‘LET ME DRAW YOUR ATTENTION TO SLEEP’

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ABSTRACT

Psychophysiologic insomnia (PI) is a disorder of somatized tension and learned sleep-preventing associations (ICSD-R). Numerous authors contend that PI is the result of numerous psychological factors. Accumulated evidence supports the notion that PI are preoccupied with sleep, and the impact of not sleeping, and that this drives various behaviours, e.g. selective attention (SA) and monitoring, that result in worrisome thoughts and excessive anxiousness. Indeed, the concept of SA is incorporated into many of the proposed models of insomnia. SA, more commonly termed Attention Bias (AB), can be measured objectively using computerised cognitive probe tasks where information processing speed is taken as a proxy for biases in attention. These tasks use salient and neutral word or picture stimuli within an experimental test paradigm, and most identified AB effects have been attributed to perceived threat. Direct assessment of AB in PI is limited. At The outset of this PhD, namely Experiment 1 and 2, novel AB paradigms were used to confirm the existence of AB in PI to sleep related stimuli. Indeed, AB effects in PI do not appear to be an idiosyncratic finding. Experiment 3 attempts to underpin the components of attention, i.e. engagement or disengagement processes, which are responsible for the AB data of PI, through the use of a Modified Posner Paradigm. The data suggest that delayed disengagement away from salient stimuli are responsible for the AB effects captured. Finally, Experiment 4 differentiates the experimental stimuli in terms of emotional salience, i.e. positive or negative, to assess whether all stimulus valences generate the AB effect in PI. Here the data suggest that negative sleep-related stimuli are most salient to PI in capturing attention. All finding are discussed in relation the existing research on PI and attention bias.

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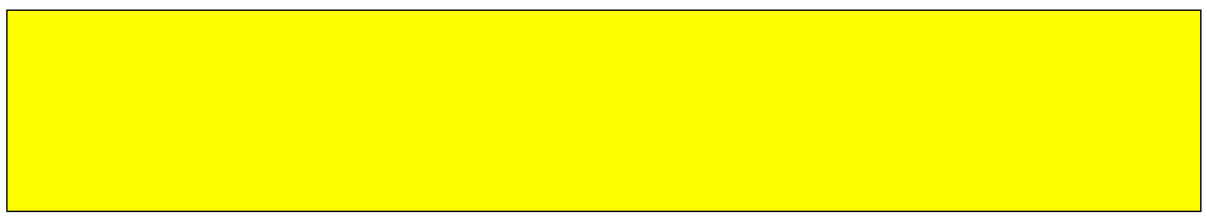
Declaration

I declare that this thesis is my own work carried out under the normal terms of supervision.



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Publications

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Experiments 2-4 are currently being written-up for publication.

Other publications involving this work

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Thomas, Margaret, John and Catherine

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ABBREVIATION LIST

PI	Psychophysiologic Insomnia
DSPS	Delayed Sleep Phase Syndrome
GS	Good Sleeper
AB	Attention Bias
SA	Selective Attention
IPB	Information Processing Bias
SOL	Sleep Onset Latency
TST	Total Sleep Time
WASO	Wake Time After Sleep Onset
SE	Sleep Efficiency
SCN	Suprachiasmatic Nucleus
RT	Reaction Time
PSQI	Pittsburgh Sleep Quality Index
BDI	Beck Depression Inventory
STAI	Speilberger State Trait Anxiety Inventory
ICB	Inducing Change Blindness Flicker Paradigm
OS	Original Stimulus
CS	Changed Stimulus

CHAPTER 1

AN INTRODUCTION TO SLEEP DISORDERS

1. 1 INTRODUCTION

Sleep is a natural state of physical and mental rest, identified by reduced body movement and decreased awareness of surroundings. Sleep is regulated by two body systems: *sleep/wake homeostasis* and the *circadian biological clock.*

When we have been awake for a long period of time, sleep/wake homeostasis tells us that a need for sleep is accumulating and that it is time to sleep. It also helps us maintain enough sleep throughout the night to make up for the hours of being awake. If this restorative process existed alone, it would mean that we would be most alert as our day was starting out, and that the longer we were awake, the more we would feel like sleeping. In this way, sleep/wake homeostasis creates a drive that balances sleep and wakefulness. Our internal circadian biological clocks, on the other hand, regulate the timing of periods of sleepiness and wakefulness throughout the day. The circadian rhythm dips and rises at different times of the day, so adults' strongest sleep drive generally occurs between 2:00-4:00 am and in the afternoon between 1:00-3:00 pm, although there is some variation depending on whether you are a "morning person" or "evening person." The sleepiness we experience during these circadian dips will be less intense if we have had sufficient sleep, and more intense when we are sleep deprived. The circadian rhythm also causes us to feel more alert at certain points of the day, even if we have been awake for hours and our sleep/wake restorative process would otherwise make us feel sleepier.

This sleep-wake cycle is vital to our health and well-being, yet sleep disturbances are not unusual. American polls reveal that up to 60 % of adults

report having sleep problems a few nights a week or more, with women representing a higher percentage than males (National Sleep Foundation, 2003). National health organisations have identified over one hundred daytime and night time sleep disorders (National Commission on Sleep Disorder Research). The effects of poor sleep result in over 40% of adults experiencing daytime sleepiness, at least a few days each month, severe enough to interfere with daily activities (National Sleep Foundation, 2003). Tragically, for example, fatigue contributes to more than 100,000 police-reported vehicle crashes, causing 71,000 injuries and 1,500 deaths each year in the United States alone (NHTSA-National Highway Traffic Safety Administration, 2002). Closer to home, it has been reported that 15-20% of road crashes in the United Kingdom are caused by driver sleepiness (Horne & Reyner,1995a; Reyner, Flatley & Horne, 2001).

Sleep disturbances are characterised by differing traits, e.g. sleep onset difficulties, sleep maintenance difficulties, night terrors etc, that have allowed researchers to discriminate them from one another. In some cases researchers have identified the genesis of specific sleep disorders, and reasons behind their maintenance. Indeed research to date has reported that sleep disturbances can be genetic in origin (Jones et al. 1999), physiological in origin (Monroe 1967, Haynes et al., 1974, Freedman et al. 1982, Adam 1986) or present with cognitive over activity at bedtime as a major defining feature (Evans 1977, Lichstein et al. 1980, Espie 2002, Harvey 2002).

1.2 DISORDERS OF CIRCADIAN RHYTHM

Every living organism, unicellular or multi-cellular has a biological rhythm. The rhythm refers to a cyclic process, within a biological system, that

constantly reoccurs. The pattern of expression of these rhythms can be very high (once every fraction of a second) or very low (once every year). Heart rate, blood pressure, menstrual cycle, body temperature, feeding patterns and the sleep/wake cycle are all examples of biological rhythms.

The earth rotates on its axis relative to the sun, and therefore cycles continuously from light to dark. This period of rotation is approximately 24 hours and creates the passage of night into day. Humans have adapted to this environmental change by exhibiting daily physiological and behavioural rhythms. Indeed, the sleep/wake cycle is an immensely important circadian rhythm, which has adapted to this approximate 24-hour cycle. This cycle is governed by the Suprachiasmatic Nucleus (SCN), located at the base of the third ventricle and above the optic chiasm, which functions as the circadian clock in mammals (Ralph et al. 1990). The SCN can be synchronised to a variety of photic and non-photic stimuli. The environmental light-dark cycle provides the principle-entraining stimulus for the circadian rhythm of most species, however non-photic cues, such as behavioural activity, have also been shown to effect the phase of the circadian clock (Mistlberger, 1994; Mrosovsky, 1988; Turek, 1989).

1.2.1 Circadian Rhythm Sleep Disorders

Delayed Sleep Phase Syndrome (DSPS) and Advanced Sleep Phase Syndrome (ASPS) result from a conflict between the patient's internal biological clock and the external environment. Unlike jet lag, this desynchronization is not activated by travel or change in external environment. Rather, the patient's propensity to fall asleep is simply "delayed" or "advanced" in relation to that of

the general public. Subsequently, a patient who is experiencing DSPS or ASPS is out of phase with the routine that governs most of his or her life.

i) Delayed Sleep Phase Syndrome (DSPS)

The cardinal feature of DSPS is difficulty in falling asleep at the socially expected time. Instead, they find that sleep does not arrive until the early hours of the morning, with rising time subsequently delayed, often until midday or later; consequently leading to social and occupational problems. The sleep period of the DSPS sufferer is relatively unbroken and refreshing, with the cause of problems lying with de-synchrony between the sleep-wake cycle of the individual and that of the outside world.

Subsequently, the main difficulty for a person with DSPS or ASPS (discussed in more detail below) is meeting the expectations of society. A person with DSPS may lose jobs or fail courses in school. Therefore, this syndrome affects individuals on a social level at the same time that it compromises their health and hygiene. Patients with DSPS may initially refer to their symptoms as insomnia. Again, the significance that society places on traditional sleep-wake patterns usually dictates what is normal and as soon as people deviate from a normal sleep pattern, they tend to assume they are not capable of sleep at all. However, as previously discussed, those who suffer from DSPS are able to get plentiful sleep; it's just postponed. If they can sleep until they are ready to wake, patients with DSPS can experience rewarding sleep. The reality is that DSPS usually makes it hard to wake up, as the patient simultaneously indulges his or her late night sleep routine and yields to the wake routine of society.

Diagnosis of DSPS is based on sleep history and treatment for the disorder ranges from learning to respond differently to external indicators of sleep-wake time to practising new and proper sleep habits. The exact prevalence of delayed sleep phase syndrome is unknown. In one recent study, DSPS accounted for 40% of disorders involving sleep-wake schedules among 5,000 participants. It is estimated to affect 7% of teenagers and to be the cause in 10% of chronic insomnia cases. Although DSPS usually surfaces in childhood, it is seen most frequently in young adults, especially men, which suggest a concurrence with lifestyle. However, Ebisawa and colleagues have found an association between the human period 3 gene (Ebisawa et al. 2001) and DSPS that is supported by a group led by van Schantz who have found that normal preferences for 'morningness' and 'eveningness' are associated with variations in the same gene (Archer, 2003). Indeed many sufferers of DSPS have an immediate family member who suffers the same debilitating complaint. This information suggests that there may indeed be two forms of DSPS, one that exists as a result of social factors, i.e. late nights socialising during university, and the other that may exist as a genetically inherent disorder.

ii) Advanced Sleep Phase Syndrome (ASPS)

ASPS patients display opposite behaviour to DSPS patients by falling asleep in the early evening and waking up in the early hours of the morning. Again such patients' have good sleep quality and duration, however it is out of synch with the conventional world. Researchers have identified a gene, the Period2 gene, which appears to contain a mutation in ASPS patients (Toh et al. 2001). However, this mutation does not explain all ASPS cases as it was found in

only one of the forty families the study was focusing on. Subsequently, Louis Ptacek and colleagues of the University of California are now sequencing all of the known clock genes in each of the other families in the hope of finding other mutations related to the syndrome (cited in nature).

At trait level, most individuals usually describe themselves as either a 'morning' person or an 'evening' person, or a Lark or an Owl as commonly termed. Although these traits mimic those of advanced and delayed sleep phase disorders, they are less extreme and simply highlight the variance of human individual difference. Furthermore, preferences for morningness and eveningness can be influenced by lifestyle, and occasionally more extreme lifestyle changes can result in the development of a specific disorder (e.g. university student example given above).

1.2.2 Jet Lag

Jet lag, or desynchronosis, is a temporary condition that some people experience following air travel across several time zones in a short period of time (meridians demarcate geographic position in relation to the Earth's poles and, ultimately, define time zones). This causes the traveller's internal clock to be out of sync with the external environment. People experiencing jet lag have a difficult time maintaining their internal, routine sleep-wake pattern in their new location, because external stimuli, like sunshine and local timetables, dictate a different pattern. For this reason, one can feel lethargic one moment and excited the next. Jet lag creates a double bind for vacationers and business people who must cross several time zones to reach their destination, but who are also intent on maximizing sightseeing or productivity. As travellers attempt to adjust their

internal clock to a new external environment, symptoms result with varying intensity (National Sleep Foundation, 2003).

1.2.3 Shift Work

Shift work is associated with numerous negative effects, the most prominent of which is disturbed sleep (Akerstedt, 1990). These sleep problems are mainly due to a disruption of the normal sleep/wake rhythm, of the normal circadian REM sleep rhythm and of the rhythm of REM/non-REM sleep patterns. Thus, the sleep problems of shift workers are partly a circadian one (Ohayon et al. 2002). More specifically, it has been demonstrated that the daytime sleep of night workers is 2-4 hours shorter than that obtained by night (Tilley et al. 1982; Walsh et al. 1981), and polysomnographic studies have demonstrated various differences in daytime sleep architecture compared with nocturnal sleep, including reductions in REM and Stage 2 sleep, increases in Stage 1 sleep, shorter REM latencies and premature awakenings (Tilley et al. 1982; Weitzman et al. 1970; Akerstedt et al. 1982, respectively). The negative consequences of night workers' sleep disruption can impinge on both the health of the worker (Moore-Ede & Richardson, 1985) and on public safety (Lyznicki et al. 1998).

1.3 INSOMNIA

Insomnia is perhaps the most frequent health complaint, in general practice, after pain. Insomnia is a heterogeneous complaint reflecting reduced quality, duration, or efficiency of sleep (Morin et al. 1999). Insomnia is distinct from a circadian rhythm disorder, in which timing of the major sleep period is

out of alignment with the local clock. It has been differentiated from parasomnia, in which behavioural events occur in association with sleep (e.g. sleep walking), disorders such as sleep apnoea, a respiratory impairment associated with sleep and narcolepsy, an excessive sleeping complaint.

The generic term 'insomnia' is applied to describe disorders of initiating and/or maintaining sleep in relation to three commonly used terms; initial, maintenance and terminal. These terms reflect the portion of the sleep period most affected by wakefulness. In initial insomnia, sleep onset latency (SOL) is significantly greater than for normal sleepers with an average of 60-90 minutes as compared to 0-15 minutes, respectively (Turner & Ascher, 1979a). Thus it is the initial initiation of sleep that is difficult. However, in maintenance insomnia the time spent awake after the initial onset of sleep is of major concern. More specifically, after the patient has initiated sleep, they repeatedly awake, thus struggling to maintain the sleep process. Indeed many individual suffer from both initial and maintenance sleep problems and thus their sleep efficiency (SE), which is the ratio of total time slept to time spent in bed, expressed as a percentage, is less than 85% which is the cut off SE score for sleep to be assessed as 'good'. Finally, terminal insomnia, also called 'late' insomnia or 'early morning wakening' refers to a final, and sometimes abrupt arousal occurring during the night with a subsequent failure to return to sleep prior to what is considered to be the usual wakening time for the individual.

1.3.1 Secondary Insomnia

Many medical disorders are associated with insomnia e.g. chronic pain, arthritis, neurological disease etc. Historically, sleep disruption presenting with

other medical disorders has been termed 'secondary insomnia'. 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). For example, drug treatments for depression are known to have stimulant effects that interfere with the sleep process.

Insomnia is also widely accepted as a symptom of many psychiatric and psychological disorders e.g. anxiety, depression, post traumatic stress disorder (Breslau et al. 1996). 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 and until recently such thinking has been widely accepted throughout psychiatry (Harvey, 2002).

However, recently an NIH consensus statement acknowledged that the limited understanding of mechanistic pathways in chronic insomnia precludes drawing firm conclusions about the nature of the associations between sleep disturbance and medical disorders and about the direction of causality (NIH consensus and state-of-the-science statements, 2005). Furthermore, they propose that the term 'secondary insomnia' is promoting under treatment and that 'comorbid insomnia' is a more appropriate term. Indeed the Research and Diagnostic Criteria (RDC) acknowledge that there is a debate over the adequacy of the 'secondary' status given to insomnia that presents simultaneously with other medical disorders (Morin, 2000).

In 2002, Harvey reviewed the data relating to three senses in which insomnia may not be secondary to medical disorders. Harvey reports that insomnia; (i) develops first (ii) when successfully targeted results in the remission of the co-morbid disorder and (iii) may be a risk factor for the

development of other disturbances (not just mental disorders). Such evidence opposes the original psychiatric position agreeing that insomnia, in comorbid presentations, is the secondary disorder (Harvey, 2002).

A robust finding in the literature is that sleep disturbance precedes the development of depression. In two independent studies, Eaton et al. (1993) and Ford et al. (1989) concluded that insomnia develops in many cases, prior to depression. Others have validated this pattern of findings in both the development of depression and anxiety disorders (Breslau at al. 1996 ; Livingston et al. 1993; Breslau et al. 1997 and Ford et al. 1989). As originally stated, the general belief 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, reported 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. Taken together, these results strongly indicate that a sleep-focused intervention is not only effective in reducing the sleep disturbance, but also reduces the accompanying pathology.

Thus, in support of the statement made by the NIH, this evidence suggests that the acceptance of insomnia as a secondary complaint, in all comorbid presentations, may be promoting under administration of treatments

that might not only improve insomnia, but also may reduce symptoms associated with the assumed 'primary' disorder.

Therefore, it is important that current research aims to discover and evaluate the genesis and maintenance of insomnia, as a primary disorder, with the purpose of identifying successful treatment routes to reduce the disorder.

1.3.2 Primary Insomnia

Primary insomnia is reported by up to 30% of the population, with prevalence of chronic insomnia estimated at 10-15% (Ohayon, Caulet, & Guilleminault, 1997). There are two main classification systems for Primary Insomnia, namely the DSM IV and ICSD-R. The DSM IV defined Primary Insomnia as difficulty initiating 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). The ISCD-R, however sub-divides the disorder further into sleep disturbances that are either, initial, maintenance or terminal insomnia, with cognitive and/or physiological components.

Psychophysiologic Insomnia (ICSD-R), which emphasises the behavioural and cognitive processes that are believed to underpin this disorder, is the most common form of PI in the ICSD-R nomenclature. This definition denotes that psychophysiological insomnia is a disorder of somatized tension and learned sleep-preventing associations (ICSD-R). 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. The belief that PI is

primarily a psychological disorder is underpinned by the widespread success of cognitive behavioural approaches to treatment (Morin et al. 1989; Morin et al. 1992; Lacks & Morin, 1992; Espie, 1991; Lacks & Powlishta, 1989; Murtagh & Greenwood, 1995; Pallesen et al. 1998).

A substantial body of psychological research has yielded important evidence on the genesis and maintenance of insomnia and there are many theories that focus on sleep-interfering processes (Monroe, 1967, Espie at al. 1989, Broman et al. 1994, Harvey, 2002, Espie, 2002). Most attribute insomnia to some kind of arousal that is assumed to interfere with sleep, such as behavioural, physiological, emotional or cognitive arousal.

The following section of this chapter aims to provide an overview of the research, and subsequent theories, presented for insomnia. Table 1 highlights the main perspectives to be discussed. Furthermore, the following section attempts to highlight the themes that are generic across all the perspectives, and provide explanations for why the body of research, incorporated in this PhD, speaks directly to these common themes.

Table 1. Main Theoretical Perspectives Presented for Insomnia

Authors	***Perspective***	***Key Words***
Bootzin 1972/1981	Behavioural	Stimulus Control
Spielman 1987	Behavioural	Sleep Restriction
Espie 1991	Cog/Beh/Phys Interaction	Paradoxical Intention
Morin 1993	Cog/Beh/Phys Interaction	Hyper-arousal
Perlis 1997	Neurocognitive	Cortical arousal through classical conditioning
Lundh 2000	Cog/Beh/Phys Interaction	Dysfunctional sleep-interfering and sleep-interpreting processes
Espie 2002	Cog/Beh/Phys Interaction	Psychobiological Inhibition Model
Harvey 2002	Cognitive	Cognitive Model of Insomnia

1.3.3 Behavioural Perspectives

The most simplistic introduction to the behavioural perspective of insomnia is that an individual's behaviour governs their sleeping patterns. The behavioural model of insomnia was first proposed by Bootzin (1972) and posits that insomnia exists through the adopting of maladaptive sleep habits. 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. Bootzin proposed that stimulus control treatment instructions, such as lying down to sleep only

when sleepy, getting up if unable to sleep within 25 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, successful sleep promoting behaviour and sleep related somatic cues. Indeed, this may serve to further strengthen the awareness of positive and negative sleep-related cues that denote successful or unsuccessful sleep, respectively.

Two studies have attempted component analysis of stimulus control treatment. Tokarz and Lawrence (1974) separated the stimulus control components (those instructions focusing upon making the bed and bedroom more discriminative for sleep) from the temporal control components (regularising the sleeping pattern). They found that both sets of procedures equally and significantly reduced self-report sleep latencies in their student sample. Zwart and Lisman (1979) conducted a more comprehensive study of 47 undergraduate subjects who were assigned to either stimulus control (all instructions), temporal control (lie down to sleep only when sleepy, rise at the same time every day, do not nap in the daytime), non-contingent control (a fixed number of arising within 20 minutes of retiring), counter control (sit up in bed, read, watch TV, etc. if unable to sleep) and no treatment. The authors reported that the counter control strategy was as effective as the complete set of stimulus control instructions. The authors proposed that the surprising effectiveness of the counter control procedure was that it ensured contingent disruption of bed and bedtime as cues for mental arousal. Indeed this theory gains support from the work of Turner and Ascher (1979b) who reported that subjects found that stimulus control instructions served to "break up lying in bed and thinking behaviour". Thus, the

counter control procedure then may act as a distraction technique by purposefully occupying the pre-sleep period for the insomniac group.

A further behavioural technique that bears some relationship to the temporal management aspects of stimulus control is Sleep Restriction Therapy. This intervention is based on the suggestion PI spend excessive periods of time awake in bed. Thus, sleep efficiency (SE) is reduced. Sleep restriction compresses sleep toward greater continuity, reduces wakefulness in bed, and increases sleep efficiency. Thus patients are encouraged to reduce bedtime hours to approximate time in bed (TIB) closer to actual sleep duration (Spielman 1987). 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.

Sleep restriction therapy involves the establishment of a wakening time each morning in accordance with the daytime schedule needs. Average subjective total sleep time (TST) as recorded over one or two weeks of self-monitoring is then used to arrive at the amount of time that should be spent in bed. Retiring time at night may then be calculated and set to the nearest 15 minutes, such that time spent in bed equals this prescribed sleep period. The patient is instructed to follow this revised sleep schedule and retiring time may be altered only if greater that 85% sleep efficiency is achieved over a series of nights (Espie, 2001).

Spielman et al. (1987) conducted a treatment outcome study of sleep restriction on a sample of 35 subjects who had an average duration of insomnia

of 15.4 years. Half of the sample presented both sleep onset and sleep maintenance difficulties and the remainder presented mostly sleep maintenance problems alone. One the first night of treatment, time in bed was restricted to a level that was on average 140 minutes below the baseline mean value. Thus, in the early stages of treatment TST reduced very considerably below baseline values. However, by the fourteenth day of treatment it had increased again the pre-treatment level and thereafter steadily increased throughout treatment. During the final treatment week, TST was 23 minutes greater than at baseline, although subjects were on average spending 86 minutes less in bed. Mean SE, increased, therefore, from 67 to 87%. The amount of time spent awake during the night also decreased significantly (from 159 minutes at base line to 50 minutes at post treatment). A follow-up conducted at 36 weeks after completion of treatment indicated that sleep improvements were maintained.

The initial sleep loss at the start of treatment in sleep restriction therapy is similar to that of stimulus control therapy. In both, the partial sleep deprivation may consolidate sleep directly, by producing daytime fatigue that dampens a chronic state of hyperarousal, or reduces maladaptive conditioning because less time is spent lying awake in bed. Spielman et al. regarded the clinical efficacy of their treatment as related to the reduction of nocturnal wakefulness, i.e. greater continuity of sleep which became reliable on a night to night basis. In conclusion, the synergy between stimulus control and sleep restriction is evident, and they are often now presented together (Espie 2002). Indeed, the term "sleep scheduling" refers to the combination of both techniques (Espie et al. 1989, 2001b; Morin & Espie, 2003).

As previously stated earlier in this chapter, interest in the control that sleep-related environments and objects might have over successful sleep is long established. Indeed all research focusing on these behavioural approaches agree that within a conditioning framework, these external 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.

With this in mind, it is proposed that cognitive paradigms, assessing information processing in PI, may be well suited to investigating the influence of the bedroom environment on cognitive processes. Furthermore, such paradigms provide the opportunity for direct comparisons between good and poor sleeper groups on a quantitative level. I will discuss in more detail the reasoning behind this claim at the end of this current chapter.

1.3.4 Physiological Perspectives

Research into the differences between good and poor sleepers, at a physiological level, has a long history. 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,

replications of these results are inconsistent. For example, numerous authors have failed to find associations of insomnia 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 one possible explanation for these inconsistencies over studies is that the involved physiological systems may differ from one patient to another. The authors suggest that a global measure, such as metabolic rate (whole body oxygen use), will show more consistent results. Thus in a study of patients with primary insomnia, these authors found that metabolic rate (whole body VO2) was consistently elevated in insomniacs, as compared to normal controls, not only during the night but also at all measurement points during the day (Bonnet, et al., 1995). Their findings are that these patients suffer from a general disorder of hyper-arousal that is responsible for both daytime symptoms and nocturnal poor sleep and that this kind of hyper arousal may be the result of biological conditions (e.g. genetic factors, caffeine, etc.), psychological (cognitive and emotional) processes, or an interaction of both.

In keeping with the more behavioural perspective, previously discussed, Perlis et al. (1997) proposed a neurocognitive model stating that; acute insomnia is precipitated by life stress, that persistent insomnia occurs because of the engagement of maladaptive 'coping' strategies and that chronic insomnia occurs as a result of conditioned arousal. However, the perspective of these authors focused on one form of conditioned arousal: cortical arousal (i.e. arousal detected in the brain by EEG signals). In their neurocognitive model, cortical arousal is 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 (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 1987; Mercia & 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). More specifically, as one develops chronic insomnia (via behavioural contingencies), there is an increase in high frequency EEG activity at or around sleep onset. In transient insomnia such activity may occur in association with stress induced worry and/or rumination (Perlis et al. 1997). In chronic insomnia, high frequency EEG activity occurs as a result of classical conditioning, i.e. high frequency EEG is elicited in response to the visual and/or temporal cues usually associated with sleepiness and sleep and that this occurs in the absence of situational stressors (Perlis et al. 1997).

Thus, this theory unpicks the notion that the information absorbed from the environment leads to changes in cognitive activity, reflected by the physiological changes in the brain, that are detected through cognitive-neuroimaging techniques. As discussed above, in chronic insomnia, sleep related temporal cues propel levels of cognitive activity, which is inconsistent with promoting good sleep. Thus, perhaps the internal/external monitoring, of the chronic insomnia patient, discussed previously, result in identification of sleep-related stimuli that have become conditioned to represent unsuccessful sleep, and thus promote a cognitive over activity through ruminating worry and emotional

distress. Indeed, the cognitive perspectives outlined below would support this theory and its neuroimaging data.

1.3.5 Cognitive Perspectives

Studies have repeatedly demonstrated that sleep-onset insomniacs report cognitive arousal, e.g. (Broman & Hetta, 1994; Espie, Brooks & Lindsay, 1989; Evans, 1977; Lichstein & Rosenthal, 1980; Nicassio, Mendlowitz, Fussel & Petras, 1985), as being of greater concern than physiological arousal (e.g.too hot/cold, increased heart rate, high energy levels etc). Investigation of the relative influence of cognitive and physiological arousal, consistently associates the former more strongly with PI, and having an 'overactive mind' has been the attribution of poor sleep rated most highly, both by insomniacs and non-insomniacs (Broman & Hetta, 1994; Espie, Brooks & Lindsay, 1989; Evans, 1977; Lichstein & Rosenthal, 1980; Nicassio, Mendlowitz, Fussel & 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, insomniacs 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 and 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 insomniacs; 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.

Although cognitive arousal is most usefully defined in terms of an engagement in cognitive activity, which interferes with sleep, and is operationalized in terms of self-report. In the literature, however, the term "cognitive arousal" is often used in a way which overlaps with emotional arousal, since what is measured is not only the cognitive activity as such, but negatively valenced thoughts or worries, in this case concerning sleep or consequences related to insufficient sleep. People with insomnia have more negative thoughts than good sleepers do at bedtime (Nicassio et al. 1985, Van Egeren et al. 1983, Kuisk et al. 1989), and such thinking is even reported when wakened from light sleep (Borkovec et al. 1981). The thoughts of PI may be dependent on emotional state, (Espie, 2000) reflecting the finding that affect-laden (emotionally charged) cognitions are the ones most likely to interfere with the sleep process (Espie,

2001; Morin, 1993; Espie & Wicklow, 2000; Coyle & Watts, 1991). A Cambridge based group have reported two factor analytic studies in insomnia. In the first Coyle and 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 and 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. Insomniacs may also feel less in control of their thinking (Watts et al. 1995). Gendron et al. (1998) reported that insomniacs with comorbid generalised anxiety disorder, evaluated their thoughts as more intrusive and worrisome, and attempted cognitive avoidance strategies more frequently.

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 insomniacs' cognition's 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. An extensive study by Fichten et al. (1998) detailing the thoughts of older adults (mean age 68 years) during wakeful periods in bed yielded a three factor solution of generalised positive thinking, generalised negative thinking and thoughts related to sleep. They suggested that insomniacs might use positive thinking as a 'buffer' in an attempt to combat more negative intrusions. More recently, Wicklow & and 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 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).

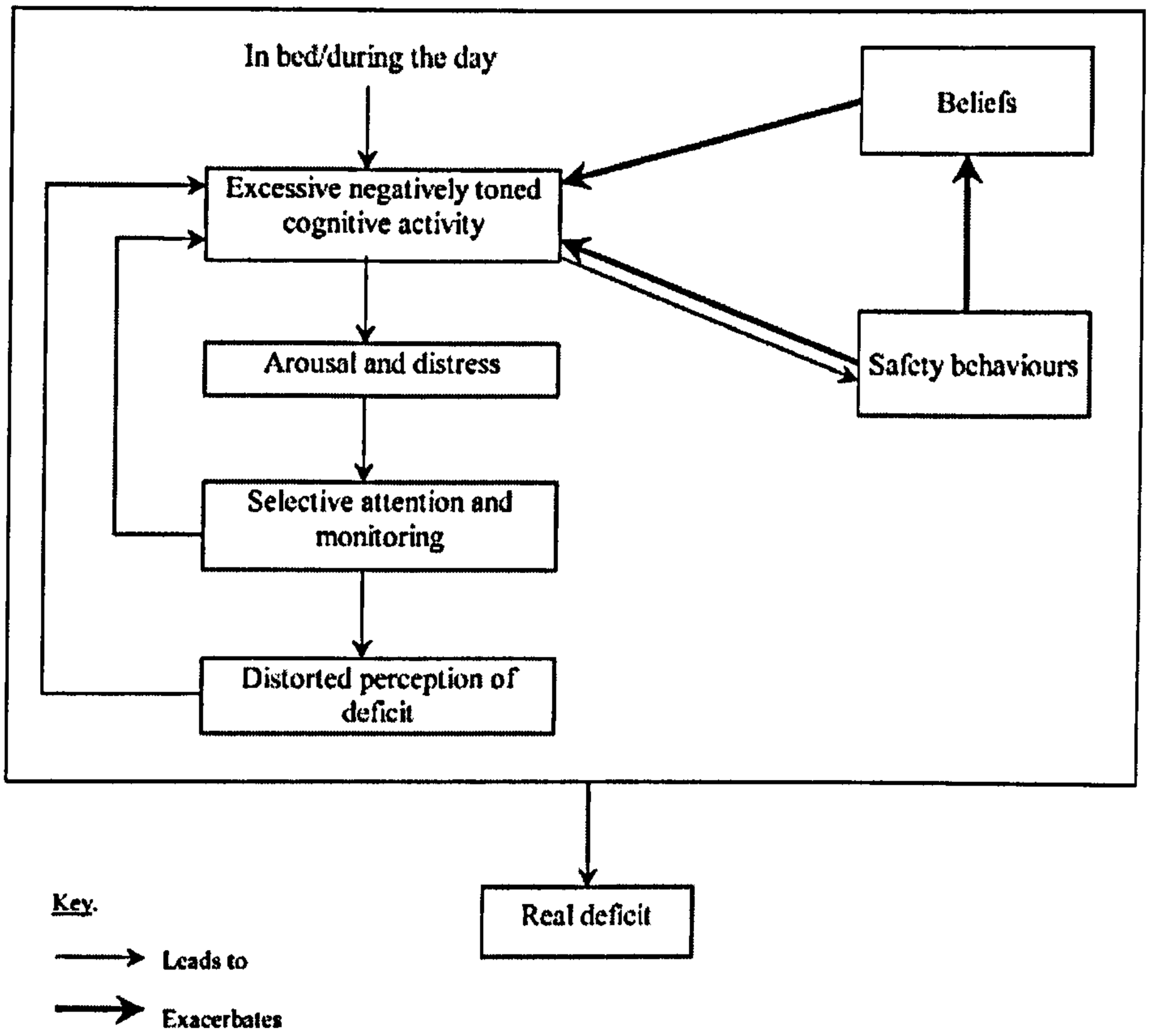
In support of these finding, Lundh and Broman presented a theoretical model which posited that psychological vulnerability factors may predispose the person with insomnia to 1) respond with sleep-interfering processes to stressful life events i.e. with cognitive over arousal at bedtime, excessive worry etc, and 2) to engage in dysfunctional sleep-interpreting processes, such as sleep-related beliefs, attitudes, and perfectionist standards. Indeed the consideration of these dysfunctional sleep interpreting processes was incorporated into the subsequent cognitive models of insomnia that are outlined below.

In 2002, Harvey proposed a Cognitive Model of Insomnia emphasising the excessive negatively toned cognitive activity of the insomniac when initiating sleep. Indeed the entry point of this model, shown below in Figure 1, holds that it is this excessive negatively toned cognitive activity about getting enough sleep and about the subsequent impact the sleep disturbance is having on health and/or daytime functioning that allow poor sleep to maintain (Harvey, 2002). Harvey proposes that the anxious state displayed by insomniacs precipitates attentional narrowing and preferential allocation of attentional resources to sleep-related threat cues (Harvey 2001). Indeed, this thinking would draw support from the literature on anxiety related disturbances, where attention bias toward stimuli of a threatening nature has been detected by cognitive paradigms (see Mogg & Bradley, 1998; Williams et al. 1997). Monitoring for both internal and external threat cues, such as indicators of not getting enough sleep or not functioning well during the day, and detection of such threat cues provides further reason for worry and concern. More specifically, Harvey reports that based on clinical practice, people with insomnia selectively attend to and monitor for sleep-related cues, and a table of the examples of monitoring, provided by Harvey, is presented below in Table 2.

Table 2 Examples of Monitoring and Sleep Related Threats, given with permission, from Harvey (2002) Behaviour Research and Therapy, 40 pp 876).

Type of monitoring	Examples
During the night	
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 1.25 a.m....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 2 a.m....that means I'll only get 4 hours of sleep tonight!"
On waking	
Body sensations for signs of poor sleep	Heavy feeling in the head, heavy and tired eyes
The clock to calculate how many hours of sleep were obtained	"It's 7 a.m....I finally got to sleep at 2 a.m. and then woke up two more times...so that means I got about 4 and a half hours of sleep!"
During the day	
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"

Figure 1. A Cognitive Model of the Maintenance of Insomnia, given with permission, from Harvey (2002) Behaviour Research and Therapy, 40 pp 872).



Of course, the identification of monitoring as a major role in the maintenance of PI, relates to our discussion in the behavioural perspectives, previously mentioned. As PI monitor for indicators compatible with sleep, they are just as likely to identify 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, and the threatening salience of such stimuli are strengthened. Thus, we propose that over time these stimuli become concrete representations of sleep incompatible indicators, and subsequently generate the cognitive activity (worry, rumination) that may be being captured in the cognitive neuroimaging studies described previously.

However, faulty conditioning is not the only distortion that occurs with the insomnia patient. Harvey acknowledges that PI consistently demonstrate a marked distortion of time perception. Moreover, PI have a tendency to overestimate the amount of time it took to fall asleep and markedly underestimate the total amount of time slept, as compared to good sleepers (Bonnet, 1990). Furthermore, safety behaviours are also adopted by people with insomnia in an attempt to avoid a feared outcome i.e. not getting to sleep. Harvey highlights that safety behaviours were first discussed in the anxiety literature and are thought to be problematic for patients as they prevent disconfirmation of the unrealistic belief and make fears more likely to occur. In the case of insomnia, Harvey suggests that such safety behaviours are working in a similar, negative, way, exacerbating the excessive cognitive activity and preventing disconfirmation of erroneous beliefs.

1.3.6 Cognitive/Emotional/Physiological Interaction Model

Two further considerations are important. Firstly, cognitive arousal, in itself, represents a form of physiological arousal, although an arousal of the central nervous system (CNS) rather than the autonomic nervous system (ANS); the distinction between physiological arousal and cognitive/emotional arousal has its basis in that the latter is most often operationalized in terms of self-report, whereas the former is operationalized in terms of somatic indices (e.g. heart rate, adrenaline levels, cortisol levels etc). Secondly, cognitive, emotional and autonomic arousal may interact in various ways. For example, it is possible that cognitive processes of worry or problem solving may interfere with sleep either directly, by preventing certain kinds of cognitive processes that are conducive to sleep, (e.g., if these more verbal processes of worried thinking prevent the development of a sleep-conducive hypnagogic imagery), or indirectly by producing an emotional arousal in the form of anxiety, dysphoria, remorse, enthusiasm that interferes with sleep. Conversely, a physiological induced arousal (e.g., by means of caffeine) may also produce an increase in cognitive activity, which might add to the sleep interfering process.

