement and disengagement at a longer presentation time of 250ms.

9.1 Method

The methodology employed in this experiment has been described in the previous 100ms clock chapter. However, to provide a brief overview, both PI and GS completed the modified Posner paradigm within which the clock cue was presented for 250ms followed by the target to which the participant had to react providing a reaction time measurement of attentional engagement and disengagement. The clock cue presented either a sleep time or day time.

- **Research question:** Do PI maintain their gaze on sleep times and therefore continue to show delayed disengagement at a longer presentation time?

Hypotheses:

- PI will have longer reaction times on invalid sleep time trials compared to invalid day time trials.
- PI will have longer reaction times on invalid sleep time trials compared to GS.

- PI will have shorter reaction times on valid sleep time trials compared to valid day time trials.
- PI will have shorter reaction times on valid sleep time trials compared to GS.

9.2 Results

9.2.1 Subjective measures

The PI and GS groups did not differ in age or sex profile; the PI group had a mean age of 22.3 years (range 18-35) with 20 females and the GS group's mean age was 24.1 years (range 19-38) with 10 females.

The table below shows that PI and GS differ significantly from each other on all subjective measures. As with the 100ms clock study, PI have poorer sleep quality and a more severe insomnia complaint as measured by the PSQI and ISI. In contrast to the 100ms clock study, PI and GS significantly differ on the measure of daytime impairment with PI subjectively expressing a higher impairment and attributing this more to sleep. PI were also more anxious and depressed, however, as discussed previously those participants scoring above threshold on the HADS were not screened out as relationships between anxiety, depression and poor sleep are now documented.

	PI N=22		GS N=25		MEAN DIFF.	SIG.
	MEAN	SD	MEAN	SD		
PSQI	12.8	2.9	3.8	1.8	9.07	.0001
ISI	18.9	3.3	4.2	3.3	14.7	.0001
DFSAS PART 1	31.1	6.7	19.6	6.4	11.5	.0001
DFSAS PART 2	34.8	9.5	18.3	5.9	16.5	.0001
HADSA	9.5	3.8	6.3	3.9	3.2	.009
HADSD	6.1	4.0	2.9	3.1	3.2	.007
DBAS	86.5	21.5	54.8	25.9	31.7	.0001
SPS	126.8	24.4	71.2	27.2	55.6	.0001

Table 9.1 The mean scores and standard deviations of PI and GS on 8 subjective measures of sleep, daytime impairment related to sleep, anxiety and depression.

9.2.2 Posner task

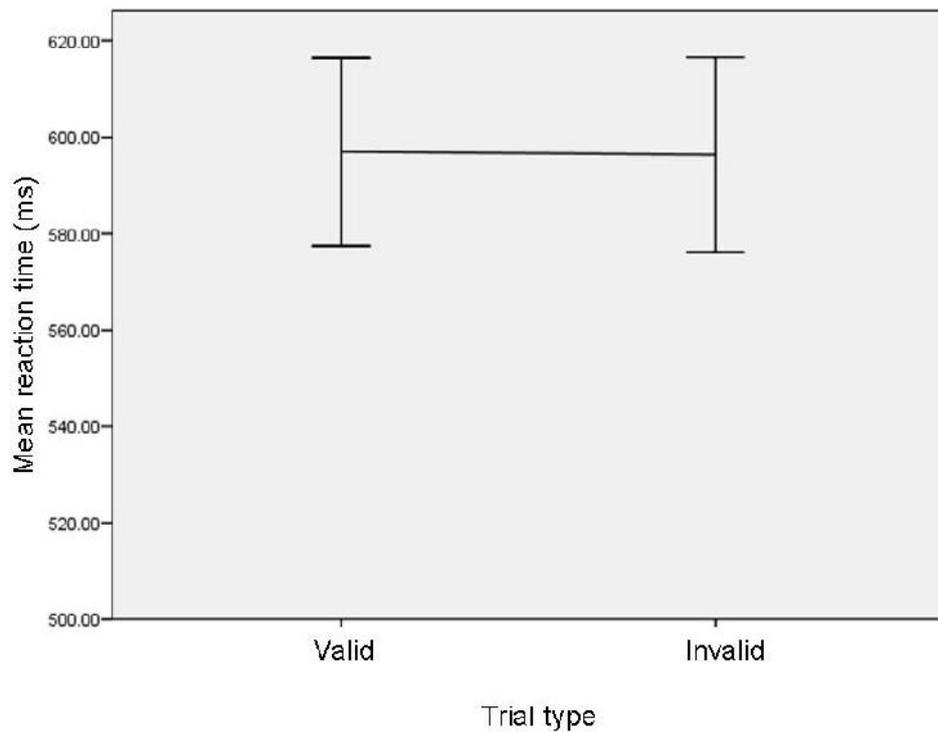


Figure 9.1 Reaction times irrespective of time displayed on clock cue for valid and invalid trials. Mean reaction time data can be found in Appendix P.

With regard to differential performance on valid and invalid trials, we first performed an ANOVA which confirmed what can be seen in the graph above, that valid and invalid trials did not significantly differ from one another $F(1,175)=0.002$, $p=0.97$. Since the expected delay on invalid compared to valid is not found, the other relevant factors can now be included in the analysis.

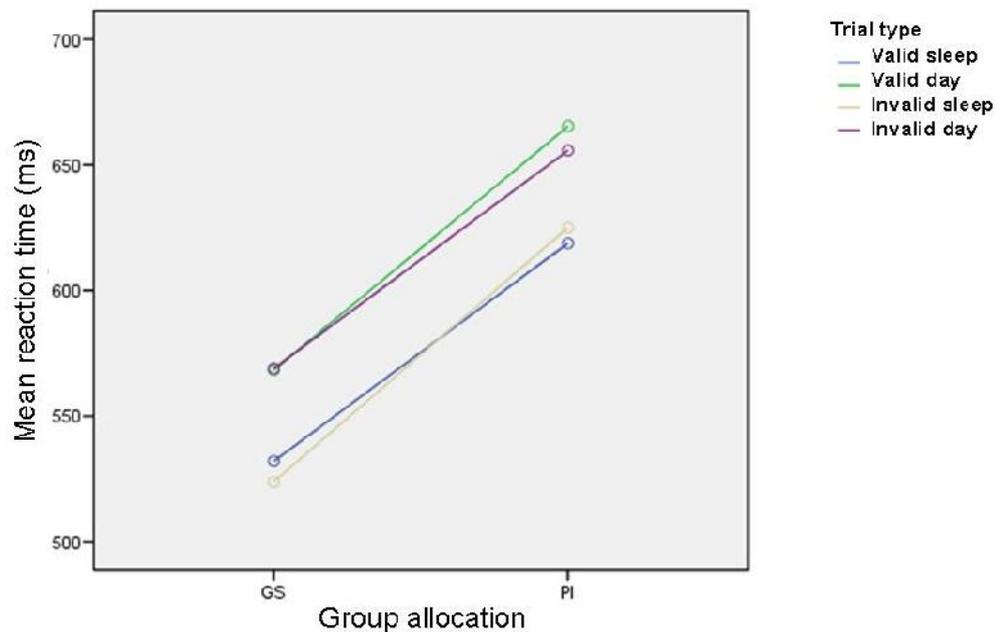


Figure 9.2 Reaction time (ms) of PI and GS on valid and invalid trials when day times and night times are presented on the clock cue.

The graph above suggests there is a delay in reaction time seen in PI over all trial types compared to GS as well as the conformity of reaction times in both groups. As was suggested earlier, there appears very little effect of validity as well as time presented on the clock cue. The formal analysis confirms this with a significant main effect of sleep quality $F(1,46)=18.7, p<0.000$ but no main effect of trial type $F(3,138)=0.13, p=0.9$ or significant interaction between sleep quality and trial type $F(3,138)=0.55, p=0.6$.

9.2.3 Effect sizes

As we have now increased the presentation time from 100ms to 250ms and produced a different pattern of results, it would be valuable to quantify these

differences by calculating effect sizes, both between and within PI and GS. As the graphs below show, the effect of trial type is minimal but sleep quality has a huge effect on performance on this task further linking sleep quality and differential processing compared to GS. Based on these findings, it would seem reasonable to hypothesize that positive relationships would exist between sleep quality as measured by the PSQI and ISI and performance on this task over all trial types.

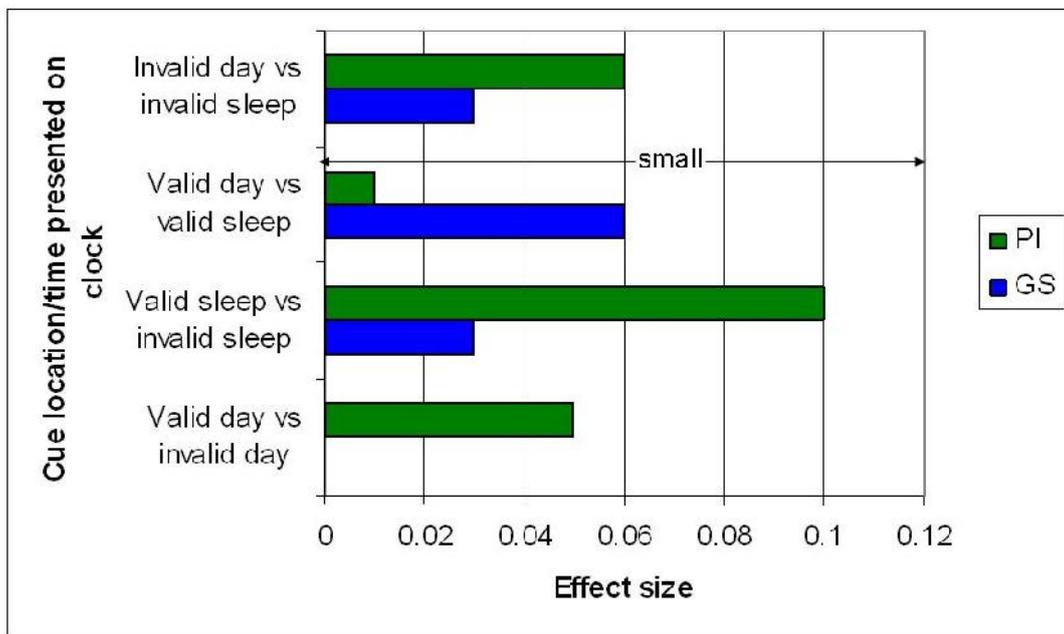


Figure 9.3 Within group effect sizes comparing trial types with relative sizes highlighted (Cohen's d effect sizes taken from Cohen, J Psychological Bulletin, Vol 112 (1)).

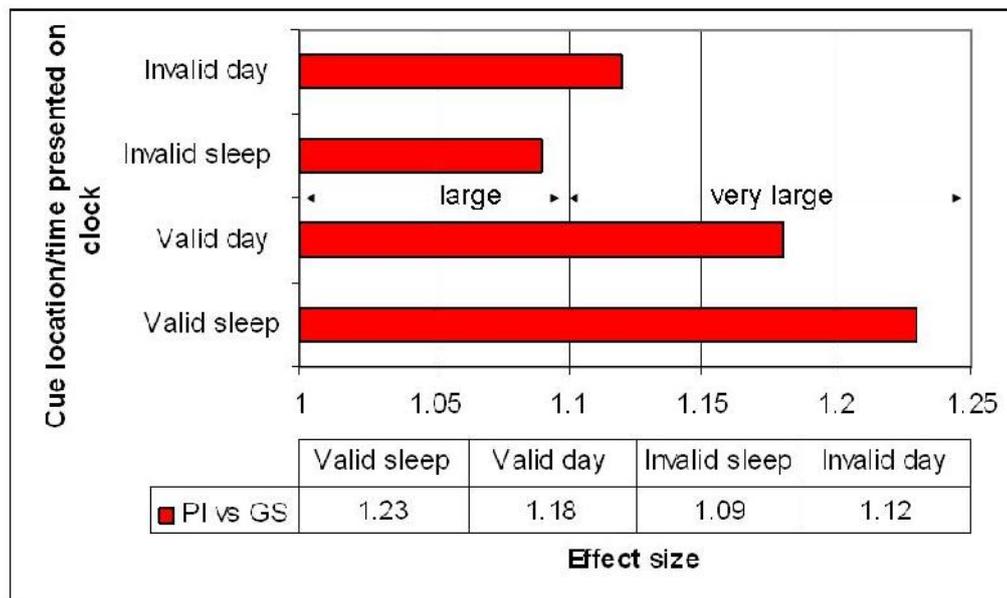


Figure 9.4 Between group effect sizes by trial type. Effect size data can be found in Appendix P.

9.2.4 Regression analyses

To attempt to quantify this relationship, four stepwise regression analyses were carried out, using the four levels of trial type (valid day, valid night, invalid day and invalid night) as separate dependent variables and the DFSAS parts 1 and 2, PSQI, ISI, HADS Anxiety and Depression measures and the SPS as predictor variables. All four models came out as significant. PSQI deviated from the null in the valid day, valid sleep and invalid sleep models accounting for 20% ($F(1,37)=10.4$, $p<0.005$. $\beta=0.47$), 22% ($F(1,37)=11.5$, $p<0.005$. $\beta=0.49$), and 15% ($F(1,37)=7.9$, $p<0.05$. $\beta=0.42$), of the variance in these models respectively. In the valid day model, the ISI deviated from the null ($F(1,37)=7.2$, $p<0.05$) accounting for 14% of the variance with a beta value of 0.4.

9.2.5 Error rates

Due to the variation in performance that is seen between the 100ms and 250ms clock studies, it may be helpful to take a look at the error rates of PI and GS. On both these experiments, if the number of errors differs between sleep groups, the delay in PI performance can be accounted for.

The graph below suggests that the largest between group differences are seen on the 250ms clock study which might be expected as this study produced the larger effects of sleep quality. In the 100ms experiment PI would appear to make more errors than GS but in the 250ms experiment, the GS appear to have a much higher error rate than the 100ms study and PI whereas PI make fewer errors than GS and the 100ms. This suggests that a statistically significant interaction will be found which is what the analysis produced, a significant interaction between sleep quality and clock study (100ms and 250ms), $F(1,102)=4.5$, $p<0.05$.

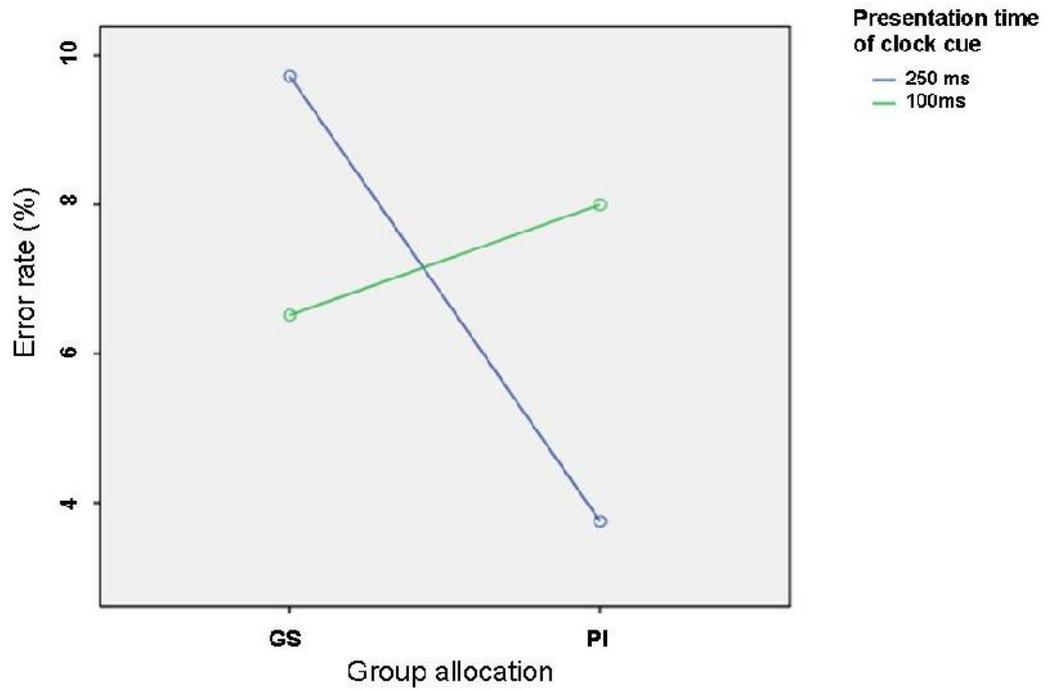


Figure 9.5 Error rates (%) of GS and PI on the 100ms and 250ms clock studies.

Since variation is seen in the error rates for GS and PI, as well as reaction time performance as described in the previous chapters, effect size calculations would allow some comparison on the effect of presentation time on error rates.

The graph below shows effect sizes both between and within groups.

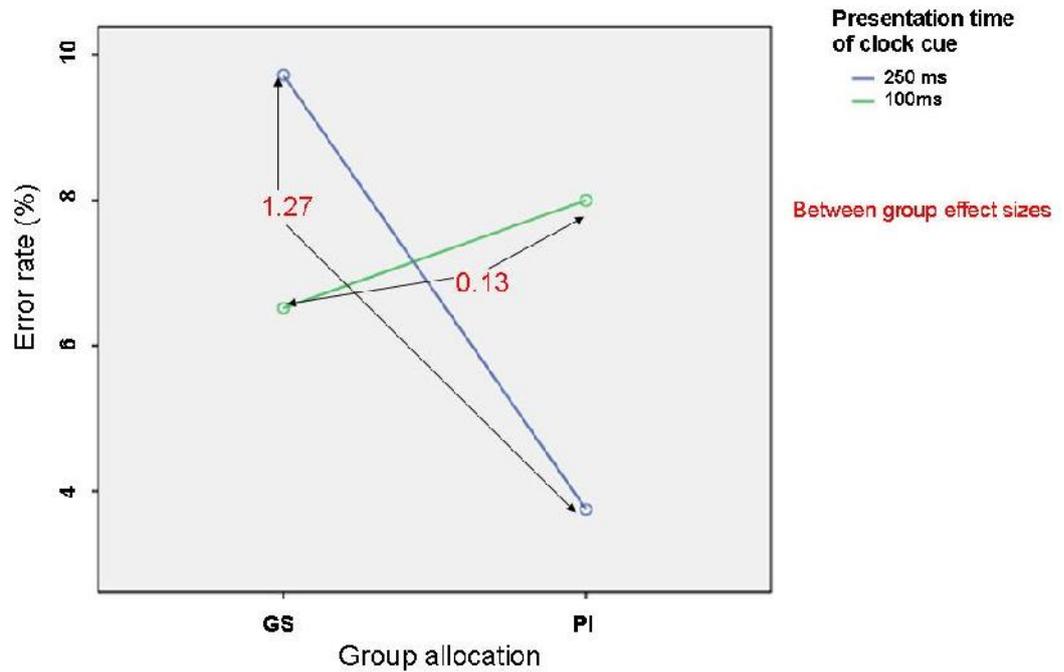


Figure 9.2 Error rates (%) with effect sizes of sleep quality on the 100ms and 250ms clock studies.