Espie's (2002) 'Psychobiological inhibition model' attempts to integrate all the existing explanations of insomnia into a conceptual framework. The central premise of this model is that 'good sleep' is maintained as long as there is insufficient inhibition to impair its presentation, but that insomnia arises when the expression and experience of normal sleep are inhibited, and there is failure to de-arouse properly. More specifically, Espie proposes that 'automaticity' and 'plasticity' (homeostatic/circadian factors central to sleep's regulatory core) are compromised, primarily by cognitive/affective processes that inhibit both mental

and physiological de-arousal (Espie, 2002). 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 individuals' behaviour, and thus the individual suffers from a performance anxiety. In paradoxical treatment counterproductive attempts to fall asleep are replaced by the intention or 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).

Recently, Espie and colleagues (2006) presented a review detailing an 'attention-intention-effort pathway' in Psychophysiologic insomnia. This pathway emphasises and delineates the major features within Psychophysiologic insomnia, and report different experimental procedures, such as attention bias paradigms and sleep effort scales, that provide direct evidence of such features existing in PI populations. Although this theoretical review relates to the models discussed in this chapter, the attention-intention-effort pathway model will be discussed in more detail later in chapter 7.

Morin (1993) also presented an integrative conceptualization of insomnia. He suggested that hyper-arousal (emotional, cognitive, physiologic) 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-arousals of these types, 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 and insomniac report though process such as catastrophising. 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).

It is also noteworthy that the psychobiological inhibition model is consistent with Spielman's (1991) model of insomnia acquisition and with Edgar's (1996) "opponent process" model. The latter proposes that circadian timing promotes wakefulness and opposes sleep drive, and that prolonged wakefulness leads to compensatory sleep responses. 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).

1.4 CHAPTER DISCUSSION

In relation to the total number of sleep disorders presented clinically, the above reviewed literature only discusses a very small proportion. However, the information selected above clearly demonstrates that sleep disorders can exist through both psychological and circadian processes and highlights the importance that treatments of each disruption should be tackled by individualised techniques.

The main aim of the first half of this chapter was to highlight the key differences between two forms of sleep disturbance i.e. those that are circadian in nature versus those that are psychological in nature. Although this current thesis is focusing on cognitive processes in insomnia, it was felt imperative that a clinical control population was included in the analysis (this will be discussed more in chapter 3). Indeed, in insomnia research circadian rhythm disorders, such as DSPS and ASPS, are rarely screened out. However, as discussed in detail above, although subjective measures taken from an insomnia population and a DSPS population are commonly similar, they exist through very different processes. Thus, previous research may have diluted the insomnia results by including a sleep disturbance mimicking insomnia, but existing through circadian rather than psychological processes.

At present the insomnia literature has proposed frameworks that describe the genesis and maintenance pathways, consisting of multiple processes, of the disorder. As highlighted throughout this chapter, shared themes can be detected across all the insomnia perspectives. For example, Espie (2002), Harvey (2002) and Lundh (2000) all highlight the existence of selective attention and faulty

information processing in insomnia. More specifically, accumulated evidence supports the notion that PI are preoccupied with sleep/sleeplessness, and the impact of not sleeping, and that this drives various behaviours such as external and internal monitoring, that result in worrisome thoughts and excessive anxiousness. Similarly, selective attention is also incorporated into the behavioural theories for insomnia, as attention bias directly relates to the phenomena of stimulus control and conditioned response paradigms. More specifically, as previously discussed, conditioned association of sleep incompatible, waking activities, with the bed and bedroom environment, promotes the salience of these environments (or stimuli within these environments) as indicators of sleeplessness and subsequent fatigue, stress, anxiousness etc).

Thus, the existence of attention bias in PI may be an important consideration in determining the origin and maintenance of the disorder, although available research that have directly assessed such an individual processes is very limited. Indeed, much of the evidence available to support selective attention at present, come as secondary observations. As will be highlighted in the following chapter, within the anxiety literature selective attention has helped explain why disorders are self-maintaining and why relapses so frequently occurs, as when stimuli are threateningly salient, anxiety responses will be generated more frequently and thus the disordered maintained. With this in mind, and taken together with the abundance of theories acknowledging the importance of attentional processes in insomnia, this body of research aims to 1) identify, through more direct cognitive methods of psychology, the existence of selective attention in PI, 2) unpick the components of the attentional system

driving selective attention in PI and 3) provide insight into which stimulus types drive this phenomenon.

The aim of the following chapter is to provide an overview of the selective attention mechanism, and review the literature focusing on selective attention in insomnia populations.

CHAPTER 2

ATTENTION BIAS & SLEEP

2.1 SELECTIVE ATTENTION

Conscious perception is selective, as human perceptions encompass only a small fraction of the information impinging upon the senses. Selectivity is apparent in most human behaviour. We constantly assign priority to partial aspects of the complex environments we encounter. For example, when reading we do not attend to all the words on the page, but only to a small set of words at a time. A second phenomenon of the human attention system, which is closely related to selectivity, is capacity limitation (Bregman, 1990). Capacity limitation can be illustrated by the fact that two tasks when performed individually pose no problem; however, when they are attempted simultaneously, they become very difficult. Thus, common conclusions hold that attention is a finite resource that can only be devoted to a subset of the total sensory input. Indeed, selectivity and capacity limitation can be viewed as two important cogs in a system, whereby selectivity prioritizes limited attentional resources to enhance the processing of important information.

Selective attention refers to a process by which specific stimuli, within the external and internal environment, are selected for further processing. Indeed, selective attention is the initial 'filtering' process at the onset of attention, rather than the continued processing of already selected information.

Traditionally, selective attention has been divided into 2 processes, automatic and controlled. The automatic process of selective attention is best described as 'the shifting of attention without conscious awareness or control' (Harvey et al. 2004). An example would be being suddenly distracted to the direction of an unexpected noise or moving object. Alternatively, selective attention can be a controlled process whereby we consciously direct our attention to a specific stimuli and hold it there for

a period of time. For example, when playing the card game 'snap'[1], we consciously attend to the cards on the deck in anticipation of a 'snap' occurring.

2.1.1 Attention Bias Research

Psychologists interested in information processing have researched human attention extensively (Broadbent, 1958, Deutsch & Deutsch, 1963, Shiffrin & Schneider, 1977, Allport, 1980, Posner, 1980). In short, stimuli that are salient are likely to attract attention, and this attentional capture has been differentially termed, either, selective attention, information processing bias (IPB) or attentional bias (AB). The latter however is arguably the most commonly used terminology and thus has been adopted as the term utilized within this body of research. Attention biases can be measured objectively using computerised cognitive probe tasks where information processing speed is taken as a proxy for biases in attention. These tasks use salient and neutral word or picture stimuli within an experimental test paradigm, and will be discussed in full later in this chapter.

Most identified attention bias effects have been attributed to perceived threat (Matthews et al, 1995, Matthews et al. 1998, Matthews et al. 2000, Fox et al. 2001). Indeed, in both in psychological disorders and substance abuse/dependence, attention biases have helped explain why disorders and dependencies are self-maintaining and why relapse so frequently occurs after apparently successful treatment (Jones et al. 2003). For example, if threatening stimuli are more readily noticed by those

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[1] Starting with the dealer, each player plays one card face up in the centre of the table making a pile of cards. This is continued until one player deals one card on top of another player's card, which is of the same colour and value, e.g. two black sevens. When this happens the first player to call out "Snap" wins. This player now adds the pile of cards in the centre of the table to the bottom of the packet of cards he already holds. The first player to win all the cards in the deck in this way is the winner of the game.

exhibiting clinical anxiety, anxiety responses will be generated more than others and the disorder maintained (Mogg et al. 1990).

Evidence for the propensity of threatening stimuli to attract attention comes from research using classic selective attention tasks in which threat and neutral stimuli are placed in competition with each other. For example, Pratto and John (1991), utilized a Stoop paradigm in which various positive and negative trait adjectives were presented in different coloured ink to a group of young adults. The authors found that colour naming was longest on the negative trait adjectives. It was concluded that the negative information captured attention of the participants at an automatic level, leading to more interference on the colour-naming task. Other studies have presented such words to highly anxious groups, both clinically and self reporting, and have replicated the effect of slower naming times toward threat-related words relative to neutral words (McLeod et al. 1986). Many theorists have assumed that these results reflect the automatic drawing of attentive processing toward threat-relevant and negative stimuli (Fox, et al. 2001).

As research methods have developed, techniques designed to assess whether visual attention is allocated toward the location of threat-related words in anxious individuals have become more advanced (Appendix 2 to 5 give visual representations of the Stoop, Dot Probe, Posner and ICB Flicker paradigms, respectively). MacLeod et al. (1986) developed a dot-probe paradigm, in which the distribution of attention is measured by a secondary task involving the detection of a small dot that can appear in a spatial location of either the top or the bottom word after the display is terminated (Tata et al. 1986). This paradigm identified that clinically anxious individuals were faster to detect the dot when it appeared in the location in which a threat-related word had previously appeared, a finding not replicated for non-anxious individuals. These

results have been replicated with non-clinical participants, with high levels of self-report anxiety (Fox, 1993) and again suggest a general tendency for negative or threat-related information to draw visual attention.

However, the Stroop and Dot-probe paradigms have one serious flaw that has attracted criticism on the efficacy of their results. More specifically, within each paradigm, the critical 'to-be-ignored' material is generally presented within foveal vision. Although foveal vision and attention is not the same thing, there is a general consensus that it is impossible not to attend to information presented within about a 1 degree radius from fixation (Eriksen & Eriksen, 1974). Indeed, although within these paradigms participants are instructed to fixate on a central fixation cross, they are still automatically attending to the cues, as the distance separating the cues from the fixation point is not great enough. Thus, with both the Stroop and Dot-probe tasks it is impossible to determine whether the threatening information draws attention or whether, once detected, threat-related information holds attention. Both these processes would account for longer colour-naming times on a Stroop task, and similarly, longer latencies on the dot-probe tasks.

Another concern of the Dot-probe paradigm, involves the length of time the critical stimuli are presented. In the majority of the available studies the stimuli were presented for as long as 500msec, thus both locations (above and below the central fixation point) are likely to receive attentive processing. Therefore it is again difficult to determine whether threat-related stimuli attract attention to themselves in the first place or whether once a threat stimulus has been detected, attention then tends to dwell at that location.

Fox et al. (2001) considered these effects in a series of experiments assessing visual attention in sub clinical anxiety. These authors suggested that the biases

detected previously, through the use of the Stroop and Dot-probe, may not be due to threat-related stimuli automatically drawing attention, but could well be due to the anxious individual's inability to disengage attention from threatening stimuli once such stimuli had been attended. Furthermore, Fox et al. demonstrated that this hypothesis could be directly investigated in accordance with a relatively new model of visual spatial attention that suggested that the human attention system is not a unitary system, but instead consists of at least three components: attentional shifting, engagement, and disengagement (Posner & Peterson, 1990).

To assess the engagement and disengagement mechanisms involved in attention bias, these authors modified a Posner paradigm (Posner et al. 1987). Within this computer task, participants are required to detect a target that may appear on the left or the right of a fixation point. On 75% of the trials, a cue highlights the area in which the target will appear (valid). However, one 25% of the trials the cue will appear in the opposite location of the following target (invalid). The typical paradigm effects reveal 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 RTs on valid trials and slower RTs on invalid trials. This effect is more commonly known as the cue validity effect.

Cue validity effects are strongest when the temporal separation between cue and target is less than 200msec and occur regardless of whether the cue is actually informative. Because of these characteristics, it is assumed that the orienting of attention by means of exogenous cues reflects an encapsulated system that cannot be affected by higher cognitive processes. However, Stolz, (1996) has challenged this assumption and shown through the use of an exogenous cueing paradigm, that the shift component of attention may be encapsulated, but that the disengagement

component is not. In this experiment participants were required to fixate on a word in the centre of a computer screen. An abrupt onset word cue was then presented either above or below fixation, followed immediately by a target to be detected in either the cued or uncued location. The results demonstrated that the semantic relation between the fixation word and the cued word had a strong influence on the related cue's ability to hold attention at the cued location. Thus, RT on invalid related trials was slowed considerably, relative to invalid unrelated trials, suggesting that a related cue hindered the disengagement process. No such differences were found between related and unrelated valid trials, suggesting a related cue did not affect the ability of the cue to draw attention.

2.1.2 Linking Attention Bias To Insomnia

As discussed in chapter 1, Harvey's (2002) cognitive model of insomnia proposes that the anxious state generated by excessive worry, negative thoughts and autonomic arousal trigger selective attention towards, and monitoring of, internal and external sleep-related threat cues (Harvey, 2002). Likewise, Espie's (2002) psychobiological model emphasises the inhibitory influence of attention and intention upon normal sleep engagement. It is argued that such processes are dysregulatory because they contravene the 'automaticity' of normal unattended sleep engagement (Espie, 2002). These theories are consistent with ICSD-2 (AASM, 2005) text describing just how marked the attention to sleep is in Psychophysiologic insomnia - *"Concerns about sleep grow progressively over months or years as sleep gradually deteriorates until the desire to obtain a good night's sleep becomes the person's major concern"* (p. I-6). This statement conveys both a sense of incrementing distress

associated with sleeplessness (c.f. threat), and a preoccupying longing for sleep (c.f. craving) that might serve as preconditions for attention bias.

Evidence of attention bias or information processing bias in insomnia can be drawn from several sources, using different methodologies. The most direct evidence comes from experimental studies utilizing more cognitive methods of psychology; i.e. paradigms specifically measuring or manipulating aspects of attention bias, like those previously discussed. Others research has provided qualitative data, questionnaires and rating scales, that have added to our understanding of selective attention and insomnia.

i. Qualitative Measures

Many of the qualitative measures have identified that insomniacs display a tendency to pre-occupy themselves with worry about sleep and the consequences of not sleeping (Nicasso & Bootzin, 1979; Morin, 1993; Fichten et al. 1998; Tang & Harvey, in press). In a prospective study of pre-sleep mentation, Wicklow and Espie (2000) obtained voice-activated audiotape recordings of spontaneous thoughts, and sleep actigraphic data from 21 poor sleepers over 3 consecutive nights. Content analysis of over 1,000 thought segments yielded eight categories of pre-sleep intrusion, and a regression model indicated that focusing on sleep and the anticipated consequences of poor sleep, along with general problem solving were the strongest predictors of objective SOL. Thought content was subsumed under one of three factors; 'active problem-solving' (e.g. rehearsing/ planning events), 'present state monitoring' (e.g. thinking about - sleep/ not sleeping, autonomic experiences, your own thinking) and 'environmental reactivity' (e.g. attending to external noises). Thirty-eight percent of thought segments represented *present state monitoring*.

The qualitative component of this study was partially replicated in a further investigation that also had a psychometric phase, leading the development of the Glasgow Content of Thoughts Inventory (GCTI: Harvey K & Espie, 2004). The GCTI was found to have good internal consistency ($\alpha = .87$) and test-retest reliability (ICC = .88) and a score of 42 discriminated PI from GS groups with sensitivity of 100% and specificity of 83%. A principal components analysis of the GCTI found that present state monitoring emerged as an important factor accounting for 38% of explained variance (Espie & Harvey K.).

Further research, assessing 5 areas of attention focus through a semi-structured interview technique, highlighted that people with insomnia relative to GS were more likely to attend to detection of sensations of falling asleep and to worries/concerns, trying to solve problems and listening to noises. Good sleepers however were more likely to attend to 'nothing in particular' (Harvey, 2000). Semler & Harvey (2004a) then reported two related studies. In the first of these, students meeting criteria for primary insomnia were compared with a GS control group using a semi-structured interview of sleep-related threat, negative thoughts, and safety behaviours. People with insomnia reported more frequent monitoring, night and day, and they engaged in more safety behaviours. A 'path analysis' suggested that monitoring might act as a driver for negative thinking in insomnia. In the second study reported in this paper some evidence emerged for the generalizability of these findings to a clinical insomnia sample.

This work was extended through the development of the *Sleep Associated Monitoring Index* (SAMI: Semler & Harvey, 2004b). This 30-item scale of sleep-related threat monitoring shows good reliability ($\alpha = .87$) and positive correlation with the PSQI. Importantly, moderate correlation ($r=0.36$) with the Penn state worry

questionnaire suggests that the SAMI score is not simply an index of generic aspects of worry. An eight-component solution was obtained following principal components analysis on a large sample (n = 400) of university students and staff. These components included monitoring for body sensations (daytime, pre-sleep, and on waking, each loaded as separate components), clock time and the environment.

ii. Experimental Measures

To date, three studies have applied the previously discussed cognitive probe tasks to insomnia and Table 3 provides an overview of each. Lundh et al's (1997) study was a pioneering piece of work because it translated the emotional Stroop task into the insomnia field and recognised the potential importance of attention in the maintenance of the disorder (Lundh et al. 1997). This modified Stroop task involved target (sleep) and control (neutral) words being presented at random in different ink colours. Subjects, 20 primary insomniacs and 20 good sleepers, were asked to respond quickly to the presented colour by pressing the corresponding coloured button on a response box. They were instructed to ignore the actual meaning of the words. Response latencies for colour identification are automatically recorded for each stimulus. Longer response latency is thought to suggest increased attention bias because automatic processing of word meaning for salient words is likely to interfere with (slow down) colour naming relative to response time for the neutral words: the so-called interference effect.

The results demonstrated that people with insomnia had prolonged response latency for sleep-related words. However, this effect was also evident in a control population of good sleepers, and there was no group difference on the Stroop interference index; a result inconsistent with the attention bias hypothesis, irrespective

of sleep problems. However, the extensive literature on the Stroop task would not predict experimental effects in normal control groups. Of course, recruited good sleepers may have a particular interest in sleep, and this might yield a bias. Also, no measure of affective state (known to influence Stroop findings) was taken, and diagnostic criteria were not reported for the insomnia group.

Taylor, Espie and White (2003) also used the Stroop task, selecting a cancer population because the primary purpose was to investigate the development of insomnia associated with stress in people who had previously been good sleepers (Taylor et al. 2003). None of the participants had insomnia prior to diagnosis; that is they were a 'true' secondary insomnia population rather than people whose (pre-existing) insomnia had been exacerbated. Two groups of people with cancer and insomnia, 0-3 months and 12-18 months after cancer diagnosis, completed the task comprising cancer-related, sleep-related and neutral word cues. Both groups demonstrated attention bias for cancer-related words but only the persistent insomnia group demonstrated attention bias for sleep-related words. The fact that interference effects were absent at 0-3 months but evident at 12-18 months, therefore, suggested that selective attention bias towards sleep may play a role in the transition from Adjustment Insomnia to Psychophysiological Insomnia.

There has been debate over whether the Stroop task measures increased vigilance or simply reflects the impact of heightened arousal interfering with information processing when salient stimuli are presented (Jones et al. 2003). The dot-probe task, has been posited as one solution to this problem (MacLeod et al. 1986). In this task, words are simultaneously presented to two areas on a computer screen. The ensuing distribution of visual attention is measured by recording detection latency for a visual probe that could appear in the spatial location of either

word, immediately after the display of that word has terminated. The trials providing the data of interest are those in which one of the words is salient. By examining the impact of sets of such words on relative probe detection latencies in the two spatial areas, it is possible to determine whether visual attention has shifted toward or away from such stimuli.

MacMahon et al. (2006)) used the dot probe task with 63 young adults across three experimental groups (PI, DSPS, GS). 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. Results supported the predictions, with 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 GS and DSPS groups did not differ from each other, suggesting that the underpinning mechanism maintaining DSPS is not an attention bias. This is consistent with DSPS as an endogenous circadian problem.

Recently, Harvey and colleagues have presented data focusing on 'real life' attentional biases in insomnia groups. Indeed, data focusing on both daytime and nighttime attentional processes have been reported. Although this data is directly related to the research outlined above, detailed discussion of this work is presented in chapter 7.

Table 3. Experimental studies investigating attention bias in insomnia using information processing paradigms

Authors	Participant characteristics	Paradigm	Findings
Lundh, Froding, Gyllenhammar, Broman & Hetta (1997)	20 patient with primary insomnia and 20 controls, matched for age, sex and education level.	Stroop task	Repeated measures ANOVA examined each of the three stimulus types. The trial effect in each analysis was significant: sleep words [$F(1,37) = 22.4$; $p<0.0001$], physical threat words [$F(1,37) = 7.12$; $p<0.05$], and colour names [$F(1,37) = 163.53$; $p<0.0001$], i.e. both insomniacs and controls responded more slowly to sleep words physical threat words and colour names than to their matched control stimuli. There was no main effect of group with regard to any stimulus type and contrary to expectations, there was no significant interaction between group and sleep words [$F(1,37) = 0.21$].
Taylor, Espie & White (2003)	33 adults (23 F/ 10 M; mean age 47 years) with sleep-onset insomnia subsequent to cancer diagnosis. Mean time since diagnosis for the acute insomnia group was 2.0 months and was 14.3 months for the persistent insomnia group	Stroop task	Independent samples *t*-tests indicated no significant difference between the two groups for the cancer interference index ($t = 0.91, p = 0.37$) but there was a significant difference on the sleep interference index ($t = -2.44, p = 0.02$). Both groups demonstrated interference for cancer words relative to neutral words, but only the persistent group exhibited interference for sleep words. Groups did not differ significantly on pre-sleep cognitive or somatic arousal (PSAS) and they used similar thought control strategies (TCQ: distraction, re-appraisal, social control).
MacMahon, Broomfield & Espie (2006)	63 adults (35 F/ 28 M; mean age 25 years). across three experimental groups (primary insomnia (PI), delayed sleep phase syndrome (DSPS), good sleeper (GS)).	Dot probe	Orthogonal contrasts of PI versus DSPS and GS indicated a significant difference ($t = 1.88, p = .03$), suggesting that participants with PI showed a greater attention bias to sleep related words than those with DSPS or GSs. A further contrast between DSPS and GS did not indicate a significant difference between these groups ($t = 1.27, p = .10$), thereby supporting the hypothesis that attention bias plays a fundamental role in the disorder of PI. The possibility of an underlying trend in DSPS responses needs to be further investigated.

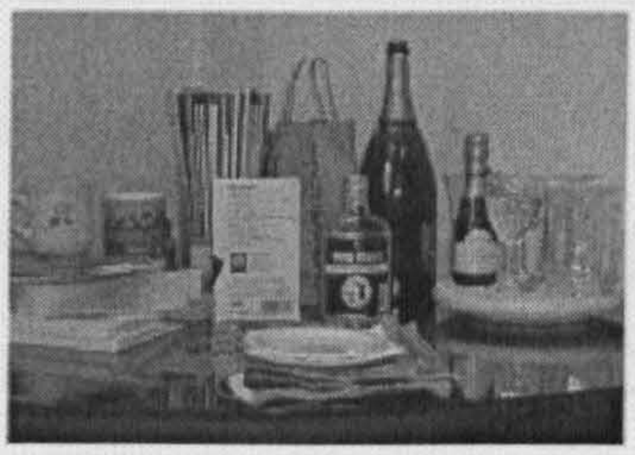
2.1.3 Experimental Studies Completed During Progression to PhD Studies

On reflection of the available research identifying attention bias in PI to sleep related stimuli, and the advantages and disadvantages of the paradigms utilized to demonstrate these effects, we considered the possibility of exploring attention bias with digitised objects (i.e. pictures, rather than words) through a novel attention paradigm.

At this time a unique paradigm, called the Inducing Change Blindness Flicker Paradigm (ICB: Simons, 2000, Rensink, 2002), had shown sufficient sensitivity in detecting attention biases to alcohol stimuli in an alcohol abuser population (Jones et al, 2003). The ICB paradigm is based on the research demonstrating that when a change is made to a visual scene (and the process of change is hidden from view), it is more difficult to detect than might be expected. In the ICB paradigm a single feature of a visual scene is changed between successively repeated brief presentations until the change is detected – essentially the ICB paradigm is a spot the difference task. Change-detection latency, measured by the number of flickers it takes for the change to be identified, is explained by object salience. For example, in the alcohol field, problem drinkers take fewer flickers to detect an alcohol-related change and are faster to detect such changes than control subjects (Figure 2 gives an example of the original Flicker experiment by Jones et al. 2003).

Figure 2. The Original ICB experiment; Original stimulus and the two versions of 'changed stimulus'; alcohol-neutral (video) and alcohol-related (whisky bottle)

Originating stimulus, OS



Alcohol-neutral change, CS

Alcohol-related change, CS

Taken from JONES, B. T., JONES, B. C., SMITH, H. and COPELY, N. (2003) A flicker paradigm for inducing change blindness reveals alcohol and cannabis information processing biases in social users. *Addiction*, **98**, 235-244. Printed with permission from the authors.

Tracking back to chapter 1, I discussed the behavioural perspectives of insomnia that posit, 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. On review of this we proposed that the ICB paradigm might be well suited to investigating the influence of the bedroom environment on sleep.

Thus in 2003/4 our laboratory group suggested that if attention is implicated in the development of persistent insomnia it might be expected that a systematically changing attention bias might be observed, not just at the clinical pole. In our ICB study (Jones et al. 2005), 192 participants (mean age 32 years) were selected for this totally between subjects experiment. Participants completed the 15-minute ICB task, after which they were assessed for sleep quality using the Pittsburgh Sleep Quality Index (PSQI). Importantly, therefore, retrospective group assignment was blind to the dependent variable of the analyses, change detection latency. The hypothesis tested was that participants with PSQI-indicated sleep problems will detect a change made to one of a collection of sleep-related objects quicker than a change made to one of a collection of neutral objects, as compared with participants with fewer PSQI-indicated sleep problems. An entirely between subjects design was adopted with the following three factors: nature of change to be detected (two levels: sleep-related and neutral), gender (two levels: male and female), and PSQI-indicated sleep quality (three levels: good, moderate and poor). The dependent variable was change detection latency. The results revealed significant differences in change detection latency between Poor, Moderate and GS for the sleep-related change. Only the poor sleepers, who detected sleep-related change quicker than neutral change, demonstrated selective attention bias for sleep salient stimuli. Moderate sleepers showed a trend in the same direction.

By contrast, GS detected the change with the neutral objects significantly quicker. Hierarchical regression was then applied to test the relationship between change detection latency and a continuous representation of the global PSQI score. This evidenced a systematically changing effect of sleep quality upon attention bias, independent of age, gender and depressive symptom level.

It is this original ICB experiment that I consider as the foundations for my PhD research. The uniqueness of the paradigm and the sensitivity to detecting attention bias provide good opportunity to test more specific populations. Furthermore, the ICB paradigm escapes the shortcoming of the Stoop and Dot-probe paradigms previously discussed.

The chapter 3 provides a clearer explanation of the specific aims of this PhD research and an introduction to the specific methodologies utilized.

CHAPTER 3

AIMS & OBJECTIVES

3.1.1 Introduction to PhD Studies

As discussed in chapter 2, the accumulating evidence leans toward the existence of attention bias in PI. However, there are self-confessed limiting factors to each of the reported studies. First, MacMahon et al. acknowledge that the dot –probe task has been described as a 'relatively fragile index of anxiety related attentional biases in non-clinical studies, particularly when using word stimuli that have relatively mild threat value' (Mogg et al. 2000, pp 1074). Indeed the possible lack of emotional salience within the word base stimuli in this experiment could have minimised the overall vigilance effect. The authors suggest that future studies might consider the use of a pictorial dot-probe paradigm to maintain the degree of experimental control available from the dot-probe, but maximise the possibility of uncovering processing bias by increasing the salience of stimuli (Mogg et al. 1999). Second, within our own experiment (Jones et al. 2005) we acknowledged that the findings relate, only, to one single sleep-related and neutral-related change, and that this might compromise generalising the findings and conclusions to other bedroom and neutral objects. Although our methodologies report that extreme care was taken to ensure that both sets of objects were highly representative of their set, we do suggest that future research should extend the range of objects used.

3.1.2 Aims of PhD Studies

Thus, the following body of research aims to expand and strengthen the available evidence for the existence of attention bias in PI, by investigating attention processes though utilizing a number of different attentional paradigms, some which are novel to the sleep domain. Indeed this research aims to

- vigorously test attention bias in PI
- identify the components of attention that may account for any AB effects e.g. engagement, shift and disengagement discussed previously by Fox et al. 2001.
- provide insight into which stimulus types are driving AB in PI.

The following 4 chapters explain in detail the methods employed, results obtained, and the reasons behind the progression from one research question to the next. The table 3, below, gives a brief overview of each experimental chapter.

Table 3. Overview of the Experimental Chapters Comprising this PhD

CHAPTER/EXPERIMENT and TILE	RESEARCH QUESTION	PARADIGM
Chapter 4 – Experiment 1 **An Investigation of Attention Bias In Psychophysiologic Insomnia**	When compared with GS and DSPS, do PI display an attention bias towards sleep-related stimuli as compared to sleep-neutral stimuli ?	Inducing Change Blindness Flicker Paradigm
Chapter 5 – Experiment 2 **An Investigation of Attention Bias, to Individually Presented Pictorial Stimuli, in Psychophysiologic Insomnia**	Do individually presented pictorial sleep-related stimuli capture an attention bias effect in PI?	Attention Competition Task
Chapter 6 – Experiment 3 **An Investigation into the Components of Attention (Engagement/Disengagement) Driving Attention Bias, To Sleep-Related Stimuli, In Psychophysiologic Insomnia**	Do sleep-related stimuli modulate the engagement component of attention and/or the disengagement component of attention?	Modified Pictorial Posner Paradigm
Chapter 7 – Experiment 4 **An Investigation of Delayed Disengagement Of Attention from 'Negative' Sleep-Related Stimuli**	Do both positive and negative sleep-related stimulus types generate attention bias responses in PI?	Modified Semantic Posner Paradigm

3.1.3 Strategy for Methodology

The series of experiments that comprise the scientific work within this PhD have both shared and unique features. For example, they share a common paradigm (computerised appraisal of Attention Bias, AB), but differ in their selection of the probe task (Inducing Change Blindness Flicker Paradigm (ICB), Attention Competition Task, Modified Semantic and Pictorial Posner Paradigm). Other aspects of methodology also reflect this generic plus specific mix. Consequently, it may be helpful to outline first, the core generic methods that were consistent across studies; and second, to report the experimental studies sequentially thereafter, making careful reference to specific methodological issues in relation to each. Hopefully this approach will also highlight the ways in with the experimental programme developed over time in response to the research findings.

3.2 CORE GENERIC METHODOLOGIES

3.2.1 Participant Selection

All data were derived form from students and staff at the University of Glasgow. In each study the appraisal of individual sleeping patterns was conducted in three phases.

i. Phase I

Phase I of screening was based upon the information gained from an initial mass advertisement email, which, after giving a brief introduction about the Glasgow Sleep Lab asked five specific questions…

1) Are you someone who struggles to sleep at night? 2) Once asleep do

you regularly wake during the night? 3) How many hours on average do you sleep per night? 4) Would you describe yourself as a lark or an owl? 5) Are you a good sleeper, who falls asleep as soon as your head touches the pillow and wakes up feeling refreshed in the morning? The email concluded by stating… *if you feel you would gain from being part of a sleep experiment and would like to learn a little more about your sleeping patterns, and ways to improve them, we would like to hear from you. Please send information about your sleep pattern based on the questions above.*

ii. Phase II

Respondents whose symptoms reflected traits similar to PI and DSPS were subsequently emailed for a second time (Phase II) to obtain further details of their sleep patterns. For PI specific traits included;

- Difficulty initiating sleep >3 nights per week
- < 7 hours of sleep per night
- Difficulty initiating sleep
- Awakening during the night/difficulty maintaining sleep
- Early morning awakenings
- Unhappy about sleep

For DSPS specific traits included;

- Difficulty initiating sleep
- Late sleep onsets
- Trouble rising from bed in the morning
- Normal sleep duration (approx 7-8 hours)

- Few awakenings during the night

This second email asked more specific questions.... *how many minutes does it take you, on average, to fall asleep at night...once asleep do you wake at regular intervals throughout the night...How long do you predict you are awake during the night...On average how many hours of sleep do you get per night...Do you view your sleep as a problem...What time do you get up in the morning... Do you feel refreshed?* Those who responded to this second email, and were assessed to be potentially suitable were invited to take part in the experiment concerned. Examples of responses that were assessed to be potentially suitable for the PI group were;

- " I can lie awake after going to bed for up to 2 hours"
- "I wake up about 4 or 5 times most nights and lie awake for hours"
- "I wake up around 4am and cannot get back to sleep"
- "I only sleep around 4 hours a night"
- "I lie awake thinking about how tired I am but just can't sleep. I constantly feel exhausted"

Examples of responses that were assessed to be potentially suitable for the DSPS group are;

- "I go to bed about 11pm but don't sleep until about 2am. Once I'm asleep however, I sleep all night for about 8 hours".

- I'm not tired until the early hours of the morning but struggle to get up in the morning"

- "I struggle to get out of bed in the morning but I'm just not tired at night. Its usually about 1am before I feel the need to sleep"

iii. Phase III

The third and final phase (Phase III) of screening took place after each experiment, and applied rigorous criteria and prospective diary and actigraphic assessment to confirm group allocations. The procedure for Phase III is described below under section **3.2.2**, and detailed descriptions of each assessment tool utilized is reported in section **3.2.3**.

3.2.2 Inclusion/Exclusion Criteria

Participants were required to meet combined DSM-IV and ICSD-R criteria for primary insomnia of the PI type or DSPS, and those with PI were required to score > 5 on the PSQI, with a sleep disruption of greater than 6 months. PI exclusion criteria included active psychological or drug interventions for sleep problems, or when sleep disorder was suspected as being the result of

substance misuse or physical ill health. The same exclusion criteria applied for DSPS. Good sleepers were required to score < 5 on the PSQI, report themselves as being 'good' sleepers, and meet no criteria for a sleep disorder at the present time or in the past. For all three participant groups, scoring above the cut off markers for depression (BDI SF - 5) resulted in exclusion from analyses. Spielberg State Trait Anxiety Inventory was used as a descriptive, not diagnostic, measure and ensured that the PI populations recruited for this research were similar to those recruited for previous PI research (Jones et al. 2005). No participant was excluded based on high STAI scores as this is a common feature of the PI population. Actigraphy was used to confirm participants' subjective account of their sleeping complaint and to aid in the differentiation between PI and DSPS in those reporting sleep difficulties. Table 4 provides an overview of the inclusion/exclusion criteria.

Table 4. Summary of Inclusion/Exclusion criteria for PI, DSPS and GS

GROUP	INCLUSION	EXCLUSION
Primary Insomnia	Meet combined DSM-IV and ICSD-R criteria for PI PSQI score >5 BDI score <5 Self reporting sleep problem Concurrence of self-report with actigraphy data Sleep problem > 6 months	Psychological or Drug intervention for sleep disturbance Alcohol/Drug misuse Physical ill health
Delayed Sleep Phase	Meet combined DSM-IV and ICSD-R criteria for DSPS BDI score <5 Concurrence of self-report with actigraphy data	Psychological or Drug intervention for sleep disturbance Alcohol/Drug misuse Physical ill health
Good Sleep	PSQI score <5 Self reporting good sleeper BDI score <5	Alcohol/Drug misuse Physical ill health

3.2.3 Procedure for Diagnostic Ascertainment

i. Assessment of Sleep

First, each participant was interviewed to evaluate his/ her typical sleeping patterns. The interview proposed questions relating to the DSM-IV and ICSD-R criteria for PI and DSPS, following the GGSRL structured interview format (Espie, 2000; Morin & Espie, 2003 - see Appendix E).

Second, the Pittsburgh Sleep Quality Index (PSQI; (Buysse et al. 1989) was completed (Appendix F). The PSQI provides a reliable, valid, and standardized measure of sleep quality; 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. Recent, independent study has validated this cut off and confirmed reliability (Cronbach's α = .85, test re-test r = .84; (Backhaus et al. 2002).

Third, participants wore an actigraph and completed a standard sleep diary (Espie & Tweedie, 1991) for seven nights following the experiment. The sleep diary is a short questionnaire that is completed upon wakening (Appendix G). This questionnaire provides important information about the participant's personal subjective account of their previous nights sleep. The sleep diary provides information to the experimenter about the participants subjective Sleep Onset Latency (SOL – how long it takes participants, in minutes, to fall asleep at night), Total

Sleep Time (TST – How long, in hours, did the participant sleep in total during the night) and Wake Time After Sleep Onset (WASO – How long the was the participant awake for during the night, in total). The actigraph is a small, non-intrusive watch-like device, which records objective rest/activity periods based on wearer movements (Appendix H). For the use of actigraphy in insomnia patients, epoch by epoch agreement rates range from 78% to 90% (Jean-Louis et al. 1997, Sadeh et al. 1995) The inclusion of actigraphy was to help determine the sleep/wake (circadian) patterns of each participant (Sadeh et al. 1995) rather than to estimate sleep continuity variables. Therefore, this measure assisted differential diagnosis of PI and DSPS and confirmed group status. In particular, we used van Someren et al's (Van Someren et al. 1999) non-parametric circadian rhythm analysis (NPCRA) to calculate the L5 component (onset of the first 5 hours of lowest movement period) , and the M10 component (onset of the first 10 hours of highest movement). This analysis gives an estimate of circadian sleep phase onset.

ii. Assessment of Psychopathology

The revised version of the Spielberger State and Trait Anxiety Inventory (STAI; (Spielberger et al. 1983), a reliable and valid scale, was completed (Appendix I). Alpha coefficients (STAI-S α =. 93 & STAI-T α = .90) reflect strong internal consistency. Construct, concurrent, divergent and convergent validity have been demonstrated (Spielberger, 1970). Additionally, the Beck Depression Inventory – Short Form (BDI-SF: Beck & Steer, 1987) is a 13 item self-report form covering symptoms

of depression. The BDI (21 scale) demonstrates high internal consistency, with alpha coefficients of .86 and .81 for psychiatric and non-psychiatric populations, respectively. The 13-item short form, adopted for this experiment, has shown similar reliabilities (Beck et al. 2000) (Appendix, J).

3.2.4 Apparatus

All experiments were implemented using a Dell optiplex GX270 laptop and the experiment-generation package SuperLab Pro 2.02 (Cedrus Corporation). The size of the screen was 28cms diagonal with stimuli positioned centrally; the viewing distance was approximately 45cm.

3.2.5 Test Location

Participants were tested in an assessment room in the Department of Psychology, University of Glasgow. The room contained a single desk with a Dell optiplex GX270 laptop on top. The participants' chair was placed 40com from the laptop, and the experimenter's chair was positioned adjacently to the participants' chair. All experiments were performed on the same laptop. Additionally, all aspects of the experiment were performed within this testing room, with the door securely closed to ensure silence during testing.

At the end of testing the participants had the opportunity to discuss their sleeping patterns and were offered a copy of the Good Sleep Guide.[2] (Appendix K)

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[2] Prepared by Colin A. Espie for guidance report (NPAC, 1993) and now recommended by the British Sleep Society.

3.2.6 Data Analysis

All data derived from the study (questionnaire, AB score, actigraphy) was stored in the statistical programme SPSS for windows (version 12.0.1). At the beginning of testing all participants were assigned a number e.g. the first participant tested was number 001. All subsequent information gained from this participant was stored under this number in SPSS. Names were not stored on our database.

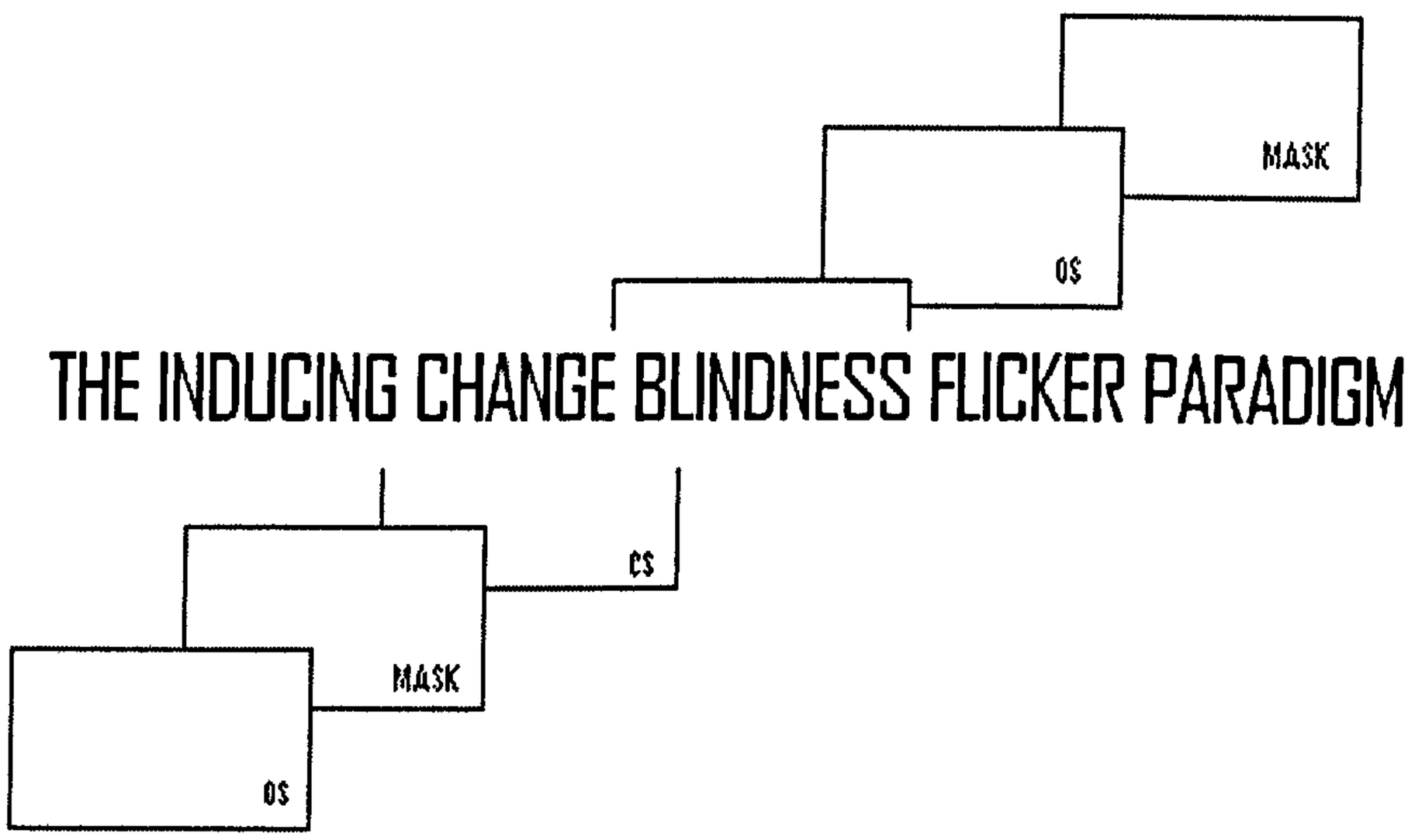
All questionnaire data were hand scored by the primary investigator and entered under the appropriate heading in the SPSS database. During the interview sessions, notes of the participants' responses were made in the corresponding boxes (again, see Appendix E) and a preliminary group allocation was made at the end of the session. However, the notes were later re-read to ensure concurrence with the initial grouping. The actigraph devices used were Actiwatch AW4 (Cambridge Neurotechnology and the data downloaded using the Actiwatch Sleep Analysis 2002, version 4.04. In particular, Van Someren et al. (1999) non-parametric circadian rhythm analysis (NPCRA) was used to calculate L5 (first 5 hours of lowest movement period) and M10 (first 10 hours of highest movement period) components. All data was then entered into the SPSS spreadsheet.

During the first meeting with each participant the initial questionnaires were completed i.e. STAI (state and trait), PSQI, BDI, thus there was no missing data on these measures. On the second meeting however, participants were asked to bring with them the sleep diary that had been completed over the previous week. On occasions where data had not been completed after a certain night, an average was taken from the available nights and entered into the database. It is

worth noting however that this was a rare occurrence as at the initial meeting session participants were made aware of the importance of the diary data and encouraged to complete it as soon as waking. All data was present for the actigraphy measure.

Analyses of all data were performed on the statistical package Experstat (1991,1997, Robin Stevens, version 2.30). Initial ANOVAs were performed, followed, where appropriate, by Scheffe Post Hoc analyses.

CHAPTER 4



THE INDUCING CHANGE BLINDNESS FLICKER PARADIGM

EXPERIMENT 1: AN INVESTIGATION OF ATTENTION BIAS IN PSYCHOPHYSIOLOGIC INSOMNIA

4.1 INTRODUCTION

As previously discussed, three of the four available attentional bias probe tasks have been reported to discriminate cognitive arousal in insomnia. Indeed, the most recent experiment to date, Jones et al. 2005, utilizing an ICB flicker paradigm, presented convincing data that evidenced a systematically changing effect of sleep quality upon attentional bias that was independent of age, gender and depressive symptom level. Thus, this paradigm is a useful research tool.

4.2 METHODS

4.2.1 Aims and Hypotheses

Experiment 1 extends the research in this field in two ways. First, this ICB experiment uses novel sleep-related and sleep-neutral objects to undergo a 'change' in the sequence. Jones et al. (2005) measured the response to one sleep-related change and one sleep-neutral change; I wanted to rule out the possibility of an idiosyncratic effect and to improve generalizability. Second, an additional, clinical control group of DSPS is included. DSPS involves initial insomnia, similar to sleep-onset psychophysiological insomnia, but with no presumed psychological mechanism. Thus, DSPS would not be expected to exhibit cognitive arousal as an explanatory mechanism for their continued wakefulness. Comparison with normal sleepers, without a clinical control, leads to problems in

testing the specificity of insomnia phenomena. Therefore, inclusion of a DSPS group will permit the assessment of differences in selective attention between sleep disorders that are behaviourally similar in terms of sleep initiation problems.

It is hypothesised that;

1) people with psychophysiological insomnia (PI) will detect a sleep-related change significantly quicker than good sleepers (GS) or DSPS participants;

2) with a neutral change there should be slower response in PI, because their attention will be drawn to the 'sleep area' of the visual scene, and will have to shift to notice a neutral change;

3) there will be no significant difference between detection latencies to sleep-related versus sleep-neutral changes by the two control groups, GS and DSPS; and

4) there will not be significant differences in detection latencies between GS versus DSPS, at either level of change.

4.2.2 Design

A 2 (experimental condition) x 3 (group) entirely between-participants design was employed. Reaction Time (RT) to response acted as the dependent variable. The necessary confirmation process (making explicit the semantic nature of the stimulus carrying the change) means that a within participant factor cannot be implemented in ICB experiments. This is because any explicit

knowledge carried over by participants as a result of the confirmation process would compromise the main objective, exploring their implicit knowledge of the stimuli contained in the scene (Jones et al. 2003). Therefore, each group (GS, PI, DSPS) was split in half at random (n=15) to represent the nature of the change introduced in the photographic image (sleep-related, or sleep-neutral).

Allocation of sleep-related stimuli to the left and sleep-neutral to the right side of the visual field was chosen at random. This orientation of the stimulus presentation was kept constant in all conditions. Including mirror reversal to control for differences in scene processing, due to left-right placement, was not considered necessary because no differences have found been between normal and reversed scene processing in previous ICB flicker paradigm experiments (Jones et al. 2003).

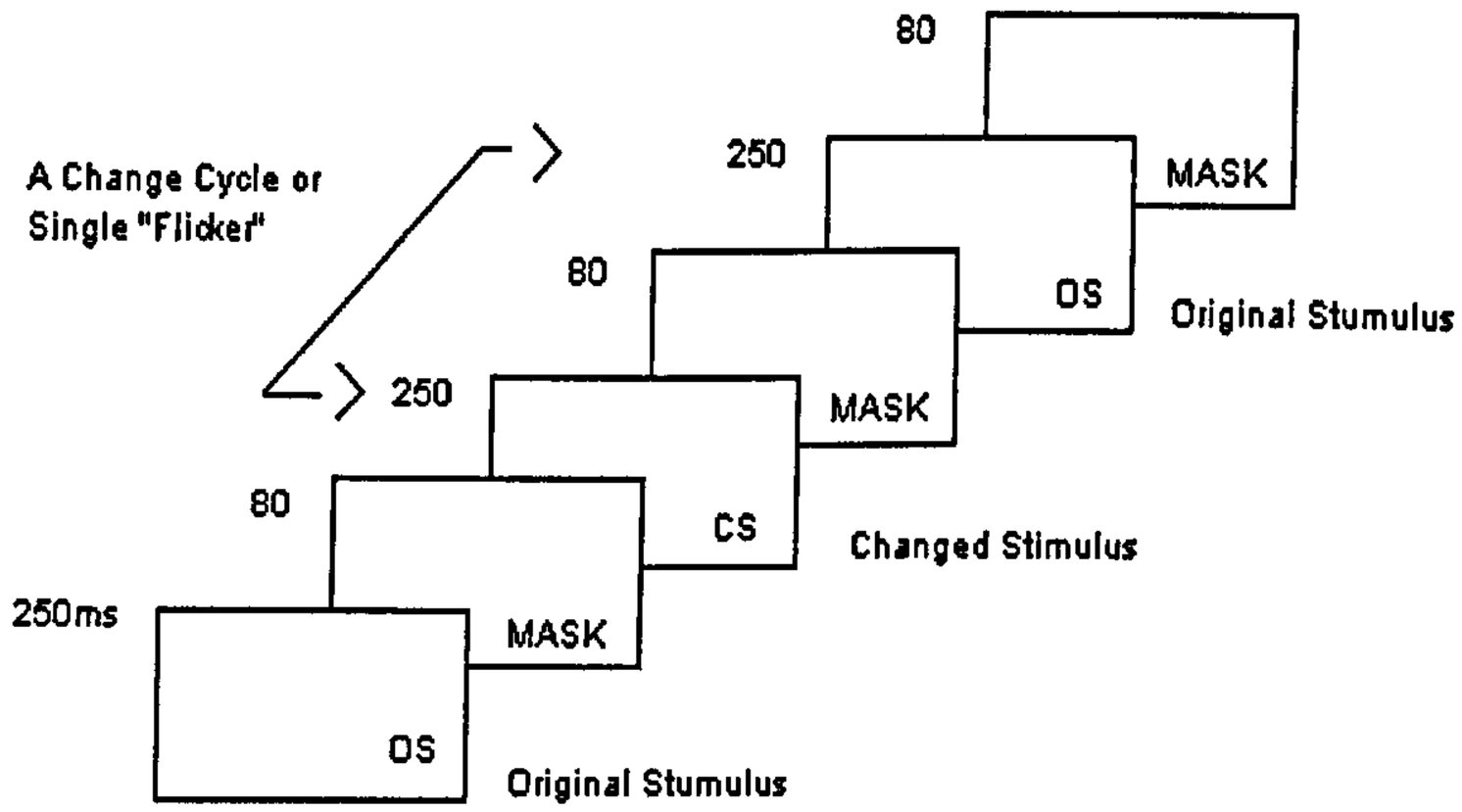
4.2.3 Participants

Thirty PI, thirty, DSPS and thirty GS were included in analyses. Selection of participants' followed the protocol described in chapter 3. At Phase I, two hundred and fifty three individuals responded. Of these 253 respondents, 189 reported symptoms similar to PI and DSPS and were subsequently emailed for a second time (phase II) to obtain further details of their sleep patterns. From the 189 individuals emailed in phase II 157 responded, and 97 were assessed as potentially suitable (refer to chapter 3, pages 63-66 for overview) and subsequently completed the ICB procedure. The third and final phase (phase III), after the experiment, resulted in 90 of the 97 tested being included in analyses.

4.2.4 Experimental Protocol and Apparatus

The flicker ICB paradigm was employed. An originating stimulus (OS) was presented for 250 ms - then a mask for 80 ms - then the changed stimulus (CS) for 250ms - and finally a mask for 80 ms (Figure 3). This 4-presentation cycle was repeated seamlessly until change-detection. Thus, the combined number of transitions between OS-CS and CS-OS (the 'flickers' to change-detection) was the dependent variable.

Figure 3. A Flicker Paradigm for Inducing Change Blindness illustrating a change cycle or single "flicker". OS = original stimulus: CS= changed stimulus.



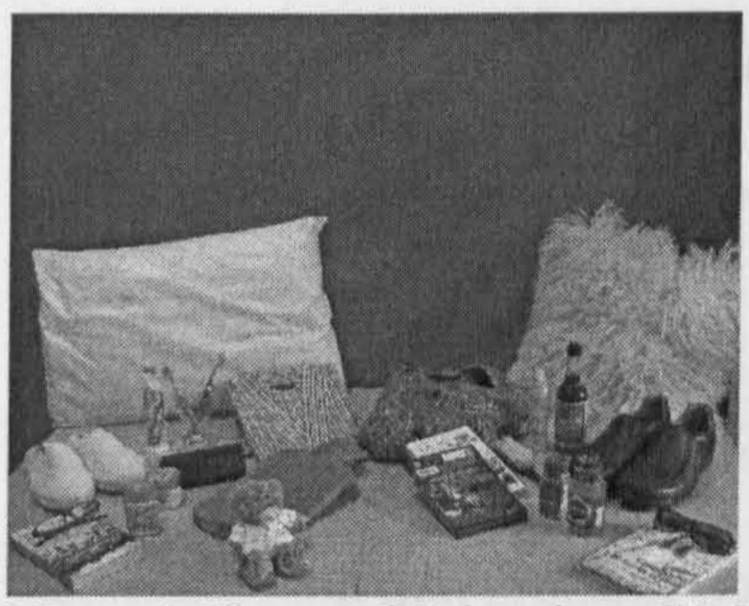
Sleep-related stimuli were chosen from a questionnaire distributed, randomly, to 60 individuals (Appendix L). This questionnaire was devised for the Jones et al. (2005) study. People were asked to list 5 or more objects that they associated with sleep and going to bed. Evaluation of the lists yielded 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, 5.6) was used, and paired with an entirely sleep-neutral item (a mug).

A different flicker pair of stimuli was used for each of the two levels of the factor; nature of change (sleep-related and neutral). Each pair contained the same OS comprising 7 sleep-related objects and an equal number of neutral objects arranged in two collections on either side of the scene midline. The second stimulus (CS) of each pair was identical to the OS but for one small change: a sleep-related change (removing the teddy bear) or a neutral change (removing the mug). The two stimuli of a pair were then presented in continuous succession (each replacing the other) until the change was detected. A brief 'mask' was inserted in between the flicker pairs to suppress visual transients. The experiment was implemented using a Dell optiplex GX270 laptop and the experiment-generation package SuperLab Pro 2.02 (Cedrus Corporation). The size of the screen was 28cms diagonal with stimuli positioned centrally; the viewing distance was approximately 45 cm. Stimuli were full colour photographs (1280 x 960 pixels) taken in natural daylight. Figure 4 shows the originating stimulus, and the two changed stimuli; one with the sleep-related change (the teddy bear is taken away) and the other with the sleep-neutral change (the mug is taken away).

Figure 4. Grey scale versions of the full colour used; original stimulus (OS) and the two changed stimuli for each of the two levels of the nature of change factor – sleep-related change (CS-S) and neutral change (CS-N)

Original Stimulus, OS



Neutral Changed Stimulus, CS-N
Stimulus, (mug removed)

Sleep-Related Changed
CS-S (teddy bear removed)

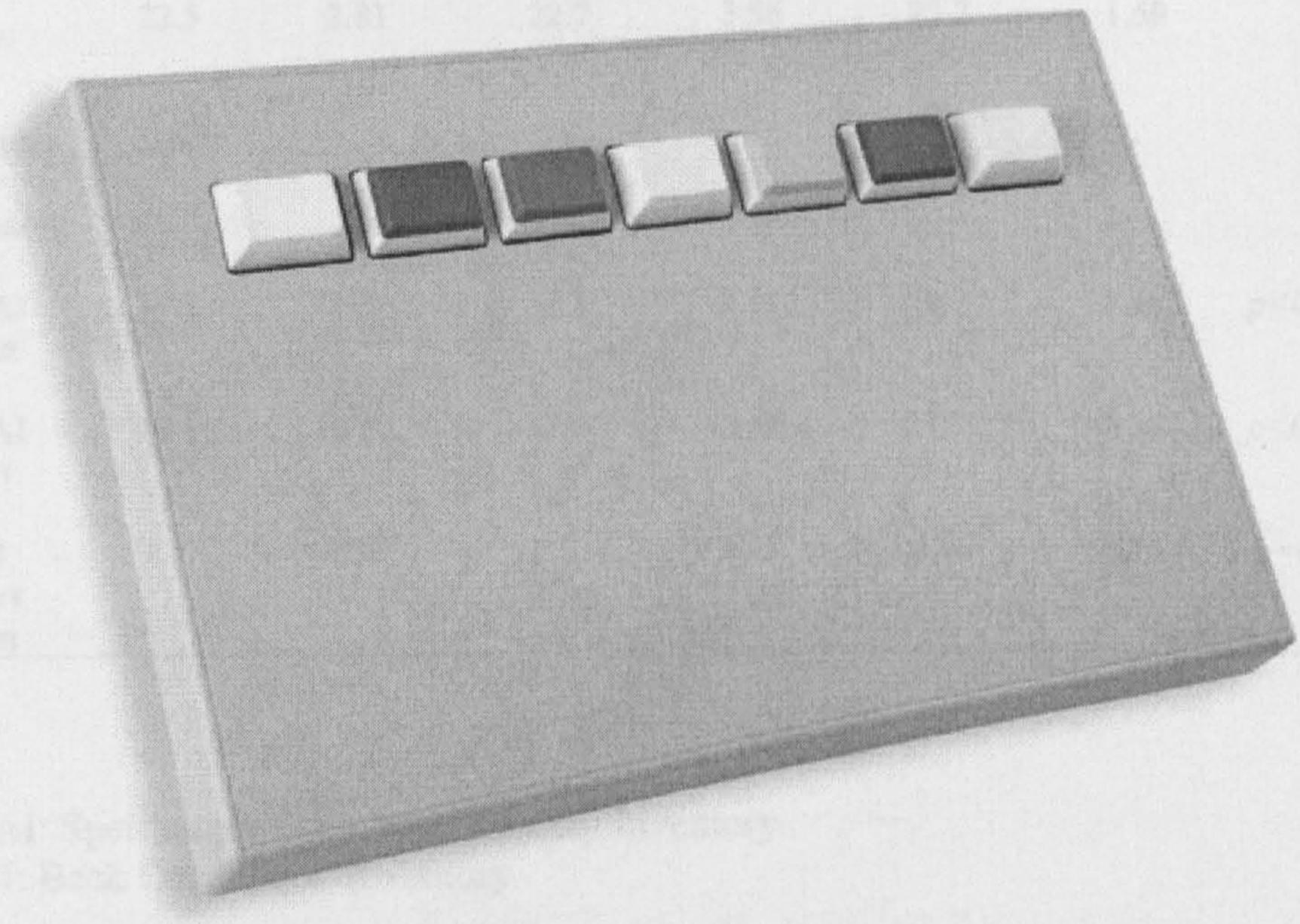
4.2.5 Procedure

As previously describe in chapter 3, contact was first made with participants by email, inviting individuals to contact the first author with details of their sleeping patterns in answer to a brief set of questions. These questions enabled the experimenter to screen and select the responses that best resembled the traits of the various sleep groups involved in the study, invite the suitable candidates to give more specific information about sleep quality and subsequently invite them to take further part in the study. Thus, group allocation was 'largely known' to the experimenter before testing commenced.

To ensure an equal distribution of participants across the experimental conditions (sleep and neutral), assignment to conditions followed an 'alternating' system. Thus, if one participant completed the 'sleep-related condition', the following would complete the 'sleep-neutral condition'. Furthermore, an identical instruction protocol was presented to participants upon arrival, regardless of their group allocation. To begin, participants pressed any button on the response button box (Figure 5) to display instructions which said that (when they pressed it again) they would "*see a scene on the screen that would be switched on and off repeatedly – each appearance and disappearance lasting less than a second*". They were instructed that they had to "*spot a change made to the visual scene at some point in the series of 'flickers' and to indicate this detection by immediately pressing the button on the response box*". After the participants had detected and responded to the change, they were asked to "*confirm what the change was*". Only those who had correctly identified the

change were retained as having completed the task[3]. They were verbally asked if they understood the instructions and were invited to continue (without additional practice). The experiment then commenced. After completion of the flicker task each participant immediately underwent the third and detailed assessment phase. This comprised of three components described in chapter 3.

Figure 5. Illustration of the RB-730 Response Box (Cedrus Corporation)



[3] In this study all participants who completed the ICB flicker paradigm identified the correct change in the scene.

4.3 RESULTS

4.3.1 Demographic and Clinical Data

Table 6.Demographic and clinical summary data (mean;SD) for PI, DSPS and GS groups participating in the ICB Flicker Paradigm Task.

	Primary Insomnia N = 30		**Delayed Sleep Phase Syndrome N = 30**		**Good Sleep N = 30**		**Between Group Analyses**
	M	***SD***	***M***	***SD***	***M***	***SD***	
Age	22.5	2.81	22.7	3.56	23.2	1.69	NS
Gender (No female)	16	-	14	-	15	-	NS
STAI state	37.2	7.83	32.8	8.74	24.3	5.35	*p<0.0001*
STAI - trait	48.1	10.52	37.9	11.40	27.3	9.15	*p<0.0001*
BDI – short form	4.7	3.52	2.3	3.32	2.9	2.36	*p=0.053*

STAI: Speilberger State Trait Anxiety Inventory
BDI: Beck Depression Inventory

Inspection of subjective and objective sleep quality data revealed that 7 of the original participants did not meet inclusion criteria for PI, DSPS or GS; 5 cases due to sleeping disorders other than PI or DSPS, one case due to the patient receiving medical intervention for sleep disruption and one due to lack of sleep caused by a broken collar-bone. On five occasions, individuals believed to have PI from the general information given in response to the email advertisement, were subsequently re-allocated to the DSPS group on the basis of the more thorough interview, questionnaire measures and actigraphy data. The experimental population as a whole consisted of 45 females and 45 males with an average age of 22.8 years. Table 6 shows the demographics of the experimental population for each sleep quality group.

Table 6 also presents summary scores for the other clinical questionnaire data. There was a significant effect of group at both levels of STAI; Trait: [F (2,87) = 30.12, p<0.0001] State: [F (2,87) = 23.43, p <0.0001]. Scheffe post hoc tests revealed that PI were generally (trait) and situationally (state) more anxious than GS, *p<0.0001 and p<0.0001*, respectively. Similarly, on both STAI scales, PI scored significantly higher than DSPS *p<0.001* trait, *p<0.001* state, respectively. DSPS scored higher on the state measure of anxiety, *p<0.001*, than GS, but were not significantly different on trait measures of anxiety, *p=1.92*. There was no significant main effect of group for the BDI data, which revealed low mean scores in all groups [F (2,87) = 3.04, p = 0.053], although the trend in the data was for PI to score higher than either GS or DSPS.

Table 7. Sleep Summary Data (mean;SD) for PI, DSPS and GS groups participating in the ICB Flicker Paradigm Task.

	Primary Insomnia		**Delayed Sleep Phase Syndrome**		**Good Sleep**		**Between Group Analyses**
	M	***SD***	***M***	***SD***	***M***	***SD***	
PSQI	9.5	2.4	4.8	3.2	2.6	1.7	*p<0.0001*
Diary TST (hrs/mins)	4.83	1.83	8.33	1.81	8.27	1.46	*p<0.0001*
Diary SOL (mins)	47.63	17.85	17.57	9.09	7.9	4.57	*P<0.0001*
Actigraphy L5 (24h clock)	01:10	0.9	04:00	1.9	N/A	N/A	*p<0.0001*

PSQI: Pittsburgh Sleep Quality Index
TST: Total Sleep Time
SOL: Sleep-Onset Latency
L5: Onset of lowest 5 hours of motor output

4.3.2 Sleep Data

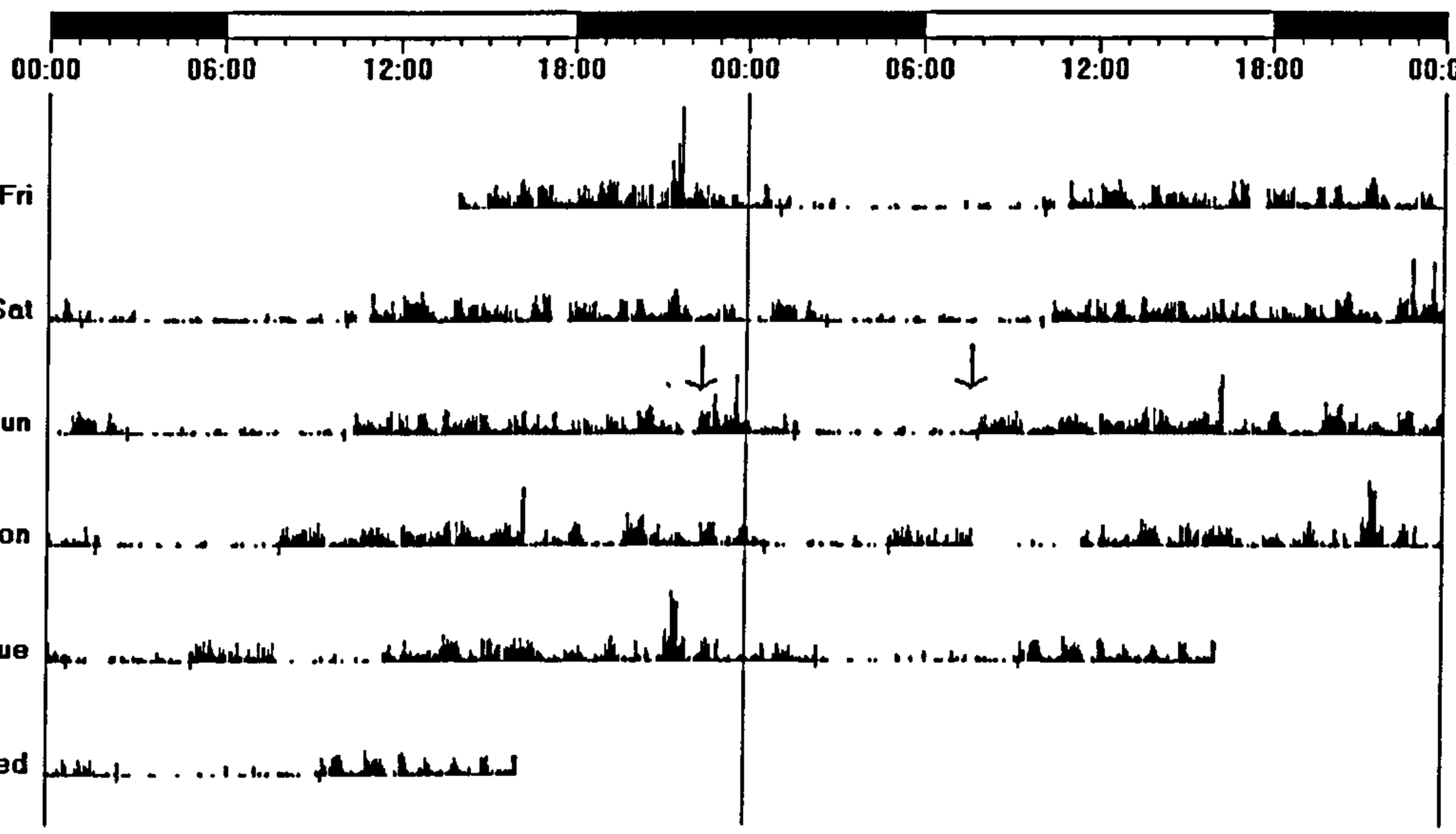
Table 7 summarises mean and standard deviation data for the PSQI, and selected sleep diary and actigraphic measures. Analyses revealed a significant effect of group at the level of PSQI [F (2, 87) = 61.07, p<0.0001]. Scheffe post hoc analyses revealed that PI scored significantly higher than both GS *p<0.0001*and DSPS, *p<0.0001*, and DSPS scored significantly higher then GS, *p<0.01*.

On the sleep diary, total sleep time (TST) was significantly different between the three sleep quality groups [F (2,87) = 41.22, p<0.0001], with PI participants reporting less than 5 hours sleep, compared with greater than 8 hours for DSPS and GS (*p< 0.0001* and *p< 0.0001* respectively). There was no difference in TST between GS and DSPS groups. Sleep onset latency (SOL) was also significantly different between groups [F (2,87) = 88.36, p<0.0001], with PI taking significantly longer to fall asleep than DSPS (*p<0.0001*), and GS (*p<0.0001*), and DSPS taking significantly longer to fall asleep than GS (*p<0.05*).

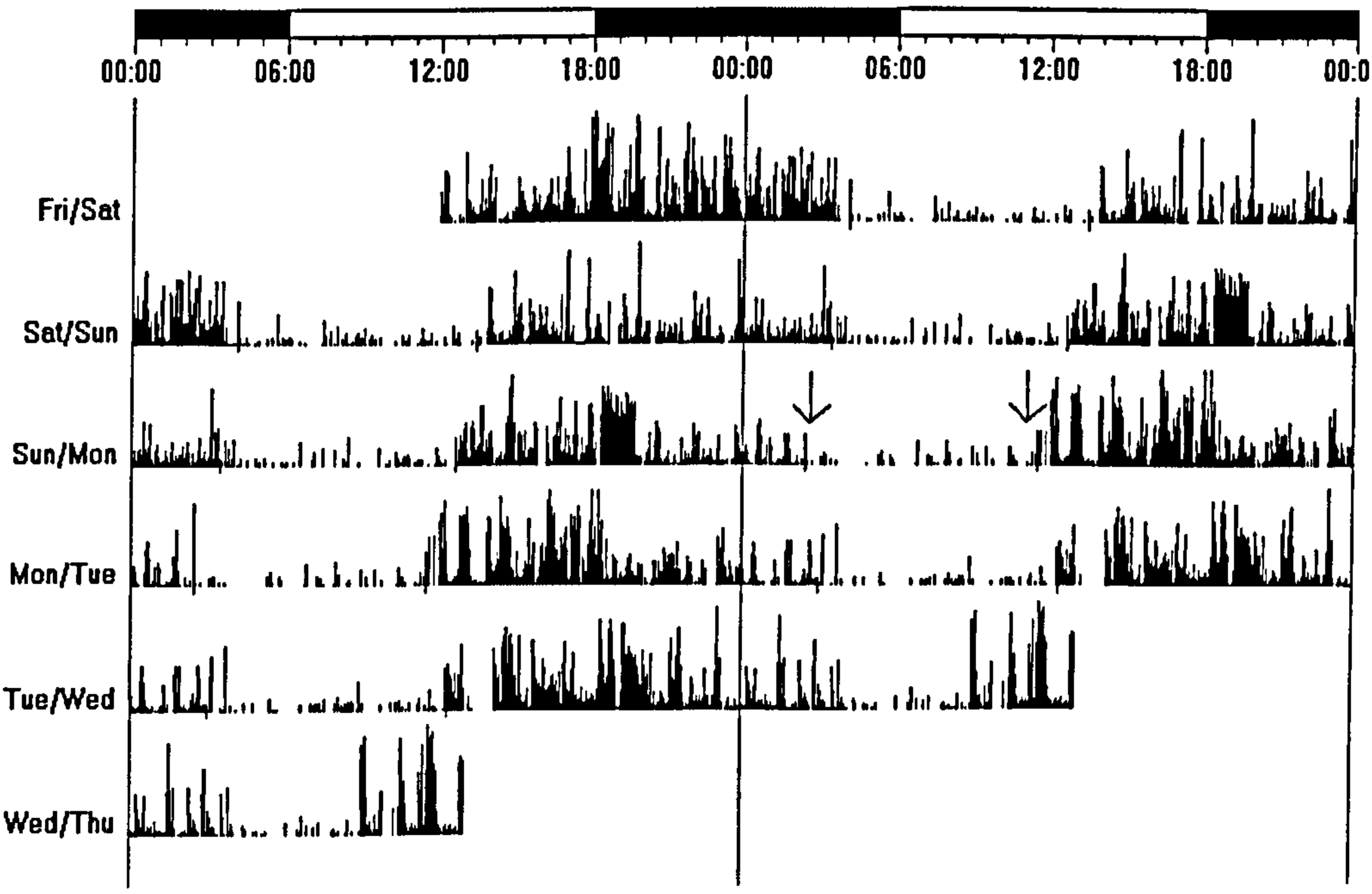
Analysis of actigraphy data using NPCRA software revealed a significant main effect of group on L5 data, [F(1,58) = 45.81, p< 0.0001], with DSPS lowest peak of activity beginning significantly later than PI. These data indicate a sleep-onset phase delay of 2.9 hours in DSPS relative to PI. Figure 6 gives an example of actigraphy data for DSPS and PI.

Figure 6.

a) Actogram of PI actigraphy data (arrows indicate in bed and get up times)



b) Actogram of DSPS actigraphy data (arrows indicate in bed and get up times)



4.3.3 Change Detection data

Hypotheses;

1) **people with psychophysiological insomnia (PI) will detect a sleep-related change significantly quicker than good sleepers (GS) or DSPS participants**
2) **with a neutral change there should be slower response in PI, because their attention will be drawn to the 'sleep area' of the visual scene, and will have to shift to notice a neutral change**
3) **there will be no significant difference between detection latencies to sleep-related versus sleep-neutral changes by the two control groups, GS and DSPS**
4) **there will not be significant differences in detection latencies between GS versus DSPS, at either level of change**

Table 8 summarises the RT means (number of flickers) and standard deviations for each sleep group. To test our hypotheses, a stimulus change/ sleep quality (2 x 3) between participants ANOVA was carried out. As predicted the stimulus change/ sleep quality interaction was significant [$F(2,84) = 51.7$, $p<0.0001$] with a significant effect of change [$F(1,84) = 70.3$, $p<0.0001$] and quality [$F(2,84) = 3.5$, $p< 0.05$]. There was a significant main effect of change at the level of PI [$F(1, 84) = 171.5$, $p<0.0001$] that was not present at the level of GS [$F(1,84) = 0.004$, $p = 0.95$] or DSPS [$F(1,84) = 2.2$, $p = 0.14$]. Significant

main effects of quality were revealed for both the sleep change [F (2,84) = 28.6, p<0.0001] and neutral change [F (2,84) = 26.7, p<0.0001].

Table 8. RT (number of flickers) to categorisation response of PI, DSPS and GS

	Sleep-Related Change		Neutral Change	
	M	***SD***	***M***	***SD***
PI	4.7	2.14	18.2	1.64
DSPS	9.7	3.50	11.2	3.14
GS	12.4	2.99	12.3	3.10

Figure 7. Mean sleep-related and sleep-neutral change detection latencies(No of flickers) for PI, DSPS and GS.