The effect sizes between presentation times of the clock cue are small within the PI group and medium within the GS group but the between group differences on the 250ms task are very large compared to the 100ms task where the between group differences are negligible.

9.3 Discussion

To summarise, in this study presenting the clock cue for 250ms, PI are significantly slower than GS over all trial types. Cue position and time presented appear to have more influence at 100ms than 250ms as no differences and very small effect sizes are seen with the trial types at the longer presentation time. Large effect sizes are seen between PI and GS in this study as well as the PSQI and ISI accounting for between 14% and 22% of the variance on performance on this task over all cue

locations and times presented. As large between group and much smaller within group effects sizes are seen, a post-hoc power calculation based on a 1.15 between group effect size with an alpha level set at 0.05, as achieved in this experiment, produced a power of 1.0. As the research question sets out to establish performance differences related to sleep quality, this suggests that the experiment achieved satisfactory power.

This experiment therefore provides a very clear influence of sleep quality on reaction time with a negligible effect of validity or time presented on the clock cue.

9.3.1 Results in context of A-I-E model

With the current experiment's hypotheses based on Woods et al (2009), the findings are unexpected yet interesting within the context of the A-I-E model. Two contributory studies to the A-I-E model will be discussed comparatively with the 250ms clock study; Marchetti (2006) and MacMahon et al (2006).

Firstly, we can make comparisons with other Posner studies such as Marchetti (2006) who used a modified semantic Posner paradigm with the aim of assessing attentional engagement/disengagement with sleep-related words in PI, GS and Delayed Sleep Phase Syndrome. Marchetti (2006) found that PI took significantly longer to disengage from negative sleep-related stimuli than both GS and DSPS suggesting that negative sleep stimuli are more salient to PI in holding attention than both GS and DSPS. However, this effect was only detected at the negative sleep word valence; no such effect was observed at the positive sleep word valence. Thus, it is proposed that, within the constraints of that experiment, negative sleep related stimuli are more salient to PI than positive sleep-related stimuli, and thus hold attention for longer.

In this 250ms clock experiment, PI show a delay in reaction time overall trials which could suggest that all times presented on the clock are seen as negative and therefore increase reaction time. The lack of difference between valid and invalid trials is different to the Marchetti (2006) study which found slower reaction times on invalid trials indicative of delayed disengagement. This would imply that enhanced engagement or delayed disengagement due to saliency of the stimuli is not relevant in this clock study.

The next possible explanatory factor is the presentation time and to draw comparisons with MacMahon et al (2006) who presented sleep and neutral words for 500ms in a dot probe paradigm. This length of cue presentation time is long in the attention bias field but is worth considering at this point as it is an increased presentation time in comparison to studies carried out by Marchetti (2006). MacMahon et al (2006) found that PI had a faster reaction time to sleep cues compared to GS on the dot probe task which, these authors concluded, indicated PI had an increased vigilance, or attention bias, towards sleep stimuli. The results of the 250ms clock study are not in line with the results of MacMahon and colleagues as an increased vigilance for sleep over day times is not seen. It can not therefore be concluded that as presentation time increases, vigilance for sleep becomes evident.

It would appear that the results from presenting sleep and day times to PI and GS for 250ms that 'traditional' attention bias is not found and that some other mechanism which differentiates PI and GS becomes apparent. Due to the absence of influence of cue location and time presented but the presence of a between group difference, it may be that a general performance deficit with PI is being seen on this task. At a longer presentation time of 250ms, PI are slower to react than GS over all trials. As the A-I-E model was developed from attention bias work carried out in the

anxiety field, it is relevant to discuss the current findings within this context as well as sleep attention bias work. Fox et al (2001) found at a presentation time of 100ms, high and low state anxious individuals did not perform significantly differently on a modified Posner task presenting threatening faces but significant differences were found at a longer presentation time of 250ms with high state anxious individuals demonstrating delayed disengagement from threatening cues on invalid trials. Significant differences were not seen for neutral or happy face cues suggesting that threatening stimuli are more salient for anxious individuals. This suggests that most people would show some attentional bias towards threatening cues at short presentation times but as the presentation gets longer, anxious individuals will maintain this difficulty in moving their attention away from salient (threatening) cues.

The differential responses to the cues being presented in the Fox et al (2001) study is relevant within the context of the current study in that at 250ms the pattern of responses of both GS and PI are not significantly different, it is just that PI are delayed in their responses compared to GS. The absence of differential responses on valid and invalid trials suggests that delayed disengagement does not explain their delayed responses but a more general performance deficit. These results also suggest that anxiety does not account for these group differences as delayed disengagement on invalid trials with perhaps a speeded engagement on valid trials would be the expected pattern of responses with an anxious response (Fox et al 2001).

9.3.2 Other considerations

Although the time presented did not statistically significantly effect performance on this task, it is worth noting that sleep times are reacted to marginally faster than day times by both sleep groups. This is consistent with the results of the

previous 100ms study in which a significant effect of time was found i.e. sleep times were reacted to faster by both PI and GS. These findings would suggest that sleep times are an ecologically valid stimulus in that, irrelevant of sleep quality, sleep times facilitate response whereas day times produce slower responses.

To further understand the very large between group error rate difference on the 250ms study, the reaction time results should be considered alongside. The reaction time data from this study shows that PI demonstrate a general delay in reacting compared to GS. It would therefore be intuitive to attribute this to a higher error rate; PI take longer to react correctly on this task because they are making more errors but by comparing the error rates of PI and GS, it would appear that this is not the case but GS are actually making more errors than PI.

One explanation for this could be a speed-accuracy trade off in GS. The instructions given to all participants at the start of the experiment were to react as fast as possible and so perhaps GS have followed instructions but have made more mistakes as a result. PI have taken longer to react overall but as a result have made fewer mistakes. These results reflect data obtained in a study conducted some time ago by Hines (1979) comparing the effects of feedback on reaction time and error rates on a letter classification task with younger (mean age 20.7 years) and older (mean age 63.6 years) participants. Older participants took significantly longer to react than younger participants but feedback only increased the error rate of the older subjects by 1.5% compared to 4.9% in the younger group. By introducing the extra dimension to the task, the performance of the younger participants suffered more than older as their faster reaction time was based on the speed/accuracy trade off.

The 250ms clock study would appear similar to this study by Hines (1979). The GS react faster but less accurately whereas PI are more cautious and therefore

slower to react but more accurate. The question now arises whether this is due to an interference effect of the clock cue in PI or a more general performance deficit. Since there is no suggestion of a variation in performance over the different trial types, the data would suggest that PI are more cautious on this task i.e. take longer to react generally. The absence of a between group difference on error rate for the 100ms clock study suggests that the effects found here are due to the varying performance dependent on sleep group and time presented on the clock.

The findings reported in this thesis so far have been insightful and challenging within the field of attention bias in insomnia and how it is influenced by saliency, timing and length of disorder. To utilise these findings constructively, it would be useful to move away from snapshots of processing and implement a strategy to allow a more consistent picture of attention allocation over time.

Chapter 10

Experiment 4

Semantic Eye Tracking

At this point in this thesis, cognitive probe paradigms which have been used in different guises to obtain reaction time measurements are being put to one side and a methodology which is new to sleep research is being introduced.

The reason for developing this methodology is to obtain a continual record of where the eyes, and therefore attention, are being allocated with regard to sleep and non-sleep stimuli presented simultaneously.

- Research question: On presenting sleep stimuli, do poor sleepers approach and then avoid or approach and maintain their gaze? These different responses reflect an anxious response or craving response, respectively.
- Hypothesis I: PI will show faster engagement for sleep related words compared to GS
- Hypothesis II: PI will remain fixated on negative sleep words for longer than positive sleep and neutral words.

10.1 Methods

10.1.1 Apparatus and Stimuli

Two words were presented on a computer screen for 3 seconds, one being an actual word and the other a pseudoword (Figure 10.1). Participants were asked to ignore the pseudoword and focus on the actual word. The stimuli were presented to participants on a 22" computer monitor (total viewing area 20" / 51cm) connected to a Dell Windows XP-based PC, with all words presented in 28 point Courier font. Actual and pseudowords were used in this experiment, in line with Scheepers et al (2008), as

pseudowords provide a comparative, baseline measure of interpretation strength compared to the actual word. Time is taken to discriminate between the actual and pseudoword with any variation of this discrimination time over the categories of words presented (sleep negative, sleep positive and neutral) providing a measure of saliency.

Some preliminary analysis has been carried out as this technique is novel within insomnia research. When designing this experiment, I was aware that the number of word stimuli we were using was rather limited compared to other eye tracking studies. The words used here were taken from previous sleep research on selective attention (Taylor et al, 2003 and MacMahon et al, 2006) and had been generated from Wicklow and Espie (2000), a study of the content of pre-sleep cognitions. Thus, they have a high degree of salience to individuals with sleep difficulties. The words used in this experiment can found in Appendix S.

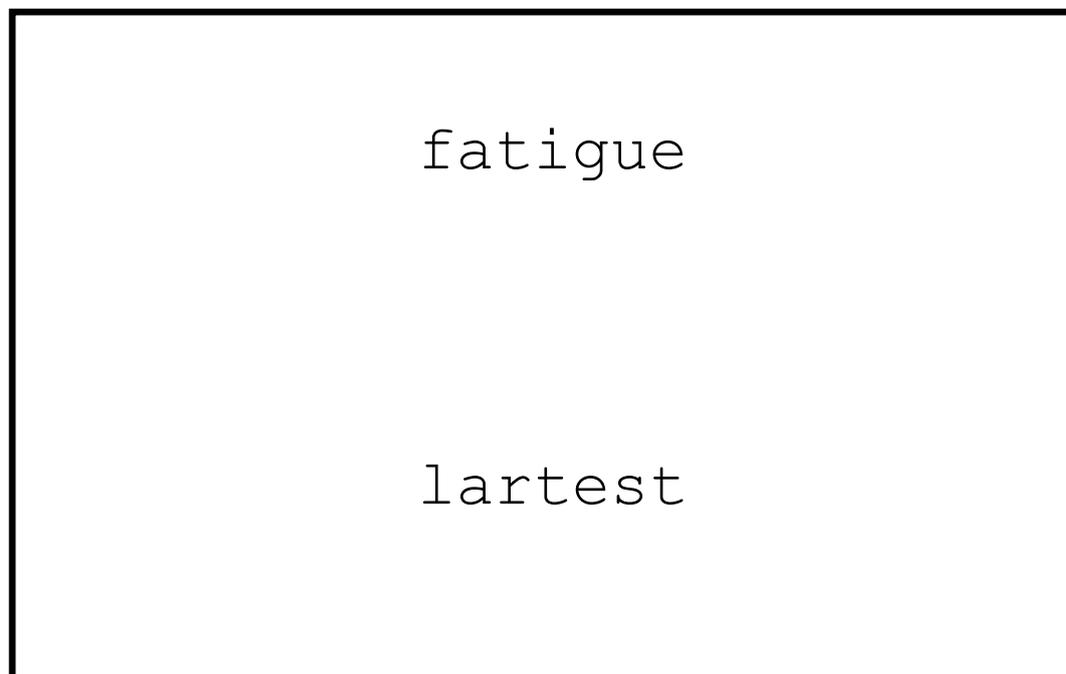


Figure 10.1 Example of stimulus presented during semantic eye tracking experiment.

An Eyelink II (SR Research, Ontario, Canada) eye-tracking system set in 500Hz mode was used to monitor the eye movements of participants to the stimuli. The eye-tracking system consists of two head-mounted cameras to sample pupil location and pupil size for each eye at the rate of 500Hz, in addition to two infrared LEDs to illuminate each eye. The resolution of eye-position is 15 seconds of arc, and gives a spatial accuracy of approximately 0.5 degrees. A head-tracking camera, mounted to the centre of the headband is also used in order to measure the head position of each participant, and four LEDs which are attached to each corner of the computer screen are viewed by the head-tracking camera whilst the participant is facing the computer screen. The eye-tracking system can compensate automatically for possible head motions by detecting movements of the four LEDs, and the compensation is better than 1 degree over the acceptable range of head motion. For each trial, areas of interest were identified for the area occupied by target and non-target words on each trial, enabling accuracy, approach and fixation parameters to be determined with respect to the areas of interest. A DOS-based PC was used to record eye-movement data. Although binocular registration of eye-movement is possible, in this study monocular registration of eye-movement was conducted by tracking the dominant eye of the participant, as determined by a subjective test carried out at time of participation.

10.1.2 Procedure

All participants were tested in a quiet room with controlled lighting and underwent a simple test in order to determine their dominant eye, and participants were then seated in a height adjustable chair in front of the eye-tracker, and placed into a fixed chin rest placed approximately 50cm from the computer monitor. The

eye-tracking cameras were then adjusted in order to best capture the measurements of each participant's dominant eye, and a 9-point calibration cycle was then completed to ensure that the recording of eye movements fell within better than 1 degree of visual angle for each calibration point. A 9-point validation cycle was then completed in order to verify the calibration cycle, and if necessary, both of these cycles were repeated until accurate measurements were obtained.

Prior to starting the experiment, participants were instructed to focus on the actual word presented to them and to completely ignore the pseudoword presented to them. Prior to the start of each trial, a fixation cross was displayed on the centre of the computer monitor in order to ensure that the starting location of their gaze was standardised, and after each group of 26 trials, the calibration cycle and validation cycle was again repeated in order to ensure that the eye-tracking system was accurately recording eye movements. Throughout the task, the experimenter made as little noise as possible and monitored both the stimulus presentation and eye-tracking measurements. Following the computer task, the questionnaires were completed and the experiment explained.

10.1.3 Eye tracking analysis

In order to prepare the eye-tracking data for subsequent statistical analysis, standard EyeLink criteria were used in order to distinguish between fixations and saccades. A fixation is defined in terms motion (in degrees), velocity (in degrees/sec), and acceleration (in degrees/sec²) thresholds for saccades i.e. everything that is not a saccade is a fixation. The default saccade threshold settings in the EyeLink software are:

saccade_motion_threshold = 0.15 degrees

saccade_velocity_threshold = 30 degrees per second

saccade_acceleration_threshold = 8000 degrees/second².

Any continuous samples exceeding these thresholds are saccades. Fixations which were considered to be out-of-range through having invalid x/y co-ordinates were removed from the data altogether, and following this, fixations on the areas of interest for each participant were recorded at a sampling rate of 100ms.

Bitmap templates were created for each experimental display which identified the distracter word, the target word and the background. The regions of interest were defined in terms of rectangles containing the relevant words within which any fixation was attributed as toward that word. Fixations shorter than 80 ms were combined with the previous or following fixation if within 0.5° of visual angle. The time period between the onset of the word pair and the end of the trial was divided into 100ms time slots. For each time slot, the number of fixations on the target word were counted and converted into fixation probabilities.

Scheepers et al (2008) developed the Logistic Power Peak (LPP) function as the best description of the variance both between and within conditions. The function comprises 3 separate parameters (Figure 10.1) which describe different characteristics of the probability differences over time:

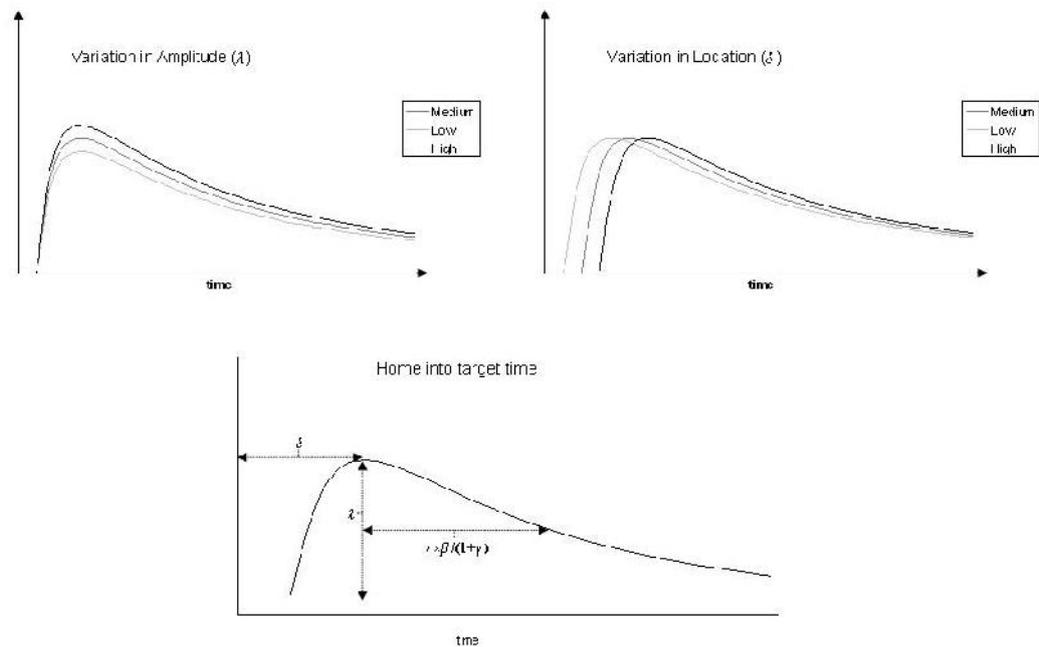


Figure 10.2 Illustration of the logistic power peak (LPP) parameters used to fit the probability difference distributions.

10.1.3.1 Parameters

A camera is used to track the movement of the darkest part of the eye, the pupil, which provides information on where the individual is looking in relation to the actual- and pseudoword presented on the screen. The output of data is then filtered to extract the factors of interest i.e. fixations on target and distracter words which we can the carry out our analysis on:

Amplitude: captures variation in overall interpretation strength.

Location: index of peak location in time. Lower values imply faster processing

Home in on target time: time point where half of peak amplitude achieved in right tail of plot. A higher value implies a slower rise and slower decline from peak.

The following 2 parameters were also analysed in relation to first fixation.

Onset: time that elapses between the onset of the visual stimulus presentation until the eye has landed on the target word for the first time.

Duration: onset of first fixation – offset of first fixation, time between start of the first fixation on target until fixation moves or trial finishes.

Figure 10.2 below highlights the parameters of interest and the probability of fixating on the distracter word (pseudoword). Each coloured line represents either GS or PI and their probability of looking at distracter when a sleep negative (neg), sleep positive (pos) or neutral (neu) target (actual) word is presented. The independent variables in this experiment were word condition (sleep negative, neutral and sleep positive) and target word position (above or below the distracter/pseudoword).