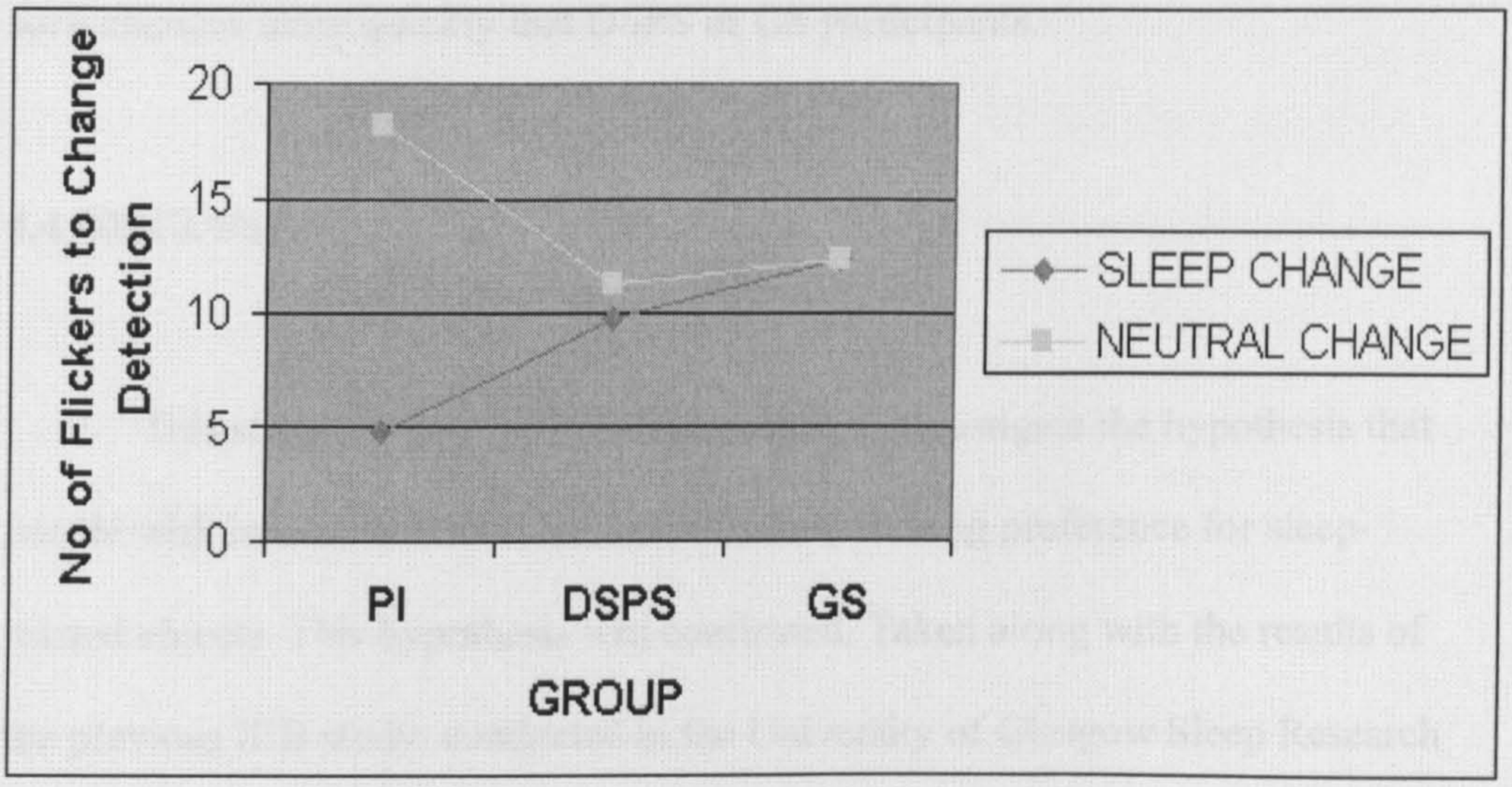


Figure 7 illustrates the tests for simple main effects that permit the following interpretation of the stimulus change/ sleep quality interaction. PI participants detected a sleep-related change significantly quicker than a sleep neutral change, [$t(15) = 13.10, p<0.0001$]. No such differences were observed in either the GS [$t(15) = 0.06, p=0.95$], or DSPS groups [$t(15) = 1.5, p=0.14$]. For the sleep-related change, responses of PI were significantly quicker than GS [$t(15) = 7.5, p<0.0001$] and DSPS [$t(15) = 4.8, p<0.0001$]; and responses of DSPS were significantly quicker than those of GS, [t$(15) = 2.66, p<0.01$]. By comparison for sleep-neutral changes, responses of GS and DSPS were significantly quicker than PI {[$t(15) = 5.7, p<0.0001$]; [$t(15) = 6.8, p<0.0001$]} respectively. No difference was observed between GS and DSPS at the neutral change.

These main effects and simple effects appear consistent with the hypotheses that sleep-related stimuli would be more salient to PI, and thus detected quicker than sleep-neutral stimuli, and that PI participants would detect such changes more quickly that DSPS or GS participants.

4.4 DISCUSSION

This study applied the ICB flicker task to investigate the hypothesis that people with insomnia exhibit an attentional monitoring preference for sleep-related objects. This hypothesis was confirmed. Taken along with the results of the previous ICB study, conducted in the University of Glasgow Sleep Research Laboratory, which used different sleep-change stimuli (Jones et al., 2005), it is suggested that this is not an idiosyncratic finding. These are the first experiments to apply a visual attention task to insomnia, and the show that people with insomnia are selectively attentive to common environmental sleep cues. Indeed, it is worth noting that the sleep stimuli were incidental and innocuous (this study: a teddy bear; Jones et al.: a slipper). They were simply objects that people associate with sleep or preparation for bed, and because people with insomnia have a heightened interest in this domain, they respond selectively to such stimuli when they are presented experimentally. Therefore, although not designed for the purpose of testing conditioned responses, these results lend weight to models of sleep-related arousal conditioning in insomnia; (Bootzin, 1972, Perlis et al. 2001) whilst emphasising the importance of the cognitive component.

In this study DSPS were included as a control condition for the insomnia group. It is important that the hypotheses were confirmed, not only in comparison to normal good sleepers, but also against these clinical controls. This result replicates the finding, using the Dot Probe task, of attention bias in PI relative to GS and DSPS (MacMahon et al. in press). Insomnia is undoubtedly a multifactorial disorder, and one that can present heterogeneously. Separating out psychophysiological insomnia from circadian disorder in relation to difficulty initiating sleep, helps to ensure good discrimination on the independent variable. In addition, however, the contrast between PI and DSPS is that of disorders that are primarily seen as psychological and circadian respectively. It is interesting, therefore, that in the present study significant differences between good sleepers and DSPS were observed, with the latter exhibiting greater attention bias to the sleep stimulus change. This effect was not replicated in the Dot Probe experiment.

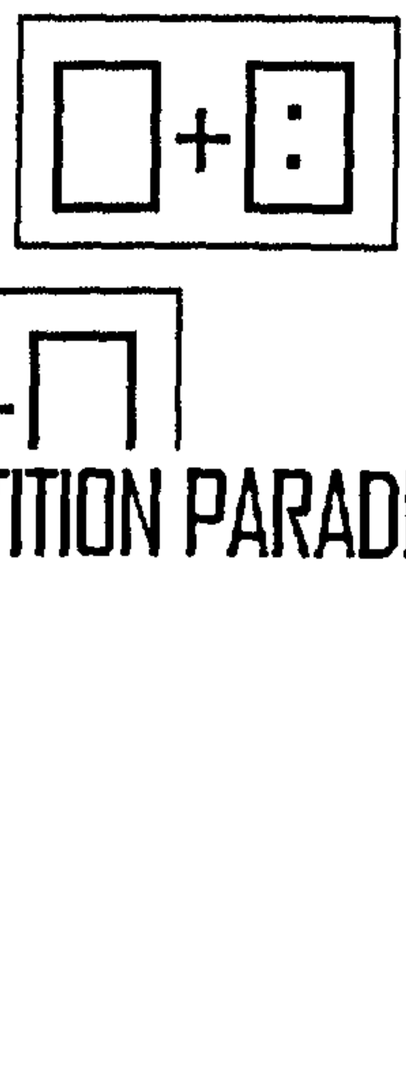
Several factors may account for the DSPS effect in this study. DSPS, particularly in younger people, may comprise two sub-populations, a socially driven DSPS and an inherent/ genetic DSPS, whose responses to attentional measures may differ. As discussed within chapter 1, evidence of an association between and the human *period 3* gene has been presented (Ebisawa et al. 2001) although other evidence has suggested 'social' cues that may result in a delayed circadian rhythm. Indeed, the mechanisms that trigger DSPS in some individuals are often precipitated by life or social events and the effects of DSPS may lead to increased pre-sleep arousal when individuals with DSPS try to reset their clocks by attempting to sleep 'out of phase'. This could theoretically precipitate symptoms of, or the onset of PI (Lack & Bootzin, 2003). Conversely, of course,

when individuals with PI are unable to fall asleep, they may inadvertently entrain their sleep to a later time, resulting in an element of DSPS. The results may also depend on whether our DSPS participants were sleeping in phase or out of phase at the time of the experiment. In the latter case one might expect more insomnia symptoms. With this information in mind, a retrospective assessment on the DSPS data was performed. Variations were evident with respect to the length of time the individuals, who were all university students, reported onset of the complaint. Some reported disorder onset in childhood and several reported a family history of DSPS, while the majority had suffered the disorder for as little as a year, and many of these reported problems occurring at the start of university. Furthermore, during the experimental period there was variation within and between DSPS subjects in terms of sleeping in and out of phase. Consequently, the results are likely explained by a combination of social, genetic and sampling frame factors, but there are insufficient numbers to separate the participants into sub-groups for the purposes of analysis. What is clear from the findings is that there may be a degree of overlap between psychological (PI) and circadian (DSPS) factors in young adult populations.

4.5 CONCLUSION

In conclusion, the results of this study are consistent with other recent experiments suggesting the existence, and potentially important role, of selective attention bias in insomnia. A computerized cognitive probe paradigm, using tasks such as the ICB, may offer insomnia research a much-needed objective index of sleep-related mental arousal.

CHAPTER 5



THE ATTENTION COMPETITION PARADIGM

EXPERIMENT 2: AN INVESTIGATION OF ATTENTION BIAS, TO INDIVIDUALLY PRESENTED PICTORIAL STIMULI, IN PSYCHOPHYSIOLOGIC INSOMNIA

5.1 INTRODUCTION

The ICB flicker paradigm studies are the first experiments to apply a pictorial attention task to insomnia, and show that people with insomnia are selectively attentive to common environmental sleep cues. Indeed Experiment 1's replication of the original flicker data, using different sleep-change stimuli, suggests that this is not an idiosyncratic finding.

One explanation for the success of the ICB paradigm in detecting attentional biases concerns the nature of the stimuli used. It has been previously suggested that pictorial stimuli can evoke responses that are more likely to mimic those in real life situations as compared to semantic representations of the same stimuli. Townshend et al. (2001) demonstrated that pictorial and semantic versions of a Dot-probe task, given to the same experimental population, resulted in inconsistent data. More specifically, IPBs for alcohol-related stimuli were revealed in heavy social drinkers when using a pictorial version of the dot-probe, a result that was not replicated when using the written word version. The authors suggest that this may be due to the fact that pictures, which in this case represented concrete rather than abstract alcohol-related representations, are more sensitive in generating attentional bias (Townshend et al. 2001).

Another explanation for the success of the ICB flicker paradigm, in capturing attention bias, considers the fact that the sleep-related and sleep-neutral

stimuli are presented in a collective manner (i.e. in a scene) rather than as individually presented entities. Thus, perhaps the accumulated effect of all the sleep-related objects together within the scene promoted a much stronger sleep association than if each were presented alone. Indeed, within the available semantic attention paradigms (e.g. Stroop, Dot-Probe, Posner), multiple stimuli cannot be presented simultaneously. So perhaps the actual set-up of the experimental paradigm may not allow for stimuli to evoke a 'significant threshold' level of anxiety that generates an attentional bias strong enough to be detected by the paradigm (Mathews & Mackintosh, 1998).

Thus, is it pictorial stimuli per se, or the accumulated effects of collectively presented pictorial stimuli that account for the captured attention bias in Experiment 1? The second experiment in this PhD attempts to answer this question. In this experiment, individually presented pictorial sleep-related stimuli will compete for attention, in PI, with individually presented pictorial sleep-neutral stimuli.

5.2 METHODS

5.2.1 Aims and Hypotheses

Experiment 2 aims to identify whether individually presented pictorial sleep-related stimuli capture an attention bias effect in PI. This study aims to do this through the use of an attention competition task in which individually pictured sleep and neutral stimuli are simultaneously presented and compete for attention. If the argument holds, that individually presented pictorial stimuli per se are effective enough in generating attentional bias responses as discussed by

Townsend et al. (2001), it is hypothesised that sleep-related pictorial stimuli will capture attention when they compete for processing priority with matched sleep-neutral pictorial stimuli, in PI, more specifically

1) RT to targets following a sleep-related stimulus cue will be significantly quicker than RT to targets following a sleep-neutral stimuli cue, in PI.

2) No such differences will be detected in GS or DSPS.

5.2.2 Design

A 3 x 2 x 2 mixed design was employed. Sleep quality (PI, DSPS, GS) acted as the between group variable and stimulus type (sleep-related/sleep-neutral), and target position (right/left) acted as the within group variables. Reaction time (RT) of a categorisation response, for one of two possible target stimuli acted as the dependent variable. Accordingly, latencies to detect targets was used to index the extent to which these groups selectively attended to either sleep-related or sleep neutral pictures.

Previous attention paradigm research has relied on a *localisation* response i.e. does the target appear on the left or right side of the screen. However, one potential problem with this task is that the cue might directly activate a response (right or left) and therefore a motor preparation effect rather than an attentional effect might drive an observed pattern in results (Fox et al. 2002). Moreover, a further problem with a location-based response is that, in principle, the information required to identify the probe location exists equally in both possible

screen locations, rather than only in the location of the target itself. Thus, responses could be made by simply attending to one side of the screen, and subsequently making a 'presence/absence' response. For these reasons, in this current experiment a target *categorisation* task is presented, such that participants had to press one key for one target type and another for a different target type (details in experimental protocol and apparatus). This feature is important, as any effects cannot be attributed to response preparation effects, because the locations of the cues are not associated with the correct response. In addition, information required to make the appropriate response can only be obtained by processing the target itself in a target categorisation task.

5.2.3 Participants

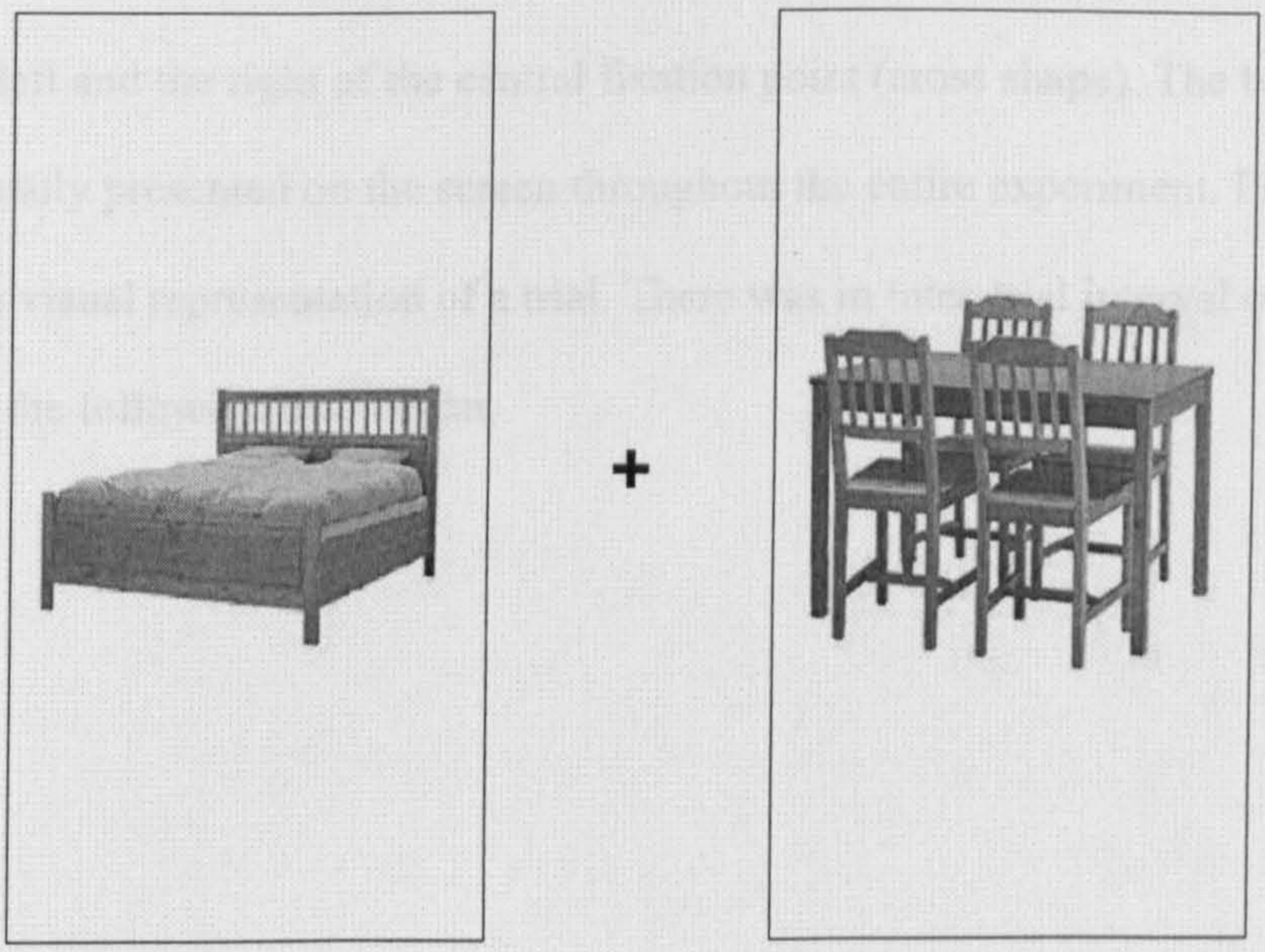
Twenty-two PI, twenty-two DSPS and twenty-two GS were included in analyses. Selection of participants followed the protocol described in Chapter 3. At Phase I, 213 individuals responded. Of these 213 respondents, 111 reported symptoms similar to PI and DSPS and were subsequently emailed for a second time (phase II) to obtain further details of their sleep patterns. From the 111 individuals emailed in phase II 89 responded and 74 were assessed as potentially suitable and subsequently completed the attention competition task. The third and final phase (phase III), after the experiment, resulted in 66 of the 74 tested being included in analyses.

5.2.4 Experimental Protocol and Apparatus

An attention competition task was employed. Seventy-two digitised single stimuli pictures represented our entire experimental stimulus set. Of these,

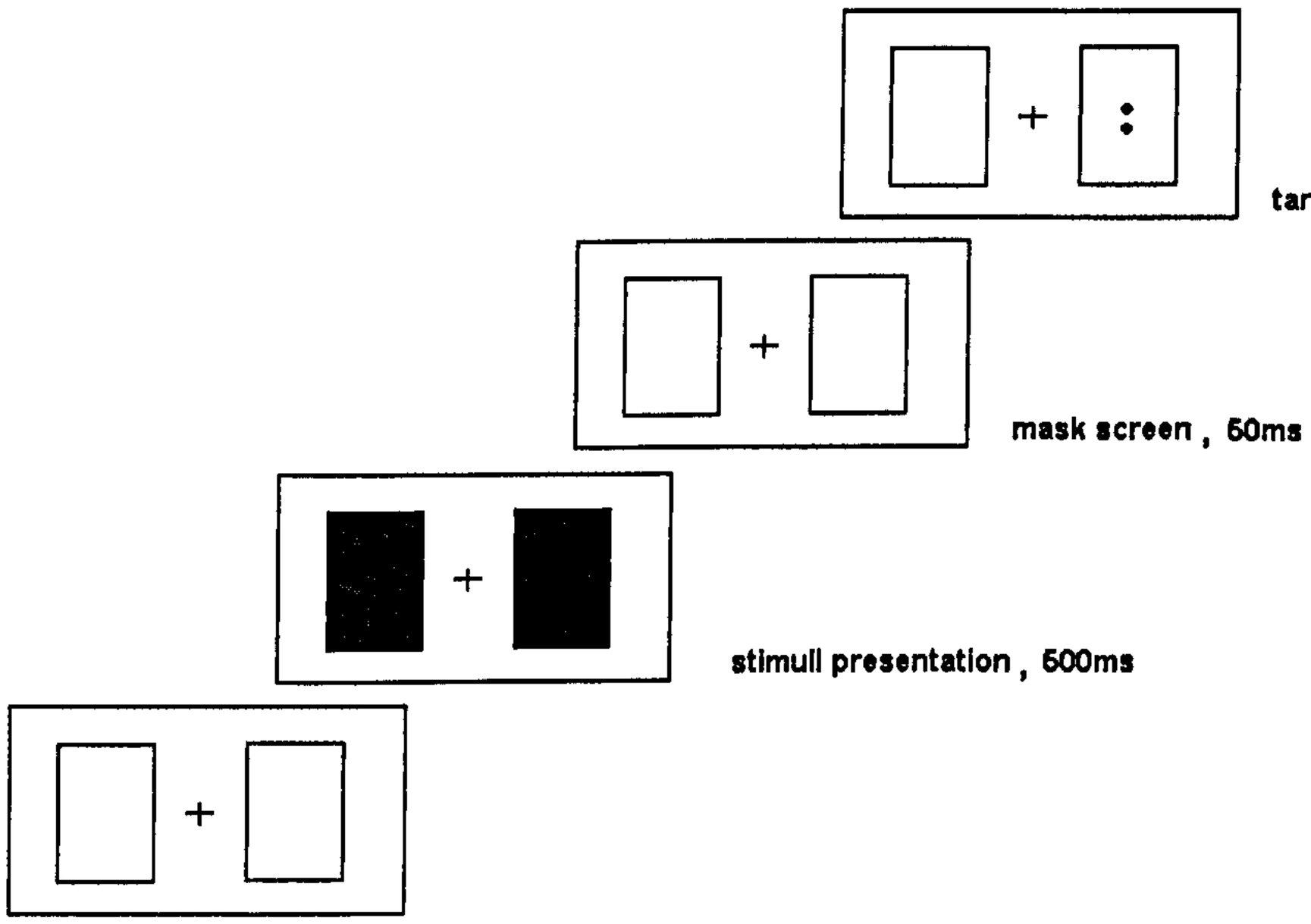
24 were sleep-related and 48 sleep-neutral. Sleep-related stimuli were chosen from a questionnaire distributed, randomly to 30 individuals. A similar questionnaire that had been devised for a previous sleep attentional bias study (Jones et al., 2005, used in Experiment 1- Appendix L) was used as a template for the new questionnaire. It asked participants to list at least 10 objects that they would strongly associate with sleep and going to bed. Evaluation of these lists resulted in the top 24 being included in our experimental sleep-related stimulus set. It was decided that neutral objects should follow an overall related pattern, and subsequently kitchen objects were chosen to represent the sleep-neutral stimulus set as no kitchen objects were listed on the sleep-related questionnaire.

Figure 8. Sleep-Related (bed) versus Sleep- Neutral (kitchen table) trial



Each sleep-related picture was matched to a sleep-neutral picture of similar orientation, brightness and level of detail as judged by the naked eye, Figure 8. Additionally, 12 "filler" (neutral vs. neutral) pairs were similarly matched and included in the experimental set. The 36 experimental pairs were repeated randomly four times within the paradigm, creating a total of 144 experimental trials. In addition, 4 practice trials and three buffer trials were also included using stimuli that were not included in the main experiment. Individual trials consisted of a fixation cross (+), presented at the centre of the screen and remaining there for the duration of the trial, followed by a picture pair displayed for 500 ms. To prevent forward masking, the cue was then blanked out for 50ms. A target (: or ..) was then presented in the left or the right box (central to where one of the pictures was positioned) and remained on the screen until a response was made. This gave a cue-target onset asynchrony of 550ms. Target stimuli consisted of either a vertical or horizontal colon. 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). The boxes were continually presented on the screen throughout the entire experiment. Figure 9 gives a visual representation of a trial. There was in inter-trial interval of 50ms before the following trial began.

Figure 9. Visual Representation of Single Trial



5.2.5 Procedure

As in Experiment 1, contact was first made with participants by email, inviting individuals to contact the first author with details of their sleeping patterns in answer to a brief set of questions. These questions enabled the experimenter to screen and select the responses that best resembled the traits of the various sleep groups involved in the study, invite the suitable candidates to give more specific information about sleep quality and subsequently invite them to take further part in the study. Thus, group allocation was 'largely known' to the experimenter before testing commenced.

To ensure an equal distribution of participants across groups, post-testing reviews of participants' subjective and objective data was performed. Thus, if more participants were allocated into the PI group an effort was made to invite those who had responded at the email phase, with descriptions mimicking DSPS, to complete the task and final screening phase.

All participants completed the same version of the attention competition task and an identical instruction protocol was presented to all participants upon arrival, regardless of their subsequent group allocation. On arrival, initial instructions were already displayed on the computer screen and read – *"Before we commence with our investigation we would like you to complete a short computer test. When you press any of the coloured buttons on the response box in front of you an instruction screen will appear on this monitor. Read these instructions carefully as these will inform you of your task in the test. If you are ready to view the instructions to the test, and are happy to do so, press any button now."* The following instruction screen informed them "*You are going to be presented with a cross at the centre of the screen that will be flanked by two*

empty boxes. After a very brief moment 2 objects will appear, one in each of the boxes. These objects will suddenly disappear and a target (either a vertical or horizontal colon) will appear in the centre of one the boxes, taking up the same position as one of the previously presented objects. Your task is to identify the orientation of the target. If the target is vertical (**:**) ***press the red*** *button and if it is horizontal* (**..**) ***press the green*** *button* (see chapter 4, page 80 for illustration of response box). *Make this decision as quickly as possible but try to be accurate".* Participants were also instructed "*during the entire duration of the experiment you are to attend to the central fixation cross (+) at the centre of the screen. Even when the objects and targets appear in the boxes, always maintain your focus to the central fixation cross".* The instructions concluded by stating that if they understood, they were to press the response box to begin a number of practice trials. After the practice trials a second instruction screen appeared, and stated, *"if you are happy with the procedure you have just practiced please press any button on the response box to begin. Please note that half way through the test there is a rest period, to begin again, after you feel you have had a sufficient rest, press any button to re-start the test".*

On four occasions, participants did not fully understand this instruction protocol. On these occasions the main experimenter verbally read out the same instructions and asked them to begin if ready. Each of these participants understood the instruction protocol after this verbal explanation. Immediately after completion of the task each participant underwent the third and detailed assessment phase, described in chapter 3.

5.3 RESULTS

5.3.1 Demographic and Clinical Data

Table 9. Demographic and clinical summary data (mean;SD) for PI, DSPS and GS groups participating in the Attention Competition Task.

	Primary Insomnia (n=22)		**Delayed Sleep Phase Syndrome (n=22)**		**Good Sleep (n=22)**		
	M	***SD***	***M***	***SD***	***M***	***SD***	**Between Group Analyses**
Age	23.5	1.82	25.7	2.19	22.2	2.09	NS
Gender (No female)	13	-	9	-	11	-	NS
STAI state	35.64	8.96	25.15	3.22	23.5	3.22	*p<0.0001*
STAI - trait	40.23	9.92	28.53	3.34	27.89	4.02	*p<0.0001*
BDI – short form	3.27	1.72	2.10	1.77	2.91	1.54	*p=0.069*

STAI: Speilberger State Trait Anxiety Inventory
BDI: Beck Depression Scale

Inspection of subjective and objective sleep quality data revealed that 8 of the original participants did not meet inclusion criteria for PI, DSPS or GS; 5 cases due to sleeping disorders other than PI or DSPS, 2 due to active medical intervention for a sleeping complaint and 1 due to pregnancy.

On three occasions, individuals believed to have DSPS from the general information given in response to the email advertisement, were subsequently re-allocated to the PI group on the basis of the more thorough interview, questionnaire measures and actigraphy data. The experimental population as a whole consisted of 33 females and 33 males with an average age of 23.8 years. Table 9 shows the demographics of the experimental population for each sleep quality group.

Table 9 also presents summary scores for the other clinical questionnaire data. There was a significant effect of group at both levels of the STAI, Trait: [F (2,63) = 90.56, $p<0.0001$] State: [F (2,63) = 53.58, $p<0.0001$]. Scheffe post hoc tests revealed that PI were generally (trait) and situationally (state) more anxious than GS, ($p<0.0001$ *and* $p<0.0001$, respectively). Similarly, on both STAI scales, PI scored significantly higher than DSPS ($p<0.0001$ trait, $p<0.0001$ state, respectively). No significant differences were observed between GS and DSPS at any level of the STAI, trait ($p=1.85$) state ($p=0.69$). There was no significant main effect of group for the BDI data, [F (2,63) = 4.05, $p = 0.064$], although the trend in the data was for PI to score higher than either GS or DSPS.

Table 10. Sleep Summary Data (mean;SD) for PI, DSPS and GS groups participating in the Attention Competition Task.

	Primary Insomnia (n=22)		**Delayed Sleep Phase Syndrome (n=22)**		**Good Sleep (n=22)**		
	M	***SD***	***M***	***SD***	***M***	***SD***	**Between Group Analyses**
PSQI	10	2.71	7.10	1.69	2.32	1.17	*p<0.0001*
Diary TST (hrs/mins)	5.42	1.00	8.29	1.21	7.27	1.30	*p<0.0001*
Diary SOL (mins)	57.0	22.4	25.27	14.18	9.17	1.0	*P<0.0001*
Actigraphy L5 (24h clock)	00:42	1.07	02:40	1.16	N/A	N/A	*p<0.0001*

PSQI: Pittsburgh Sleep Quality Index
TST: Total Sleep Time
SOL: Sleep-Onset Latency
L5: Onset of lowest 5 hours of motor output

5.3.2 Sleep Data

Table 10 summarises mean and standard deviation data for the PSQI, and selected sleep diary and actigraphic measures. Analyses revealed a significant effect of group at the level of PSQI [F (2, 63) = 58.06, $p<0.0001$]. Scheffe post hoc analyses revealed that PI scored significantly higher than both GS $p<0.0001$and DSPS, $p<0.0001$, and DSPS scored significantly higher then GS, $p<0.01$.

On the sleep diary, total sleep time (TST) was significantly different between the three sleep quality groups [F (2,63) = 27.39, $p<0.0001$], with PI sleeping significantly less than DSPS and GS, ($p< 0.0001$ and $p< 0.0001$ respectively). No such differences existed in TST between GS and DSPS groups. Sleep onset latency (SOL) was also significantly different between groups [F (2,63) = 57.73, p<0.0001], with PI taking significantly longer to fall asleep than DSPS ($p<0.0001$), and GS ($p<0.0001$), and DSPS taking significantly longer to fall asleep than GS ($p<0.001$).

Analysis of actigraphy data using NPCRA software revealed a significant main effect of group on L5 data, [F(1,42) = 42.03, $p< 0.0001$], with DSPS lowest peak of activity beginning significantly later than PI. These data indicate a sleep-onset phase delay of approx 2 hours in DSPS relative to PI.

Table 11. RT means and SD, in milliseconds, to Target Categorisation of PI, DSPS and GS.

	Primary Insomnia		**Delayed Sleep Phase Syndrome**		**Good Sleep**	
	M	***SD***	***M***	***SD***	***M***	***SD***
Sleep Cue	484.09	49.05	531.27	49.62	529.73	48.44
Neutral Cue	568	27.43	534.09	52.1	529.73	50.61
Within Group Analysis	*p<0.001*	-	*NS*	-	*NS*	-

5.3.3 Reaction Time Data

Hypotheses;

1) **RT to targets following a sleep-related stimulus cue will be significantly quicker than RT to targets following a sleep-neutral stimuli cue, in PI.**

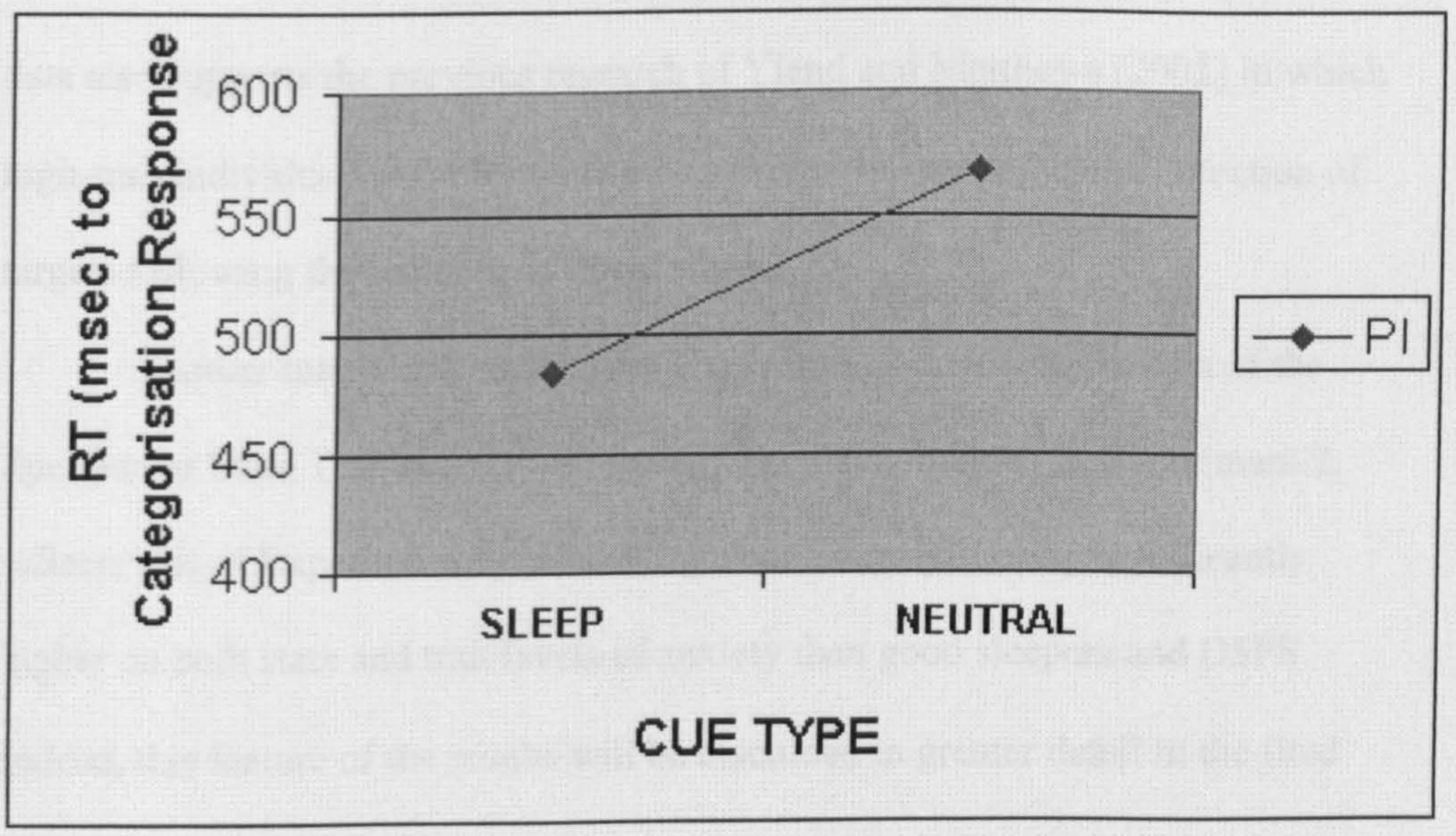
2) **No such differences will be detected in GS or DSPS.**

Table 11 summarises the RT means (time in msec to categorisation of target) and standard deviations of each sleep group. Trials with errors and trials with RT latencies greater than 1,100msec were excluded from analyses.

A mixed design analysis of variance (ANOVA) was then carried out, with sleep quality group as the between subjects variable and picture type and target position as within subjects variables.

As predicted, the interaction between group, stimulus type and target position was significant, [F (2,63) = 9.57, $p<0.001$]. Conducting separate analyses for each group decomposed this 3-way interaction. For PI, the picture position by target position interaction remained significant, [F (2,63) = 6.59, $p<0.001$]. Scheffe Post Hoc analyses revealed that PI were significantly quicker to respond to targets replacing sleep-related pictures as compared to sleep-neutral pictures, ($p< 0.001$) (Figure 10). No such differences were found for GS or DSPS.

Figure 10. PI mean reaction times to targets following a sleep-related cue and sleep-neutral cue.



5.4 DISCUSSION

This study applied an attentional competition task to investigate the hypothesis that individually presented sleep-related pictorial stimuli will capture the attention of PI when they compete for processing priority with matched sleep-neutral pictorial stimuli. This hypothesis was confirmed.

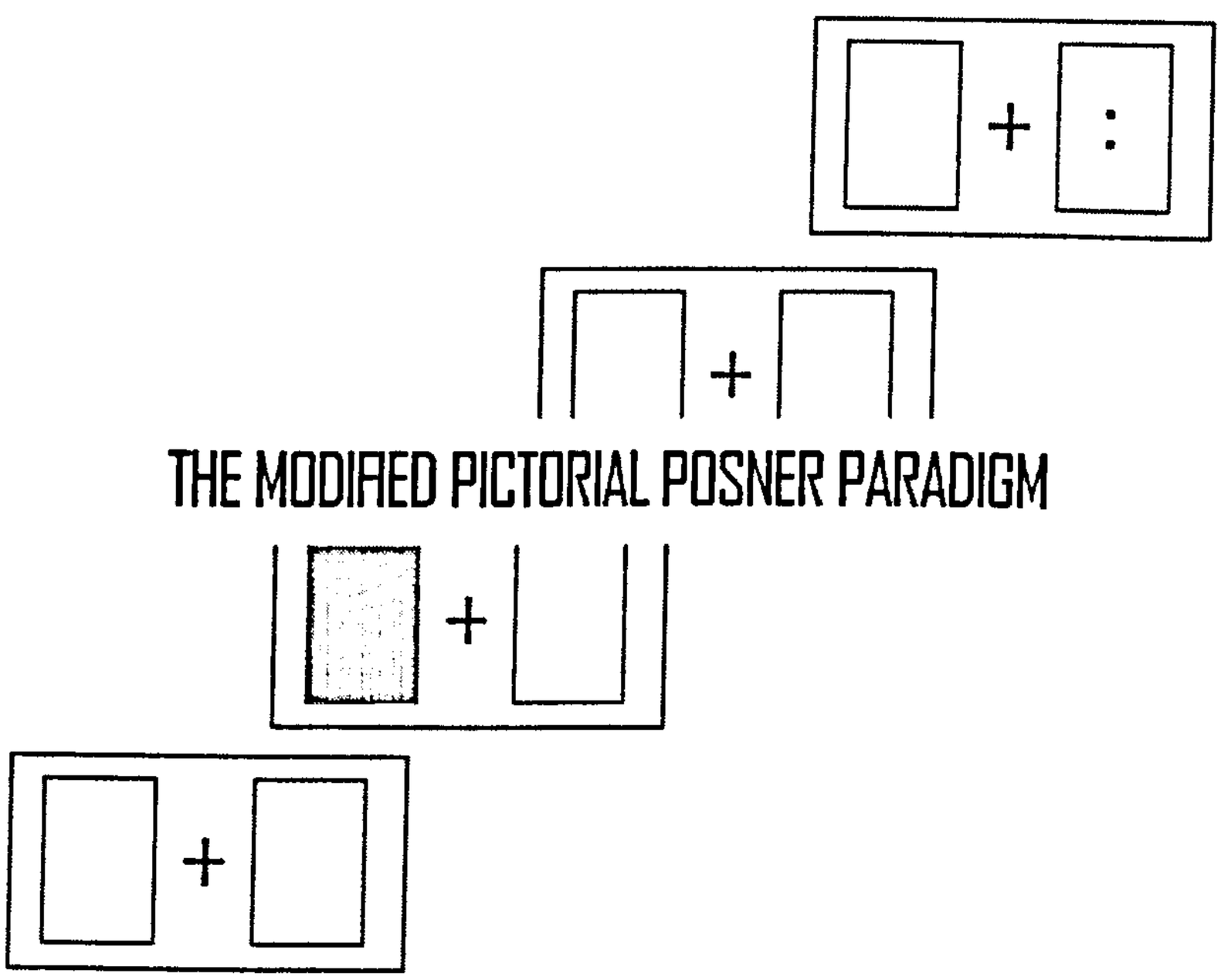
Experiment 2 confirms the expectation, and is the first study to demonstrate, that sleep-related pictorial stimuli per se are capable of evoking an attention bias response that can, subsequently, be detected by the Attention Competition Paradigm. Indeed these data are consistent with previous research showing that clinically anxious groups, or high-trait anxious students under examination stress, were found to be significantly speeded in detecting targets in the location of threat words (Matthews & MacLeod, 1994). Furthermore, this data also supports the previous research of Yiend and Matthews (2001) in which high-trait individuals were found to be significantly speeded in the detection of targets following the location of threat words.

Another interesting result from Experiment 2 concerns the data of the Speilberger State Trait Anxiety Inventory. The data obtained in Experiment 2, reflects that of Experiment 1, in that PI are consistently scoring significantly higher on both state and trait levels of anxiety than good sleepers and DSPS. Indeed, this feature of the results will be discussed in greater detail in the final discussion in Chapter 8.

The result of Experiment 2 adds strength to the data of Experiment 1, which demonstrates the existence of attention bias in PI to sleep-related stimuli using the ICB flicker paradigm. Experiment 2 has also demonstrated anxiety data

and sleep scores that are consistent with those of Experiment 1. Indeed, this will be discussed in greater detail in the final chapter (Chapter 8). However, the most important consequence of these data is the possibility that other attention paradigms, relying on individually presented pictorial stimuli, may provide investigative opportunities in the sleep and attention bias domain. Indeed, Experiment 3, within the following chapter, attempts to explain this is more detail.

CHAPTER 6



EXPERIMENT 3: AN INVESTIGATION OF THE COMPONENTS OF ATTENTION (ENGAGEMENT/DISENGAGEMENT) DRIVING ATTENTION BIAS, TO SLEEP-RELATED STIMULI, IN PSYCHOPHYSIOLOGIC INSOMNIA

1.6 INTRODUCTION TO A MODIFIED PICTORIAL POSNER PARADIGM

The experimental research within this PhD has identified attention biases in PI to sleep-related stimuli through the use of two attentional paradigms with different internal features. Indeed, the data derived in Experiments 1 and 2 suggest that attention paradigms relying on both collectively and individually presented stimuli are effective in capturing attention bias in PI. However, although the sensitivity of these attentional tasks is successful in capturing the attentional bias effects, none are capable of providing insight into which components of attention are driving the attention bias effects. Previously, Yiend and Matthews suggested that perhaps attention is preferentially held at the spatial location in which salient stimuli occur, and thus the speeded advantage observed toward target following salient stimuli (as in Experiment 2) is actually better understood in terms of a RT disadvantage when detecting targets on the opposite location to the salient cue (i.e. targets replacing the sleep-neutral picture). More specifically, attention is initially directed and held at the salient stimulus

position, thus having to undergo an attentional shift to the opposite location, in order to detect the target. In such trials RT would be significantly slow.

Indeed, as described in chapter 2, 'disengagement' is one of the three attention components that have been discussed in association with attention bias; the others being engagement and attentional shift. Furthermore, Fox et al. (2001) considered these effects in a series of experiments, assessing visual attention in sub clinical anxiety, using a modified Posner paradigm (Posner et al. 1990) (see chapter 2 page 46 for review). Within this computer task, participants are required to detect a target that may appear on the left or the right of a fixation point. On 75% of the trials, a cue highlights the area in which the target will appear (valid). However, one 25% of the trials the cue will appear in the opposite location of the following target (invalid). The typical paradigm effects reveal 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 RTs on valid trials and slower RTs on invalid trials. This effect is more commonly known as the cue validity effect.

Fox et al. demonstrated that, in terms of attention bias to salient vs less salient objects (in this experiments case threatening vs non threatening stimuli, respectively), RT on invalid salient trials are slowed considerably, relative to invalid less salient trials, suggesting that a increased salience of the cue hinders the disengagement process. No such differences were found between salient and less salient valid trials, suggesting that an increase in salience of the cue does not affect the ability of the cue to draw attention.

The nature of the Posner paradigm used by Fox et al. (2001) is relevant to the questions raised by the first experiments within this thesis, as it is capable of directly assessing the components of attention that could account for the attention biases recorded; i.e. attentional shift, engagement and/or disengagement. Neither of the utilized attention paradigms within this research so far has provided more than suggestions for the attention components accounting for the observed biases. The Posner paradigm provides the opportunity to assess whether salient cues provide a RT advantage in detecting subsequent targets (*engagement benefits*) or a RT disadvantage, by slowing *disengagement* away from salient cues.

6.2 METHODS

6.2.1 Aims and Hypotheses

It is still unclear whether the attention bias detected by the ICB flicker paradigm and the attention competition task result from differential attentional engagement at relevant locations, and/or differential difficulty in disengagement when changes/targets appeared at a different location. Either effect alone or both acting together, could account for differential latencies to detect targets at the location of sleep-related versus non-sleep-related stimuli. Thus, this current experiment aims to incorporate Posner's attention model in attempts to develop a modified paradigm that can determine whether sleep-related stimuli can attract attention i.e. modulate the engagement component of covert attention, and/or hold attention, i.e. modulate the disengage component, in PI. Based on the evidence presented by Fox et al. 2001, it is hypothesised that;

1) When collapsed across all groups, RT following valid trials will be significantly quicker than RT following invalid trials, thus demonstrating the *global posner paradigm cueing effect.*

2) On invalid trials, PI will be significantly slower to respond to targets following a sleep-related cue as compared to a sleep neutral cue.

3) On invalid trials, PI will be significantly slower to respond to targets following a sleep-related cue than GS and DSPS.

4) On valid trials, there will be no significant difference in RT of PI to targets following a sleep-related cue as compared to a sleep neutral cue.

5) There will be no significant difference between RT of PI, GS and DSPS on valid sleep trials.

6.2.2 Design

A between group (PI, GS, DSPS) by within group mixed design was employed, whereby each group completed a modified Posner paradigm. Sleep quality (PI, DSPS, GS) acted as the between group variable, and all factors associated with the Posner paradigm (Picture type, Picture Location, Target location) were the within group variables. Reaction time (RT) to a categorisation response, for one of two possible target stimuli acted as the dependent variable, as latencies to detect these targets was used to index the extent to which these groups selectively attended to either sleep-related or sleep neutral pictures (see chapter 5, page 96 for overview on localisation vs. categorisation responses)

6.2.3 Participants

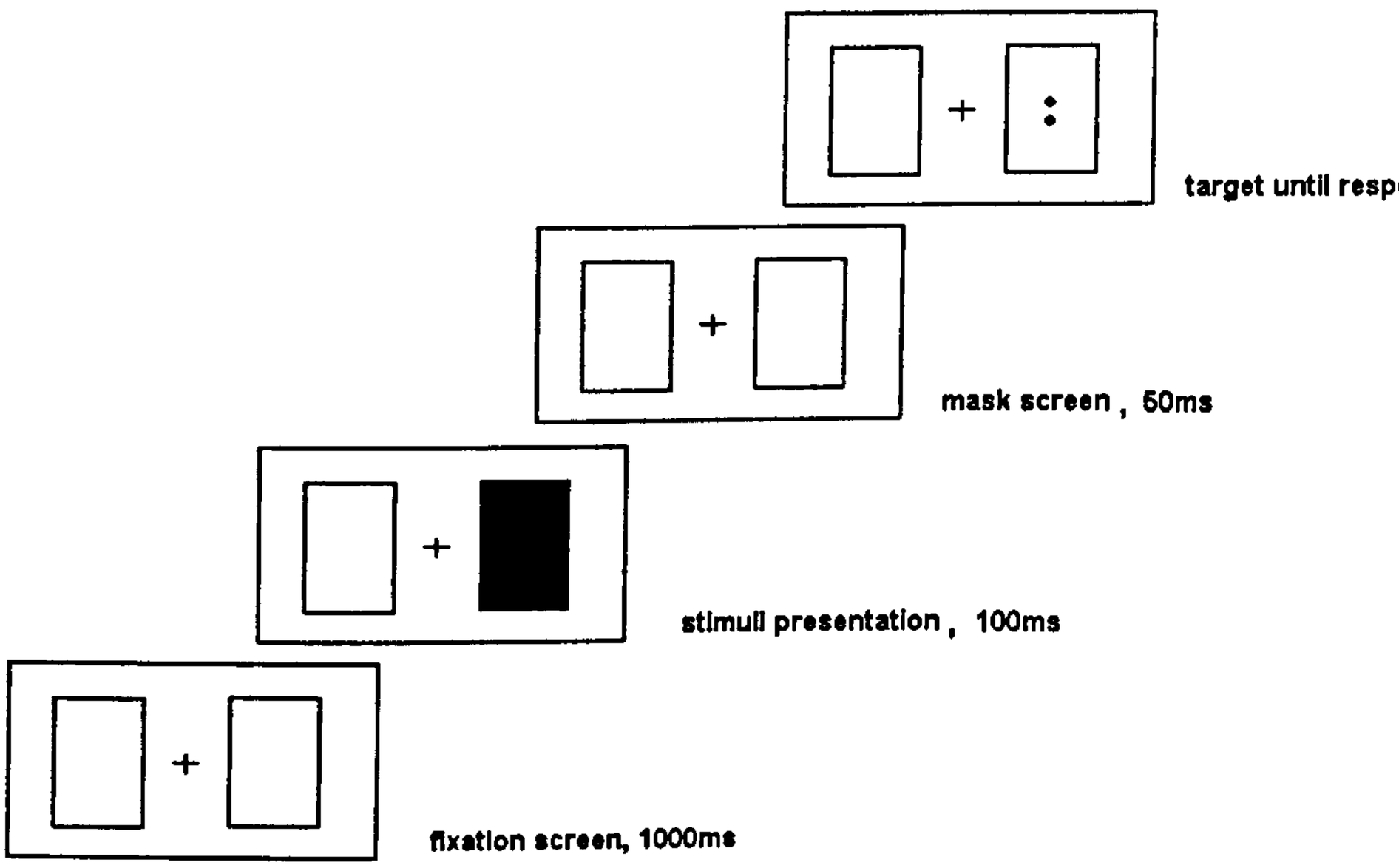
Twenty-two PI, twenty-two DSPS and twenty-two GS were included in analysis. Selection of participants followed the protocol described in Chapter 3. At Phase I, 142 individuals responded. Of these 142 respondents, 102 reported symptoms similar to PI and DSPS and were subsequently emailed for a second time (phase II) to obtain further details of their sleep patterns. From the 102 individuals emailed in phase two 76 responded, and 70 were assessed as potentially suitable and subsequently completed the attention competition task. The third and final phase (phase III), after the experiment, resulted in 66 of the 70 tested being included in analyses.

6.2.4 Experimental Protocol and Apparatus

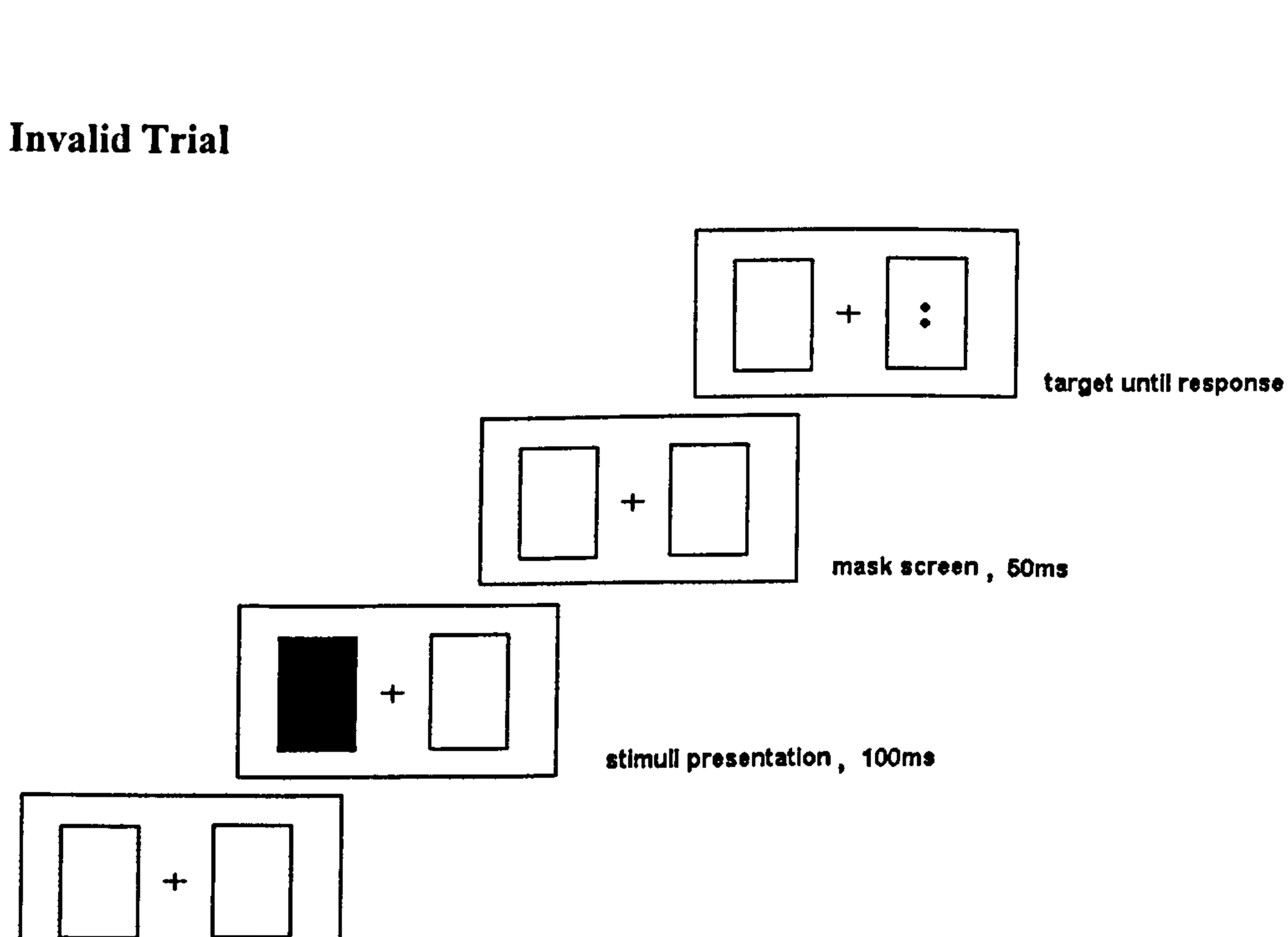
A modified pictorial Posner paradigm was employed. A fixation cross (+) - flanked by two rectangular boxes was presented at the centre of the screen for the duration of each trial. 1000msec after the onset of the fixation point, an individually photographed picture cue (sleep or neutral) was presented in either the left of the right box for 100msec. The cue was then blanked out, and 50msec later the target (either vertical or horizontal colon) was presented in the centre of either the left or right box until the participant responded. This gave a cue-target onset asynchrony of 150ms. There was in inter-trial interval of 1000ms before the following trial began. Figure 11 gives a visual representation of this sequence.

Figure 11. Illustration of single valid and invalid trial in the Modified Pictorial Posner Paradigm.

a) Valid Trial



b) Invalid Trial



Forty-eight digitised single stimuli pictures represented the entire experimental stimulus set. Critical picture stimuli were drawn from the same set as those used in Study 2, but each picture was presented individually as a cue. Again, an effort was made to match sleep and neutral stimuli for orientation, brightness and level of detail, as judged by the naked eye. 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). As in Experiment 2, 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). This unbalanced ratio of trials is routine in emotional cue-target paradigm studies (Stormark et al. 1995, 1997).

6.2.5 Procedure

All participants completed the same version of the modified Posner paradigm task and an identical instruction protocol was presented to all participants upon arrival, regardless of their subsequent group allocation. This instructions protocol is similar to that of Experiment 2, thus below we give a brief overview of the aspects that are identical, and a more thorough description of the aspects new to this study.

On arrival, initial instructions were already displayed on the computer screen and read... *"You are going to be presented with a cross at the centre of the screen that will be flanked by two empty boxes". "After a very brief moment a picture will appear in the centre of one of the boxes". "The object will suddenly disappear and a target (either a : or a ..) will appear in the centre of one the boxes...it could take the place of the previous picture of take the place of the opposite box to where the picture appeared".* The instructions explained... *"Your task is to identify the orientation of the target dots i.e. are they horizontal or vertical...If the target is* ***horizontal (..) press the red button*** *and the target is* ***vertical (:) press the green button*** *...make this decision as quickly as possible but try to be accurate. During the entire duration of the experiment you are to attend to the central fixation cross at the centre of the screen Even when the objects and targets appear in the boxes, always maintain your focus to the central fixation cross".*

The instructions concluded by stating that if they understood the above instructions, they were to press the response box to begin a number of practice trials. After theses practice trials a third instruction screen appeared. This third instruction screen was identical to that of Experiment 2.

Immediately after completion of the task each participant underwent the third and detailed assessment phase, described in chapter 3.

6.3 RESULTS

6.3.1 Demographic and Clinical Data

Table 12. Demographic and Clinical Summary Data for PI, DSPS and GS groups.

	Primary Insomnia N = 22		**Delayed Sleep Phase Syndrome N = 22**		**Good Sleep N = 22**		**Between Group Analyses**
	M	***SD***	***M***	***SD***	***M***	***SD***	
Age	22.4	2.1	21.2	2.2	23.2	1.3	NS
Gender (No female)	11	-	12	-	10	-	NS
STAI state	38.36	6.45	32.27	6.3	27.54	4.2	*p<0.01*
STAI - trait	43.77	8.2	35.41	7.42	29.55	5.0	*p<0.0001*
BDI – short form	3.96	1.6	3.68	2.6	3.91	3.3	*p=0.061*

STAI: Speilberger State Trait Anxiety Inventory
BDI: Beck Depression Scale

Inspection of subjective and objective sleep quality data revealed that seven of the original participants tested did not meet inclusion criteria for PI, DSPS or GS; 2 cases due to incomplete data, and 5 due to diagnosis of sleep disruption other than PI or DSPS. The experimental population as a whole consisted of 33 females and 33 males with an average age of 22.3 years. Table 12 shows the demographics of the experimental population for each sleep quality group.

Table 12 also presents summary scores for the other clinical questionnaire data. There was a significant effect of group at both levels of STAI; Trait: [F (2,63) = 24.82, $p<0.0001$] State: [F (2,63) = 10.31, $p<0.001$]. Scheffe post hoc tests revealed that PI were generally (trait) and situationally (state) more anxious than GS, $p<0.0001$ *and* $p<0.001$, respectively. On the STAI trait scale, PI scored significantly higher than DSPS $p<0.001$. DSPS scored higher on the trait measure of anxiety, $p<0.05$, than GS, but no such differences were found at the state measure of anxiety. There was no significant main effect of group for the BDI data, which revealed low mean scores in all groups [F (2,63) = 0.059, $p = 0.94$].

Table 13. Sleep Summary Data (mean;SD) for PI, DSPS and GS groups participating in the Modified Pictorial Posner Paradigm Task.

	Primary Insomnia N = 22		**Delayed Sleep Phase Syndrome N = 22**		**Good Sleep N = 22**		**Between Group Analyses**
	M	***SD***	***M***	***SD***	***M***	***SD***	
PSQI	**11**		**8.14**		**1.95**		*p<0.0001*
Diary TST (hrs/mins)	**6.33**		**9.31**		**8.09**		*p<0.0001*
Diary SOL (mins)	**42.26**		**35.31**		**7.17**		*P<0.0001*
Actigraphy L5 (24h clock)	**01:08**		**04:17**		**N/A**	**N/A**	*P<0.01*
Actigraphy M10 (24h clock)	**8.23**		**13.36**		**N/A**	**N/A**	*P<0.0001*

PSQI: Pittsburgh Sleep Quality Index
TST: Total Sleep Time
SOL: Sleep-Onset Latency
L5: Onset of lowest 5 hours of motor output

6.3.2 Sleep Data

Table 12 summarizes mean and standard deviation data for the PSQI, and selected sleep diary and actigraphic measures. Analyses revealed a significant effect of group at the level of PSQI [F (2, 63) = 97.59, $p<0.0001$]. Scheffe post hoc analyses revealed that PI scored significantly higher than both GS $p<0.0001$ and DSPS, $p<0.001$, and DSPS scored significantly higher then GS, $p<0.0001$.

On the sleep diary, TST was significantly different between the three sleep quality groups [F (2,63) = 16.39, $p<0.0001$], with PI participants reporting less than 6.5 hours sleep, compared with around 9 hours for DSPS and around 8 hours for GS ($p< 0.01$ and $p< 0.001$ respectively). There was no difference in TST between GS and DSPS groups. SOL was also significantly different between groups [F (2,63) = 22.67, $p<0.0001$], with PI and DSPS taking significantly longer to fall asleep than GS ($p<0.001$), ($p<0.001$), respectively, and PI taking significantly longer to fall asleep than DSPS ($p<0.01$).

Analysis of actigraphy data using NPCRA software revealed a significant main effect of group on L5 data, [F(1,42) = 39.89, $p< 0.0001$], with DSPS lowest peak of activity beginning significantly later than PI.

6.3.3 Posner Reaction Time Data

The rationale behind separating the RT analysis into disengagement and engagement ANOVAs was largely driven by the work by Fox et al. (2001), discussed previously in this chapter. Furthermore, the analyses within this experiment were hypothesis driven. Thus, the manipulation check was considered to be the preliminary analysis, the disengagement analysis the primary analysis and the engagement analysis the secondary analysis. Each, therefore, were conducted and reported separately.

All response errors constituted 2% of the critical trial and were excluded. RT latencies greater than 750ms or less than 100ms, totalling 1% of the data, were excluded as outlying responses on the basis of a box plot.

i) Paradigm Cueing Effects

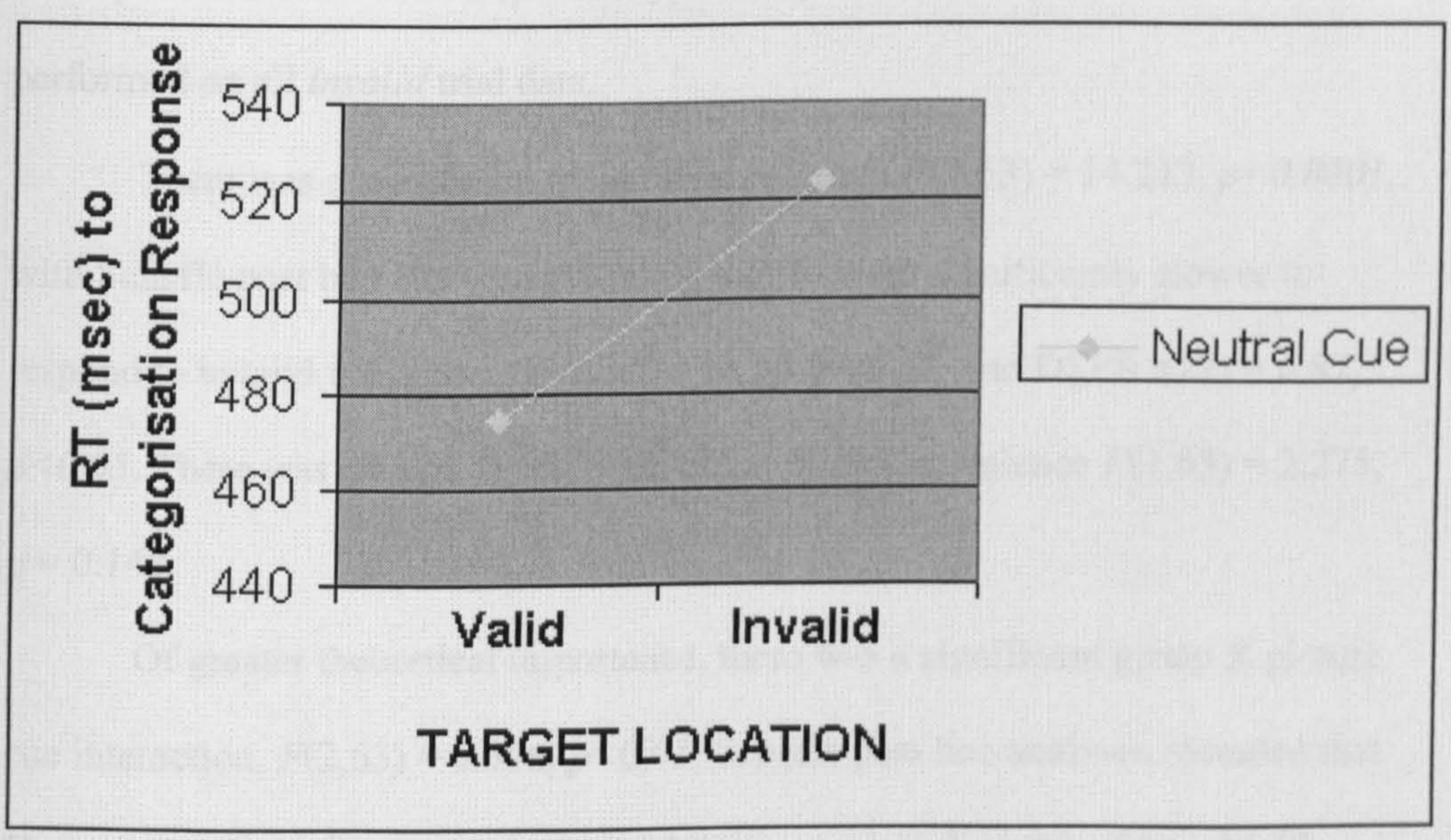
Hypothesis

1)* when collapsed across all groups, RT following valid trials will be significantly quicker than RT following invalid trials, thus demonstrating the *global Posner paradigm cueing effect.

First, to assess the effectiveness of our Posner paradigm (i.e. demonstrate the posner paradigm cueing effect), a 3 (PI, DSPS,GS) X 2 (Valid, Invalid) ANOVA was performed; comparing RT between levels of cue location (valid vs. invalid) and group, at the neutral picture cue type. This analysis allowed me to assess RT to stimuli that should be equally salient across both groups, thus escaping confounding variables that may affect RT results.

As predicted there was a significant effect of location $F(1,130) = 11.53$, $p<0.001$, with participants responding approx 50ms faster on valid relative to invalid trials (Figure 12). There was no significant effect of group $F(2,63) = 0.435$, $p = 0.584$ and no significant group x location interaction $F(2,63) = 0.625$, $p = 0.852$.

Figure 12. Valid vs. Invalid trials at Neutral Picture Type (Neutral Cue) for all experimental groups (PI, DSPS and GS)



ii) Disengagement Effects

Hypotheses

***2)* on invalid trials, PI will be significantly slower to respond to targets following a sleep-related cue as compared to a sleep neutral cue.**

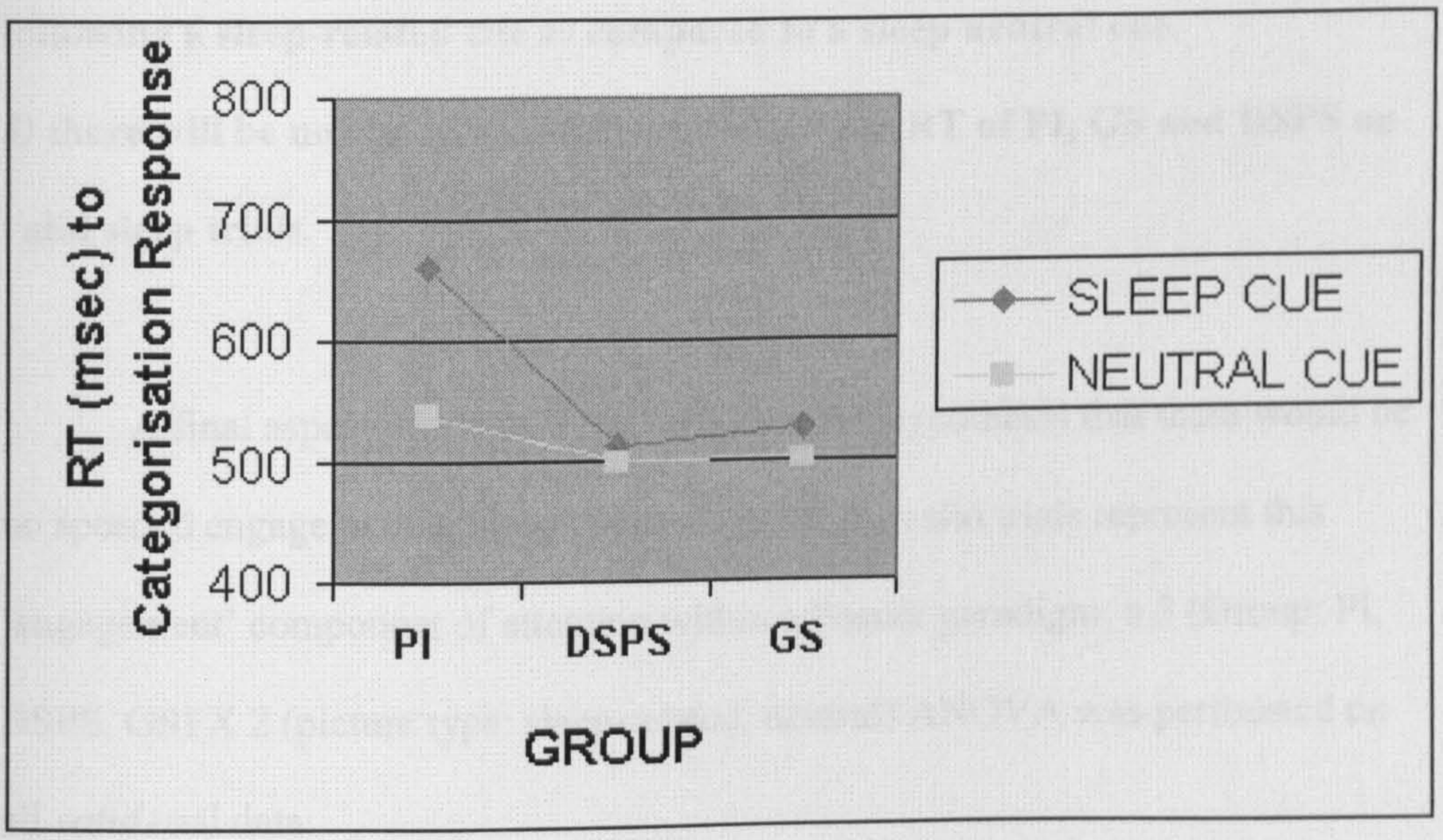
***3)* on invalid trials, PI will be significantly slower to respond to targets following a sleep-related cue than GS and DSPS.**

Based on the previous finding of Fox et al., 2002, our first hypotheses concerned the disengagement component of attention. As invalid trials represent this 'disengagement' component of attention within a Posner paradigm, a 3 (Group: PI, DSPS, GS) X 2 (picture type: sleep-related, neutral) ANOVA was performed on all *invalid* trial data.

There was a significant main effect of group $F(2,63) = 14.213$, $p<0.0001$, with Scheffe post hoc analyses revealing that PI were significantly slower to respond to invalid trials than GS $t(22) = 14.30$, $p<0.01$, and DSPS $t(22) = 6.87$, $p<0.05$. There was no significant main effect of picture valence $F(1,63) = 2.275$, $p = 0.148$.

Of greater theoretical importance, there was a significant group X picture cue interaction, $F(2,63) = 8.396$, $p<0.01$. Scheffe post hoc analyses revealed that PI were significantly slower on sleep picture cue trials than GS $t(22) = 10.52$, $p<0.01$, and DSPS $t(22)= 8.36$, $p<0.01$. No differences were found for the neutral picture cue trials. Furthermore, within themselves, PI were significantly slower to respond following sleep picture cue trials than neutral picture cue trials $t(22) = 18.56$, $p<0.001$. Figure 13 illustrates the above data.

Figure 13. PI, DSPS and GS mean reaction times to categorisation response of sleep and neutral target stimuli on invalid trials



iii) Engagement Effects

Hypotheses

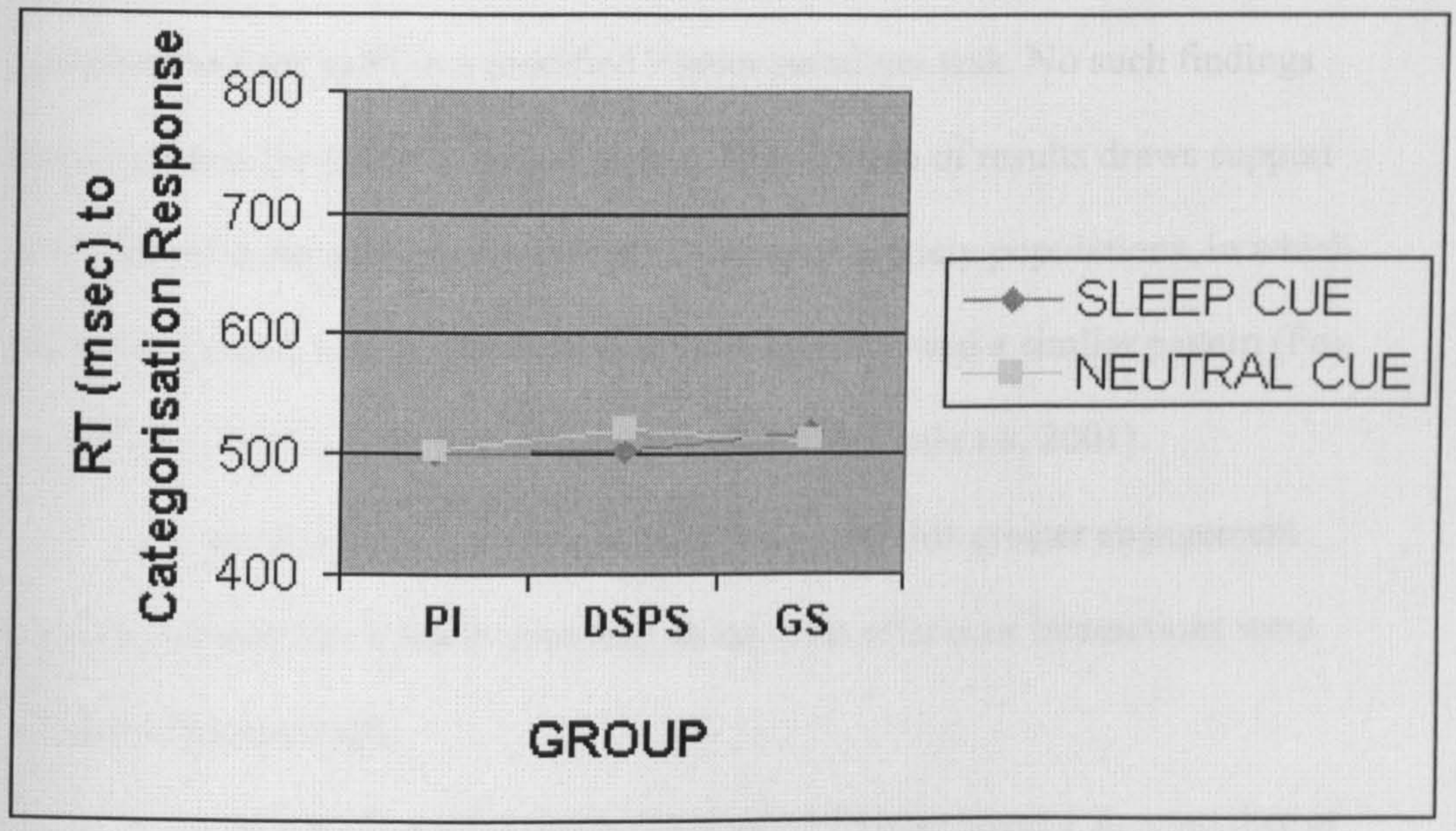
***4)* on valid trials, there will be no significant difference in RT of PI to targets following a sleep-related cue as compared to a sleep neutral cue.**

***5)* there will be no significant difference between RT of PI, GS and DSPS on valid sleep trials.**

A final aspect of our analysis considers the hypothesis that there would be no speeded engagement to sleep cue trials in PI. As valid trials represent this 'engagement' component of attention within a Posner paradigm, a 3 (Group: PI, DSPS, GS) X 2 (picture type: sleep-related, neutral) ANOVA was performed on all *valid* trial data.