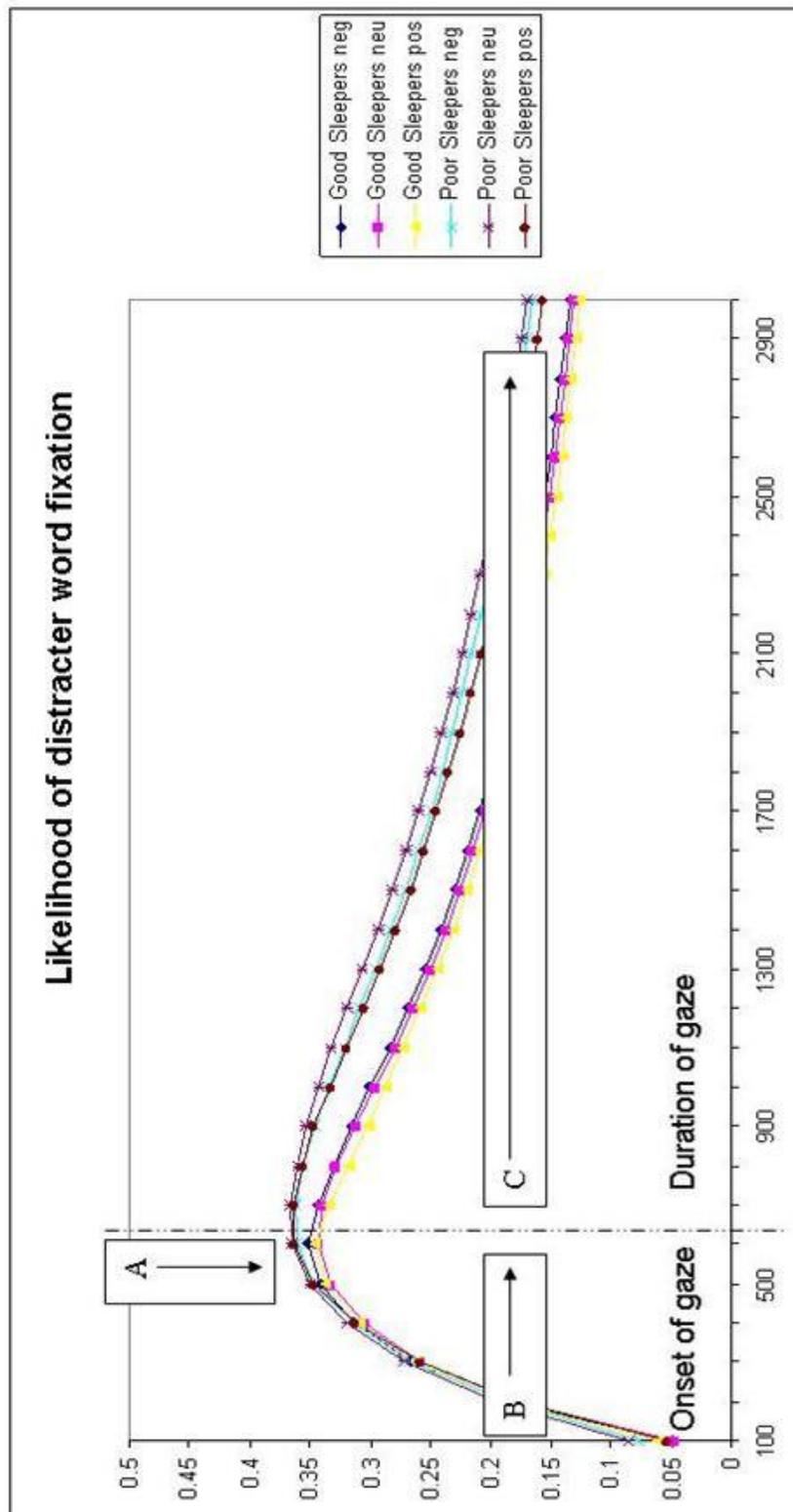


Figure 10.3 Probability of fixating on distracter word with parameters of interest highlighted; A= amplitude of peak and interpretation strength, B= location of peak in time and speed of processing and C= home in on target time. Parameters on left of dotted line are used to address the vigilance question and those on right are to address maintenance of gaze.

10.2 Results

10.2.1 Subjective measures

First, as with the other experiments in this thesis, we must confirm correct allocation to sleep quality group and substantiate the profile of each group. Table 1 below outlines the mean scores on the subjective measures for both PI and GS.

	PI		GS		MEAN DIFF.	SIG.
	MEAN	SD	MEAN	SD		
PSQI	12.3	2.3	3.9	2.1	8.4	.000*
ISI	15.9	3.8	3.3	2.5	12.6	.000*
HADSA	11.1	4.2	6.2	2.8	4.9	.000*
HADSD	6.2	3.4	2.1	1.6	4.1	.000*
DBAS	85.4	27.6	54.2	17.4	31.2	.0000*
MEQ	1.4	0.5	1.3	0.5	0.1	.52
APSQ	64.8	19.2	26.2	10.3	54.5	.000*
SES	10.0	1.5	2.5	2.1	7.9	.000*

Table 10.3 The mean scores and standard deviations of PI and GS on 8 subjective measures of sleep, anxiety, depression, circadian preference, sleep related cognitions and sleep effort. * signifies a significant effect.

As the table above shows, PI and GS significantly differed on all measures with the exception of the Morningness Eveningness Questionnaire (MEQ). The MEQ was included in this experiment to possibly identify any influence of circadian preference on performance. However, the sleep quality groups did not differ on this measure and therefore any differences seen on task performance within this experiment are not likely to be due to suboptimal timing issues with regard to

circadian phase and administration of the task. PI, appropriately, had poorer sleep quality and a more severe insomnia complaint as well as higher anxiety and preoccupation about sleep and employed more effort regarding sleep. They were also more anxious and depressed than GS although remained sub-threshold on both these measures.

We will now consider each of our hypotheses separately with the relevant analysis for clarity. The effects of interest are sleep quality (PI and GS), word condition (sleep positive, sleep negative and neutral) and target position (target word above and below distracter). With regard to target position, the graphical representations of the target word above distracter is presented as reading from top to bottom is the natural state, just as from left to right, and therefore produced the most interesting results. Mean reaction time data can be found in Appendix T.

10.2.2 Vigilance for sleep words

The following show the results of the analyses carried out in respect of the first hypothesis; PI will show higher vigilance for sleep related words compared to GS. The three parameters which will be analysed are Onset of first fixation, Location of Peak and Amplitude of Peak. * indicates significant effect.

Onset

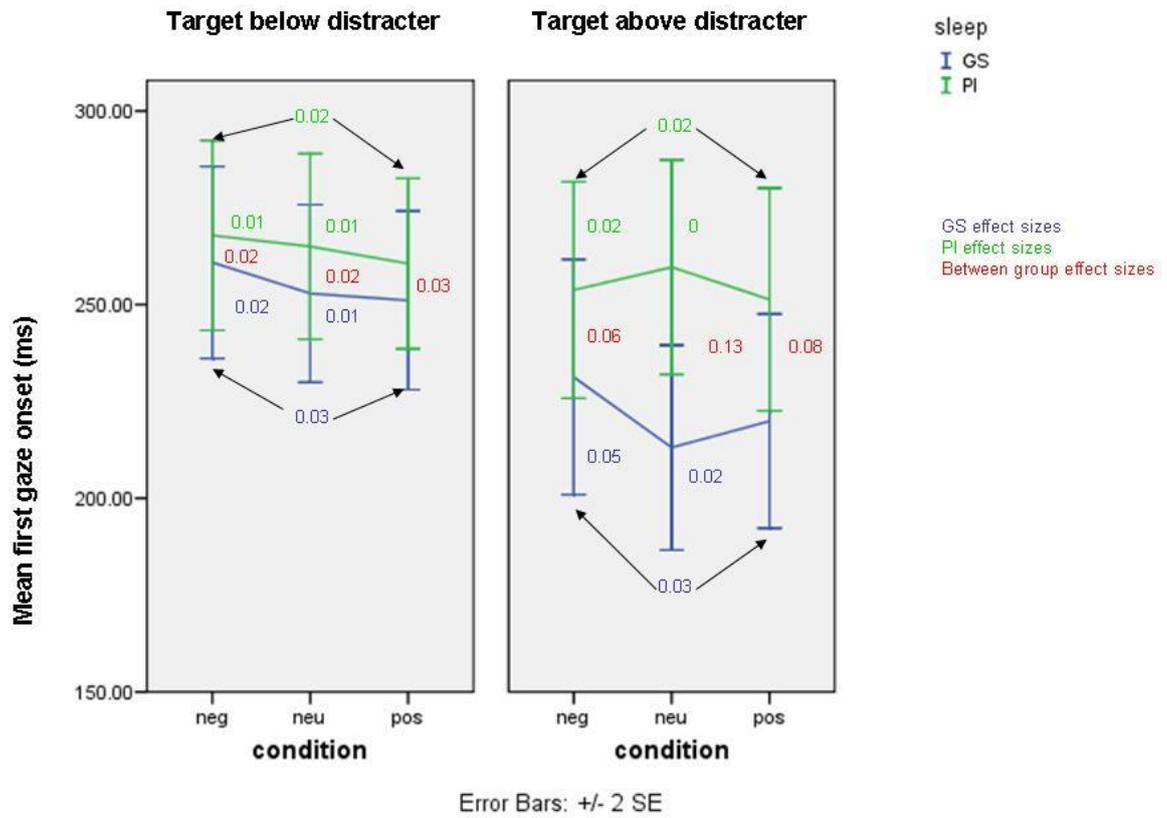


Figure 10.4 Mean onset of first fixation (ms) with within and between group effect sizes.

Two significant effects are found here; target word position $F(1,8654) = 8.33$, $p < 0.005$ and sleep quality $F(1,8654) = 8.32$, $p < 0.005$. Target word position is expected as mentioned previously, we read from top down, therefore when looking at an effect of time, position of target word will have an effect. With regard to sleep quality, the graph above suggests that PI started their first fixation on the target word later than GS and this was irrespective of word salience. This observation is confirmed in the analysis. There is no significant effect of word condition $F(2,8654) = 0.39$, $p = 0.68$ or interactions between sleep quality and word condition $F(2,8654) = 0.325$, $p = 0.72$, sleep quality x word condition x target position $F(1,8654) = 0.139$,

$p=0.87$, word condition x target position $F(2,8654) = 0.009$, $p=0.99$ or sleep quality x target position $F(1,8654) = 2.57$, $p=0.11$.

Location

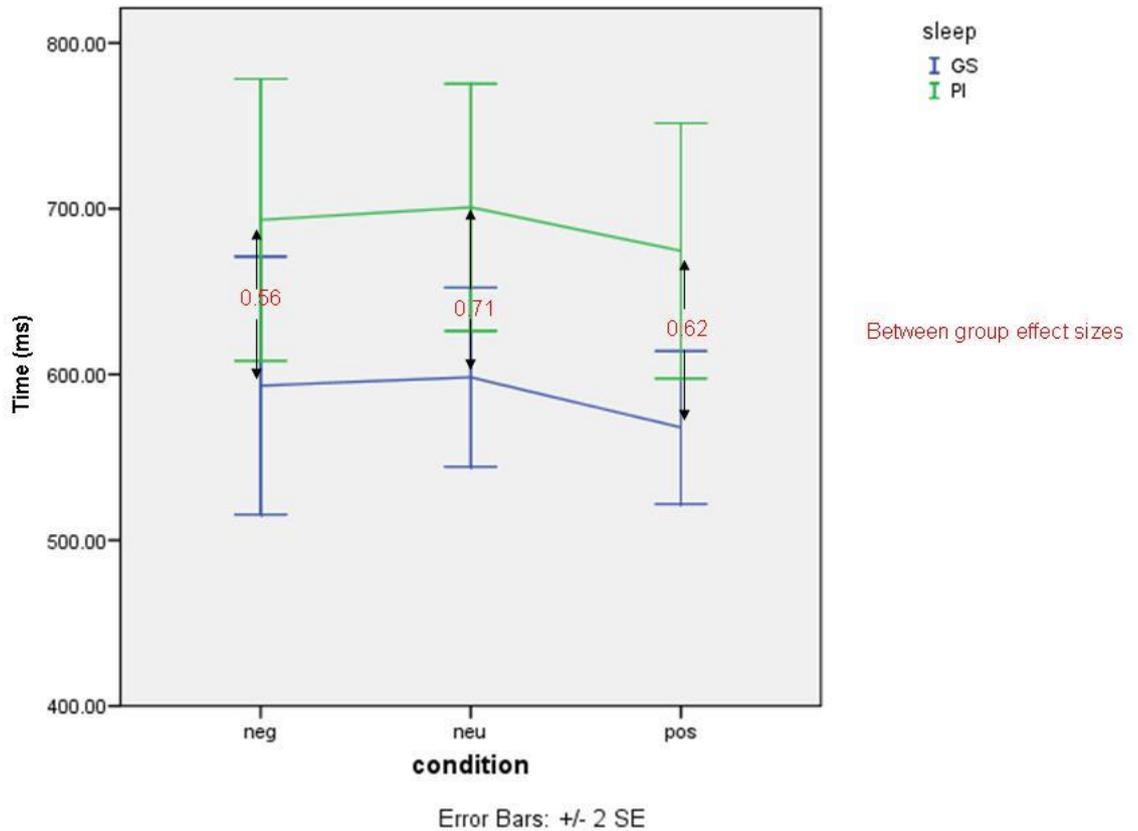


Figure 10.5 Mean location of peak for GS and PI for negative sleep (neg), positive sleep (pos) and neutral (neu) words.

The graph above suggests that the time taken for the target and distracter words to be discriminated between remains constant over word conditions but PI take consistently longer to achieve this discrimination. This observation is again confirmed by the formal analysis with a significant main effect of sleep quality $F(1,39)=5.0$, $p<.05$. Of note here also, are the much larger effect sizes between PI and GS on all 3 word conditions as compared to the effect sizes obtained for onset of first fixation (see

Graph 4). No significant effect was found for word condition $F(2,78)=1.4$, $p=0.25$ or a word condition x sleep quality interaction $F(2,78)=0.02$, $p=0.98$.

Amplitude

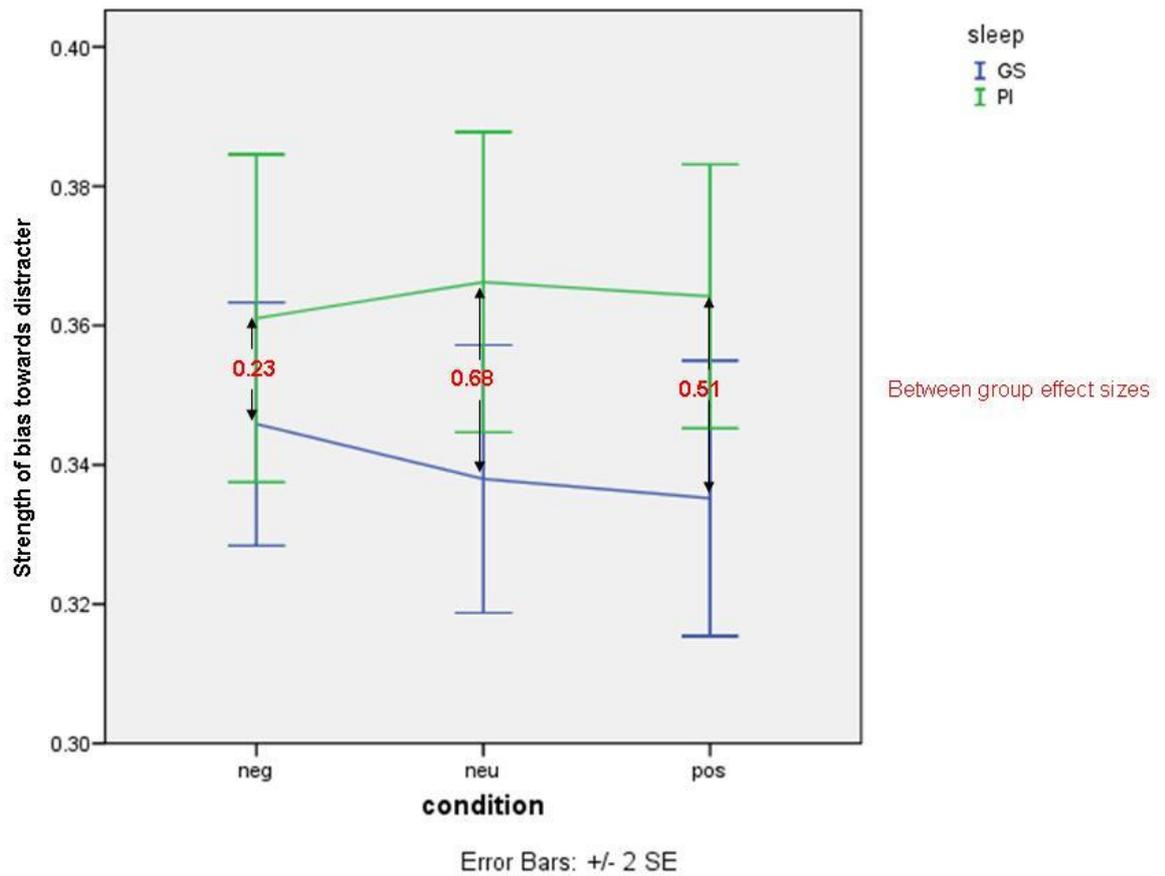


Figure 10.6 Mean peak amplitude of GS and PI for negative sleep (neg), positive sleep (pos) and neutral (neu) words.

With regard to the strength of bias towards distracter word, the graph above shows that PI have a much stronger bias towards the distracter compared to GS. This is borne out in the formal analysis with a significant difference between sleep quality group difference $F(1,39)=4.6$, $p<.05$. The effect of word condition remains pretty stable over all 3 conditions with a suggestion of variance of performance with the negative sleep words and a decrease of the effect size within this condition but no

main effect is seen of word condition $F(2,78)=0.12$, $p=0.88$ or interaction between sleep quality and word condition $F(2,78)=0.51$, $p=0.60$.

Hypothesis I summary

In summary, with regard to the question of initial vigilance for sleep related stimuli, onset of first fixation, location of peak and amplitude suggest that PI are actually delayed on fixating on target word compared to GS and more likely to look at distracter irrespective of word condition/salience. Now turn to our second hypothesis which we aim to address using duration of fixation and time to home in on target word.

10.2.3 Maintenance of attention

The following show the results of the analyses carried out in respect of the second hypothesis; PI will remain fixated on negative sleep words for longer than positive sleep and neutral words. The two parameters which will be analysed are Duration of first fixation and Home in to target time. * indicates significant effect.

Duration

Duration = onset of first fixation – offset of first fixation, time between start of the first fixation on target until fixation moves or trial finishes.