Analysis revealed, no significant main effect of group $F(2,63) = 0.532$, $p = 0.485$, no significant main effect of picture valence $F(1,63) = 0.258$, $p = 0.652$, and no significant group x valence interaction, $F(2,63) = 0.684$, $p = 0.863$. Figure 14 provides a visual representation of this data.

Figure 14. PI, DSPS and GS mean reaction times to categorisation response of sleep and neutral target stimuli on valid trials



6.4 DISCUSSION

Consistent with the global Posner paradigm cueing effects, there was a large cue validity effect, such that participants were faster in categorising a target that appeared in a validly cued location relative to an invalidly cued location. As a target categorisation task was implemented, this cue validity effect can be attributed to an attentional mechanism, rather than a response preparation mechanism, as the location of the cue was not predictive of the required response.

Secondly, consistent with our initial hypotheses, PI showed delayed disengagement away from sleep-related cues, as compared to sleep-neutral cues,

on invalid trials, resulting in slowed RT to targets. This confirms the prediction that attentional dwell-time (i.e. time until attentional disengagement from stimulus) increases when salient stimuli, in this case sleep-related objects, are presented as cues to PI in a modified Posner paradigm task. No such findings were revealed for the two control groups. This pattern of results draws support from other Posner paradigm research focusing on anxiety populations, in which attentional-dwell time to threatening stimuli has followed a similar pattern (Fox et al. 2002., Broomfield & Turpin, 2005, Yiend & Mathews, 2001).

As predicted, there is no evidence to suggest that greater engagement with salient pictures leads to speeding, as no main effects or interactions were revealed on valid trials.

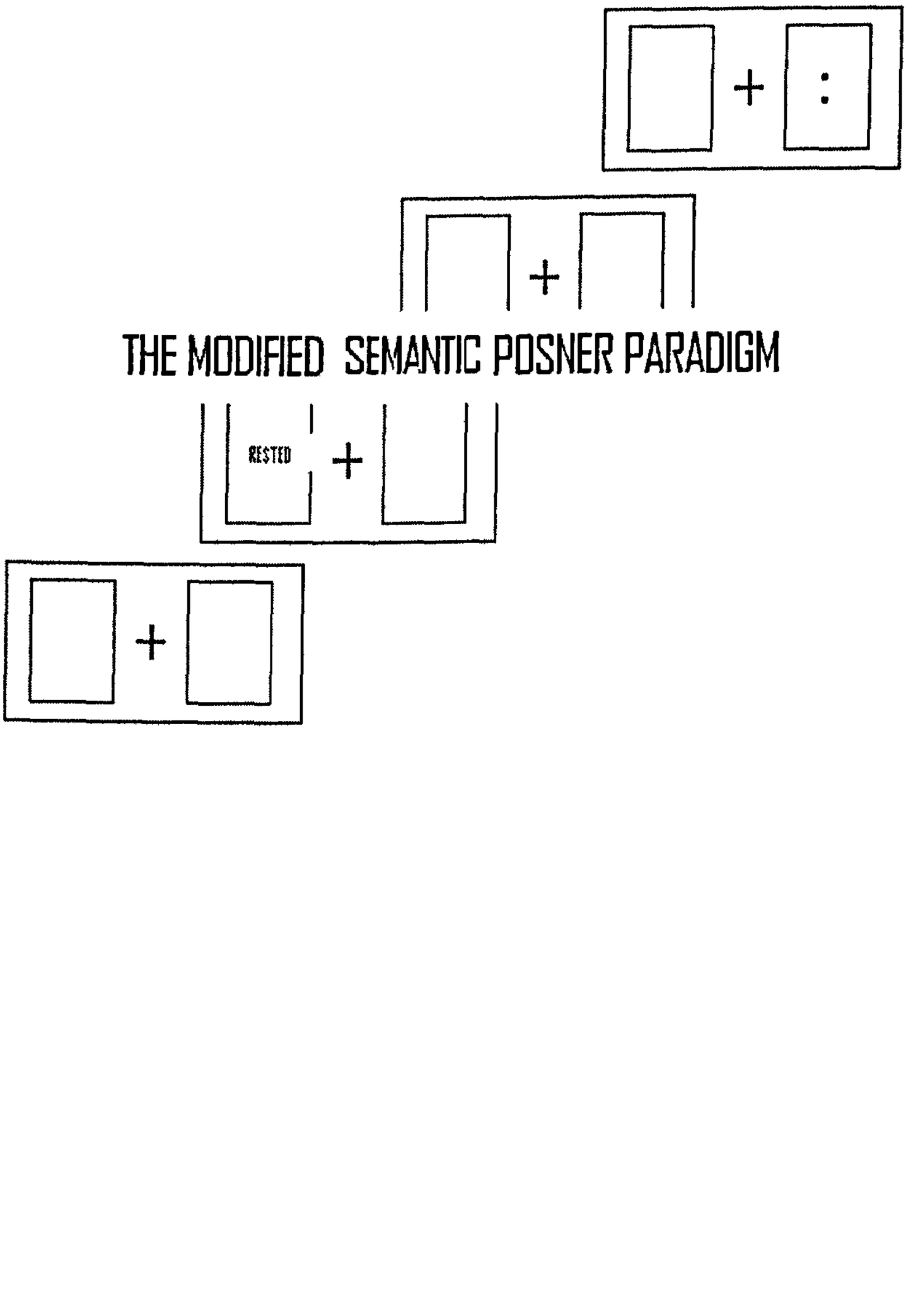
Experiments 2 and 3 support the results of Experiment 1 in a number of ways. First, the data presented from the attention competition task, demonstrating speeded RT to targets following sleep-related stimuli, are consistent with the speeded RT to change detection on sleep-related changes within the ICB paradigm. Thus, PI display attention bias to sleep-related stimuli, irrespective of presentation type (collectively or individually), or paradigm type (ICB, Attention Competition). Indeed, these experiments, taken together with the original ICB experiment (Jones et al. 2006), suggest that attention bias, measured by computerised probe tasks, may be an effective and useful index of cognitive arousal in insomnia.

Second, the results taken from Experiment 3, the modified Posner paradigm task, are the first to provide evidence that a single component of attention, disengagement, may be driving the observed attention bias effects. These data provides concrete explanations for the pattern of results reported in

Experiment 1. One striking feature of the results in Experiment 1 is the marked difference in 'neutral change' RT data between PI and the two control groups. On initial thought, one might have hypothesised that PI would not show significant differences in RT to the control groups during this condition, as the objects here should have held an equal salience to each group. However, when considering the results of this Experiment 3, we can easily attribute the markedly slowed RT of PI at the neutral change to a delayed disengagement from (or enhanced dwell-time to) the sleep-related stimuli. Furthermore, although the data in this current experiment does not suggest that greater engagement to salient stimuli leads to speeding – one can now appreciate that on the 'sleep change' condition within the flicker paradigm, PI's attention will naturally be drawn toward the salient area, but additionally held there (delayed disengagement) making it more likely that the change taking place being detected quickly.

In comparison, GS and DSPS randomly assigned their attention to either side of the scene and therefore have an equal probability of initially attending to the side embedding the changing object. More specifically, on the trials where attention is initially assigned to the 'changing' side of the screen RT will be relatively fast, but on the other trials where attention is assigned to the opposite side of the scene RT will be relatively slow, and this pattern would be true irrespective of condition (i.e. sleep-related change or sleep-neutral change) as both sets of stimuli have equal salience to good sleepers and DSPS. Indeed, this random allocation of attention in GS and DSPS would reflect the absence of RT differences over the sleep-related and sleep-neutral conditions, and the RT constant that their mean data display.

CHAPTER 7



THE MODIFIED SEMANTIC POSNER PARADIGM

EXPERIMENT 4: AN INVESTIGATION OF DELAYED DISENGAGEMENT OF ATTENTION, FROM 'NEGATIVE' SLEEP-RELATED STIMULI, IN PSYCHOPHYSIOLOGIC INSOMNIA

7.1 INTRODUCTION

Throughout the body of this thesis I have highlighted the interest psychologists have given to attention bias and have outlined various examples of the extensive research into human attention. In short, we now know that stimuli that are *salient* to an individual are likely to attract attention. That is, there is an *information processing bias* toward salient stimuli (Espie at al. 2006). For example, consider the simple situation of buying a new car. Before making the purchase you probably didn't notice the high numbers of cars on the road were of this make and model. However, after purchasing it, you are suddenly aware that many of the cars on the road are this type; you may even feel that there are more cars of this type on the road than there were previously. However this is most likely not the case, but instead, all the cars that are identical to yours have become more salient to you, in comparison to all the others, as they now have a personal relevance to your everyday life.

As discussed in chapters 2 and 5, much support has been found implicating attention bias in the perpetuation of a wide range of anxiety-related psychological disorders and concerns including panic disorder (Clark, 1985), hypochondriasis (Barsky et al. 1988), eating disorders (Cooper & Fairburn,

1992), obsessional disorders (Salkovskis, 1999), GAD (Rapee, 1991) and PTSD (Bryant & Harvey, 1995) (see Mogg & Bradley, 1998; Williams et al. 1997 for reviews). Attention biases detected within these disorders have largely been attributed to perceived threat, and it has been argued that attention bias toward threat may have a causal role in anxiety disorders (Mathews & McLeod, 2002).

The classic Beck cognitive model of emotional disorders assumes that attention biases (as well as mnemonic and interpretive biases) are driven by negative beliefs stored in long-term memory. When activated these 'schema' guide information processing, including attention, toward stimuli congruent with them (Beck et al. 1997). Conviction in negative automatic thoughts is thereby increased, and hypervigilance is promoted. The anxious individual therefore remains preoccupied with danger and threat. (Beck & Clark, 1997). In support of this, recent evidence has converged that anxious individuals are indeed characterised by selective attention bias favouring threat (eg. Mogg & Bradley, 1998).

When considering the implications of this theory with regard to insomnia, it becomes apparent that many characteristics if the insomnia lie within Beck's framework. For example, it has long been established that insomniacs experience excessive negatively toned cognitive intrusions and worry about sleep and the consequences of not sleeping. At the outset, the Cognitive Model of Insomnia proposes that this excessive worry generates autonomic and emotional distress as a result of sympathetic nervous system activation (Harvey, 2002). More specifically, the individual enters a mode of anxiety that may precipitate attentional narrowing and preferential allocation of attentional resources to sleep-

related threat cues, which include bodily sensations consistent with falling asleep or not falling asleep, environmental monitoring and clock monitoring.

An abundance of recent research has considered this relationship between anxiety in insomnia and threat monitoring and accumulating evidence is emerging in favour of internal and external monitoring being a cause of exacerbated perceived threat (Harvey, 2001, MacMahon et al. 2006). Tang and colleagues presented two 'real world' experiments investigating an association between clock monitoring, pre-sleep worry and sleep. In their former experiment, where poor and good sleepers were instructed to either 'monitor' or 'not monitor' a clock while initiating sleep, the clock monitor group reported more pre-sleep worry and experience greater SOL. In a second experiment, extended over 3 consecutive nights (1 adaptation night, 1 baseline night, 1 experimental night), 38 diagnosed primary insomniacs were instructed to either monitor a digital clock or a digital display unit (a control monitoring task) as they were trying to initiate sleep. The results demonstrated that whilst display unit-monitors experienced less pre-sleep worry, the clock-monitors experienced more pre-sleep worry and reported a longer SOL on the experimental night, relative to baseline (Tang et al. in press). The authors concluded that their findings were consistent with clinical observations, and provide empirical support for the hypothesis that monitoring for sleep-related threat, in this case the clock, contribute to insomnia by fuelling pre-sleep worry and exacerbating misperception of sleep (Harvey, 2002).

On a similar note, *daytime* monitoring in primary insomnia has also been investigated. Semler and Harvey (in press), provide evidence showing that monitoring for sleep-related threat during the day, triggers a cycle of cognitive processes that include increased negative thinking, increased use of safety

behaviours, increased perceived impairment in functioning, and increased self-reported sleepiness. Primary insomniacs were randomly assigned to a Monitoring Group (instructed to monitor their body sensations), a No-Monitoring Group (instructed to distract from their body sensations), or a No-Instruction Group. These manipulations were administered immediately on waking and participants were asked to continue the manipulation throughout the experimental day. The results revealed that the Monitoring Group reported more negative thoughts, the use of more safety behaviours, and more sleepiness during the day relative to the No-Instruction Group.

Semler and Harvey (in press-b) assigned 51 participants meeting DSM-IV criteria for insomnia to a self-focus group (viewing themselves on a TV monitor), to a monitoring group (similar to above but also focusing on thoughts and mood) or to a no instruction group. Participants were then exposed to a 60-minute neuropsychological test battery. The purpose of the study was to index the effect of attentional focus on real versus perceived performance. As hypothesized, no differences were observed in the former comparison. However, the self-focus group perceived their performance as significantly worse on the majority of tasks than the no instruction group, providing limited confirmation of the potential role of self-focusing as a contributory factor to the perceived daytime impairments of people with primary insomnia. By contrast, the monitoring condition did not differ from the no instruction group on any subjective performance rating. The authors suggest that the self-monitoring condition, unlike the video-TV condition, in this experiment may have resulted in insufficient self-focused attention.

Consideration of this evidence in favour of a link between anxiety and insomnia has led me to consider the nature of the stimuli incorporated into the attention paradigms used within this current research so far. Interestingly, none of the experimental stimuli utilized have been intrinsically commanding of attention, nor emotive or threatening. However, as presented above, the literature on attention bias in psychological disorders leans toward a threat-monitoring model, whereby words and images are presumed to grab attention because they are *emotionally* salient. Through conditioned association this may indeed be possible, even with everyday sleep objects, and this would support Harvey's cognitive model of insomnia that is based largely on responses to perceived threat.

However, at this juncture it may be too early to judge what may be motivating attentional processing within the attentional bias tasks. Indeed, at this stage we can assert is that attentional biases to sleep-related stimuli exist in primary insomnia. Nothing more. The experimentation thus far gives no insight into the driving processes behind it.

Indeed, it is important to observe that attention biases do not only operate in the context of threat. Habit, expertise and personality may also mediate selective attention (e.g. Waters & Feyerabend, 2000; Dalgleish 1995; Segerstrom, 2001, respectively). Returning to the earlier illustration, noticing more cars of a certain type may be motivated by positive interest in making a purchase. Attention bias has been implicated in the perpetuation of substance abuse/ dependence disorders as well as in psychological disorders. These include alcohol (Sharma, et al. 2001), heroin (Franken, et al. 2000) and nicotine (Waters & Feyerabend, 2000). The stimuli used in such experiments were clearly salient

(related to the dependence) but if anything were reinforcers rather than threats. To take the example of alcohol, selective attention bias to behaviourally relevant word or picture stimuli has been found in alcoholics and problem drinkers, but not in social drinkers (Stormark et al. 1997; Lusher et al. 2004; Waters & Green, 2003; Ryan, 2002; Jones & Reed, in press). It seems that problem drinkers are more likely to notice alcohol-related stimuli in the environment, that this attention bias 'reminds' them of drinking, and that it may even mediate the maintenance of their addiction by producing 'craving' (Lusher et al. 2004).

Our sleep research lab recently published a theoretical review into the development pf Psychophysiological Insomnia (Espie et al., in press). Here we acknowledge that the ICSD-2 criteria for PI conveys both a sense of incrementing distress associated with sleeplessness (c.f. threat), and a preoccupying longing for sleep (c.f. craving) that might serve as preconditions for attention bias. We suggest that the person with 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. Just as food is more of a reinforcer when we are hungry, in PI we might expect that a much higher than normal value would be placed upon sleep. Indeed under this framework, the desire for sleep of good quality may become a 'craving' (Espie et al., in press).

However, we also acknowledge 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. There is also another sense in which being unable to sleep

might be experienced as a threat. Taking our principle of automaticity into account, people who sleep well do not usually know how they do so. Ask any normal sleeper what they do to sleep and they will probably appear rather bewildered. Sleep is not in this sense an enacted operant (c.f. Bootzin, 1972), but rather it is passive and effortless. On the assumption that the person with PI started out as a normal sleeper, one can understand that to have apparently lost the capacity to sleep, not really knowing how you managed to sleep successfully before, might be rather threatening. Indeed, studies need to be conducted that specifically manipulate the emotional valence of presented stimuli.

7.2 METHODS

7.2.1 Aims and Hypotheses

Experiment 4 extends the previous Posner paradigm experiment (Experiment 3) by manipulating the valence of the experimental sleep stimuli by separating them into two groups; positive and negative. The aim of Experiment 4 is to determine the principal driving force behind the attentional biases detected in PI. By differentiating the positive and negative word types into separate groups, I aim to identify whether both threatening (negative valence) and craving (positive valence) processes are accounting for the attentional biases detected in PI.

As with all effective Posner paradigms, it is predicted that invalid trials will be slower than valid trials due to cueing effects. Furthermore, based on the results from Experiment 3, delayed disengagement effects at invalid trials are expected. Within these invalid trials five tentative hypotheses are proposed;

1) PI will be slower to disengage from negative sleep cues than neutral sleep cues

2) PI will be slower to disengage from positive sleep cues than neutral sleep cues

3) PI will be slower to disengage from negative and positive sleep cues than both GS and DSPS

4) There will be no significant differences in response times between GS and DSPS at any word valence.

5) We do not expect any speeded engagement effects on valid trials at any of the sleep quality groups.

7.2.2 Pilot Study

Before this experiment could commence it was necessary to effectively differentiate sleep words into three distinct groups: positive negative and neutral. A pilot questionnaire (see Appendix M) was randomly distributed to 30 university students. This questionnaire listed all the sleep words that have been previously used in various sleep related attention studies, along with other sleep related words generated by an on-line thesaurus. The questionnaire has three specific instructions. First, individuals were asked to consider all the words on the list and assign a rating to each one. The instructions read '*the rating you have to assign is Positive, Negative or Neutral. Try not to think about your responses too much, just put down the initial response that comes to you'*. The second task asked individuals '*in your view; how closely to sleep would you related each of these words? 1 meaning not at all related to sleep and 10 meaning very related to sleep'*. Finally, individuals were asked *'Based on your response to question 1,*

please give a rating on how positive or negative you view each word in relation to sleep. 1 being a very negative sleep word and 10 being a very positive sleep word. Please remember this is only for the words that you rated as positive or negative in task 1'.

7.2.3 Pilot Results

The responses to question 1 identified words that were assigned a positive or negative rating by all individuals. Thus, the only words that were included for further analysis were those that all participants viewed as positive or negative (n=27) – see appendix 22.

Ratings from all participants to questions 2 and 3, for this condensed list of generic positive and negative words, were averaged to give an overall sleep rating and an overall positive or negative rating for each word. Cut of score for each words sleep rating was 5.5. No word that scored below this rating was included in the study. Of these 25 words whose sleep score was above 5.5, twenty-two were included in the study as words were only included if the average positive rating was between 6.5 and 10 and the average negative rating was between 1 and 3.5. This resulted in 11 positive sleep words and 11 negative sleep words. This final experimental word set was then matched with neutral words for size and frequency in the English language. This was done using matched words from previous studies, (Wicklow & Espie 2000; Taylor et al. 2003; MacMahon et al., in press) as well as use of the frequency tables constructed from the British National Corpus (BNC), a representative sample of present-day spoken and written British English (http://www.comp.lancs.ac.uk/ucrel/bncfreq/).

7.2.4 Design

A between group (PI, GS, DSPS) by within group mixed design was employed, whereby each group completed a modified Posner paradigm. Sleep quality (PI, DSPS, GS) acted as the between group variable, and all factors associated with the Posner paradigm (Word Valence, Word Location, Target Location (valid/invalid)) were the within group variables. Reaction time (RT) to a categorization response, for one of two possible target stimuli acted as the dependent variable, as latencies to detect these targets was used to index the extent to which these groups selectively attended to either positive or negative sleep-related words or sleep neutral words.

7.2.5 Participants

Fifteen PI, fifteen DSPS and fifteen GS were included in analyses. Selection of participants followed the protocol described in Chapter 3. At Phase I, 98 individuals responded. Of these 98 respondents, 75 reported symptoms similar to PI and DSPS and were subsequently emailed for a second time (phase II) to obtain further details of their sleep patterns. From the 75 individuals emailed in phase two 61 responded, and 55 were assessed as potentially suitable and subsequently completed the attention competition task. The third and final phase (phase III), after the experiment, resulted in 45 of the 55 tested being included in analyses.

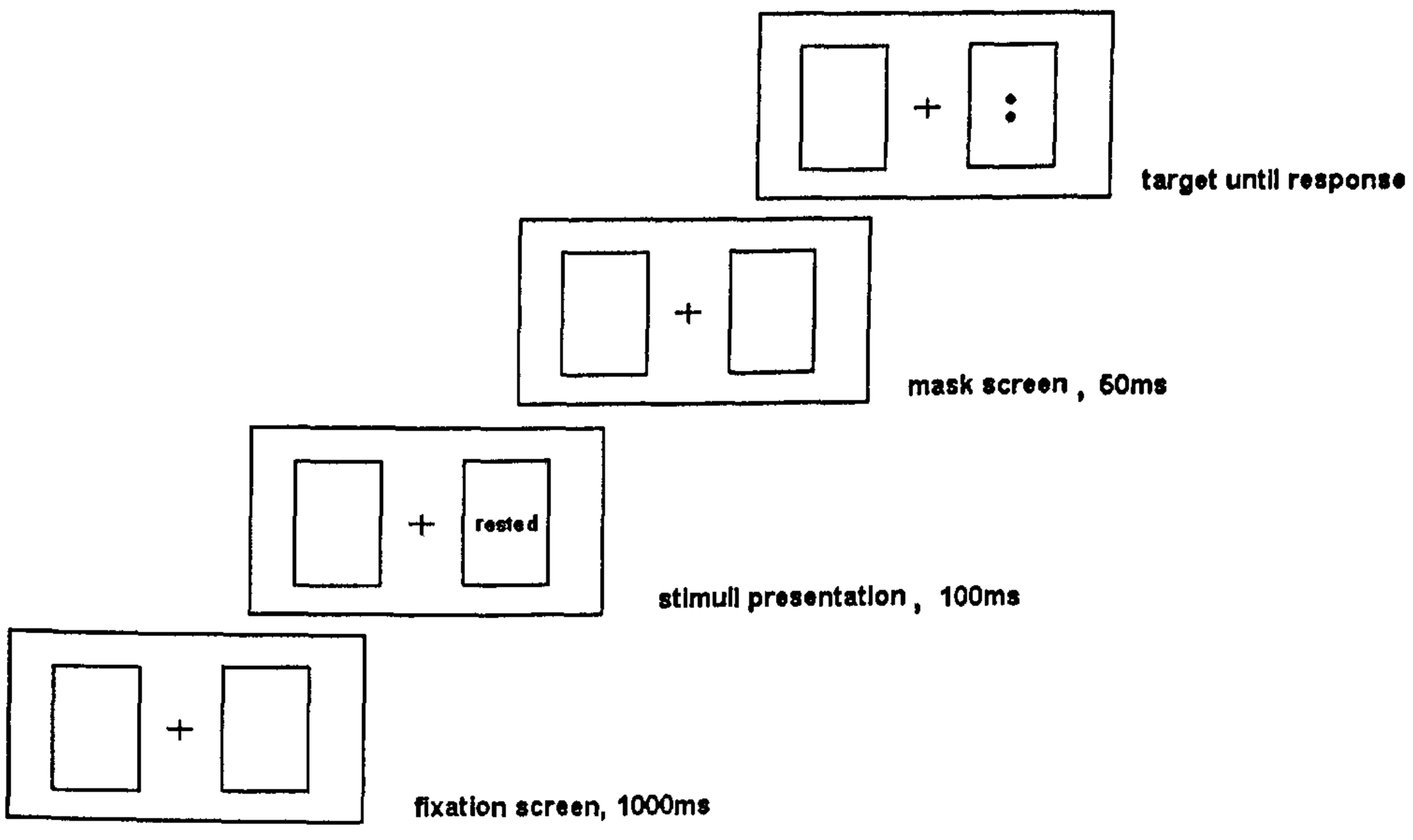
7.2.6 Experimental Protocol and Apparatus

As a modified Posner paradigm was employed, both experimental protocol and apparatus follow a similar structure to that of Experiment 3, with exception of the experimental stimuli (i.e. words instead of pictures).

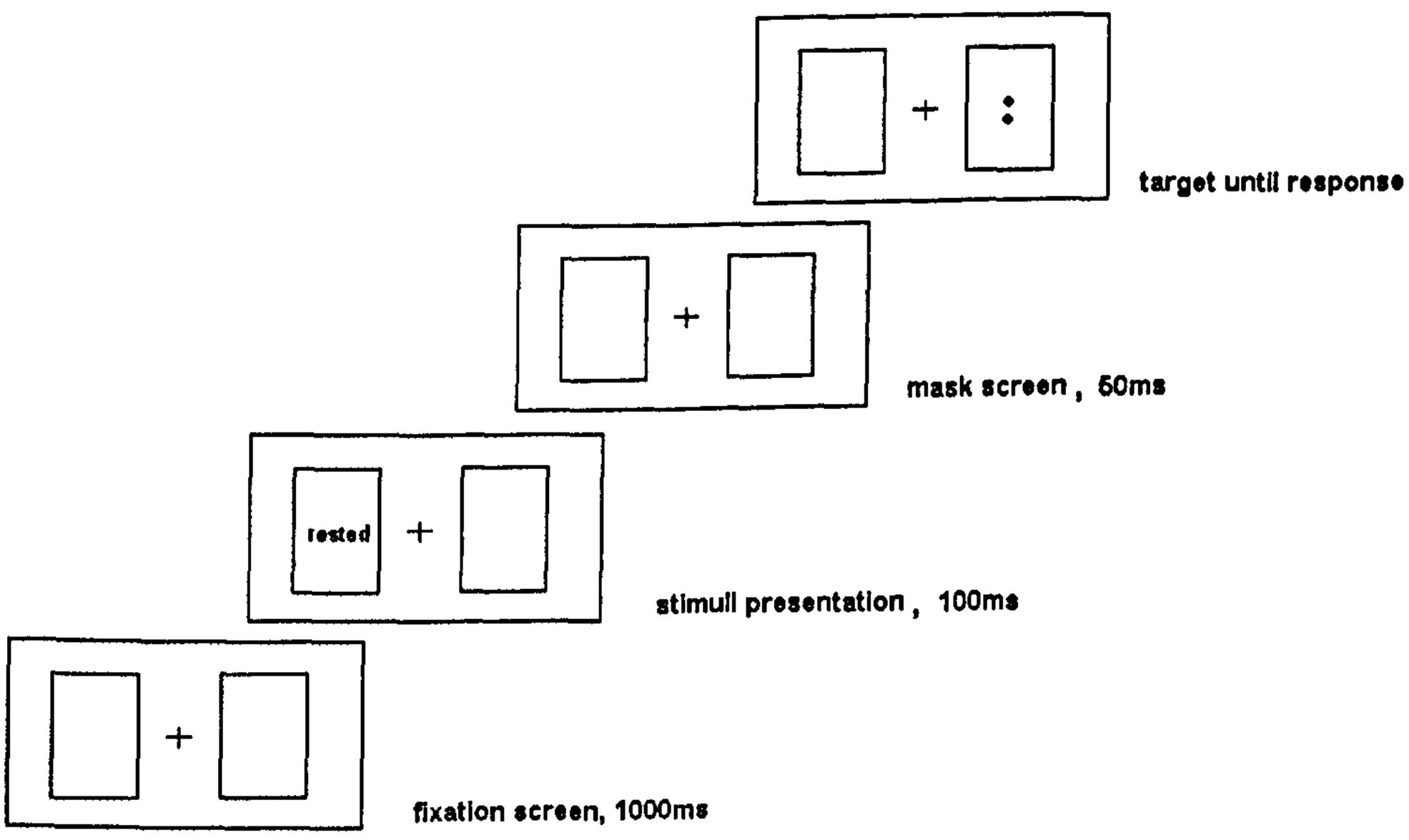
A fixation cross (+) - flanked by two rectangular boxes was presented at the centre of the screen for the duration of each trial. 1000msec after the onset of the fixation point, an individually photographed word cue (positive or negative sleep, or neutral non sleep) was presented in either the left of the right box for 100msec. The cue was then blanked out, and 50msec later the target (either a horizontal or vertical colon) was presented in the centre of either the left or right box until the participant responded. This gave a cue-target onset asynchrony of 150ms. There was in inter-trial interval of 1000ms before the following trial began. Figure 15 gives a visual representation of this sequence.

Figure 15. Illustration of single valid and invalid trial in the Modified Semantic Posner Paradigm.

a) Valid Trial



b) Invalid Trial



Forty-four words (font 24, bold black ink) represented the entire experimental stimulus set. Each word was presented individually as a cue. There were 11 positive sleep-related word stimuli, 11 negative sleep-related word stimuli and 22 non-sleep related word stimuli. Presentation of each word was repeated randomly 4 times generating a total of 176 critical trials. As in Experiment 3, there were four catch trials (no target) to prevent participants developing an automated response (see Stormark et al. 1997). Additionally, target stimuli again consisted of either a (:) or a (..). 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 previously described, this unbalanced ratio of trials is routine in emotional cue-target paradigm studies (see Stormark et al. 1995, 1997).

7.2.7 Procedure

All participants completed the same version of the modified Posner paradigm task. An identical instruction protocol to Experiment 3 (although changing picture for word) was presented to all participants. See Experiment 3 procedure. Immediately after completion of the task each participant underwent the third and detailed assessment phase, described in chapter 3.

7.3 RESULTS

7.3.1 Demographic and Clinical Data

Table 14. Demographic and Clinical Summary Data (mean;SD) for PI, DSPS and GS groups participating in the Modified Pictorial Posner Paradigm Task..

	Primary Insomnia (n=15)		**Delayed Sleep Phase Syndrome (n=15)**		**Good Sleep (n=15)**		
	M	***SD***	***M***	***SD***	***M***	***SD***	**Between Group Analyses**
Age	24.7	1.81	23.5	2.17	25.1	1.76	NS
Gender (No female)	8	-	8	-	8	-	NS
STAI state	35.2	6.31	31.3	6.5	28.0	4.8	*p<0.01*
STAI - trait	43.5	8.3	35.5	7.02	29.0	4.9	*p<0.0001*
BDI – short form	4.6	3.3	3.7	3.3	2.4	2.3	*p=0.063*

STAI: Spielberg State Trait Anxiety Inventory

BDI: Beck Depression Inventory

Inspection of subjective and objective sleep quality data revealed that 10 of the original participants did not meet inclusion criteria for PI, DSPS or GS; 6 cases due to incomplete data, 3 cases due to the participant receiving medical intervention for sleep disruption and one due to diagnosis of sleep disruption other than PI or DSPS. The experimental population as a whole consisted of 24 females and 21 males with an average age of 24.4 years. Table 14 shows the demographics of the experimental population for each sleep quality group.

Table 14 also presents summary scores for the other clinical questionnaire data. There was a significant effect of group at both levels of STAI; Trait: [F (2,42) = 16.778, p<0.0001] State: [F (2,42) = 5.603, p <0.01]. Scheffe post hoc tests revealed that PI were generally (trait) and situationally (state) more anxious than GS, *p<0.0001 and p<0.01*, respectively. On the STAI trait scale, PI scored significantly higher than DSPS *p<0.05*. DSPS scored higher on the trait measure of anxiety, *p<0.05*, than GS, but were not significantly different on state measure of anxiety, *p=3.33*. There was no significant main effect of group for the BDI data, which revealed low mean scores in all groups [F (2,42) = 0.235, p = 0.79], although the trend in the data was for PI to score higher than either GS or DSPS.

Table 15. Sleep Summary Data (mean;SD) for PI, DSPS and GS groups participating in the Modified Semantic Posner Paradigm Task.

	Primary Insomnia (n=15)		**Delayed Sleep Phase Syndrome (n=15)**		**Good Sleep (n=15)**		
	M	***SD***	***M***	***SD***	***M***	***SD***	**Between Group Analyses**
PSQI	10.87	2.4	7.73	1.5	2.07	0.9	*p<0.0001*
Diary TST (hrs/mins)	6	2.5	8.12	1.9	8.24	1.2	*p<0.0001*
Diary SOL (mins)	36.67	12.6	17.27	14.6	6.88	5.0	*P<0.0001*
Actigraphy L5 (24h clock)	02:10	1.21	03:33	1.18	N/A	N/A	*P<0.01*

PSQI: Pittsburgh Sleep Quality Index
TST: Total Sleep Time
SOL: Sleep-Onset Latency
L5: Onset of lowest 5 hours of motor output

7.3.2 Sleep Data

Table 15 summarizes mean and standard deviation data for the PSQI, and selected sleep diary and actigraphic measures. Analyses revealed a significant effect of group at the level of PSQI [F (2, 42) = 52.31, p<0.0001]. Scheffe post hoc analyses revealed that PI scored significantly higher than both GS *p<0.0001* and DSPS, *p<0.01*, and DSPS scored significantly higher then GS, *p<0.0001.*

On the sleep diary, total sleep time (TST) was significantly different between the three sleep quality groups [F (2,42) = 8.82, p<0.001], with PI participants reporting less than 6 hours sleep, compared with around 8 hours for DSPS and GS (*p< 0.01* and *p< 0.01* respectively). There was no difference in TST between GS and DSPS groups. Sleep onset latency (SOL) was also significantly different between groups [F (2,42) = 11.72, p<0.0001], with PI and DSPS taking significantly longer to fall asleep than GS (*p<0.001*), *(p<0.001)*, respectively.

Analysis of actigraphy data using NPCRA software revealed a significant main effect of group on L5 data, [F(1,28) = 8.21, p< 0.01], with DSPS lowest peak of activity beginning significantly later than PI. These data indicate a sleep-onset phase delay of 1.23 hours in DSPS relative to PI.

7.3.3 Posner Reaction Time Data

All response errors constituted 1.7% of the critical trial and were excluded. As with Experiment 3, RT latencies greater than 750ms or less than 100ms, totalling 2.1% of the data, were excluded as outlying responses on the basis of a box plot.

i) Paradigm Cueing Effects

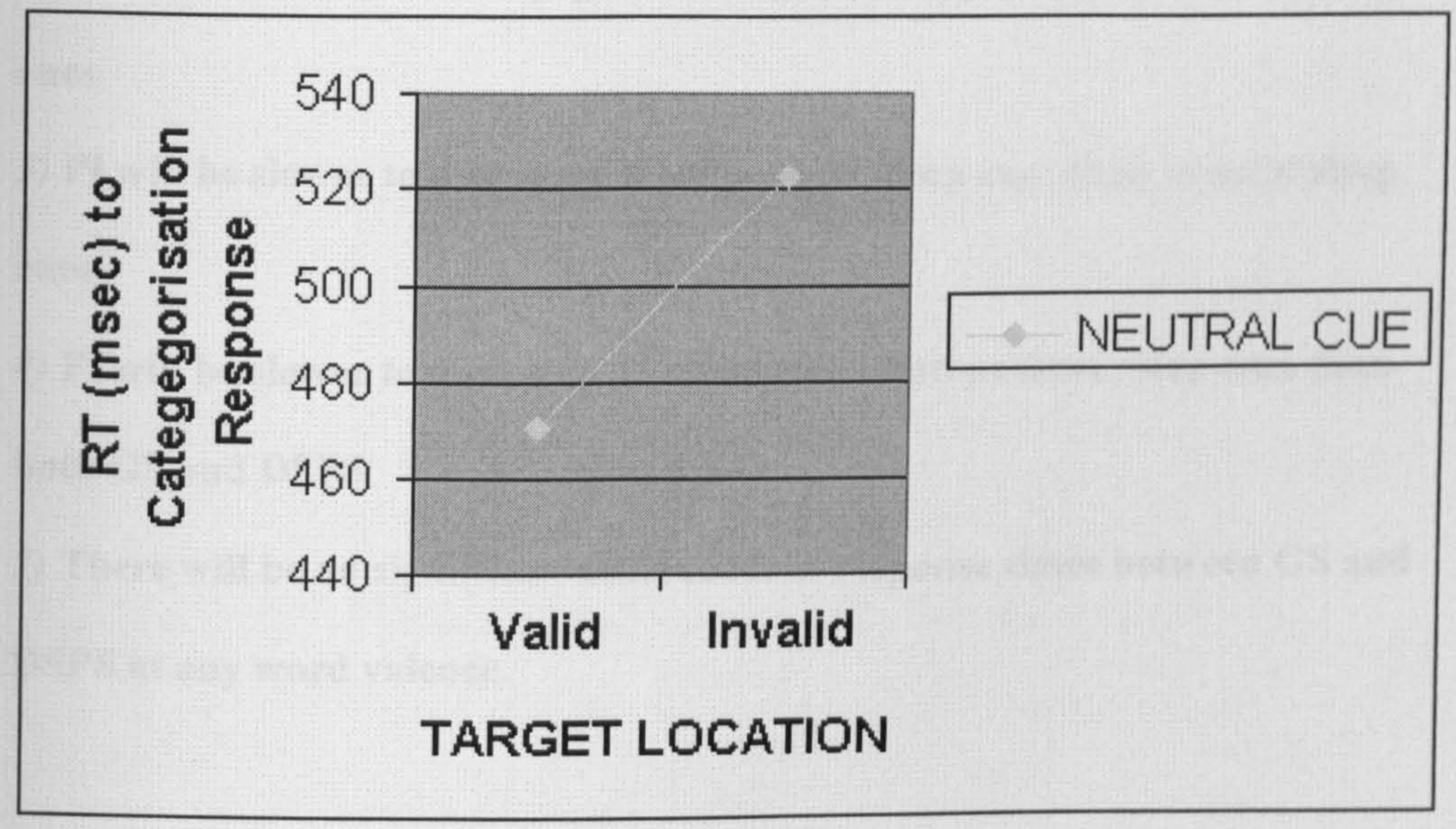
Hypothesis;

***1)* when collapsed across all groups, RT following valid trials will be significantly quicker than RT following invalid trials, thus demonstrating the *global Posner paradigm cueing effect*.**

First, to assess the effectiveness of the Posner paradigm (i.e. demonstrate the Posner paradigm cueing effect), a 3 (PI, DSPS,GS) X 2 (Valid, Invalid) ANOVA was performed; comparing RT between levels of cue location (valid vs. invalid) and group, at the neutral word cue type.