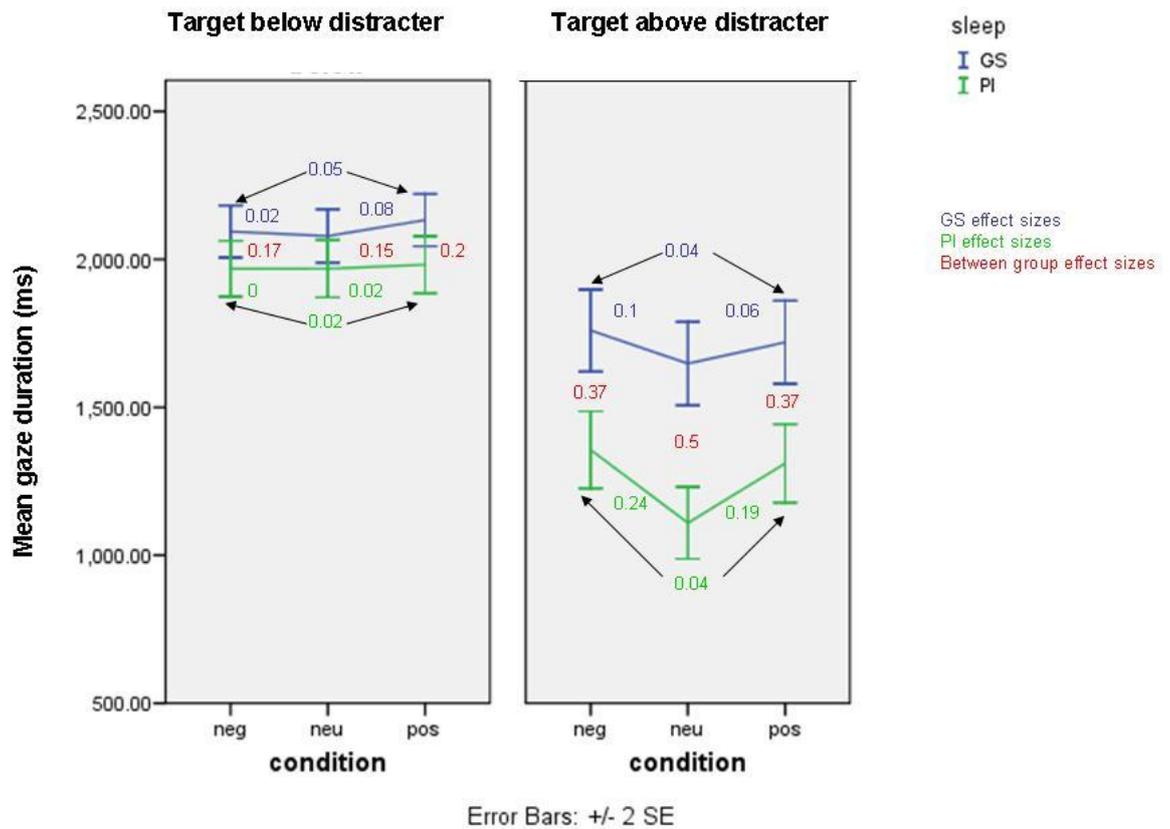


Figure 10.7 Mean fixation or gaze duration (ms) and effect sizes for GS and PI to negative sleep, neutral and positive sleep words when target below and above distracter word.

Duration of first fixation provides the first effect of word condition

$F(2,3167) = 3.22, p < 0.05$ that we see within this experiment. The graph above suggests that this is due to a longer gaze duration on sleep positive and negative words compared to neutral, particularly with the PI group. The effect size is greatest between the GS and PI with neutral words and the analysis provides a significant effect of sleep quality $F(1,3167) = 75.8, p < 0.005$. There is also a significant effect of target position $F(1,3167) = 276.7, p < 0.005$ as well as a significant sleep quality x target position interaction $F(1,3167) = 23.3, p < 0.005$. This interaction is illustrated in the graphs by the exaggeration of an effect of condition when the target word is above

the distracter particularly for PI. There is no significant interactions between sleep quality and word condition $F(2,3167) = 0.29, p=0.746$, word condition x target position $F(2,3167) = 2.25, p=0.11$ or sleep quality x word condition x target position $F(2,3167) = 0.649, p=0.52$.

Home in to target time

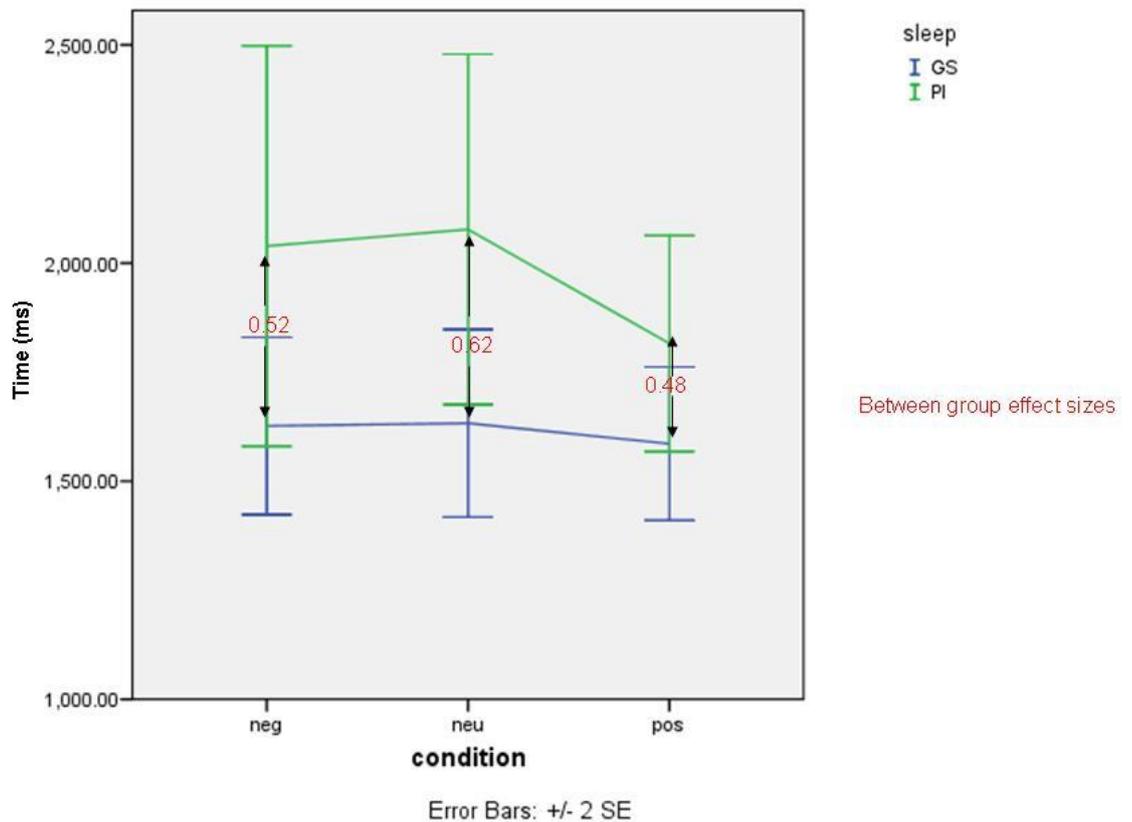


Figure 10.8 Mean ‘home in on target’ times (ms) for GS and PI by word salience.

The graph above shows the now familiar delay of PI compared to GS to, in this case, home in on the target word. With GS, the home in on target word is stable over the 3 conditions but less stable with PI who would appear to be faster to home in

on the target word when that word is sleep positive. This is illustrated by the decrease in effect size seen between GS and PI with sleep positive words. Our analysis shows that although there is suggestion of an effect of condition here, it is not significant $F(1.5,58)=1.3$, $p=0.28$ and there is no significant interaction with sleep quality $F(2,78)=1.4$, $p=0.25$ but there is a significant effect of sleep quality $F(1,39)=3.9$, $p<0.05$.

Hypothesis II summary

To summarize with regard to maintenance of attention, duration of first fixation and time to home in on target word show us the delay found with initially looking at the target word persists with maintenance of gaze and continues over the period of the trial. PI maintain their first fixation on the target word for less time than GS as well as take longer to home in on the target word.

This experiment suggests that PI actually contradict our initial hypotheses in that instead of actually showing increased vigilance for and engagement with sleep related words, we find that PI show a deficit in performance on this task compared to GS in that they take longer to fixate upon and maintain that fixation for less time on target words and are more likely to look at distracter. This is a relevant finding within the context of neurocognitive deficits in PI.

10.3 Discussion

In this study, several parameters were analysed. Firstly, we analysed three parameters measuring overall interpretation of the semantic stimuli presented. It was found that PI were more likely to look at the distracter versus the target word for

longer than GS. These between group differences on amplitude, location of peak and home in on target time have medium effect sizes and are therefore relatively robust.

Secondly, two parameters were analysed with regard to fixations; onset of first fixation and duration of that fixation. Within these parameters, position of target word i.e. above or below distracter produced a significant effect in that participants were faster when the target was above the distracter. This is as expected due to a natural tendency to read from top to bottom just like left to right (Duggan and Payne 2009). With regard to onset, PI took longer to begin their first fixation on the target word than GS with this delay particularly evident when the target is above distracter. Effect sizes showed the largest effect between PI and GS was with neutral words in the target above condition. This larger effect size for neutral words can be attributed to a differing pattern of response between PI and GS in that PI are slower to start fixating on neutral words compared to both negative and positive sleep words whereas GS are faster with neutral but slower with positive and negative sleep words.

The duration of first fixation produced more significant effects than onset. Again, differences between GS and PI are particularly evident when the target is above the distracter and with neutral words with PI fixated on the target word for a shorter length of time than GS. The pattern of responses with PI and GS were similar with duration of gaze on positive and negative sleep words longer than neutral but this is emphasized in PI.

This data would therefore suggest that PI are delayed compared to GS to start fixating on the target and once their gaze is engaged on target, they do not sustain their gaze for as long as GS and spend more time moving their gaze to and fro between the target and distracter.

10.3.1 Results in context of A-I-E model

To understand our data we can make comparisons with other semantic studies such as Marchetti (2006) who used a modified semantic Posner paradigm with the aim of assessing attentional engagement/disengagement with sleep-related words in PI, GS and Delayed Sleep Phase Syndrome. PI took significantly longer to disengage from negative sleep-related stimuli than both GS and DSPS suggesting that negative sleep stimuli are more salient to PI in holding attention than both GS and DSPS. Marchetti's (2006) result supports our prediction that PI find it difficult to disengage from sleep related stimuli based on this and other studies contributing to the A-I-E model (Espie et al 2006). However, a similar effect as Marchetti (2006) was only detected at the negative sleep word valence; no such effect was observed at the positive sleep word valence. Thus, it is proposed that negative sleep related stimuli are more salient to PI than positive sleep-related stimuli, and thus hold attention for longer. This is relevant within the context of the A-I-E model as it suggests PI attend to the negative representations of their inability to sleep which fuels their arousal and rumination.

The Marchetti et al (2006) study, which provided this eye tracking study with the words, also informed our hypotheses in that it was expected that PI would show delayed disengagement from, or sustained attentional engagement toward, sleep words, particularly negative sleep words. However, by using eye tracking, we find that PI take longer to engage with the target word and sustain attention on that word for less time than GS although, within PI, negative sleep and positive sleep words are fixated upon for longer than neutral. The semantic Posner study (Marchetti 2006) would suggest that PI engage then have difficulty in disengaging their attention from the negative sleep word compared to GS whereas the current study suggests that PI

have difficulty in *engaging* in a sustained manner with all words. Due to the nature of the experiments, the modified semantic Posner was more of a snapshot of attentional engagement at 100ms presentation time of the word as a cue followed by a target compared to this eye tracking study which presented the target and distracter word for 3000ms so we are therefore gathering more information regarding engagement over a longer period of time although first fixation did occur within a 100ms time frame.

As this is another unexpected outcome and the hypotheses are rejected, the results have to be considered in the context of the A-I-E model (Espie et al 2006). The studies discussed above suggest that the sleep, particularly negative sleep, words would be engaged with faster than neutral words. As the words have been used previously in a modified Posner task (Marchetti 2006), the nature of this eye tracking task may provide some insight into the difference in outcome between the studies.

Compared to the other semantic studies carried out in the attention bias in insomnia field, this eye tracking study presents the target sleep negative, sleep positive and neutral words simultaneously with a pseudoword. Whereas Marchetti (2006) presented sleep or neutral trials separately, this eye tracking paradigm always presented a pseudoword distracter which competed for interpretation. As would be expected, the interpretation of the target actual word became stronger as time progressed while interpretation of the distracter pseudoword diminished. This introduces another new concept to this attention processing in insomnia research field as it would appear that the nature of the task, i.e. discriminating between a target and distracter, influences the performance of PI compared to GS in a way that has not been seen before. This is an important consideration in interpretation of the results within the A-I-E model. PI will not always selectively attend to sleep or sleep negative stimuli, wherever or whenever it is presented.

10.3.2 Other considerations

To move away from semantic tasks specifically and look at performance of PI on cognitive tasks more generally, Altena et al (2008) undertook a study of vigilance to investigate whether PI show performance deficits as compared with healthy GS controls. Participants were administered both a simple and complex vigilance task with the simple task involving responding to the appearance of an asterisk and the complex involving participants reacting to a target letter and ignoring a distracter. An interaction was found between task and sleep status in that PI perform faster than GS on the simple task but slower on the complex task which the authors attribute to a larger 'complexity cost' in PI. With this variable reaction time seen in PI, an explanation of reduced awareness is suggested but, the authors conclude, points to a disturbance of brain processes involved in higher aspects of information processing (Bastien et al. 2003).

With regard to our current study within the context of Altena et al (2008), we had thought the task prescribed to our participants was a very simple one in that they were not measured in terms of reaction time but only where they were looking over the 3 second presentation time. However, Altena et al (2008) classed discriminating between a target and distracter letter as complex which is similar to the instruction given to participants in the current study which was to look at the actual word and ignore the pseudoword. Perhaps there is more complexity involved in discriminating between target and distracter than expected or this particular type of task involving vigilance and attention is able to tease out deficits in PI. This would offer a possible explanation for the delay in engaging and prolonging engagement as would be expected following on from the Marchetti (2006) study where the task instruction was to press the appropriate key in response to dot orientation i.e. there was no instruction

to be vigilant for but ignore other stimuli which could be compared to the simpler task in Altena et al (2008). Therefore, the vigilance and engagement for the negative sleep stimuli in Marchetti (2006) could have been helped to the surface by the level of cognitive load required.

Consistent evidence of a cognitive deficit in insomnia is lacking. Shekleton et al (2010), in their clinical review paper, summarise that generally, when performance on a task is measured by accuracy rather than speed, PI were more likely to perform more poorly than control groups. The authors also note that studies which showed a performance deficit in PI included distracter stimuli and required participants to make a response choice, much like the Altena et al (2008) study. These statements are in line with the results from the current study in that the PI group in this study was more likely to look at the distracter and took longer to discriminate between the target and distracter words.

The significant effect of condition in gaze duration in this study suggests that positive sleep and negative sleep words produce a different response compared to neutral words. GS looked at positive sleep and negative sleep words slightly longer than neutral and this response pattern was exaggerated in PI with an increase in effect size. As everyone sleeps and sleep is something that is salient to everyone, albeit in different ways and to varying degrees, the responses of participants over the three word types suggests that sleep words are more salient than neutral but this is much stronger in PI.

To address explanations for these findings, it would be expected that if avoidance was a possible underlying cause for this differing pattern between PI and GS, it would be seen particularly with the positive and negative sleep words and there would be no difference between the groups on neutral words. The salience of the

words presented is significant with gaze duration only in that PI and GS fixate on positive and negative sleep words for longer than neutral but this is a stronger effect in PI. This could be due to the ecological validity of sleep stimuli which is exaggerated in PI compared to GS although PI have a shorter first gaze duration than GS over all three word types. This delay in onset of first fixation, shorter first gaze duration followed by a higher number of eye movements between target and distracter words is more suggestive of a delay or disruption in processing rather than an enhanced engagement with salient stimuli.

Chapter 11

General Discussion.

‘The feeling of sleepiness when you are not in bed, and can't get there, is the meanest feeling in the world.’

Edgar Watson Howe

The table below provides an overview of each experiment included in this thesis including the outcomes.

Experiment	Research Question	Paradigm/Methodology	Outcome
1. Attentional bias (AB) as a maintaining factor within chronic insomnia.	Is attentional bias to sleep in PI a maintaining factor leading to chronic insomnia and not an initiating factor leading to an acute period of insomnia?	Modified Pictorial Posner presenting stimuli representative of sleep and non-sleep (kitchen).	By drawing comparisons with data provided by Marchetti (2006), these results suggest that AB to sleep is a maintaining factor specific to the chronic condition as no similarities were found with the acute insomnia group.
2. Sleep times as a focus of selective attention in insomnia.	Is it sleep times presented on the clock that individuals with insomnia are attending to or the clock itself?	Modified Pictorial Posner presenting stimuli showing a digital alarm clock displaying either a sleep or day time.	Unexpectedly, AB to sleep times was not seen in PI but day times influenced performance on task with significant relationships with sleep quality and daytime impairment. This is informative within the context of the A-I-E model where sleep is proposed as the salient entity.
3. Sleep times as a focus of maintained attention in insomnia.	Is delayed disengagement to sleep times still seen at a longer presentation time of 250ms?	Modified Pictorial Posner presenting stimuli showing a digital alarm clock displaying either a sleep or day time.	Again, sleep times did not selectively influence PI performance on this task as expected. Over all trials, PI were significantly delayed compared to GS. This is interesting within the context of the A-I-E model as well as highlighting a possible more general performance deficit in PI.
3. Pattern of eye movements to sleep in PI: avoidance or engagement?	On presenting sleep stimuli, do individuals with PI approach and then avoid or approach and maintain their gaze? These different responses reflect an anxious response or craving response, respectively.	A sleep positive, sleep negative or neutral word presented simultaneously with a pseudoword on a computer screen while participants are eye tracked.	A more general performance deficit is seen in PI which is not influenced by the target word representing sleep on a positive-negative continuum. This suggests a general performance delay in PI. This is discussed in the context of the discriminatory nature of the task influencing performance which is informative to the A-I-E model.

Table 11.1 Thesis overview by experiment.

11.1 Thesis findings

The overall picture to be drawn from this thesis is that PI perform slower than GS when the presentation time is longer than 100ms i.e. a more overt presentation.

This delay in reaction times is generally irrespective of the saliency of stimuli presented; delay is seen with sleep and day times as well as positive sleep, negative sleep and neutral words. This leads into consideration of possible causes of delayed reaction times in PI such as prefrontal cortex impairment as discussed below.

When the presentation time of the stimulus is more covert (100ms) we see a more differential pattern of reaction times in PI compared to GS with validly cued day times producing the largest effect in that PI react slowest on these trials. It is argued that this is due to day times having high saliency, possibly more than sleep times and informs the model of insomnia presenting as a 24 hour disorder and not necessarily only during the night.

The third notable finding of this thesis is the support for attention bias as an indicator for the chronic disorder as the experiment comparing reaction times of acute insomnia and GS found no significant difference between these groups with a trend for those with acute insomnia to react faster than GS which could be attributed to hyperarousal.

11.2 Current findings in relation to previous research

The work carried out in this thesis started from research on attention bias towards sleep in psychophysiological insomnia to a variety of stimuli representing sleep and attempted to further understand the underlying mechanisms of such a processing bias. The first study included day times with the sleep times which had been presented in a previously published study carried out in this lab, Woods et al (2009). In line with this previous research, a significant effect of trial type and trial type x sleep group interaction was found. However, the day times presented on the clock cue appeared to have more of an effect on processing in PI than sleep times with

most delay in reaction time being seen with validly cued day times. There is also a significant relationship between performance on these trials, sleep quality and perceived daytime impairment suggesting a heightened response towards sleep generally and an avoidance of day times resulting in the speeded reaction times seen in PI on invalid day time trials compared to valid.