As predicted there was a significant effect of location $F(1,88) = 10.09$, $p<0.01$, with participants responding approx 64ms faster on valid relative to invalid trials, (Figure 16). There was no significant effect of group $F(2,42) = 0.626$, $p = 0.645$ and no significant group x location interaction $F(2,88) = 0.485$, $p = 0.754$.

Figure 16. Valid vs. Invalid trials at Neutral Word Valence (Neutral Cue)for all experimental groups



ii) Disengagement Effects

Hypotheses;

***2)* PI will be slower to disengage from negative sleep cues than neutral sleep cues**

***3)* PI will be slower to disengage from positive sleep cues than neutral sleep cues**

***4)* PI will be slower to disengage from negative and positive sleep cues than both GS and DSPS**

***5)* There will be no significant differences in response times between GS and DSPS at any word valence.**

Second, as invalid trials represent the 'disengagement' component of attention within a Posner paradigm, a 3 (Group: PI, DSPS, GS) X 3 (word: positive, negative, neutral) ANOVA was performed on all invalid trial data. Here there was a significant main effect of group $F(2,126) = 6.363$, $p<0.01$, with Scheffe Post Hoc analyses revealing that PI were significantly slower to respond to invalid trials than GS, $F = 10.32$, $p<0.01$, and DSPS $F = 8.70$, $p<0.05$. There was no significant main effect of word valence $F(2,126) = 2.95$, $p = 0.056$.

A significant interaction between group and word valence was revealed, $F(2,126) = 3.803$, $p<0.01$. Scheffe Post Hoc analyses revealed that PI were significantly slower on invalid negative word trials than GS, $F = 20.31$, $p<0.0001$, and DSPS $F = 13.58$, $p<0.01$. Furthermore, within the PI group, PI were significantly slower to respond to negative word trials than neutral word trials $F = 17.15$, $p<0.001$. Figures 17-18 illustrate the above data and highlight

where the significant differences lie. No such differences were found for the positive and neutral levels of word valence.

Figure 17. PI, DSPS and GS mean reaction times to categorisation response of positive-sleep, negative-sleep and neutral target stimuli on invalid trials

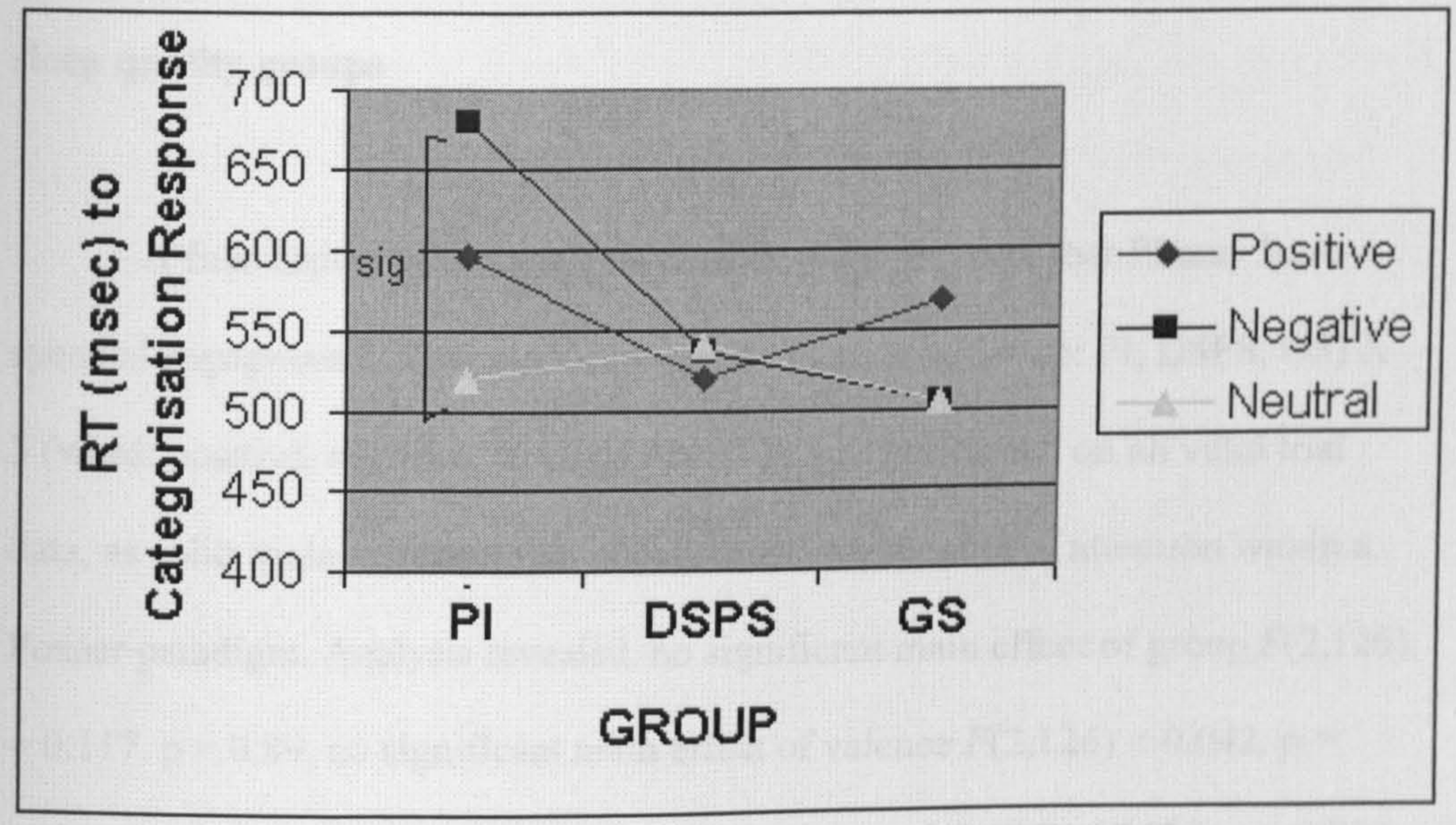
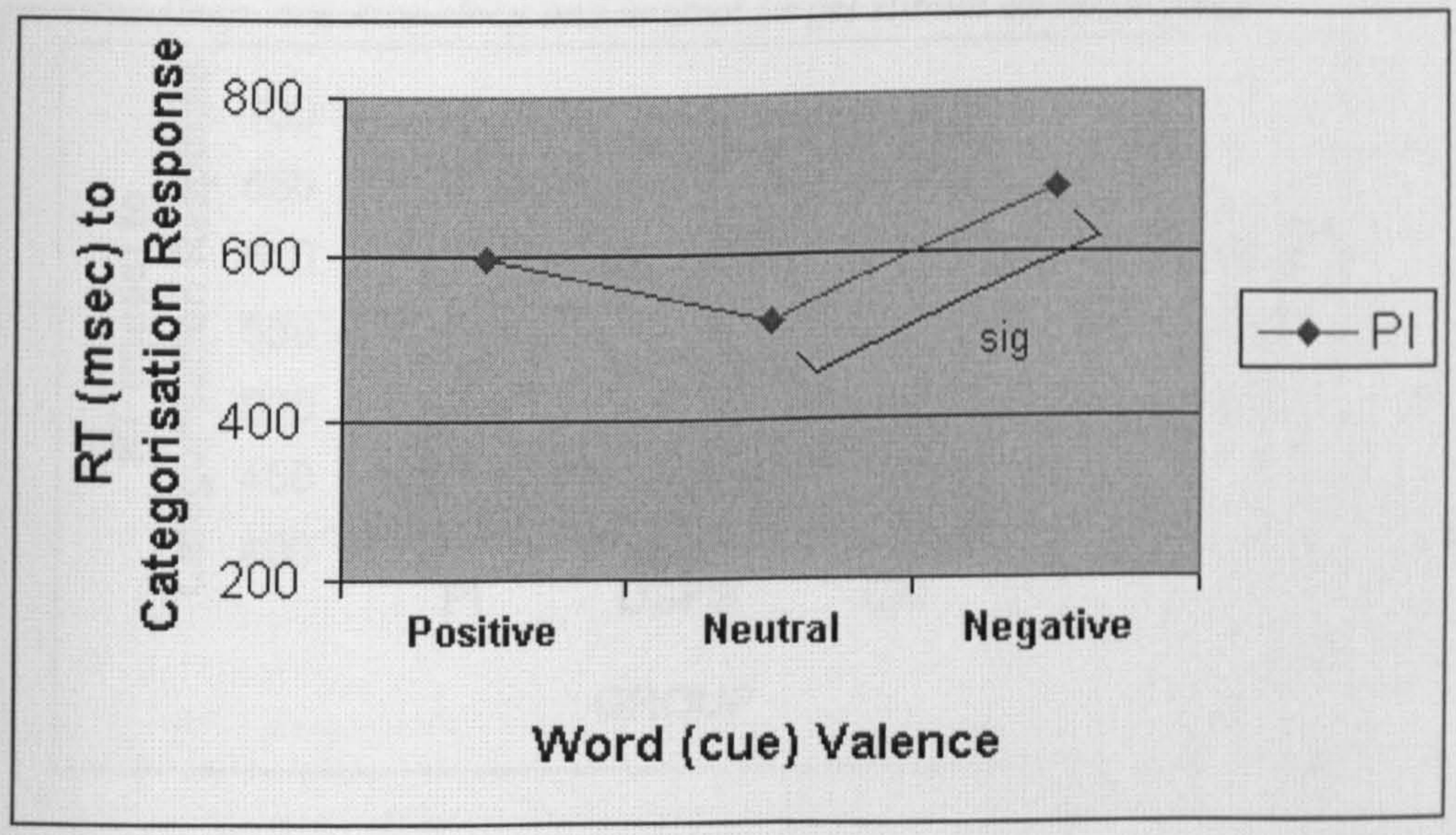


Figure 18. PI 's RT to Positive, Negative and Neutral Word Valence on Invalid Location Trials



iii) Engagement Effects

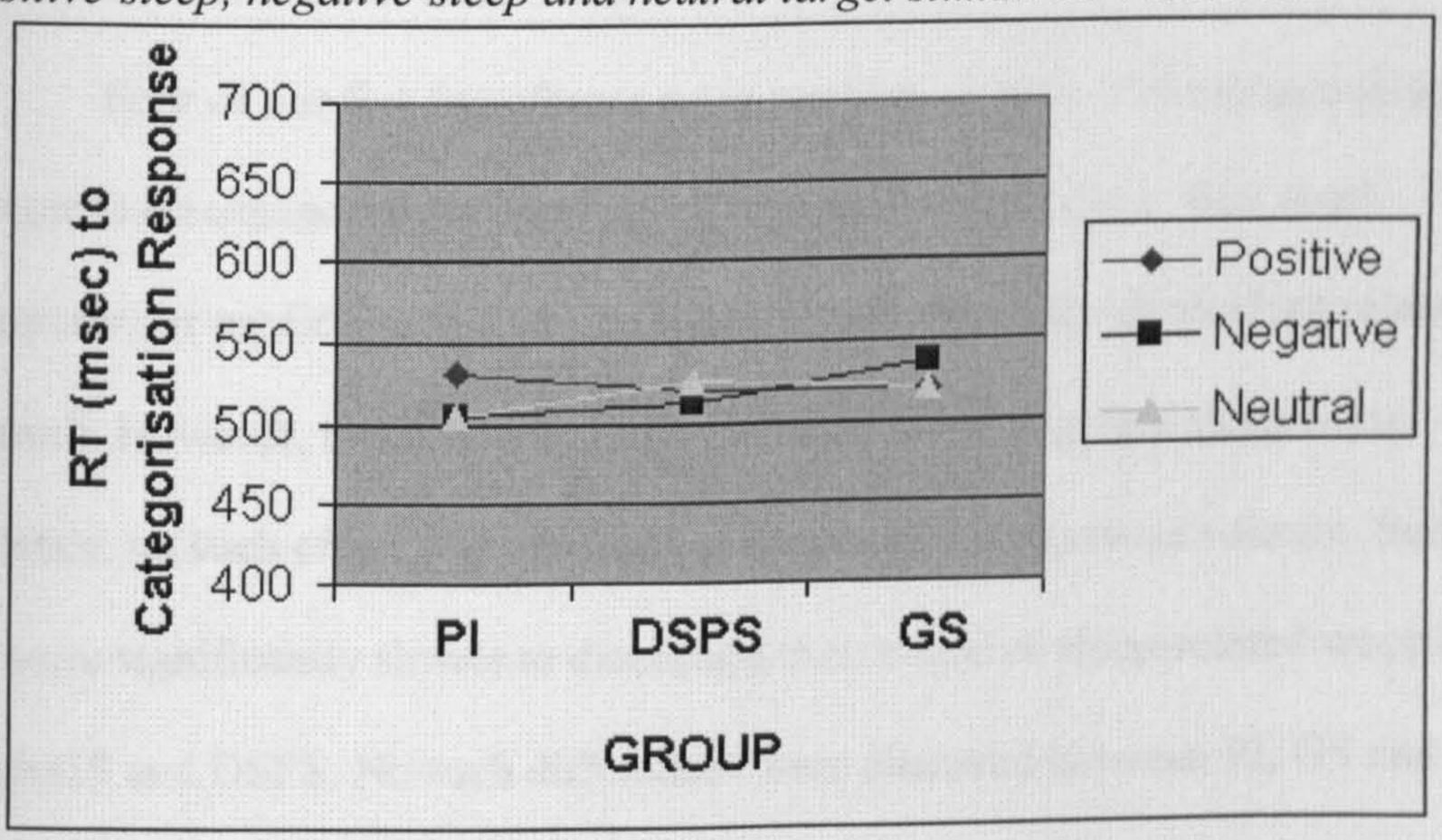
Hypothesis;

***6)* There will be no speeded engagement effects on valid trials at any of the sleep quality groups**

A final aspect of our analysis considers the prospect that PI may have speeded engagement to certain sleep words. Thus, a 3 (Group: PI, DSPS, GS) X 3 (word: positive, negative, neutral) ANOVA was performed on all valid trial data, as valid trials represent the 'engagement' component of attention within a Posner paradigm. Analysis revealed, no significant main effect of group $F(2,126) = 0.117$, $p = 0.89$, no significant main effect of valence $F(2,126) = 0.042$, $p = 0.96$, and no significant group x valence interaction, $F(2,126) = 0.208$, $p = 0.934$.

Figure 19 illustrates the above data.

Figure 19. PI, DSPS and GS mean reaction times to categorisation response of positive-sleep, negative-sleep and neutral target stimuli on valid trials



7.4 DISCUSSION

Experiment 4 applied a modified Posner paradigm to people with Psychophysiologic insomnia, DSPS and GS, with the aim of assessing attentional orienting toward different groups of sleep-related words. This study has demonstrated a successful Posner paradigm effect. More specifically, when taken at the neutral word valence only, and collapsed across group, valid trials were responded to significantly more quickly than invalid trials. This confirms the efficiency of our modified Posner paradigm, as smaller valid RTs compared to invalid RTs is a global Posner paradigm effect.

At the outset of this experiment we proposed five tentative hypotheses; 1) PI will be slower to disengage from negative sleep cues than neutral sleep cues 2) PI will be slower to disengage from positive sleep cues than neutral sleep cues 3) PI will be slower to disengage from negative and positive sleep cues than both GS and DSPS 4) there will be no significant differences in response times between GS and DSPS at any word valence 5) there will be no speeded engagement effects on valid trials.

Four of our five hypotheses were confirmed. First, PI took significantly longer to disengage from sleep-related cues than neutral cues. This result supports our prediction that PI find it difficult to disengage from sleep related stimuli. However, this effect was only detected at the *negative* sleep word valence; no such effect was observed at the *positive* sleep word valence. Second, PI were significantly slower to disengage from negative sleep-related words than both GS and DSPS. No such differences were observed between PI, GS and DSPS for positive or neutral words. Third, there were no significant differences

in response times between, or within, GS and DSPS at any word valence. Finally, as predicted, there were no speeded engagement effects on valid sleep-related trials.

As highlighted at the beginning of this chapter, the literature on attention bias in psychological disorders leans toward a threat-monitoring model, whereby words and images are presumed to grab attention because they are emotionally salient. However, it was also acknowledged that other factors such as, habit, expertise, personality or 'craving' might also mediate attention bias. Although experiment 1 to 3 demonstrate the existence of attention bias in PI, and Experiment 3 provides insight into the components of attention affected by salient stimuli i.e. disengagement, none considered the intrinsic valence of the experimental stimuli used. Indeed, on reflection, none of the experimental stimuli used in Experiments 1-3 were intrinsically commanding of attention, emotive or threatening. Thus, this current experiment (Experiment 4) is the first attempt to differentiate the sleep-related stimuli, used within an attention paradigm, in terms of emotional valence.

The results of Experiment 4 have supported the data of Experiments 1 and 2 and in particular Experiment 3, by demonstrating that PI had significantly slower RT in invalid sleep-related word trials as compared to neutral word trials. Thus, identical to Experiment 3, PI had difficulty in disengaging their attention away from sleep-related cues. However, the finding of interest in Experiment 4 is that when differentiated into positive and negative sleep-related words, only the negative sleep-related words resulted in a significant slowing of disengagement processes as compared to neutral words. No such differences were observed for positive sleep-related words.

This suggests that, within the constraints of this experiment, negative sleep-related words are more salient to PI than the positive sleep-related words. These finding supports the theory that attention biases in PI are driven, primarily, by perceived threat. Furthermore, these data draw support from the literature on attention bias in other psychological disorders.

However, an interesting component of the PI reaction time data concerns the trend that appears between the positive sleep-related words and neutral words. Indeed, although the difference does not reach significance, RTs between positive sleep-related words and neutral words is in the predicted direction. More specifically, RTs following invalid positive sleep-related trials are generally slower that RTs following invalid neutral word trials, suggesting some level of delayed disengagement. Therefore, it is likely that although not statistically confirmed, positive-sleep related words might be more salient to PI than neutral words.

In support of this theory, it is important to consider the argument made in Experiment 2. Here, the nature of stimuli used within attention paradigms was considered a key predictor for the success of the paradigm. More specifically, previous research had demonstrated that semantic attention paradigms do not result in the same attention bias data as pictorial versions (Townsend et al. 2001), as pictorial stimuli can evoke responses that are more likely to mimic those in real life situations. Thus, in Experiment 4, the absence of significance between positive sleep-related words and neutral words may be, in part, due to the semantic nature of the stimuli. More specifically, perhaps the positive sleep-related words do not generate a response level that is significant enough to be captured by the attention paradigm.

However, the negative sleep-related stimuli were also presented semantically, but here significant differences were observed. If the argument holds that semantic representations are unsuccessful in generating attention bias, this result would not have been predicted. On reflection of the data it is inarguably apparent that negative sleep-related stimuli are more salient to PI than positive sleep-related stimuli, and that this 'extra' salience results in the significant difference detected between negative sleep-related words and neutral words. We also suggest, however, that if pictorial representations of the negative sleep-related words were generated and incorporated into an attention paradigm, the attention bias effects to the negative pictures would be greater than to the negative semantic versions. Thus, although it may be difficult, future research should attempt to differentiate sleep-related pictures into those that are intrinsically negative and those that are intrinsically positive.

In conclusion, Experiment 4 is the first attention bias experiment to differentiate sleep-related stimuli into positive and negative groups and show that intrinsically 'negative' sleep-related stimuli are more successful in generating attention bias in PI than positive-sleep related stimuli.

CHAPTER 8

GENERAL DISCUSSION

8.1 INTRODUCTION

Insomnia research is constrained by the absence of reliable objective markers of the phenomenology of poor sleep. Mental arousal is a hallmark feature of insomnia. It is also a symptom of other psychopathological conditions – in which context it has been captured using techniques adapted from experimental cognitive psychology, based on the measurement of information processing speed to target stimuli. The experimental chapters within this PhD, aimed to identify that attention bias, measured by computerised probe tasks, may be a useful index of cognitive arousal in insomnia. Indeed, based on the accumulating data sets, this experimental research concludes that attention bias is a cognitive process, involved in the maintenance, and perhaps the development, of PI from adjustment insomnia, and detectable after the onset of the complaint.

8.2 EXPERIMENTAL OVERVIEW

The following section aims to highlight the key findings and implications of the experimental data and Table 16 provides an overview of the key findings, main methodological issues and future research directions, generated from the experimental data sets of Experiments 1-4.

Table 16. Key findings, main methodological issues and future research directions, stemming from Experiments 1-4.

Experiment No.	Paradigm	Methodological Strength	Future Direction
1	ICB	• Short task duration • Possibility of assessing implicit attention	Assessment of AB responses in combination with physiological markers of AB i.e. cortical components
2	Attention Competition	• Simple stimulus presentations • Disconfirmation of 'accumulated salience effect'	Utilisation of other attention paradigms relying on single stimulus presentations. Highlights the opportunity to assess components of attention driving AB effects.
3	Pictorial Posner	• Isolate engagement and disengagement components of attention	Incorporation of multiple stimulus types, differentiated by salience Insight into stimulus types driving AB effects in PI.
4	Semantic Posner	• Sleep-related stimuli differentiated into intrinsically negative and positive word types	Eye tracking the semantic posner task to ensure compliance. Repetition of experiment with differentiated pictorial stimuli

Experiment 1 applied the ICB flicker paradigm task to investigate the hypothesis that people with insomnia exhibit an attentional monitoring preference for sleep-related objects. Indeed, this hypothesis was confirmed. Furthermore, taken together with the previous ICB flicker paradigm data collected by the GSRL (Jones et al. 2006), this is not an idiosyncratic finding. These experiments are the first to apply a visual attention task to insomnia, and consistently show that people with insomnia are selectively attentive to common environmental sleep cues.

Attention bias is conceived of as an initial involuntary (unconscious, implicit) process that gives rise to the voluntary (conscious, explicit) processes (Shiffrin & Schneider, 1977). In other words, pre-attentive processing guides the early, automatic capture of relevant information even when conscious access to the information is not available. A good illustration of this phenomenon was a study by Ohman and Soares (1994) that successfully elicited both psychophysiological reactivity and subjective fear in phobic participants to pictures of their feared objects when they were presented beyond their conscious awareness. It is not possible using an ICB study to address the 'level' at which processing begins, because the dependent variable within the paradigm is defined by awareness of the change. However, it was noted that after completing the experiment, many of the participants, including those with insomnia, were unaware that the experimental stimuli were particularly related to sleep or sleep disorder, yet the experimental effects were found (however this was not an official manipulation check, future research should include this as a compulsory component of testing). Thus, although it cannot be confirmed, the ICB flicker paradigm appears to be a possible measure of implicit attentional processes.

Additionally, the nature of the actual task process seems worth considering here. Specifically, the ICB paradigm relies on very short stimulus presentations i.e. each presentation last a mere quarter of a second (250msec). Although this time scale is sufficient to generate conscious awareness of stimuli, the added feature of 'spotting the change' (i.e. the actual instructed task) may actually detract from the participant's conscious (explicit) awareness. Indeed, this argument has not been discussed in terms of the ICB flicker paradigm before, however on review of Experiment 1 and the previous ICB data it is proposed that the added instructed task might aid the ICB paradigm in assessing more implicit attentional components. However, it is important to acknowledge that is attempting to capture implicit attentional processes, the paradigm of choice would most likely be an auditory task, as implicit auditory attention biases have previously been reported.

Of course, irrespective of the level of attention the ICB is capturing, Experiment 1 identifies attention bias in PI. A feature that may account for the success of this paradigm, in capturing attention bias, may be the total time duration the task takes to run from start to finish. More specifically, the ICB is the only attention bias paradigm in which total task duration for each participant is less that 1000msec. The implications of this feature can only be positive. Indeed, when considering other attention bias paradigms, such as the Stroop or Dot Probe, task duration can total approximately 40 minutes (Yiend & Matthews, 2001), and participants are required to maintain their attention for the duration of the task. However, self reports of participants after these experiments suggest that focusing attention for this period of time is very difficult, and tiredness quickly sets in. This suggests that the paradigm set-up may result in a decrease of

attentional focus; more specifically, a decrease in the cognitive process they are aiming to assess. However, due to its short task duration, as highlighted in the experimental protocol in chapter 4, the ICB escapes this drawback and is perhaps the only paradigm capable of holding focused attention for its entire task duration.

Experiment 2 adds to the consistency of the attention bias research in insomnia populations. Here, an attention competition task was presented to people with insomnia and again the insomnia population revealed an attentional preference for the same environmental sleep cues. Beyond the principle finding, i.e. capturing AB in PI through the use of a different attentional paradigm, a second key finding concerning the nature of the experimental stimuli incorporated into the attentional paradigms emerged. Specifically, although the ICB and attention competition task incorporated identical stimuli, the way in which they were presented was very different. The ICB presented a collection of sleep-related stimuli together within a scene, and it was previously hypothesised that the accumulating effects of all the sleep-related stimuli within this scene heightened the overall salience to PI. However, in the attention competition task, each sleep-related stimulus was presented individually, thus the 'accumulated salience' effect proposed for the success of the ICB was lost. Interestingly however, the attention preference for the sleep-related stimuli in the attention competition task was confirmed, suggesting that when compared to neutral stimuli, individually presented sleep-stimuli have a greater salience to PI.

Thus, a main consequence of the result of Experiment 2 was the possibility that other attention paradigms, relying on individually presented pictorial stimuli, may provide investigative opportunities in the sleep and

attention bias domain. Indeed, until this point, none of the utilised attention paradigms i.e. ICB and attention competition, gave rise to the possibility of exploring the components of attention driving the detected bias effects. In contrast however, the Posner paradigm, another attention task, had recently been reported as being successful in identifying specific components of the attention process, namely engagement, disengagement and attentional shift. Interestingly the Posner paradigm's methodology relies on individually presented semantic or pictorial stimulus presentations. Furthermore, the Posner paradigm has consistently demonstrated that attention bias effects are a result of an inability to draw attention away from salient stimuli; more specifically, a disengagement effect. Thus, based on the result of Experiment 2 showing that individually pictured stimuli generate attention bias in PI, the Posner paradigm provides investigative opportunities that could reveal important information on the components of attention involved in the attention bias data of Experiment 1 and 2.

Subsequently, Experiment 3 applied a modified pictorial Posner paradigm task to PI, DSPS and GS, to investigate the hypothesis that attention bias effects, detected in PI, are due to delayed disengagement away from salient stimuli. Indeed this hypothesis was confirmed. The results of Experiment 3 provide important information that help link the results of Experiments 1 to 3. Indeed, on review of the evidence of delayed disengagement effects, concrete explanations for the pattern of results in the ICB paradigm experiments, both Experiment 1 and Jones et al. 2006, can be proposed. Specifically, in both the ICB experiments, the reaction times of PI to changes embedded within the sleep-neutral stimuli were significantly slower than the reaction times of the two

control groups, a result that might be considered unexpected as neutral stimuli should have an equal salience to all groups. However, taken together with the results of Experiment 3, this markedly slower RT to sleep-neutral changes can be attributed to a delay in disengaging attention away from the sleep-related stimuli within the visual scene where the change is not occurring, to the sleep-neutral stimuli within the visual scene where the change is occurring.

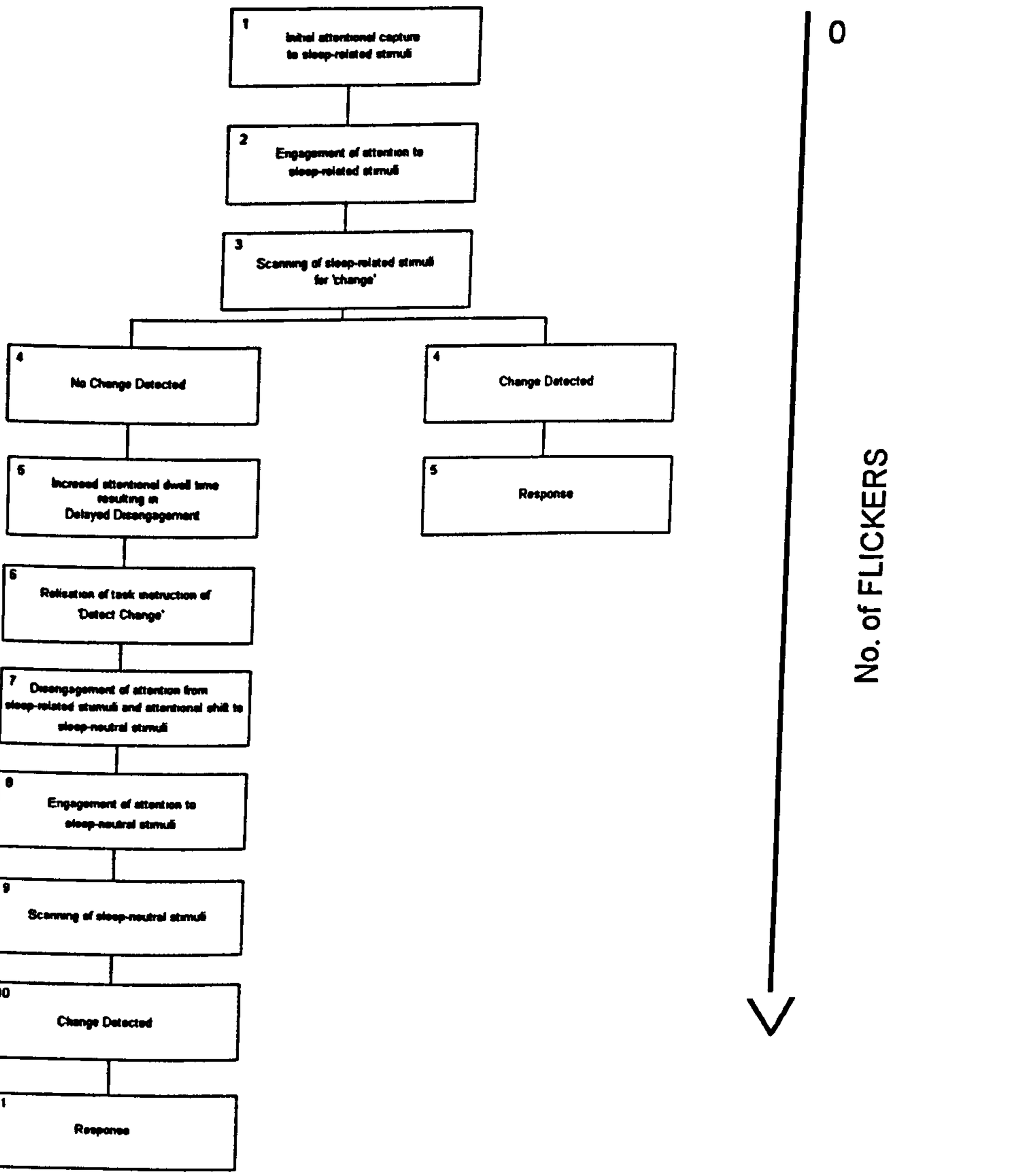
Perhaps an easier way to understand this suggestion is to look first at the sequence of events predicted to occur in the insomnia group, when the 'change' is embedded within the *sleep-neutral* stimuli.(i.e. when the RT are slowest), and the *sleep-related* stimuli (when the reaction times are fastest). Figure 20 provides an illustration of the sequences of events predicted to occur in these conditions.

The marked difference in the reaction times of PI between the two levels of 'change' (sleep-related, sleep-neutral), can be clearly explained by these predictions, as the number 'steps' in the sequence is more than halved when the change is embedded within the sleep-related stimuli. Indeed, the reduced number of flickers to change detection, observed in the PI group on such trials, directly corresponds with this theory.

However, to return to the original question, why are the reaction times of PI and the control groups different at the neutral change, the sequences of events outlined above also provide a suitable explanation. More specifically, for PI we predict that on *neutral trials* the left sequence of events, outlined on figure 20, *always occurs*. Thus, PI will always direct attention initially to the salient stimuli, hold attention there for an extended period of time (i.e. increased dwell time), and subsequently be forced to disengage attention away to detect the 'change' as the task requires. Indeed, this is a time consuming process, and the

number of flickers continues to increase throughout each step.. Alternatively, for the control populations we predict that both sets of stimuli within the scene are of equal salience and thus the *controls have an equal probability* of directing their attention to either side of the scene.

Figure 20. Sequence of events during the ICB flicker paradigm for PI, in both sleep-related and sleep-neutral change conditions.

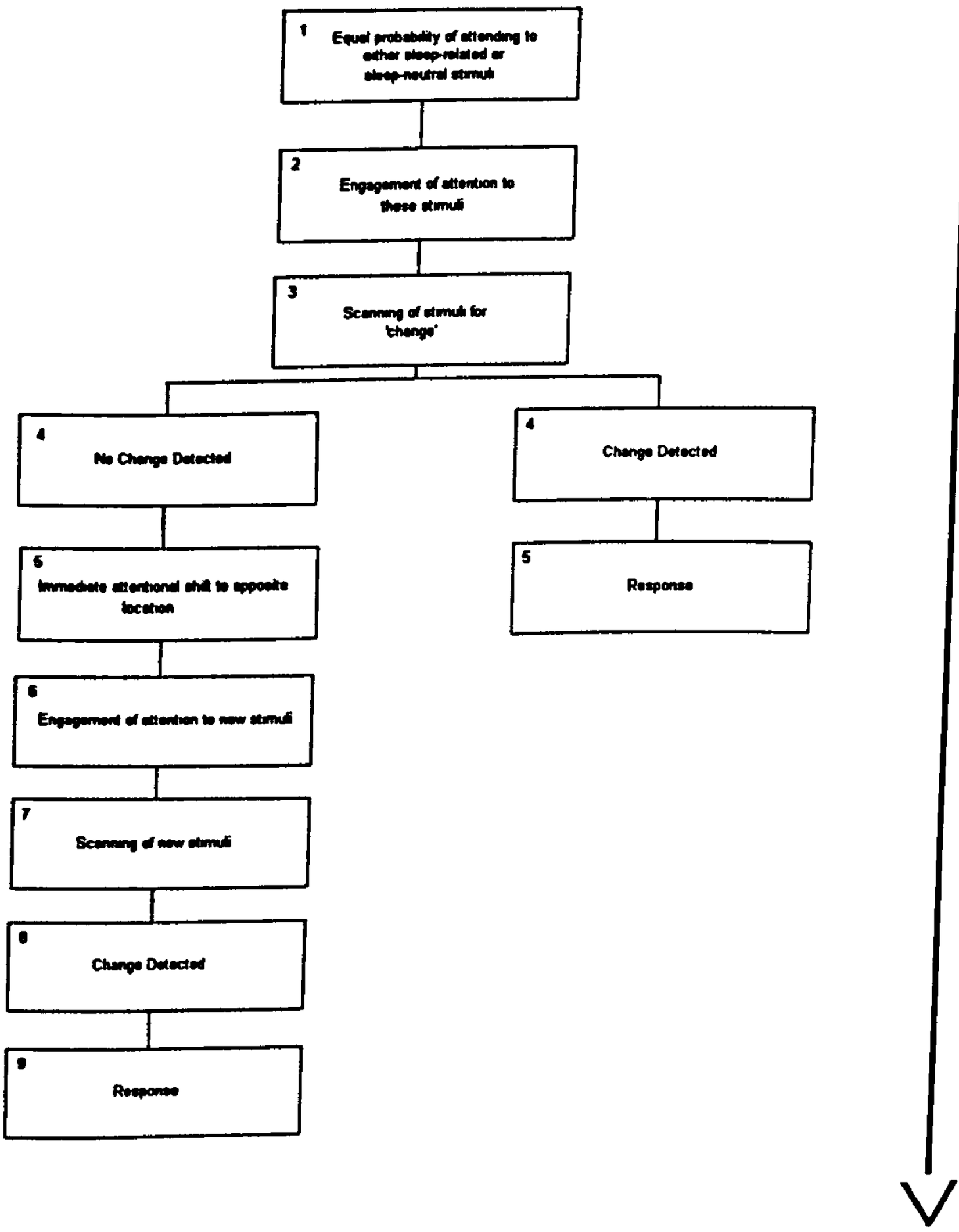


Therefore, based on the rules of probability, on half of the sleep-neutral conditions, GS and DSPS should direct their attention the sleep-neutral side of the scene where the change *is* occurring, resulting in faster reaction times than PI. However, even on the other half of the tests, where they initially direct their attention to the *opposite* location from where the' change' is occurring (the sleep-related side), they escape the attentional dwell time and thus delayed disengagement process seen in PI. Indeed, this again results in shorter reaction times. Figure 21 illustrates the two sequence of event predicted to occur in GS and DSPS.