One of the strategies used to understand the underlying mechanisms of attention bias in insomnia was to manipulate the presentation time of the salient cues to see if differences in performance maintained beyond a 100ms presentation time as had been seen in previous research carried out with anxiety populations (Fox et al. 2001). The modified Posner paradigm presenting the clock cue showing sleep and day times was repeated although the presentation time was now extended to 250ms, although this was the only difference. The results here differed from the previous clock studies in that no effect of trial type or interaction within sleep group was found but a significant between group effect was found between PI and GS in that PI were delayed in responding over all aspects of the task compared to GS. This is not in line with the anxiety literature which suggests that an attention bias towards anxiety provoking stimuli maintains at 250ms in high trait anxious individuals (Fox et al 2001). This delay in reaction time is suggestive of a more general performance deficit in PI compared to GS which could be indicative of an attention or neuropsychological deficit in insomnia. To understand this more a new methodology to insomnia research was employed.

A feature of cognitive probe tasks is that they provide a snapshot of attentional bias i.e. a reaction time measurement is obtained after the offset of the probe. This is affected by the duration of the presentation time of the probe. For example, if the

probe is displayed for shorter presentation times (100ms) then this possibly measures initial shifts in attention compared to longer presentation times (250ms) which provides a measure of maintained attention. More recent studies have used eye tracking to further understand information processing beyond a time limited snapshot and over a longer period of time. Eye tracking was used here to provide a timeline of approach/engagement and avoidance/disengagement on a semantic task and found that PI were more likely to look at the distracter word, took longer to discriminate between the target and distracter words and to fixate on the target word. PI also had a shorter first fixation as well as continuing to move between target and distracter for longer than GS. In this task, the words that were displayed were positive sleep, negative sleep and neutral but no effect of word type was found. This would be more suggestive of a general performance deficit rather than a bias towards or away from a stimulus type, although PI showed a trend towards homing in on the positive sleep target word faster than negative sleep or neutral words but this was still significantly slower than GS.

11.3 Insomnia performance deficit

In summary, the eye tracking study and 250ms clock study would suggest that at longer presentation times a general performance deficit is seen in PI but at a 100ms snapshot presenting the clock cue, PI and GS vary in their performance in that day times provoke reactions suggestive of avoidance in PI whereas sleep times affect performance in GS. The general performance deficit seen in these former studies is in line with Altena et al (2008) who undertook a study of vigilance to investigate whether PI show performance deficits as compared with healthy GS controls. Participants were administered both a simple (respond to an asterix) and complex

(react to target and ignore distracter) vigilance task. An interaction was found between task and sleep status in that PI perform faster than GS on the simple task but slower on the complex task which the authors attribute to a larger ‘complexity cost’ in PI. With this variable reaction time seen in PI, an explanation of reduced awareness but, the authors conclude, points to a disturbance of brain processes involved in higher aspects of information processing (Bastien et al. 2003). Perhaps by introducing day times into the paradigm, the task becomes more complex, similar to Altena et al (2008), by introducing discrimination between two different categories, sleep and day. The stimulus presentation times in the Altena et al (2008) study varied between 500ms and 5 seconds which would be longer than the length of time the clock cue was presented for in the present study but interestingly a deficit is seen in PI beyond a presentation time of 100ms in line with the current study.

11.4 Avoidance

With regard to the initial avoidance of the day times, 100ms is accepted as a measure of initial vigilance in cognitive probe tasks (Field and Cox, 2008). Other studies on attentional bias have indicated that shorter presentation times indicate early attentional processing rather than maintained attention. Veenstra et al (2010) investigated whether restrained eaters are characterized by enhanced engagement for and/or an impaired disengagement from food stimuli compared to unrestrained eaters. Restrained eating refers to dieting as well as the intention to diet. They found that both groups showed a pattern of avoidance of high-fat food compared to neutral stimuli at 500ms by displaying slower attentional engagement with high fat food stimuli compared to neutral. When the presentation time was extended to 1500ms no differential processing either between groups or between stimuli types was found. The

authors of this study found a similar pattern of results to the current 100ms study in that the longest reaction time recorded on the experiment was on valid trials when a particular type of stimuli were presented; with Veenstra et al (2010) performance was longest with high fat food stimuli and both restrained and unrestrained eaters and the current study showed that PI take longest to react on valid trials presenting day times.

Veenstra et al (2010) attribute their findings to avoidance and directing attention away from 'forbidden food'. Although the presentation time in their study was longer than the 100ms in the clock study, Veenstra et al's (2010) explanations of avoidance and directing of attention away is a legitimate argument that can be applied to the 100ms clock study for two reasons; faster reactions on invalid day trials and relationship between perceived day time impairment and reaction time on the valid day trials. Although the effect size was negligible between reaction time on valid and invalid day time trials with PI, invalid day trials were slightly faster than valid. This could be indicative of attention being moved from presentation site of clock displaying a day time which results in attention being deployed at the site of the invalidly cued target. The stronger suggestion of deployment of attention away from day times is that a medium effect size is found between PI and GS on these day time trials whereas the remainder of the between group differences are small. Also, nearly 20% of the variance found on performance on valid day trials is accounted for by sleep quality as measured by the PSQI and perceived daytime impairment as measured by the DFSAS. This would suggest that indeed sleep quality and complaint is linked to performance on this task, not necessarily with sleep times.

11.5 Daytime impairment

One of the recognised daytime impairments that the results of the 100ms clock study could instinctively be attributed to is daytime sleepiness. However, although sleepiness was not measured for this or the other studies conducted in this thesis, the published research on daytime impairments in insomnia show that a distinction should be made between perceived sleepiness and propensity to fall asleep. Bonnet and Arand investigated this by manipulating the sleep architecture of 10 healthy control subjects to match that of 10 insomnia patients followed by measurement of all on the Multiple Sleep Latency Test (MSLT). The healthy control group with induced insomnia showed decreased sleep latency both during the day and at night. Therefore, the MSLT appears to measure ability to fall asleep rather than perceived sleepiness and highlights the impairment in insomnia to fall asleep although high levels of sleepiness are reported. This previous research and the absence of a delay over all trial and stimuli types in responses with PI would suggest that these results are not due to general fatigue or sleepiness but due to day times specifically influencing processing and therefore performance on this task.

The results from this study would suggest then that day times are particularly salient to PI. Daytime impairment attributed to disturbed and/or poor quality sleep is one of the core diagnostic criteria for insomnia according to the ICSD 2 and DSM IV. Previous research has shown that PI report consistent detriments in mood and cognitive abilities alongside higher levels of anxiety, fatigue and physical pain/discomfort compared to GS (Kyle et al 2010). Two main points regarding daytime impairments are relevant in the current study; relevance of day times as a period where effects of poor sleep are felt and poor performance on task due to neurocognitive impairments due to poor sleep. We have discussed neurocognitive

deficits within the context of the longer presentation time on the 250ms clock study and the eye tracking study as well as research outwith our lab by Altena et al (2008). However, as an overall deficit is not seen on the 100ms clock study we move on to discuss the relevance of day times in PI. Shekleton et al (2010) discuss daytime impairments of insomnia in their review paper including sleepiness, fatigue and neurobehavioural performance. Several cognitive domains are discussed in this paper reviewing neurobehavioural performance; attention, psychomotor and processing speed, working memory, new learning and memory and executive functioning. Deficits between PI and NS have been found on attention and some aspects of executive functioning but little evidence of a single and consistent cognitive deficit among PI exists compared to controls. Shekleton et al (2010) generalise that across the domains mentioned above, when performance has been measured by accuracy rather than speed or when having to shift attention, PI tend to show a deficit compared to GS.

Edinger and colleagues (2008) conducted a study looking at performance of PI and GS on psychomotor tasks and their relationship with subjective and objective sleep measures. Participants underwent 3 nights of polysomnography (PSG) followed by daytime testing on a performance battery including a simple reaction time task, a continuous performance task and switching attention tasks. This battery of tasks was given at 4 time points, before each of 4 multiple sleep latency tests, throughout the testing day and analysis conducted on the mean reaction times and standard deviations of each participant's within-test response latencies. These authors found that PI had longer response latencies and more variability in their reaction times across several tasks, specifically, PI took longer to respond on 3 of the 4 switching attention tasks and showed significantly more variability in reaction times than GS on the simple

reaction time task and all of the switching attention tasks. The switching attention tasks in this study involved pressing specifically marked computer keys in response to stimulus presentations.

Regression analyses were also carried out using PSG and sleep diary values to predict performance on the 8 tasks administered to PI and GS. Wake time after sleep onset (WASO) from PSG came out as the best predictor of most of the performance indices although the R^2 values suggest that a small amount of variance in performance is accounted for by this sleep measure; R^2 varies from 0.04 to 0.08 over the different tasks.

The above authors discuss these findings within the context of daytime complaints of PI including difficulties in concentrating and a general lack of mental sharpness which would appear to be most challenged in the switching attention tasks over the simple reaction time and continuous performance tasks which perhaps were not challenging enough to make draw out performance deficits in PI compared to GS. Also, as Edinger et al had participants undergo MSLT, a measure of alertness is available which suggests the PI participants in this study had a longer mean MSLT latency than GS although they rated themselves as more sleepy on the Stanford Sleepiness Scale. This would suggest that despite showing poorer sleep in PSG and sleep diaries as well as reporting being more sleepy during the day, the PI group would appear to be less able to initiate sleep when given opportunity which is attributed to hyperarousal. Hyperarousal would perhaps suggest faster reaction times but only performance deficits are seen in this study.

The deficits seen by Edinger et al (2008) in the PI group reflect the delays in reaction time found in the 250ms clock and the semantic eye tracking studies as the stimulus presentation time in the Edinger study was either until a reaction was given

or 2500ms had elapsed. This would be in line with the data from the studies involving longer presentation times in this thesis although these are not as long as 2500ms.

Also, the switching attention task which requires intact concentration, attention and reaction to the orientation of a stimulus appears to be similar on many aspects to the modified Posner used in the clock studies to measure engagement and disengagement of attention by measuring reaction time to dots presented horizontally or vertically with each orientation requiring a specific response.

The clock studies in this thesis have shown that sleep quality as measured by sleep related questionnaires accounts for between 10% and 20% of the variance on performance on the tasks whereas Edinger and colleagues found between 4% and 8% of performance variance was accounted for by objective measurement of wake time after sleep onset. The possible explanations of these findings are threefold. Firstly, it may be due to the different paradigms used with the modified Posner providing a cleaner measure of engagement and disengagement of attention compared to the switching attention task involving more response inhibition and decision making. Secondly, the predictive measures used by Edinger et al in the regression models were derived from sleep diaries and PSG whereas the predictive measures entered into the models used in the analyses for the clock studies were perceived sleep quality as measured by the PSQI and ISI. Therefore, there appears to be a difference between using objective sleep measures and perceived sleep quality and complaint in accounting for differences in reaction time performance on psychomotor tasks in PI. The third factor to be considered is that the recruited insomnia group in the Edinger et al study had a mean age of 50 years (SD=17.1 years) compared to the present clock studies (experiments 2 and 3) which recruited a student population and therefore had mean ages of early 20s. This age difference highlights issues in comparing the present

studies in that deficits in performance in the Edinger et al study may be due to the participants being older than the participants recruited from the student population in Glasgow and showing age related reaction time delays as demonstrated by a study conducted out with the insomnia field by Hines (1979) which showed that older (mean age 63.6 years) performed slower than younger (mean age 20.7 years) participants.

11.6 Prefrontal cortex impairment

Miller and Cohen (2001) in 'An integrative theory of prefrontal cortex function' suggest that the prefrontal cortex is particularly important in cognitive control, is most elaborated in primates and well positioned to co-ordinate a wide range of neural processes such as when top-down processes are needed. Miller and Cohen (2001) highlight the Stroop task (Stroop 1935, MacLeod 1991) which involves subjects either reading words or naming the colour in which they are written. To perform this task, subjects must selectively attend to one attribute. This is especially so when naming the colour of a conflicting stimulus (e.g. the word GREEN displayed in red) because there is a tendency to read the word ("green"), which competes with the response to the colour ("red"). This illustrates one of the most fundamental aspects of cognitive control and goal-directed behaviour: the ability to select a weaker, task-relevant response (or source of information) in the face of competition from an otherwise stronger, but task-irrelevant one. Patients with frontal impairment have difficulty with this task (e.g. Perrett 1974, Cohen & Servan-Schreiber 1992, Vendrell et al 1995), especially when the instructions vary frequently (Dunbar & Sussman 1995, Cohen et al 1999), which suggests that they have difficulty adhering to the goal

of the task or its rules in the face of a competing stronger (i.e. more salient or habitual) response.

Altena et al (2008) conducted a study to investigate abnormalities in functional brain activation in line with subjective complaints about daytime cognitive functioning. Whilst being scanned by fMRI, GS and PI performed both a category and a letter fluency task which showed that, compared to GS, PI showed hypoactivation of the left medial prefrontal cortex and left inferior frontal gyrus for both task types. However, this compromised prefrontal brain activation occurred in the absence of a behavioural deficit. Wilson et al (2004) discuss the dorsolateral prefrontal cortex, among other areas, within the context of craving as seen in individuals associated with drug use. The authors discuss that factors such as treatment status affect processing of drug related cues and treatment seeking status influenced activation of the prefrontal cortex. The activation in the dorsolateral prefrontal cortex is reliably produced in cue-exposure studies of drug-addicted individuals who are still actively using. In contrast, these regions are rarely activated among patients preparing to quit.

Rounis et al (2006) examined effects on the dorsolateral prefrontal cortex following conditioning with 5Hz rTMS and found that reaction times on a cued choice reaction time task increased specifically on invalid trials which would suggest that manipulation in this area effects ability to disengage or reallocate attention.

In summary, prefrontal activation is influenced by sleep as hypoactivation is seen in an insomnia group when administered a word task and, in another clinical population, is implicated in craving as activation is seen when producing a 'desired' stimulus in those still using drugs but activation is not seen in a population seeking treatment. This is relevant within the context of this thesis because stimuli are relevant to PI in that sleep is something that is reported as being desirable and the individuals

would want more of but at the same time their unsatisfactory sleep is producing possible deficits in prefrontal activity. The pattern of data from the two clock studies as well as the eye tracking study suggest that any behavioural impairment is seen at presentation times beyond 100ms as the longer presentation times produce more general, less variable and stimuli related delays. Why do we see this deficit later on? One possibility is that the prefrontal hypoactivation seen in Altena et al (2008) is producing a behavioural deficit in these studies whereas a fluency test would not produce such behavioural results. This task dependent behaviour is analogous to that seen in another Altena et al (2008) study which task complexity affected reaction time of an insomnia group compared to GS. Discriminating between stimuli has been discussed previously as being a complicating factor with regard to the task and perhaps by presenting both day and sleep times or words and pseudowords, the complexity of the tasks are increasing and therefore a behavioural deficit is becoming apparent related to prefrontal hypoactivation which suggests impairment in allocating attention and maintenance of attention. A very interesting avenue of future research would be to understanding the prefrontal cortex impairment in PI and how task complexity and stimuli type effects activation in this area.

Transcranial magnetic stimulation (TMS) offers the possibility of assessing the excitability of the cerebral cortex (Kobayashi M and Pascual-Leone A, 2003). The technique involves delivering short-lived pulses of a strong magnetic field over the scalp, inducing local electrical currents in the brain through electromagnetic induction (Kujirai et al, 1993). When the coil is held over the primary motor cortex pulses lead to a muscle response that can be recorded using electromyographic recording. The size of the evoked muscle response (motor evoked potential, MEP) is then taken as a measure of excitability. Van der Werf et al (2010) conducted a study using TMS to

examine intracortical excitability before and after multimodal sleep therapy. The authors found that as the inter pulse interval (IPI) increased, a difference became apparent between PI and GS which disappeared towards longer IPIs; significant differences were found between groups at IPIs between 3ms and 13ms with PI showing higher MEPs. Overall, PI show an increased absolute excitability relative to control participants. Paired pulse TMS modulates the MEP size by preceding the test pulse with a subthreshold conditioning pulse which results in either inhibition or facilitation of the MEP. The paired pulse technique is a test of intracortical control over excitability and Van der Werf et al (2010) found that PI demonstrated a reduced intracortical facilitation as the increased MEPs are not maintained at the longer IPIs which the authors describe as the intracortical inhibition-facilitation curve having shifted to the left compared to GS; PI show maximal facilitation at IPIs shorter than healthy controls and the drop off with longer IPIs would reflect the drop off seen with GS at even longer intervals.

11.7 Overall conclusions and future research directions

In overall conclusion, an attention bias in insomnia is influenced by nature of the sleep disturbance, presentation time of the salient cues and methodology employed. With reference to the previous research on attention bias, we have found that when a salient stimulus is presented to PI, reactions are not simply measurable in terms of engagement and disengagement or approach and avoidance. Discrimination of categories of stimuli have highlighted a performance impairment in PI compared to GS which to date has proved elusive to document although PI will report debilitating effects of their poor sleep. This is alongside the saliency of times representative of daytime which was suggestive of a higher saliency than sleep times.

There are limitations to the methodologies applied in this thesis which should be discussed within the context of the findings. Firstly, sleepiness or fatigue levels were not recorded which could be presented as an explanation for the findings in Experiment 3, the 250ms clock experiment (Chapter 9) and Experiment 4, the eye tracking experiment (Chapter 10). Although there is no data with regard to sleepiness or fatigue presented here, the research to date on these measures, for example the MSLT, have found that PI do not show a greater inclination to fall asleep when given the opportunity. Also, PI do not show a delay in reacting when the cue presentation time is 100ms (Chapter 8) which would be expected to occur if sleepiness or fatigue was at play.

The words presented in the semantic eye tracking experiment were taken from Marchetti et al (2006) where the lists of positive sleep, negative sleep and neutral had been developed by asking a non-clinical student population for sleep related words and to rate them on a positive to negative dimension. What should be considered here is that the word 'bed' may be a positive word to those whose sleep is not giving them cause for complaint or even to PI who craves sleep but when considering bedtime and sleep results in both cognitive and physiological arousal, 'bed' can be a negative expression. The opportunity could be taken in future studies to obtain the participants opinions on the words presented, or even the times presented on the clock in Chapters 8 and 9. What is a negative representation to PI and what is positive? However, sleep is something that everyone does to varying degrees of success, the majority of people do not have insomnia, and by asking the general population for sleep related words, the question is being asked about a familiar concept.

The experimental populations recruited into these studies were screened for insomnia with the exclusion of other sleep disorders and were mostly from the student

population. Without further screening by a psychologist and overnight PSG stays with physiological assessment to provide objective measures of insomnia and general health in the experiments presented in this thesis, a recommendation for moving forward would be to consider a clinical population screened using PSG and medical assessment but also to measure performance on these tasks before and after treatment. Research has been carried out in the alcohol abuse research field and found that attention bias to alcohol cues decreases as alcohol consumption decreases and treatment is successful and long term if an attention bias is reduced (Fadardi and Cox, 2009).

One of the concerns raised in previous research (Marchetti et al, 2006) is the role of anxiety levels in attention bias in PI and the recommendation was made to ‘assess whether people suffering from insomnia in the absence of elevated anxiety show similar attention bias results..’. The three experiments included in this thesis involving PI, the 100ms clock study, the 250ms clock study and the semantic eye tracking study (Chapters 8-10) PI have higher anxiety levels than GS. However, this informs in another way that we see the general delay in reaction times of PI on the 250ms clock Posner (Chapter 9) and the semantic eye tracking (Chapter 10) which suggests that as an anxious response would be expected to be faster on valid and slower on invalid due to enhanced engagement and delayed disengagement. Differential responses over trials are only seen on the 100ms clock study (Chapter 8) even though anxiety levels of PI are higher than GS here as well.

Methodologically, evidence has been given for images to be more salient than words (Mogg et al, 2000) which lends support to the findings of Experiments 1, 2 and 3, but perhaps identifies potential improvements for Experiment 4, the semantic eye

tracking task. Moving forward, ideas for future experiments to take this work forward would include:

- Objectively screening participants using PSG to tighten classification of psychophysiological insomnia
- Develop a paradigm to use with the eye tracking which involves pictorial stimuli
- Objectifying a decrease in a bias towards sleep and establishing the relationship between this attenuation and attention retraining as seen in alcohol treatment research (Fadardi and Cox, 2009) or CBT-I.
- Considering sleepiness as a daytime consequence and including measurement of this factor in relation to reaction time performance or attention allocation on task.

11.8 Summation

At the beginning of this thesis, I outlined my aim as to attempt to further our understanding of the underlying mechanisms of selective attention to sleep in PI by:

- Establishing if selective attention to sleep is specific to psychophysiological insomnia or if it is seen in other populations who have a sleep complaint.
- Manipulating the presentation time of the stimuli presented within the same cognitive probe paradigm.
- Employing methodologies which are unique to the sleep research field which provide data over a longer time period.

Overall, our findings suggest that attention bias to sleep is specific to the chronic insomnia condition as it is not evident in an acute population. Also, manipulating the presentation time of the salient cue affects how PI will react on the relevant task in that longer presentation times tend to produce a slowing in reaction times across all trial types and that the nature of the task i.e. the categories of stimuli included, affects how the insomnia population react. The novel effect of day times further informs our knowledge of saliency relevant to PI. Finally, by using eye tracking over a longer than previously used presentation time, the difficulty that PI have in discriminating between categories and maintaining their attention becomes evident. This suggests more than a bias of attention towards sleep which has been seen previously and forms the basis for the A-I-E model (Espie et al 2006). This thesis presents evidence for an insomnia-related impairment in on-task discrimination and attention maintenance which has been linked to functional impairment of prefrontal cortex as proposed in the wider insomnia literature. These performance deficits provide further insight into the insomnia condition and open new avenues of research to develop the contemporary models of insomnia.

Appendix A. Psychology Department Online advertisement

The title of the study as displayed on the departmental website was 'People with insomnia wanted' and the study description read as follows:

'My research interests are sleep and particularly those who have difficulty getting and/or staying asleep. If you:

- * have difficulty getting to sleep or
- * have difficulty staying asleep and
- * one/both of these problems occur 3 or more times per week

please get in touch. We can provide advice on how to improve your sleep and are currently working towards developing a treatment package for those individuals who take part in our research.'

Appendix B

University of Glasgow Sleep Centre Preliminary Screening Interview

Personal

<i>Full Name:</i>	<i>Date of Birth:</i>	<i>Age:</i>
<i>Telephone:</i>	<i>Address:</i>	
<i>Alternative Telephone:</i>		
<i>When is a good time to call?</i>		
<i>What GP practice do you attend, and who is the GP you normally see?</i>		

Sleep

<i>Do you have difficulty sleeping at the moment? (Y/N)</i>	
<i>Have you always been a poor sleeper? (Y/N)</i>	
<i>How long have you had a sleep problem?(yr)</i>	
<i>Do you have difficulty falling asleep? (Y/N)</i>	
<i>How many nights per week do you have difficulty falling asleep? (out of 7)</i>	
<i>How long does it normally take you to fall asleep?(min)</i>	
<i>Do you have a difficulty with waking up during the night?(Y/N)</i>	
<i>How many nights per week do you have a difficulty with waking up during the night?(out of 7)</i>	
<i>How long are you normally awake during the night, in total? (min)</i>	
<i>What time do you normally go to bed? (time)</i>	

<i>What time do you normally get up?(time)</i>	
<i>How long do you normally sleep?(hr/min)</i>	
<i>Do you have any other difficulties with your sleep (e.g. restless legs, breathing problems, sleep walking)?</i>	
<i>Do you work shifts, night shifts?</i>	
<i>Roughly, how many units of alcohol do you drink per week? (Remember: One standard (175ml) glass of wine = 2 unit One pint of standard lager = 2.3 units Spirit & Mixer = 1 unit)</i>	
<i>Does your sleep disturbance affect how you feel and function during the day (e.g. fatigue, sleepiness, concentration, memory, mood, motivation, irritable, work/social functioning etc.). If yes, specify most salient.</i>	

Health

<i>Do you keep in good health physically? (Y/N)</i>	
<i>What physical health problems do you have (if applicable)?</i>	
<i>What medicines do you take for your physical health? (if applicable)</i>	
<i>Do you keep in good health mentally? (Y/N)</i>	
<i>What mental health problems do you have (if applicable)?</i>	
<i>What medicines do you take for your mental health? (if applicable)</i>	

Do you give your consent for us to contact your GP if necessary regarding your health?

If you are not suitable for any of the studies ongoing at the moment are you happy for your details to be kept on a database so that you may be contacted in the future should a suitable study start?

Appendix C Pittsburgh Sleep Quality Index (PSQI)

Subject's Initials ID# _____ Date Time __ PM

PITTSBURGH SLEEP QUALITY INDEX

INSTRUCTIONS:

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.

Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?

BED TIME _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

NUMBER OF MINUTES _____

3. During the past month, what time have you usually gotten up in the morning?

GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)

HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you . . .

a) Cannot get to sleep within 30 minutes

Not during the Less than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

b) Wake up in the middle of the night or early morning

Not during the Less than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

c) Have to get up to use the bathroom

Not during the Less than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

d) Cannot breathe comfortably

Not during the Less than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

e) Cough or snore loudly

Not during the Less than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

f) Feel too cold

Not during the Less than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

g) Feel too hot

Not during the Lss than Once or twice Three or more
past month_____ once a week_____ a week_____ times a week_____

h) Had bad dreams

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

i) Have pain

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

j) Other reason(s), please

describe _____

How often during the past month have you had trouble sleeping because of this?

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

6. During the past month, how would you rate your sleep quality overall?

Very good _____

Fairly good _____

Fairly bad _____

Very bad _____

7. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all _____
Only a very slight problem _____
Somewhat of a problem _____
A very big problem _____

10. Do you have a bed partner or room mate?

No bed partner or room mate _____
Partner/room mate in other room _____
Partner in same room, but not same bed _____
Partner in same bed _____

If you have a room mate or bed partner, ask him/her how often in the past month you have had . . .

a) Loud snoring

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

b) Long pauses between breaths while asleep

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

c) Legs twitching or jerking while you sleep

Not during the Less than Once or twice Three or more
past month _____ once a week _____ a week _____ times a week _____

d) Episodes of disorientation or confusion during sleep

Not during the Less than Once or twice Three or more
past month _____ once a week _____ a week _____ times a week _____

e) Other restlessness while you sleep; please

describe _____

Not during the Less than Once or twice Three or more
past month _____ once a week _____ a week _____ times a week _____

Buyse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ: Psychiatry Research, 28:193-213, 1989.
lmw:F5.PSQ (4/2002)

Appendix D Insomnia Severity Index (ISI)

1. Please rate the current (i.e., last 2 weeks) severity of your insomnia problem(s).

	None	Mild	Moderate	Severe	Very
a. Difficulty falling asleep:	0	1	2	3	4
b. Difficulty staying asleep:	0	1	2	3	4
c. Problem waking up too early:	0	1	2	3	4

2. How satisfied/dissatisfied are you with your current sleep pattern?

Very satisfied	Satisfied	Neutral	Dissatisfied	Very dissatisfied
0	1	2	3	4

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

Not at all interfering	A little	Somewhat	Much	Very much interfering
0	1	2	3	4

4. How noticeable to others do you think your sleeping problem is in terms of impairing the quality of your life?

Not at all noticeable	A little	Somewhat	Much	Very much noticeable
0	1	2	3	4

5. How worried/distressed are you about your current sleep problem?

Not at all worried	A little	Somewhat	Much	Very much worried
0	1	2	3	4

Appendix E Hospital Anxiety and Depression Scale (HADS)

The next questions are about how you feel. Read each item and tick the box next to the reply that comes closest to how you have been feeling in the past weeks. Don't take too long over your replies, your immediate reaction will probably be more accurate than a long, thought-out response.

1	I feel tense or 'wound up':	
	Most of the time	
	A lot of the time	
	From time to time, occasionally	
	Not at all	

2	I still enjoy the things I used to enjoy:	
	Definitely as much	
	Not quite as much	
	Only a little	
	Hardly at all	

3	I get a sort of frightened feeling as if something awful is about to happen:	
	Very definitely and quite badly	
	Yes, but not too badly	
	A little, but it does not worry me	
	Not at all	

4	I can laugh and see the funny side of things:	
	As much as I always could	
	Not quite so much now	
	Definitely not so much now	
	Not at all	

5	Worrying thoughts go through my mind	
	A great deal of the time	
	A lot of the time	
	From time to time, but not too often	
	Only occasionally	

6	I feel cheerful:	
	Not at all	
	Not often	
	Sometimes	
	Most of the time	

7	I can sit at ease and feel relaxed:	
	Definitely	
	Usually	
	Not often	
	Not at all	

8	I feel as if I have 'slowed down':	
	Nearly all the time	
	Very often	
	Sometimes	
	Not at all	

9	I get a sort of frightened feeling, like butterflies in my stomach:	
	Not at all	
	Occasionally	
	Quite often	
	Very often	

10	I have lost interest in my appearance:	
	Definitely	
	I don't take as much care as I should	
	I may not take quite as much care	
	I take just as much care as ever	

1	I feel restless as if I have to be on the move:		
1			
		Very much indeed	
		Quite a lot	
		Not very much	
	Not at all		

1	I look forward with enjoyment to things:		
2			
		As much as I ever did	
		Rather less than I used to	
		Definitely less than I used to	
	Hardly at all		

1	I get a sudden feeling of panic:		
3			
		Very often indeed	
		Quite often	
		Not very often	
	Not at all		

1	I can enjoy a good book or radio or TV programme:		
4			
		Often	
		Sometimes	
		Not often	
	Very Seldom		

Appendix F Daytime Functioning and Sleep Attribution Scale (DFSAS)

Name:.....

PART 1

Date:.....

Please complete Part 1 before moving on to Part 2.

Please rate each item below on how much of a problem it has been for you, in the **past two weeks**.
Circle the most appropriate response.

(1) Difficulty concentrating and focusing on things:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(2) Feeling irritable:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(3) Fatigue or tiredness:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(4) Feeling 'down in the dumps'/ low mood:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(5) Not performing at work/or study as well as you would like to:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(6) Feeling tense or anxious:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(7) Difficulty remembering things:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(8) Lack of energy and motivation:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(9) Aches and pains:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(10) Avoiding or cancelling social activities:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(11) Feeling sleepy during the day:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

(12) Lack of desire for physical intimacy or sex:

No problem at all Only a very slight problem Somewhat of a problem A very big problem

PART 2

Many things can determine the way we feel and behave. In your opinion, how much was **poor sleep responsible** for your answers in part 1, in the past two weeks. Please **circle** the most appropriate response for each item.

(1)
Not at all Only slightly Moderately To a large extent Entirely

(2)
Not at all Only slightly Moderately To a large extent Entirely

(3)
Not at all Only slightly Moderately To a large extent Entirely

(4)
Not at all Only slightly Moderately To a large extent Entirely

(5)
Not at all Only slightly Moderately To a large extent Entirely

(6)
Not at all Only slightly Moderately To a large extent Entirely

(7)
Not at all Only slightly Moderately To a large extent Entirely

(8)
Not at all Only slightly Moderately To a large extent Entirely

(9)
Not at all Only slightly Moderately To a large extent Entirely

(10)
Not at all Only slightly Moderately To a large extent Entirely

(11)
Not at all Only slightly Moderately To a large extent Entirely

(12)
Not at all Only slightly Moderately To a large extent Entirely

Appendix G Dysfunctional Beliefs and Attitudes about Sleep (DBAS)

Id: _____ initials: _____ date(dd/mm/yy): _____ Evaluation period : _____
14/01/2008

DYSFUNCTIONAL BELIEFS AND ATTITUDES ABOUT SLEEP

Statements reflecting people's beliefs and attitudes about sleep are listed below. Please

indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement circle the number according to your PERSONAL rating falls.

Example: If I sleep too much, I don't perform as well the next day

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE AGREE

1. I need 8 hours of sleep to feel refreshed and function well during the day.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

2. When I don't get proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

3. I am concerned that chronic insomnia may have serious consequences on my physical health.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

4. I am worried that I may lose control over my abilities to sleep.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

5. After a poor night's sleep, I know that it will interfere with my daily activities on the next day.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

6. In order to be alert and function well during the day, I believe I would be better off taking a sleeping pill rather than having a poor night's sleep.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

7. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

8. When I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

9. Without an adequate night's sleep, I can hardly function the next day.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

10. I can't ever predict whether I'll have a good or poor night's sleep.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

11. I have little ability to manage the negative consequences of disturbed sleep.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

12. When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

13. I believe insomnia is essentially the result of a chemical imbalance.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

14. I feel insomnia is ruining my ability to enjoy life and prevents me from doing what I want.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

15. Medication is probably the only solution to sleeplessness.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

16. I avoid or cancel obligations (social, family) after a poor night's sleep.

0 1 2 3 4 5 6 7 8 9 10
STRONGLY STRONGLY
DISAGREE

Appendix H Sleep Preoccupation Scale

Sleep Preoccupation Scale

Appendix

Participant ID:

Date: *week 1*

This questionnaire is designed to find out how often you think about your sleep pattern throughout the day and the kinds of thoughts that you have. Read each statement and circle the answer which best represents your feelings.

	Never	Hardly Ever	Very Infrequently	Sometimes	Quite A Lot	Almost All the Time	Always
I feel anxious about my sleep pattern	1	2	3	4	5	6	7
I feel anxious about what will happen when I try to sleep tonight	1	2	3	4	5	6	7
I feel drained because I did not sleep well last night	1	2	3	4	5	6	7
I take naps in the daytime	1	2	3	4	5	6	7
I get upset when people tell me I look tired	1	2	3	4	5	6	7
I try to get to bed early the next day after a bad night's sleep	1	2	3	4	5	6	7
I find it hard to concentrate during the day after a bad night's sleep	1	2	3	4	5	6	7
I will drink coffee throughout the day to try to keep myself awake	1	2	3	4	5	6	7
My memory appears to be worse after a bad night's sleep	1	2	3	4	5	6	7
I am more sensitive to what other people say after a bad night's sleep	1	2	3	4	5	6	7
I have to make more of an effort with my appearance after a bad night's sleep	1	2	3	4	5	6	7
I am more irritable after a bad night's sleep	1	2	3	4	5	6	7
I become frustrated when I think about my sleep pattern	1	2	3	4	5	6	7
I get upset when others talk about their 'good' sleep patterns	1	2	3	4	5	6	7
I have a lie-in after a bad night's sleep	1	2	3	4	5	6	7
I look for physical symptoms of a bad night's sleep throughout the day	1	2	3	4	5	6	7

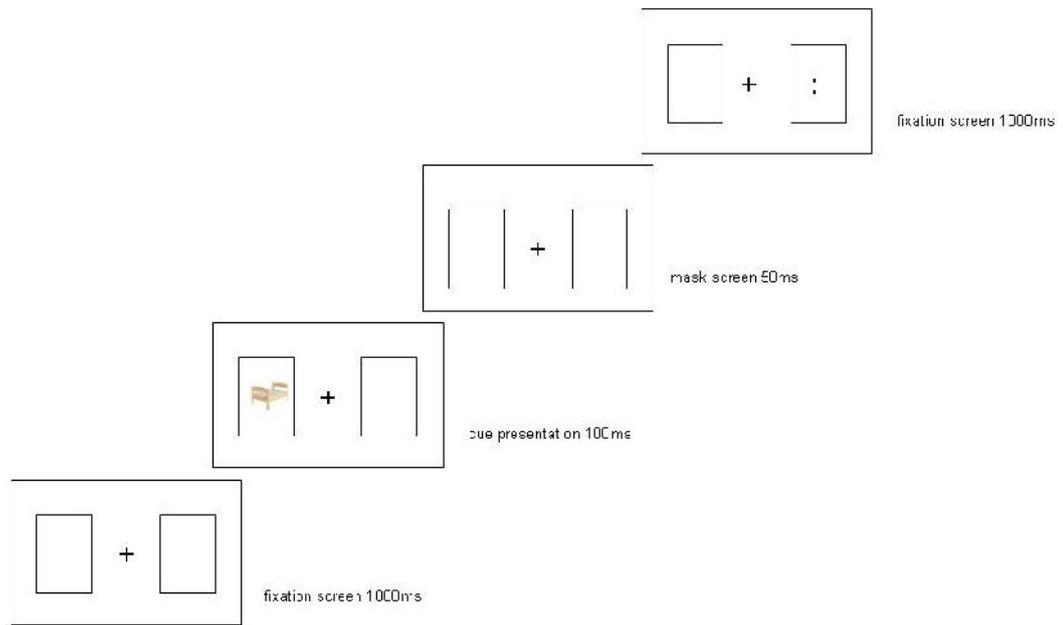
I know that if I have a bad night's sleep, I will also have a bad day	1	2	3	4	5	6	7
If I sleep badly I feel tense all day	1	2	3	4	5	6	7

Never Hardly Ever Very Infrequently Some-times Quite A Lot Almost All the Time Always

I worry about the long-term consequences of poor sleep	1	2	3	4	5	6	7
I cannot perform my daily tasks as well when I have had a bad night's sleep	1	2	3	4	5	6	7
I think of what 'good' sleep would be like	1	2	3	4	5	6	7
I feel as though I have lost control over my sleep	1	2	3	4	5	6	7
I take it easy the next day after a bad night	1	2	3	4	5	6	7
All my problems seem worse after a bad night's sleep	1	2	3	4	5	6	7
I cannot stop dwelling on thoughts of sleep during the day	1	2	3	4	5	6	7
I wonder if my sleep patterns will ever become 'normal'	1	2	3	4	5	6	7
I blame others for my sleep problems	1	2	3	4	5	6	7
I try to avoid other people when I have had a bad night's sleep	1	2	3	4	5	6	7
I get a dry feeling in my mouth when I think about sleep	1	2	3	4	5	6	7
I get angry at myself after a bad night's sleep	1	2	3	4	5	6	7
I yawn more often after a bad night's sleep	1	2	3	4	5	6	7
My eyes are more sensitive / sore after a bad night's sleep.	1	2	3	4	5	6	7

If there are any other thoughts, feelings or images you have throughout the day, due to a bad night's sleep, please list them below and how often you have them. Thank you.

Appendix I Example of Acute insomnia Posner trial



Appendix J Posner instruction screen

In this experiment you will see single pictures presented on the computer screen.

Firstly, you will see a cross (+) in the centre of the screen, flanked by two empty boxes. Following this, one picture will appear on the screen, in one of the boxes.

The picture will then disappear, and you will see either a colon (:) or a 2 horizontal dots (..) appear in one of the boxes. Press the **C** key if the target is a (:) and the **M** key if the target is a (..)

Try and react as quickly as you can, but also try and be as accurate as you can. If you press the wrong key, just press the right one as quickly as possible.

The experiment will start with some practice trials. If you have any questions ask the experimenter.

PRESS ANY KEY TO START THE PRACTICE TRIALS

Appendix K Acute insomnia Posner mean reaction time and effect size data

	Mean Acute	GS	SD Acute	GS
Valid sleep	874.5	888.9	44.8	66.1
Invalid sleep	879.5	896.7	47.2	68.3
Valid neutral	873.1	882.7	40.9	60.7
Invalid neutral	882.3	890.4	45.2	77.9

Mean and SD values (ms) for individuals with acute insomnia and GS by trial type.

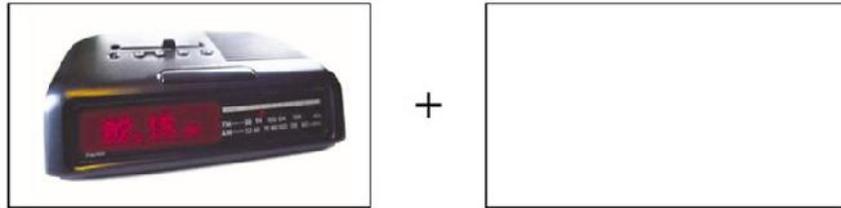
Trial type	GS	Acute insomnia
Valid sleep vs valid neutral	0.05	0.09
Invalid sleep vs invalid neutral	0.2	0.1
Valid neutral vs invalid neutral	0.2	0.12
Valid sleep vs invalid sleep	0.3	0.19

Effect sizes within the GS and acute insomnia groups comparing trial types.

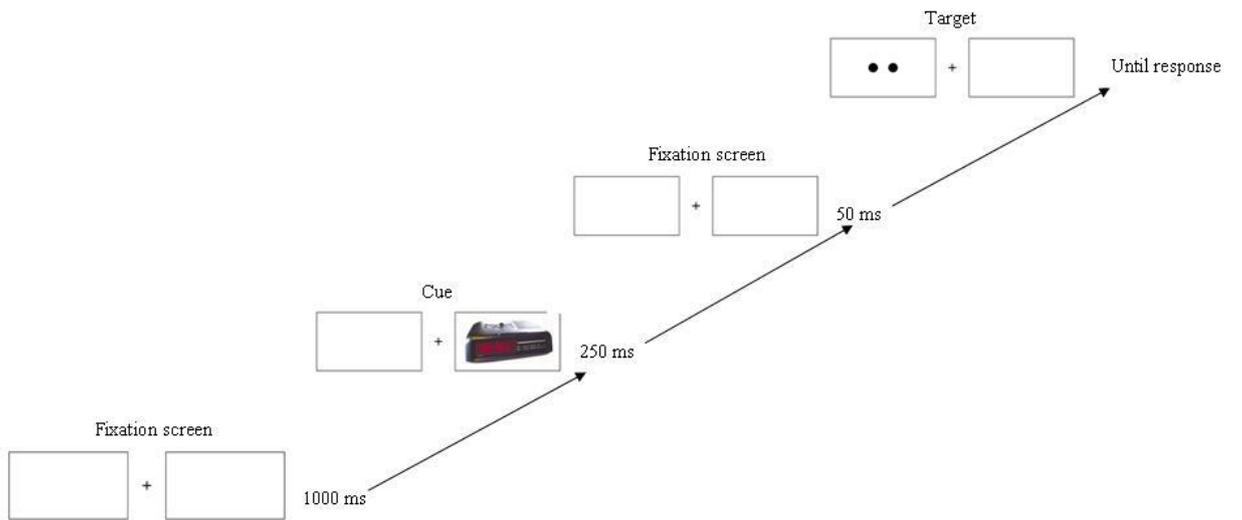
Trial type	Acute insomnia v GS
Invalid neutral	0.14
Invalid sleep	0.32
Valid neutral	0.2
Valid sleep	0.27

Effect sizes between the acute insomnia group and GS by trial type. The largest between group difference is on invalid trials presenting a sleep related cue and the smallest effect is seen on invalid trials presenting a neutral cue, however, all these effect sizes are small.

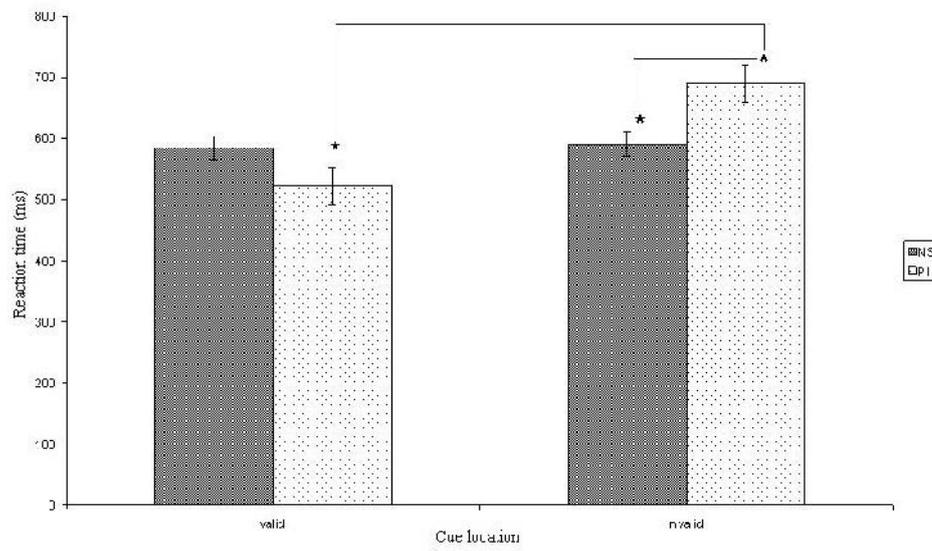
Appendix L Clock experiments stimuli



Appendix M Example of invalid Posner trial



Appendix N Graphical representation of findings of Woods et al (2009)



Significant differences were found between valid and invalid trials for PI and also between GS and PI on invalid trials. There was a non-significant trend towards a faster response by PI than GS on valid trials.

Appendix O 100ms clock mean reaction time and effect size data

	Mean PI	GS	SD PI	GS
Valid day	628.6	601.3	116.7	121.9
Valid sleep	578.0	574.8	105.5	109.6
Invalid day	617.2	600.9	97.3	103.3
Invalid sleep	577.8	581.0	102.3	116.4

Mean and SD values (ms) by trial type for PI and GS.

Cue location/time shown on clock	GS	PI
Invalid day vs invalid night	0.04	0.11
Valid day vs valid night	0.06	0.04
Valid night vs invalid night	0.41	0.5
Valid day vs invalid day	0.42	0.43

Within group effect sizes comparing trial types.

Appendix P 250ms clock mean reaction time and effect size data

	Mean PI	GS	SD PI	GS
Valid day	665.3	568.3	94.1	60.8
Valid sleep	618.8	532.1	98.8	56.7
Invalid day	655.7	568.9	90.4	68.0
Invalid sleep	625.0	524.0	92.5	73.0

Mean and SD values (ms) by trial type for PI and GS.

Cue location/time shown on clock	GS	PI
Invalid day vs invalid sleep	0.03	0.06
Valid day vs valid sleep	0.06	0.01
Valid sleep vs invalid sleep	0.03	0.1
Valid day vs invalid day	0.0	0.05

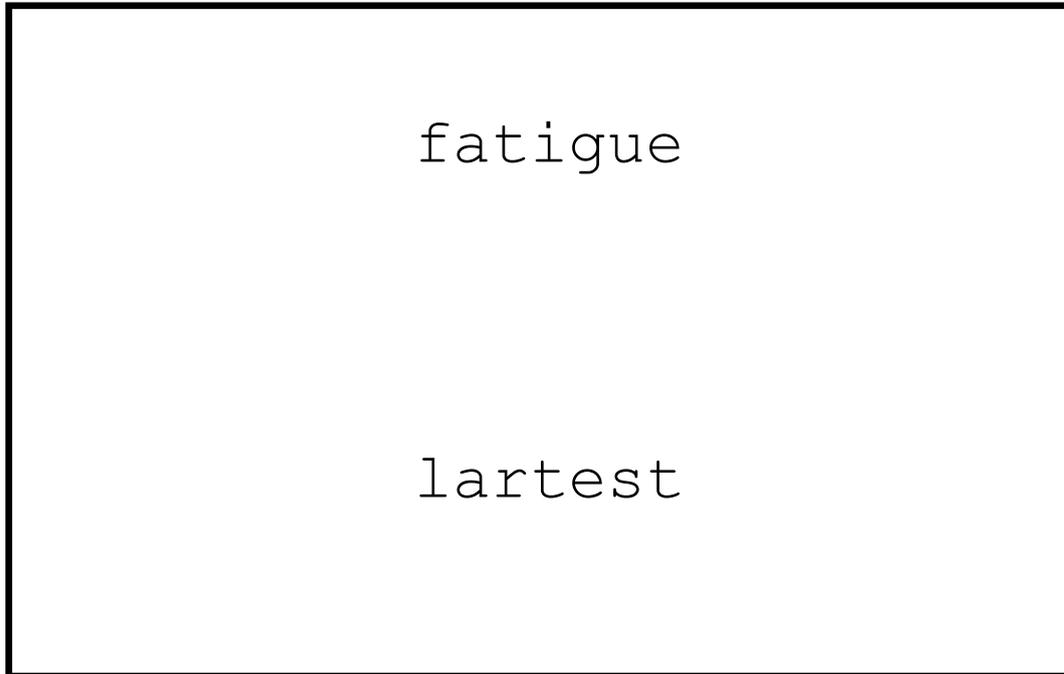
Within group effect sizes comparing trial types

Appendix Q Participant wearing eye tracker.



Appendix R

Example of stimulus presented during semantic eye tracking experiment



Appendix S Negative sleep-related, positive sleep-related and neutral target words used as stimuli in semantic eye tracking experiment with matched pseudo words.

Positive target	Pseudoword	Negative target	Pseudoword	Neutral target	Pseudoword
asleep	comset	alarm	korst	actress	mestolt
bed	ast	alert	poxer	address	lortest
bedtime	smaster	anxious	quaster	ball	lerc
comfy	hante	arousal	moosern	bath	reat
pillow	faltem	awake	fanat	bottle	shosta
dark	god	exhausted	rastlests	chord	blost
dream	stast	insomnia	estlasts	club	dost
duvet	rasts	restless	condeser	cream	shaeb
mattress	loasered	shattered	demgrased	cupboard	mearsten
naps	larp	deprived	horganed	drafted	leserst
night	blose	disturbed	bassiered	drawing	fantant
pyjamas	reasted	drained	portust	featuring	partested
quiet	wear	drowsy	loaske	grass	brosk
refreshed	goarstier	fatigue	lartest	intellect	tanabrast
relaxed	hastolt	groggy	morqed	nation	destan
rest	parn	irritable	moornadle	panorama	bartolst
rested	lotern	lethargy	entabish	pear	shos
sheets	postak	nightmare	kortenelm	pencil	sarten
siesta	goteim	nocturnal	hoarstend	playful	lackars
silence	narstel	overactive	geanstrand	sandwich	tistelid
sleepy	troste	sedative	bovaster	Saturday	lordalds
snooze	larsem	snoring	wear	sculpture	gimployed
snug	nase	tired	haint	set	mep
snuggle	treaser	tossing	hearten	signal	goadem
cosy	shan	unrefreshed	dresharened	study	larte
energy	gaster	wakeful	searpen	suit	narm

Appendix T Semantic eye tracking mean reaction time and effect size data

Eye tracking

1. Vigilance

Sleep group	Target word position	Word saliency	Mean	SD
GS	Below distracter word	Negative sleep	260.9	337.7
		neutral	252.9	311.9
		Positive sleep	251.1	311.5
PI		Negative sleep	267.9	339.7
		Neutral	265.0	331.4
		Positive sleep	260.6	308.5
GS	Above distracter word	Negative sleep	231.3	385.9
		Neutral	213.1	339.7
		Positive sleep	219.9	351.4
PI		Negative sleep	253.8	375.6
		Neutral	259.7	375.5
		Positive sleep	251.3	387.0

Mean and SD onset of first fixation times (ms) for GS and PI when the target word is situated below and above the distracter word by word salience.

Sleep group	Parameter	Word saliency	Mean	SD
GS	Location (ms)	Negative sleep	593.2	174.1
		Neutral	598.3	121.0
		Positive sleep	567.9	103.1
PI	Location (ms)	Negative sleep	693.3	194.8
		Neutral	700.8	170.8
		Positive sleep	674.6	176.6
GS	Amplitude (probability)	Negative sleep	0.35	0.04
		neutral	0.34	0.04
		Positive sleep	0.34	0.04
PI	Amplitude (probability)	Negative sleep	0.36	0.05
		Neutral	0.37	0.05
		Positive sleep	0.36	0.04

Mean and SD values for location of peak and amplitude parameters by word saliency.

2. Maintenance

Sleep group	Target word position	Word saliency	Mean	SD
GS	Below distracter word	Negative sleep	2094.0	708.0
		neutral	2078.9	722.8
		Positive sleep	2102.1	713.7
PI		Negative sleep	1968.4	778.1
		Neutral	1968.4	794.5
		Positive sleep	1982.0	795.7
GS	Above distracter word	Negative sleep	1759.7	1110.3
		Neutral	1647.9	1135.2
		Positive sleep	1719.8	1130.6
PI		Negative sleep	1356.4	1077.1
		Neutral	1109.3	1002.2
		Positive sleep	1310.5	1089.7

Mean and SD values (ms) for duration of first fixation when target word is positioned below and above the distracter by word saliency.

Sleep group	Parameter	Word saliency	Mean	SD
GS	Home in time	Negative sleep	1626.7	454.1
		Neutral	1632.8	481.2
		Positive sleep	1585.9	393.0
PI		Negative sleep	2038.8	1051.4
		Neutral	2077.1	920.1
		Positive sleep	1815.6	567.5

Mean and SD values (ms) for time taken to home into the target word for GS and PI by word saliency.

References

Adam, K., Tomeny, M. and Oswald, I. (1986). Physiological and Psychological Differences Between Good and Poor Sleepers. *Journal of Psychiatry*, 20, 301-316.

Altena, E., Van Der Werf, Y.D., Strijers, R.L.M. & Van Someren, E.J.W. (2008a). Sleep loss affects vigilance: effects of chronic insomnia and sleep therapy. *Journal of Sleep Research*, 17, 335-343.

Altena, E., Van Der Werf, Y.D., Sanz-Arigitia, E.J., Voorn, T.A., Rombouts, S.A., Kuijper, J.P. & Van Someren, E.J. (2008b). Prefrontal hypoactivation and recovery in insomnia. *Sleep*, 31, 1271-76.

Altena, E., Vrenken, H., Van Der Werf, Y.D., Van Den Heuvel, O.A. & Van Someren, E.J.W. (2010). Reduced orbitofrontal and parietal gray matter in chronic insomnia: a voxel-based morphometric study. *Biological Psychiatry*, 67, 182-185.

American Academy of Sleep Medicine. *ICSD-2 – International classification of sleep disorders: Diagnostic and coding manual*. (2nd edition). American Academy of Sleep Medicine, Westchester IL, 2005.

American Psychiatric Association. *DSM-IV: Diagnostic and Statistical Manual of Mental Disorders*. (4th edition). American Psychiatric Press, Washington DC, 1994

American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Fourth edition, Text revisions*. American Psychiatric Association, Washington DC, 2000.

Ancoli-Israel, S. & Roth, T. (1999). Characteristics of insomnia in the United States: results of the 1991 National Sleep Foundation Survey.I. *Sleep*, 22, S347-353.

Ancoli-Israel, S., Richardson, G.S., Mangano, R.M., Jenkins, L., Hall, P. & Jones, W.S. (2005). Long-term use of sedative hypnotics in older patients with insomnia. *Sleep Medicine*, 6, 107-113.

Ansfield, M.E., Wegner, D.M. and Bowser, R. (1996). Ironic Effects of Sleep Urgency, *Behaviour Research and Therapy*, 37(7), 523-531.

Ascher, L.M. and Turner, R.M. (1979). Paradoxical Intention and Insomnia: An Experimental Investigation. *Behaviour Research and Therapy*, 17, 408-411.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D. & Hohagen, F. (2002). Test–retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of Psychosomatic Research*, 53, 737-740.

Backhaus, J., Junghanns, K., Born, J., Hohaus, K., Faasch, F. & Hohagen, F. (2006). Impaired declarative memory consolidation during sleep in patients with primary insomnia: influence of sleep architecture and nocturnal cortisol release. *Biological Psychiatry*, 60, 1324-30.

Bastien, C.H., Vallières, A. & Morin, C.M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine*, 2, 297-307.

Bastien, C.H., Fortier-Brochu, E., Rioux, I., LeBlanc, M., Daley, M. & Morin, C.M. (2003). Cognitive performance and sleep quality in the elderly suffering from chronic insomnia. Relationship between objective and subjective measures. *Journal of Psychosomatic Research*, 54, 39-49.

Bastien, C.H., Vallieres, A. & Morin, C.M. (2004a). Precipitating factors of insomnia. *Behavioral Sleep Medicine*, 2, 50-62.

Bastien, C.H., St-Jean, G., Morin, C.M., Turcotte, I. & Carrier, J. (2008). Chronic psychophysiological insomnia: hyperarousal and/or inhibition deficits? An ERPs investigation. *Sleep*, 31, 887-898.

Beaulieu-Bonneau, S., LeBlanc, M., Merette, C., Dauviliers, Y. & Morin, C.M. (2007a). Family history of insomnia in a population-based sample. *Sleep*, 30, 1739-45.

Beaulieu-Bonneau, S., Fortier-Brochu, E., Ivers, H. & Morin, C. (2007b). Daytime consequences of insomnia: A factor analysis. *Sleep*, (Abstract Suppl.), 30, A230.

Bouchard, S., Bastien, C. & Morin, C.M. (2003). Self-efficacy and adherence to cognitive-behavioral treatment of insomnia. *Behavioral Sleep Medicine*, 1, 187-199.

Bonnet, M.H. & Arand, D.L. (1997). Hyperarousal and insomnia. *Sleep Medicine Reviews*, 1, 97-108.

- Bonnet, M. & Arand, D.L. (2007). Cardiovascular implications of poor sleep. *Sleep Medicine Clinics*, 2, 529-538.
- Bonnet, M.H. & Arand, D.L. (2010). Hyperarousal and insomnia: State of the science. *Sleep Medicine Reviews*, 14, 9-15.
- Bootzin, R.R. (1972). Stimulus control treatment for insomnia. Proceedings of the 80th American Psychological Society Annual Convention, 395-6.
- Bradley, B.P., Mogg, K. and Millar, N.H. (2000). Covert and Overt Orienting of Attention to Emotional Faces in Anxiety. *Cognition and Emotion*, 14(6), 789-808.
- Braun, V. & Clarke, V. (2006). Using thematic analysis in psychology. *Qualitative Research in Psychology*, 3, 77-101.
- Broman, J.E. and Hetta, J. (1994) Perceived Pre-Sleep Arousal in Patients with Persistent Physiological and Psychological Insomnia. *Journal of Psychiatry*, 48, 203-207.
- Broomfield, N.M. & Espie, C.A. (2005). Towards a valid, reliable measure of sleep effort. *Journal of Sleep Research*, 14, 401-407.
- Burgos, I., Richter, L., Klein, T., Fiebich, B., Feige, B., Lieb, K., Voderholzer, U. & Riemann, D. (2006). Increased nocturnal interleukin-6 excretion in patients with primary insomnia: A pilot study. *Brain, Behavior, and Immunity*, 20, 246-253.

Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R. & Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, 28, 193–213.

Buysse, D.J., Ancoli-Israel, S., Edinger, J.D., Lichstein, K.L. & Morin, C.M. (2006). Recommendations for a Standard Research Assessment of Insomnia. *Sleep*, 29, 1155-73.

Buysse, D.J., Thompson, W., Scott, J., Franzen, P.L., Germain, A, Hall, M., Moul, D.E., Nofzinger, E.A. & Kupfer, D. (2007). Daytime symptoms of primary insomnia: A prospective analysis using ecological momentary assessment. *Sleep Medicine*, 8, 198-208.

Carskadon, M.A., Dement, W.C., Mitler, M.M., Guilleminault, C., Zarcone, V.P. & Spiegel, R. (1976). Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *American Journal of Psychiatry*, 133, 1382-8.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Daley, M., Morin, C.M., LeBlanc, M., Gregoire, J.P. & Savard, J. (2009a). The economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep*, 32, 55-64.

Daley, M., Morin, C.M., LeBlanc, M., Grégoire, J.P., Savard, J. & Baillargeon, L. (2009b). Insomnia and its relationship to health-care utilization, work absenteeism, productivity and accidents. *Sleep Medicine*, 4, 427-438.

Davidson, J.R., MacLean, A.W., Brundage, M.D. and Schulze, K. (2002). Sleep Disturbance in Cancer Patients. *Social Science and Medicine*, 54, 1309-1321.

Drummond, S.P., Brown, G.G., Salamat, J.S. & Gillin, J.C. (2004). Increasing task difficulty facilitates the cerebral compensatory response to total sleep deprivation. *Sleep*, 27, 445– 451.

Edinger, J.D. & Sampson, W.S. (2003). A primary care “friendly” cognitive behavioural therapy. *Sleep*, 26, 177-182.

Edinger J.D., Bonnet M.H., Bootzin R.R., Doghramji, K., Dorsey, C.M., Espie, C.A., Jamieson, A.O., McCall, W.V., Morin, C.M. & Stepanski, E.J. (2004). Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine workgroup. *Sleep*, 27, 1567-1596.

Edinger, J.D. & Means, M.K. (2005). Cognitive-behavioral therapy for primary insomnia. *Clinical Psychology Review*, 25, 539-558.

Edinger, J.D., Means, M.K., Carney, C.E. & Krystal, A.D. (2008a). Psychomotor performance deficits and their relation to prior nights' sleep among individuals with primary insomnia. *Sleep*, 31, 599-607.

Edinger, J.D., Carney, C.E. & Wohlgemuth, W.K. (2008b). Pretherapy cognitive dispositions and treatment outcome in cognitive behaviour therapy for insomnia. *Behaviour therapy*, 39, 406-416.

Edinger, J.D., Olsen, M.K., Stechuchak, K.M., Means, K., Lineberger, M.D., Kirby, A. & Carney, C.E. (2009). Cognitive behavioral therapy for patients with primary insomnia or insomnia associated predominantly with mixed psychiatric disorders: a randomized clinical trial. *Sleep*, 32, 499-510.

Engeland, A., Skurtveit, S. & Morland, J. (2007). Risk of road traffic accidents associated with the prescription of drugs: a registry-based cohort study. *Annals of Epidemiology*, 17, 597-602.

Espie, C.A., Lindsay, W.R., Brooks, N., Hood, E.M. & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy*, 27, 79-88.

Espie C.A. (1991). *The psychological treatment of insomnia*. J. Wiley and Sons Ltd., Chichester, England.

Espie, C.A. (2002). Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Annual Review of Psychology*, 52, 215-243.

Espie, C.A, Broomfield, N.M., MacMahon, K.M., Macphee, L.M. & Taylor, L.M. (2006). The attention-intention-effort pathway in the development of psychophysiological insomnia: a theoretical review. *Sleep Medicine Reviews*, 10, 215-245.

Espie, C.A. (2007). Understanding insomnia through cognitive modelling. *Sleep Medicine*, 8, S3-8.

Espie, C.A., MacMahon, K.M.A, Kelly, H., Broomfield, N.M., Douglas, N.J. Engleman, H.M., McKinstry, B., Morin, C.M., Walker, A. & Wilson, P. (2007). Randomized Clinical effectiveness trial of nurse-administered small-group cognitive behaviour therapy for persistent insomnia in General practice. *Sleep*, 30, 574-584.

Espie, C.A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L.M., White, C.A., Douglas, N.J., Engleman, H.M., Kelly, H.L. & Paul, J. (2008). Randomized controlled clinical effectiveness trial of cognitive behaviour therapy compared with treatment as usual for persistent insomnia in patients with cancer. *Journal of Clinical Oncology*, 26, 4651-58.

Espie, C.A. & Kyle, S.D. (2008). Towards an Improved Neuropsychology of Poor Sleep?: commentary on Edinger et al.: Psychomotor performance deficits and their relation to prior nights' sleep among individuals with primary insomnia. *Sleep*, 31, 591-592.

Espie, C.A. (2008). Treating insomnia with CBT: should we step up to stepped care? Keynote lecture delivered at the 22nd annual meeting of the Association for Professional Sleep Societies (APSS), June 9-12, Baltimore, USA.

Espie, C.A. & Kyle, S.D. (2009). Primary insomnia: an overview of practical management using cognitive behavioural techniques. *Sleep Medicine Clinics*, 4, 559-569.

Espie, C.A. (2009). 'Stepped care': a health technology solution for delivering cognitive behavioral therapy as a first line insomnia treatment. *Sleep*, 32, 1549-1558.

Evans, F.J. (1977). Subjective Characteristics of Sleep Efficiency. *Abnormal Psychology*, 86, 561-564.

Ford, D.E. & Kamerow, D.B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *Journal of the American Medical Association*, 262, 1479-1484.

Fox, E., Russo, R. and Dutton, K. (2002) Attentional bias for threat: Evidence for delayed disengagement from emotional faces. *Cognition and Emotion*, 16(3), 355-379.

Greene, G. (2008). *Insomniac*. University of California Press.

Harvey, A.G. (2001). Insomnia: Symptom or Diagnosis? *Clinical Psychology Review*, 21(7), 1037-1059.

Harvey, A.G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, 40, 869-893.

Harvey, L., Inglis, S.J. & Espie, C.A. (2002). Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. *Behaviour Research and Therapy*, 40, 75-83.

Harvey, A.G., Tang, N.K.Y. & Browning, L. (2005). Cognitive approaches to insomnia. *Clinical Psychology Review*, 25, 593-611.

Harvey, A.G., Sharpley, A.L., Ree, M.J., Stinson, K. & Clark, D.M. (2007). An open trial of cognitive therapy for chronic insomnia. *Behaviour Research and Therapy*, 45, 2491-2501.

Jones, B.T., Macphee, L.M., Broomfield, N.M., Jones, B.C. and Espie, C.A. (2005). Sleep Related Attentional Bias in Good, Moderate and Poor (Primary Insomnia) Sleepers. *Journal of Abnormal Psychology*, Vol. 114, No. 2, 249-258.

Kyle, S.D, Espie, C.A. & Morgan, K. (2008). The impact of insomnia on occupational functioning: a 'crystallisation' of qualitative methods. *Journal of Sleep Research*, 17 (Abstract Suppl.), 304.

Kyle, S.D., Espie, C.A., Morgan, K. & Fleming L. (2009). The Glasgow Sleep Impact Index: A Patient Generated Measure for Capturing Sleep-Related Quality of Life. *Sleep*, 32 (Abstract Suppl.), 0850.

Kyle, S.D., Espie, C.A. & Morgan, K. '*...Not just a minor thing, it is something major, which stops you from functioning daily*': Quality of Life and Daytime Functioning in Insomnia. *Behavioral Sleep Medicine*, 8, 3, 123-140.

Kyle, S.D., Morgan, K. & Espie, C.A. (2010). Insomnia and health-related quality of life. *Sleep Medicine Reviews*, 14, 69-82.

Lichstein K.L., Means, M.K., Noe, S.L. & Aguillard, R.N. (1997). Fatigue and sleep disorders. *Behaviour Research and Therapy*, 35, 733-40.

Lichstein, K.L., Durrence, H.H., Riedel, B.W. & Bayen, U.J. (2001a). Primary versus secondary insomnia in older adults: subjective sleep and daytime functioning. *Psychology and Aging*, 16, 264-71.

Lichstein, K.L., Riedel, B.W., Wilson, N.M., Lester, K.W. & Aguillard, R.N. (2001b). Relaxation and sleep compression for late-life insomnia: a placebo controlled trial. *Journal of Consulting and Clinical Psychology*, 69, 227-239.

Lichstein, K.L., Durrence, H.H., Taylor, D.J., Bush, A.J. & Riedel, B.W. (2003). Quantitative criteria for insomnia. *Behaviour Research and Therapy*, 41, 427-445.

Lichstein, K.L. (2006). Secondary insomnia: a myth dismissed. *Sleep Medicine Reviews*, 10, 3-5.

MacMahon, K., Broomfield, N., Macphee, L. and Espie, C.A. (2006), Attention Bias for Sleep Related Stimuli in Primary Insomnia and Delayed Sleep Phase Syndrome using the Dot-Probe Task. *SLEEP*, 29,11.

Marchetti, L.M., Biello, S.M., Broomfield, N.M., MacMahon, K.M.A. and Espie, C.A. (2006), Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. *Journal of Sleep Research*, 15, 212-221.

Means, M., Lichstein, K.L., Epperson, M.T. & Johnson, C.T. (2000). Relaxation therapy for insomnia: nighttime and daytime effects. *Behaviour Research and Therapy*, 38, 665-678.

Merica, H., Blois, R. & Gaillard, J.M. (1998). Spectral characteristics of sleep EEG in chronic insomnia. *European Journal of Neuroscience*, 10, 1826-34.

Mogg, K., McNamara, J., Powys, M., Rawlinson, H., Seiffer, A. and Bradley, B.P. (2000) Selective Attention to Threat: A test of 2 cognitive models of anxiety. *Cognition and Emotion*, 14(3), 375-399.

Morin, C.M., Colecchi, C., Stone, J., Sood, R. & Brink, D. (1999a). Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. *Journal of the American Medical Association*, 281, 991-999.

Morin, C.M., Hauri, P.J., Espie, C.A., Spielman, A.J., Buysse, D.J. & Bootzin, R.R. (1999b). Nonpharmacological treatment of chronic insomnia. An American Academy of Sleep Medicine Review. *Sleep*, 15, 1134-56.

Morin, C.M. & Espie, C.A. (2003). *Insomnia: A Clinical Guide to Assessment and Treatment*. Kluwer Academic/ Plenum Publishers.

Morin, C.M., Rodrigue, S. & Ivers, H. (2003). Role of stress, arousal, and coping skills in primary insomnia. *Psychosomatic Medicine*, 65, 259-267.

Morin, C.M. (2003). Measuring outcomes in randomized clinical trials of insomnia treatments. *Sleep Medicine Reviews*, 7, 263-79.

Morin, C.M. (2004). Insomnia treatment: taking a broader perspective on efficacy and cost-effectiveness issues. *Sleep Medicine Reviews*, 8, 3-6.

Morin, C.M., LeBlanc, M., Daley, J.P. & Merette, C. (2006a). Epidemiology of insomnia: Prevalence, self-help treatments, consultations, and determinants of helpseeking behaviors. *Sleep Medicine*, 7, 123-130.

Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A. & Lichstein, K.L. (2006b). Psychological and behavioural treatment of insomnia update of the recent evidence (1998-2004). *Sleep*, 29, 1398-414.

Morin, C.M., Vallieres, A. & Ivers, H. (2007). Dysfunctional beliefs and attitudes about sleep (DBAS): validation of a brief version (DBAS-16). *Sleep*, 30, 1547-54.

Morin, C.M., Belanger, L., LeBlanc, M., Ivers, H., Savard, J., Espie, C.A., Merette, C., Baillargeon, L. & Gregoire, J.P. (2009). The natural history of insomnia: a populationbased 3-year longitudinal study. *Archives of Internal Medicine*, 169, 447-453.

Ohayon, M.M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews*, 6, 97-111.

Ohayon, M.M. & Reynolds, C.F. III. (2009). Epidemiological and clinical relevance of insomnia diagnosis algorithms according to the DSM-IV and the International Classification of Sleep Disorders (ICSD). *Sleep Medicine*, 10, 952-960.

Orff, H.J., Drummond, S.P., Nowakowski, S. & Perlis, M.L. (2007). Discrepancy between subjective symptomatology and objective neuropsychological performance in insomnia. *Sleep*, 30, 1205-11.

Orff, H.J., Almklov, E., Olandj, C. & Drummond, S. (2009). Insomnia patient's show increased cerebral activation when compared to good sleepers during an back working memory task. *Sleep*, 32 (Abstract Suppl.), 0779.

Perlis, M.L., Giles, D.E., Mendelson, W.B., Bootzin, R.R. & Wyatt, J.K. (1997). Psychophysiological insomnia: the behavioral model and a neurocognitive perspective. *Journal of Sleep Research*, 6, 179-188.

Perlis, M.L., Kehr, E.L., Smith, M.T., Andrews, P.J., Orff, H., & Giles, D.E. (2001a). Temporal and stagewise distribution of high frequency EEG activity in patients with primary and secondary insomnia and good sleepers controls. *Journal Sleep Research*, 10, 93-104.

Perlis, M.L., Smith, M.T., Andrews, P.J., Orff, H. & Giles, D.E. (2001b). Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep*, 24, 110-117.

Perlis, M.L., Sharpe, M., Smith, M.T., Greenblatt, D. & Giles, D. (2001c). Behavioural treatment of insomnia: treatment outcome and the relevance of medical and psychiatric morbidity. *Journal of Behavioural Medicine*, 24, 281-296.

Posner, M.I. (1980), Orienting of attention. *Quarterly Journal of Experimental Psychology*, 32A, 3-25.

Posner, M.I. and Cohen, Y. (1984), Components of visual orienting, in H. Bouma and D. Bowhuis (Eds.), *Attention and Performance X*, 531-556. Hove, UK: Lawrence Erlbaum Associates Ltd.

Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M. & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews*, 14, 19-31.

Roth, T. & Ancoli-Israel, S. (1999). Daytime consequences and correlates of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey. II. *Sleep*, 22(Suppl. 2), S354-S358.

Roth, T. (2007). Insomnia: definition, prevalence, etiology, and consequence. *Journal of Clinical Sleep Medicine*, 15, S7-10.

Semler, C.N. & Harvey, A.G. (2006). Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? *Behavioral Sleep Medicine*, 4, 85-103.

Semler, C.N. and Harvey, A.G. (2004). An Investigation of Monitoring for Sleep Related Threat in Primary Insomnia. *Behaviour Research and Therapy*, 42, 1403-1420.

Shekleton, J.A., Rogers, N.L. & Rajaratnam, S.M.W. (2010). Searching for the daytime impairments of primary insomnia. *Sleep Medicine Reviews*, 14, 47-60.

Spiegelhalder, K., Kyle, S.D., Feige, B., Nissen, C., Espie, C.A. & Riemann, D. (2010). The impact of sleep-related attentional bias on polysomnographically measured sleep in primary insomnia. *Sleep*, 33, 107-112.

Spiegelhalder, K., Espie, C.A., Nissen, C. and Riemann, D. (2008) Sleep-related attentional bias in patients with primary insomnia compared with sleep experts and healthy controls. *Journal of Sleep Research*, 17, 191-196.

Spielman, A., Caruso, L. & Glovinsky, P. (1987a). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America*, 10, 541-53.

Spielman, A.J., Saskin, P. & Thorpy, M.J. (1987b). Treatment of chronic insomnia by restriction of time in bed. *Sleep*, 10, 45-56.

Tang, N.K.Y and Harvey, A.G. (2004). Effects of cognitive arousal and physiological arousal on sleep perception. *SLEEP*, 27 (1), 69-78.

Tang, N.K.Y., Schmidt, D.A. and Harvey, A.G. (2007). Sleeping with the Enemy: Clock Monitoring in the Maintenance of Insomnia. *Journal of Behavior Therapy and Experimental Psychiatry*, 38, 40-55.

Taylor, L., Espie, C.A. and White, C.A. (2003). Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behavioural Sleep Medicine*, 1(4), 200-212.

Tyron, W.W. (2004). Issues of validity in actigraphic sleep assessment. *SLEEP*, 27, 158-165.

Yiend, J. and Mathews, A. (2001). Anxiety and Attention to Threatening Pictures. *The Quarterly Journal of Experimental Psychology*, 54A(3), 665-681.

Wicklow, A. & Espie, C.A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy*, 38, 679-693.

Woods H., Marchetti L.M., Biello S.M., Espie C.A. (2007) The clock as a focus of selective attention in those with primary insomnia. *SLEEP*, 30: A256-A256 751

Suppl.

Woods H, Kathuria P, Biello SM, Espie CA. (2008) Covert attention to sleep in those with primary insomnia: no evidence for an attentional bias. *SLEEP*, 31: A244-A244 744 Suppl.

Woods H., Steele A.J.E., Biello S.M., Espie C.A. Selective attention to sleep is not an artefact of sleep complaint in insomnia: a study with pregnant and postpartum women (2008). *Journal of Sleep Research*. Volume 17, Issue Supplement s1.

Woods H., Harvey C.J., Ellis J., Biello S.M., Espie C.A. (2008) Selective attention to sleep in heavy and light social drinkers. *Journal of Sleep Research*. Volume 17, Issue Supplement s1.

Woods H., Marchetti L.M., Biello S.M., Espie C.A. (2009) The clock as a focus of selective attention in those with primary insomnia: an experimental study using the modified Posner paradigm. *Behaviour Research and Therapy*, 47.

World Health Organization. The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines. Geneva: World Health Organization; 1992.