The results of Experiment 3 are consistent with the previous work using the Posner paradigm in which delayed disengagement from emotionally salient stimuli has been demonstrated to exist in anxious populations (Fox et al. 2001). An interesting discussion point of attention bias in other psychological disorders is that the majority of research attributes the phenomenon to perceived threat. However, as discussed in chapter 7, although experiments 1 to 3 provide convincing evidence for the existence of attention bias and the components of attention that it involves, none provide insight into the emotional processes that my be driving the phenomenon. Indeed, when compared with threatening stimuli incorporated into other attention paradigms, none of the experimental stimuli utilized in Experiments 1 to 3 were intrinsically commanding of attention, nor emotive or threatening. More specifically, in a previous study the threatening picture set was taken fro the International Affective Picture System (IAPS; Lang et al. 1995) which include items such as, wounded people, missing limbs, human corpses etc (Yiend & Matthews, 2001). In comparison with such extremes, a teddy bear or pillowcase, as incorporated in Experiments 1 –3, appear

remarkably non-threatening. Thus, at this point it remained unclear as to whether the attention biases detected in insomnia, follow a threat-monitoring model similar to that of other psychological disorders.

Figure 21. Sequence of events during the ICB flicker paradigm for GS and DSPS, in both sleep-related and sleep-neutral change conditions.



Therefore, Experiment 4 attempted to provide a testable methodology for the types of sleep-related stimuli that generate attention bias in PI. As described in chapter 7, Experiment 4 implemented a semantic Posner task in which sleep-related stimuli were differentiated into intrinsically positive or intrinsically negative word types. Interestingly, only the negative sleep-related word types resulted in the slowed disengagement effects previously demonstrated in Experiment 3. Thus, negative sleep-words appear to have the greatest salience to PI when presented together with positive sleep-related and neutral words. Indeed, due to the negative nature of the stimuli generating AB in this experiment, these data may provide support for the theory that attention biases in PI are, in part, driven by perceived threat.

It is also important to acknowledge that a trend in the data, of delayed disengagement from positive words, was also observed in the PI population although this did not reach significance. Within chapter 7 an explanation of this result in terms of the nature of the stimulus presentations is presented. Additionally however, a further explanation for the direction of these data can be proposed. More specifically, although the positive words were rated as positive by the general population in a questionnaire (see pilot study, chapter 7), PI may not view such words as intrinsically positive. Indeed, it would be interesting to embed these positive sleep words in a collection of general non-sleep positive words, and ask an insomnia group to assign each a rating in terms of valence. Indeed, this may provide evidence that, in general, any sleep word rated by a person with insomnia would be viewed as intrinsically negative, as it may not be the words per se that they are responding to, but the thoughts and feelings that are generated by them and this would reflect the trend in the data observed for

the positive sleep-related words in Experiment 4. In conclusion however, within the constraint of this experiment negative sleep-related stimuli, as rated by the general population, appear to be the most salient to PI as compared to positive sleep-related stimuli and sleep- neutral stimuli.

8.3 Relating The Results to the Literature

As previously discussed in chapter 7, our laboratory group in Glasgow (GSRL) has recently presented the attention-intention-effort pathway (A-I-E), which attempts to highlight the mechanisms through which PI develops and maintains, and highlights the importance of the attention bias data derived from the experiments comprising this PhD.

The A-I-E pathway has its origins in the psychobiological inhibition model of insomnia (Espie, 2002), which differs from most other conceptualisations in that it takes as its starting point a perspective upon normalcy rather than pathology. The model considers what it takes to upset the course of normal good sleep, and to prevent (inhibit) its recovery.

Within the 'two process' system (process S, sleep homeostatic drive, process C, circadian variation), the sleep homeostat drives the sleep-wake schedule toward a balanced requirement in that prolonged wakefulness accrues 'sleep debt', and the circadian timer modulates sleep propensity on approximately a 24 h cycle (Dijk & Czeisler, 1995). The GSRL suggest, however, that there is an implicit ancillary process that is associated with the automatic regulation of sleep-wake patterns in good sleepers. The concept of automaticity (Espie, 2002) refers to the largely involuntary nature of the well-adjusted sleep schedule, and to the over-learned associations that may form part of a good sleep stimulus control paradigm

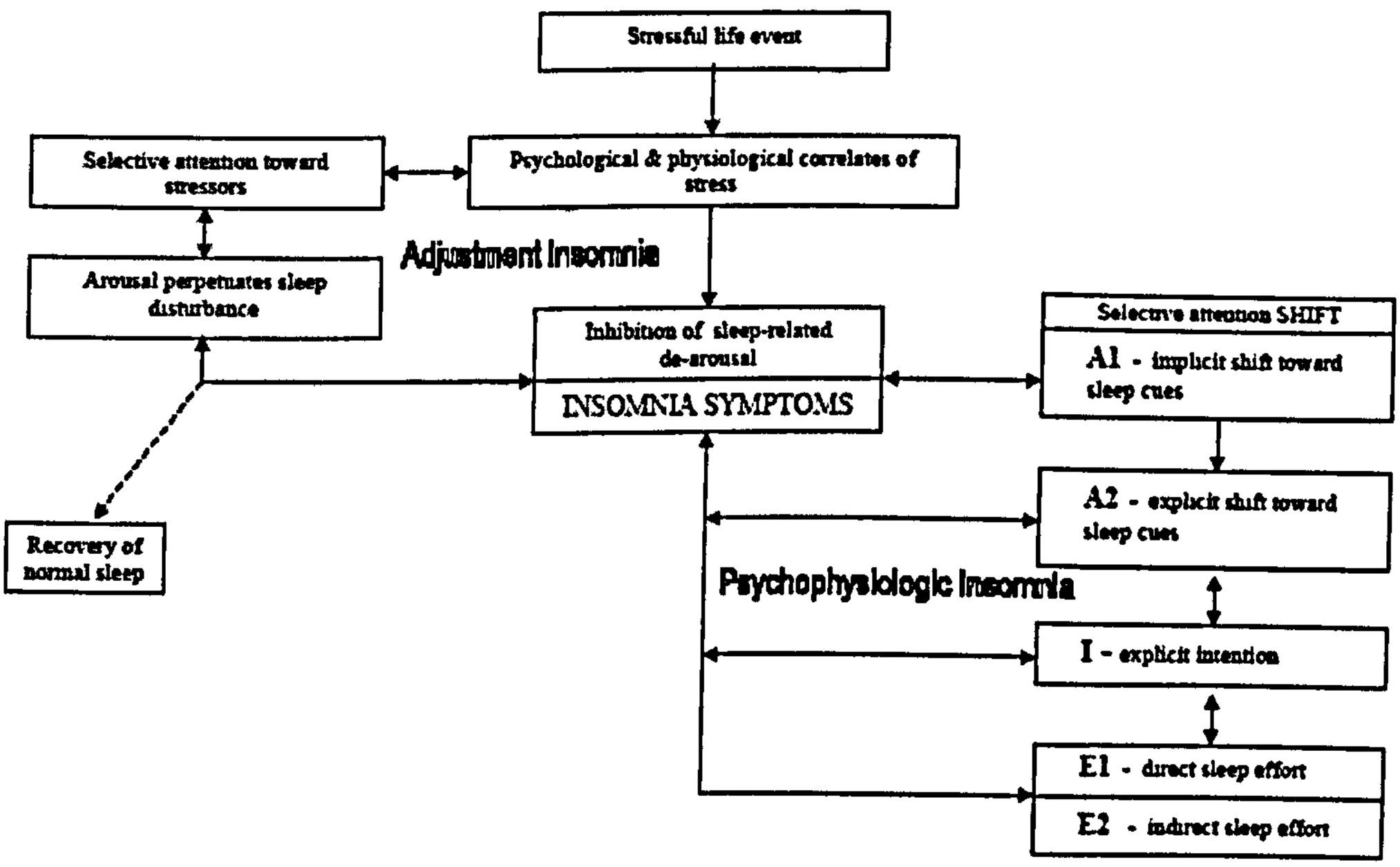
(Bootzin et al. 1991). In other words, the good sleeper as essentially passive because internal and external cues act as automated setting conditions for sleep, and these are further reinforced by rapid sleep-onset. Endogenous cues to sleep, such as physical and mental fatigue, are presumed to interact reciprocally with exogenous perhaps classically conditioned cues, in the bedroom environment; so that the good sleeper approaches sleep, just as s/he walks or talks - without thinking much about it and without a consciously explicit plan (Espie at al. 2006)

Indeed this 'third process' of automaticity may be central to PI, and it is hypothesized that, because the sleep-wake process is essentially self regulatory, de-arousal and sleep engagement may be particularly vulnerable if for any reason the process is switched out of its natural automated mode. The term inhibition has been adopted for this 'switching', for two reasons. First, main focus of the pathway is upon factors that might be preventing the expression of normal sleep and preventing its natural recovery. Second, assumptions that PI is associated causally with any particular sleep pathology are not made. Rather, it is thought that people with PI have the potential to sleep normally if inhibitory factors can be overcome.

Thus, the GSRL proposes that sleep-wake automaticity can be inhibited by selectively attending to sleep, by explicitly intending to sleep, and by introducing effort into the sleep engagement process (Figure 22). Subsequently, the experiments comprising this PhD research have provided direct evidence that selectively attending to sleep is a testable and subsequently detected factor in PI populations. Furthermore, the GSRL suggest that, close to the point of normal resolution of adjustment insomnia, selective attention, which is at this stage upon the stressor might reduce markedly. However, in circumstances where insomnia

symptoms still persist, there could be an increased risk that an attention bias towards sleep-related cues might develop. That is, attention might shift from the resolving stressor to any persisting sleep disturbance at this point. Furthermore, the transition to sleep related implicit attention bias could be prepared by the frequent prior conditioning of sleep cues with sleeplessness during preceding weeks.

Figure 22. Proposed evolution of psychophysiologic insomnia from adjustment insomnia following the A-I-E pathway.



The data from Experiment 1-4 additionally parallel that of Harvey's 'real world' experiments, in which attention is manipulated in order to test its causal role in increasing or decreasing insomnia symptoms. Furthermore, these data support the cognitive model of insomnia, which holds that the anxious state displayed by PI precipitates attentional narrowing and preferential allocation of attentional resources to sleep-related threat cues (Harvey, 2001). However, the contribution of anxiety or worry to the A-I-E model remains unclear. In these current experiments the STAI scores appeared consistent, with generally elevated scores in PI as compared to controls. Indeed, this descriptive measure highlights that the PI samples used within Experiments 1-4 were reflective of PI samples across other research studies, as PI populations are expected to be generally trait and situationally state more anxious than GS. However, the participants within this study were mot clinically anxious (i.e. did not have a diagnosis of anxiety or were receiving any intervention for anxiety), and this research was not designed t partial effects. An interesting progression to future experiments could assess the nature of attention bias in different types of PI patients. More specifically for example, Watts et al. (1994) demonstrated that insomnia sufferers could be divided into two distinct sub-groups, namely worrying ans non-worrying insomniacs. Here it would be interesting to assess whether attention bias data could differentiate these patients or whether both sets would demonstrate the attention bias effects. Additionally, as previously mentioned in chapter 4, a measure of neuroticism should also be included in future research with similar populations to identify correlations between high levels of neuroticism and the AB effects. Similarly, further studies are required to investigate the specificity of

sleep-related attention bias to PI, as opposed to other forms of sleep disorder including those that are comorbid with other psychopathological complaints.

8.4 SECONDARY OBSERVATIONS

Another important finding of the data comprising this PhD, is the stability of the sleep scores across the experimental groups. Most specifically, at the beginning of this thesis it was highlighted that the inclusion of DSPS as a control group was an important methodological feature. Indeed, as discussed in chapter 4, DSPS involves initial insomnia, similar to sleep-onset psychophysiological insomnia, but with no presumed psychological mechanism. Thus, due to the similarities of the two complaints DSPS is rarely screened out of insomnia research, however, DSPS do not exhibit the cognitive arousal mechanism observed in PI. The sleep data results of DSPS highlight the importance of this methodological decision. Specifically, across all studies DSPS were shown to have a TST similar to that of the GS group. Indeed no significant differences were observed between DSPS and GS on both TST and WASO measures. Thus, these data provide concrete evidence that DSPS have a sleep duration and quality that cannot be differentiated from that of GS. Indeed the only measure that can differentiate the two control groups in terms of sleep pattern is the actual time of sleep onset/offset, i.e. significantly later in DSPS than GS. Thus, inclusion of DSPS in the PI group would severely dilute the results, as the sleep data within this PhD has evidenced that DSPS actual sleep efficacy is that of a GS. It is also important to acknowledge however, that the PSQI scores of DSPS across the experimental groups had a high degree of variability. Indeed, this makes it

difficult to compare their results across the experimental studies. On reflection, future inclusion/exclusion criteria for this experimental population should be tightened in an attempt to capture a DSPS group that are *either* genetically or socially driven, not a combination of both, as discussed in chapter 4.

8.5 OVERALL CONCLUSIONS & FUTURE RESEARCH DIRECTIONS

In overall conclusion, the replication of attention bias data, within and across these experimental paradigms, highlights the stability of the attention bias phenomenon in insomnia. Furthermore, these data sets suggest the plausibility of a 'cognitive marker of insomnia'. Indeed, our Glasgow Sleep Research Laboratory has previously published data indicating that attention bias to sleep-word cues is absent in acute/adjustment insomnia but present in persistent insomnia, and this further reinforces the importance of how, and when, attention bias develops in relation to the complaint of insomnia.

Of course, there are a number of factors limiting the plausibility of the existence of such a diagnostic marker. First, because the experimental populations within this PhD were a recruited student population, the findings may not be generalisable to the clinical pole. Attention bias studies, therefore, require replication with clinical samples, that have been assessed for sleep quality by rigorous objective measures e.g. EEG recording along with actigraphy. Nevertheless, it should be noted that non-clinical populations do display cognitive over-activity at bedtime, excessive worry about sleep, distorted perception, and characteristic safety behaviours (cited in Harvey, 2002). Besides, processes detected at any stage of sleep disruption, particularly before the clinical

extreme, may provide crucial evidence about the mechanisms involved in the maintenance/enhancement of the disorder proper. However, if conducting future research, again with this population, concordance of self report and actigraphy measures should be validated with EEG recordings, to ensure appropriate inclusion/exclusion. Additionally, future research should aim to include other control groups beyond DSPS, such as sleep experts. Indeed this group would enable a clever comparison as this group would, arguably, have some form of preoccupation with sleep.

Second, none of the experiments within this PhD were intervention studies. It is not known whether attention bias in insomnia reduces as a result of effective intervention, as it has been shown to do following cognitive behavioural therapy (CBT) for anxiety disorders (Matthews et al. 1995; Mogg et al., 1995). Demonstrating that established psychological treatments such as stimulus control or multi-component CBT impact attention bias in PI would add strength to the argument that such biases play a critical role. Third, these results suggest an important pathway in insomnia, but cross sectional comparison of the type reported here cannot test aetiological factors. The work of Harvey and colleagues is leading the way in this regard by emphasising the application of controlled experimental methods to tease out the causal pathways associated with sleep associated monitoring (Neitzert-Semler & Harvey, in press; Tang et al. in press).

Fourth, the data concerning the anxiety scores, within this PhD, have generated speculation of whether anxiety is an important core feature for the existence of AB in PI. Future research should assess whether people suffering from insomnia in the absence of elevated anxiety show similar AB results as those presented here. Furthermore, AB effects and anxiety scores of PI patients should be tracked

over time, even after the PI episode had subsided. Indeed, this would allow for the direct comparison of AB effects at each stage of the PI episode as well as during remission. Indeed, the absence of AB effects at the remission stage of testing, as well as a decrease in anxiety scores, would provide further support for AB acting as a reliable cognitive marker of PI, and additionally support the important role anxiety plays in the AB phenomenon.

Methodologically, there are also a number of limitations. First, within experiment 4, the total umber of participants per group was only 15 and retrospective power calculations suggested that 22 participants per group would have been more sufficient for this experiment. This has quite important implications for the results of Experiment 4, as the failure of the positive sleep word to reach significance in the disengagement analysis may be due to the experiment being under powered. Future research should consider this fact and incorporate larger sample sizes of at least 22 per group. Longitudinal data has also not been collected with these paradigms and methodology. Future research should track the existence of attention bias processes over the course of the insomnia complaint. Indeed this will provide more information on, both, the stability of the AB phenomenon over time, and the reliability of the methods used.

Finally, a limitation of any experimental research utilising cognitive probe tasks concerns the 'compliance to instructions' of the participants. More specifically, as described within each experimental chapter, strict, clear instructions were given to each participant before commencing the computer tasks. Indeed, the importance of maintaining fixation to the fixation points within Experiments 2-4 was highlighted. However, unless the experimental procedure

involves an eye-tracking device, which pinpoints exactly if this fixation is maintained, one cannot be certain that this compliance was met. However, due to the consistent Global Posner paradigm effects observed across the experimental data, it appears highly likely that this instruction was followed.

8.6 SUMMATION

The data within this PhD research are consistent with other recent experiments in suggesting the potentially important role of selective attention bias in insomnia. Computerised cognitive probe paradigms, using tasks such s the ICB and Posner, may offer insomnia research a much-needed objective index of sleep-related mental arousal. The consistency of the attention bias phenomenon, demonstrated within this current research, provide solid grounds for the investigation of somatic correlates of attention bias that will aid in our understanding of insomnia as a Psychophysiologic disorder with automatic, cortical and cognitive components. More specifically, if the attention bias paradigm provides a direct measure of cognitive arousal, and skin conductance or heart rate variability offer a parallel measure of autonomic function, then comparisons of such data with quantitative EEG parameters could prove particularly informative in exploring the underpinnings of the PI phenotype.

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APPENDIX A

Example The Stroop Paradigm

Red

Blue

Green

Yellow

Red

Blue

Green

Blue

Yellow

Red

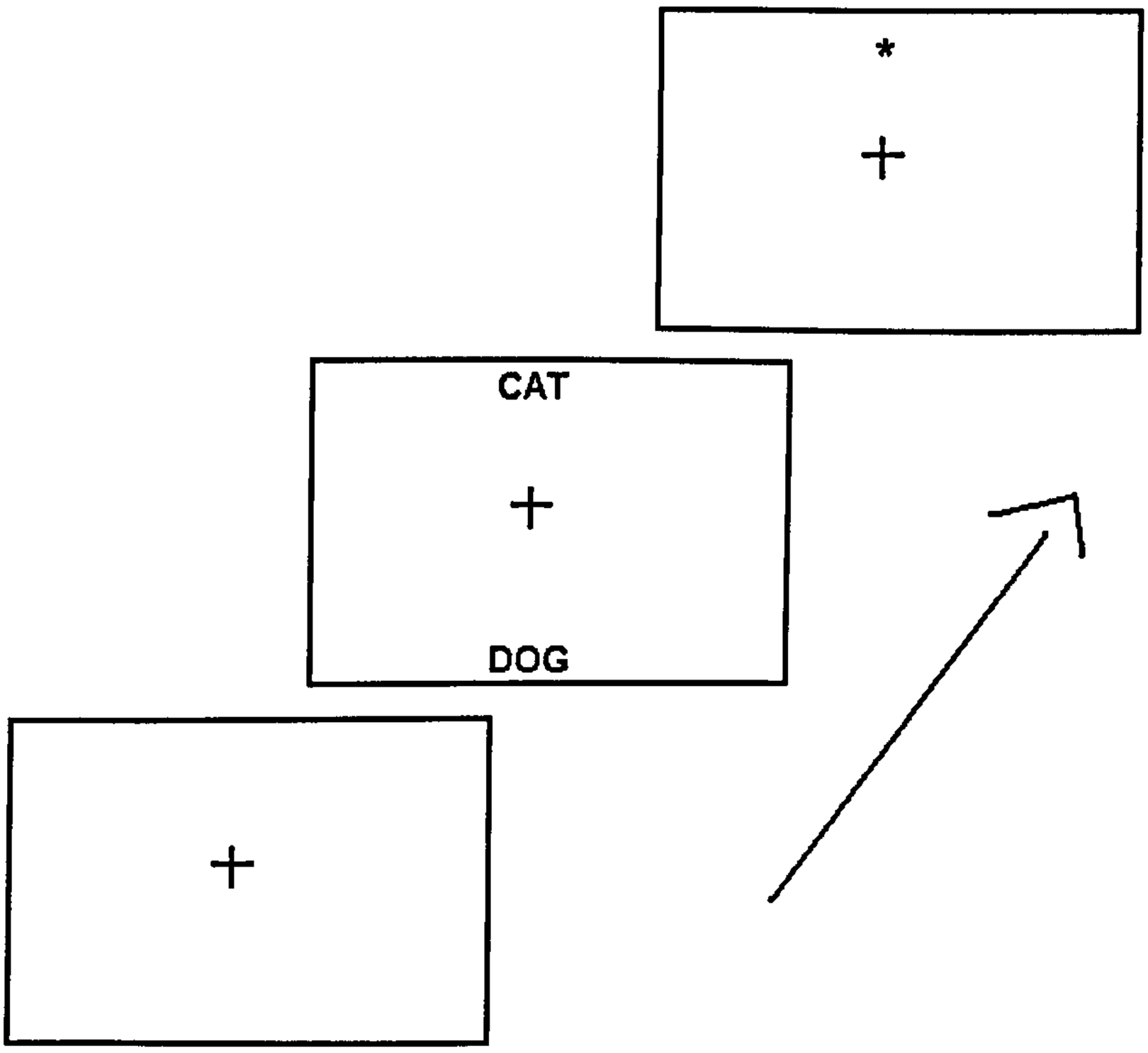
Instructions

Participants are asked to respond to the colour of the ink in which each of the words is written, and to ignore the semantic content of the word. They are asked to do this as quickly as possible. Reaction time to respond to each word is measured.

The Emotional Stroop Task is a variation of this original task. Here, emotional and neutral words are presented in different coloured ink. The instructions are identical to those above. Research has shown that when words have an emotional salience to participants' RT to the response of those words is significantly increased.

APPENDIX B

The Dot Probe Paradigm



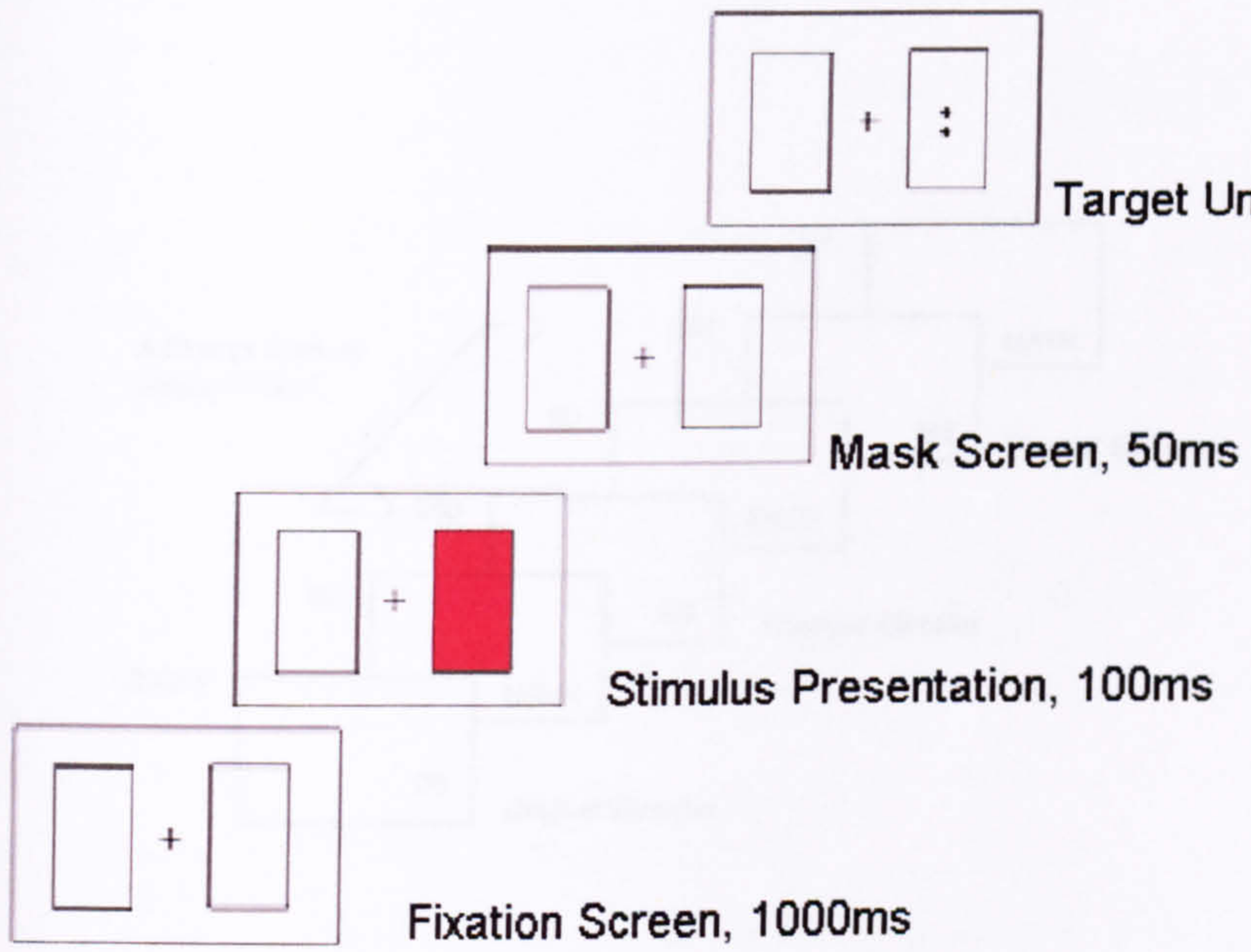
Instructions

Participants are asked to fixate on the central cross at all times. Two words appear on the screen, one above the central cross and one below. Shortly after they disappear an asterisk appears in either the position of one word or the other. Participants are asked to respond to asterisk by making a decision about its position. Reaction time to respond to each asterisk is measured.

Research has shown that when words have an emotional salience to participants' RT to the response of asterisk replacing these words are significantly faster. Furthermore, RTs to asterisk occurring in the opposite location are significantly longer.

APPENDIX C

The Posner Paradigm



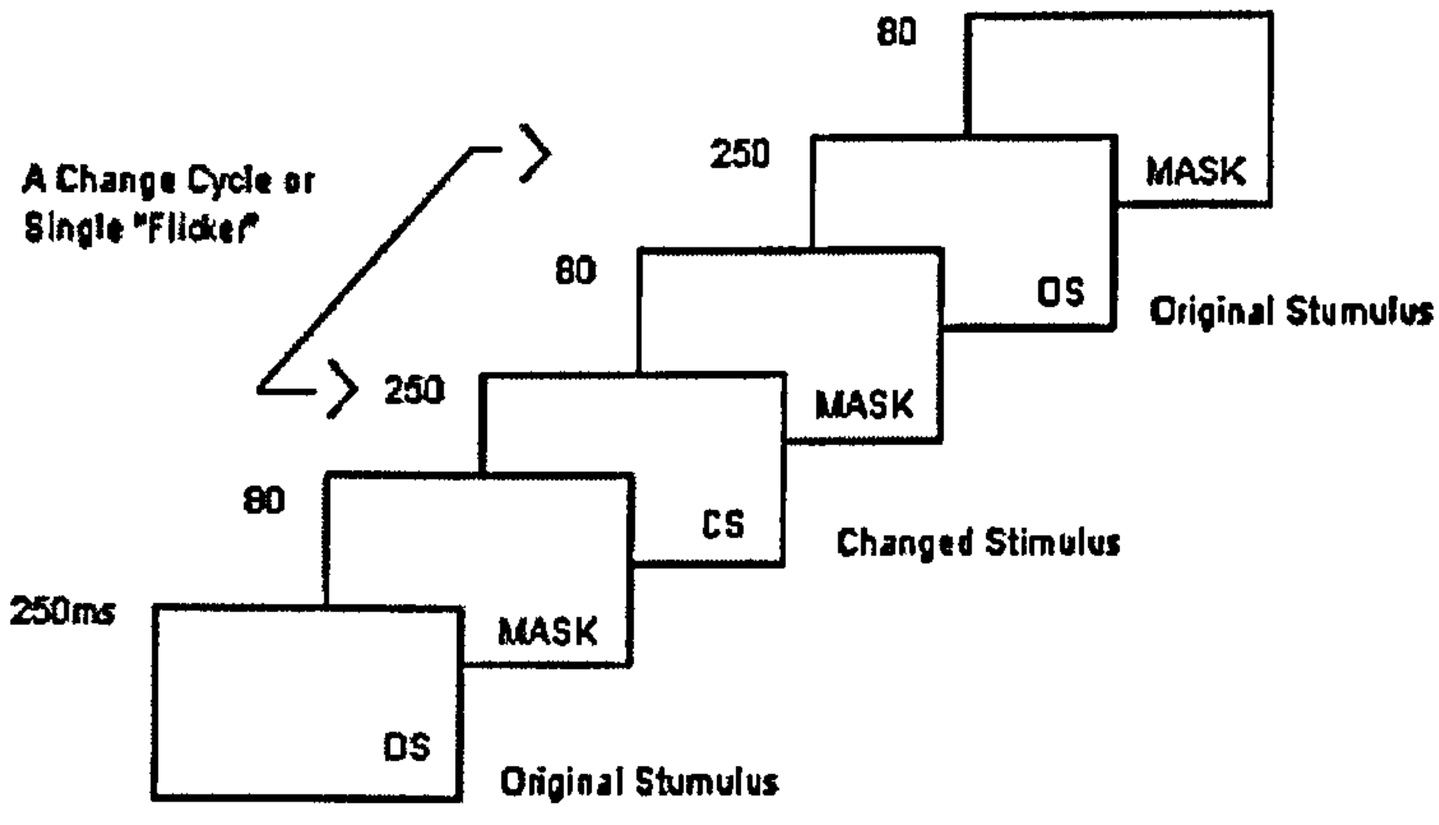
Instructions

Participants are asked to fixate on the central cross at all times. A word or picture cue appears in one of the two rectangular boxes then disappear. A taget then appears in either the same location as the previous cue (valid trials) ot in the opposite location (invalid trials). Participants are asked to respond to the target by making a decision about its location or shape. Reaction time to respond to each target is measured.

Research has shown that when compared with neutral cues, word or picture cues that have an emotional salience to participants' result in greater RT on invalid trials.

APPENDIX D

The Inducing Change Blindness Flicker Paradigm



Instructions

Participants are told that they will see a scene on the screen that will be switched on and off repeatedly – each appearance and disappearance lasting less than a second. They are instructed that they have to "spot a change made to the visual scene at some point in the series of 'flickers' and to indicate this detection by immediately pressing the button on the response box". Thus, the ICB paradigm is a RT task. After the participants detect and respond to the change, they are asked to "confirm what the change was".

Research has shown that when a change is made to an object salient to the participant, RT to change identification is fast. However, when a change is made in the opposite location to the object of high salience, RTs are significantly slower.

APPENDIX E

DSM-IV and ICSD-R interview screening

Participant ID	

ICSD-R statement of criteria for psychophysiologic insomnia	Comments	Yes	No
A. A complaint of insomnia* is present and is combined with a complaint of decreased functioning during wakefulness.			
B. Indications of learned sleep preventing associations are found and include the following:			
1.Trying too hard to sleep, suggesting an inability to fall asleep when desired, but ease of falling asleep during other relatively monotonous pursuits, such as watching television or reading.			
2. Conditioned arousal to bedroom or sleep-related activities, indicated by sleeping poorly at home, but sleeping better away from home or when carrying out bedroom routines.			
C. There is evidence that the patient has increased somatised tension (e.g. agitation, muscle tension, and increased vasoconstriction.			
D. Polysomnographic monitoring demonstrates all of the following:			
1. An increased sleep latency			
2. Reduced sleep efficiency			
3. An increased number and duration of awakenings.			
E. No other medical or mental disorders accounts for the sleep disturbance.			
F. Other sleep disorders can co-exist with the insomnia (e.g. inadequate sleep hygiene, obstructive sleep apnea, etc.			
Minimum criteria: A plus B			
Acute: 4 weeks or less; Subacute: More than 4 weeks, but less than 6 months; Chronic: 6 months or longer.			
Mild insomnia: This term descnbes almost nightly complaint of an insufficient amount of sleep or not feeling rested after the habitual sleep episode. It is accompanied by little or no evidence of impairment of social or occupational functioning. Mild insomnia often Is associated with feelings of restlessness, irritability, mild anxiety, daytime fatigue, and tiredness. Moderate insomnia: This term describes a nightly complaint of an insufficient amount of sleep or not felling rested after the habitual sleep episode. It is accompanied by mild or moderate impairment of social or occupational functioning. Moderate insomnia always is associated with feelings of restlessness, irritability, anxiety, daytime fatigue, and tiredness. Severe insomnia: This term describes a nightly complaint of an insufficient amount of sleep or not feeling rested after the habitual sleep episode. It Is accompanied by sever impairment of s0cial or occupational functioning. Severe Insomnia Is associated with feelings of restlessness, irritability, anxiety, daytime fatigue, and tiredness.			
DSM IV statement of criteria for primary Insomnia	**Comments**	**Yes**	**No**
A. Difficulty initiating or maintaining sleep or non-restorative sleep, for at least one month.			
B. The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.			
C. The disturbance in sleep does not occur exclusively during the course of another mental disorder.			
D. The disturbance in sleep does not occur exclusively during the course of another sleep disorder			
E. The sleep disturbance is not due to the direct physiological effects of a substance or a general medical condition			
ICD-10 Statement of criteria for insomnia	**Comments**	**Yes**	**No**
The complaint is either of difficulty falling asleep or maintaining sleep, or of poor quality of sleep.			
The sleep disturbance has occurred at least three times per week for at least 1 month			
ICSD-R statement of criteria for DSPS	**Comments**	**Yes**	**No**
A. The patient has a complaint of an inability to fall asleep at the desired clock time, an inability to awaken spontaneously at the desired time of awakening, or excessive sleepiness.			
C. The symptoms are present for a least 1 month.			

APPENDIX F

Pittsburgh Sleep Quality Index (PSQI)

PITTSBURGH SLEEP QUALITY INDEX (PSQI)

Name_____ ID#_____ Date_____ Age_____

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, when have you usually gone to bed at night?
USUAL 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, when have you usually gotten up in the morning?
USUAL 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 spend 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 past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____
(b) Wake up in the middle of the night or early morning
Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____
(c) Have to get up to use the bathroom.
Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____
(d) Cannot breathe comfortably.
Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____
(e)Cough or snore loudly.
Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____
(f)Feel too cold.
Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____
(g) Feel too hot.
Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more 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_____

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 (Prescribed or "over the counter") to help you sleep?

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 share a room?

No bed partner or do not share a room _____

Partner/ flatmate in other room _____

Partner in same room, but not same bed _____

Partner in same bed _____

11. If you have a bed partner or share a room, 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 past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

(d) Episodes of disorientation or confusion during sleep.

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

(e) Other restlessness while you sleep: please describe_____

_____

___

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

[Buysse DJ, Reynolds CF, Monk TH, Berman SR, DJ Kupfer (1989) The Pittsburgh Sleep Quality Index: A New Instrument for Psychiatric Practice and Research, **Psychiatry Research, 28**: 193-213].

APPENDIX G

Sleep Diary

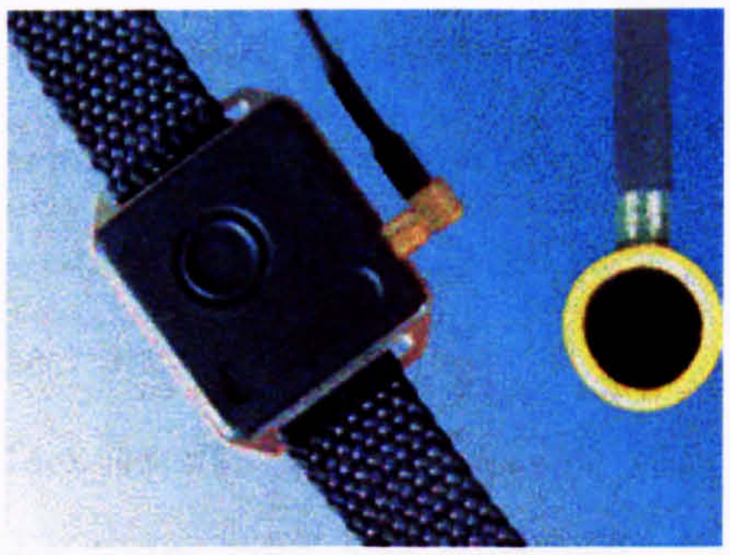
	1	2	3	4	5	6	7
1. What time did you rise from bed this morning?							
2. At what time did you go to bed last night?							
3. How long did it take you to fall asleep (minutes)?							
4. How many times did you wake up during the night?							
5. How long were you awake during the night (in total)?							
6. About how long did you sleep altogether (hours/mins)?							
7. How much alcohol did you take last night?							
8. How many sleeping pills did you take to help you sleep?							

MEASURING THE QUALITY OF YOUR SLEEP

1. How well do you feel this morning? 0 1 2 3 4 not at all moderately very							
2. How enjoyable was your sleep last night? 0 1 2 3 4 not at all moderately very							
3. How mentally alert were you in bed last night? 0 1 2 3 4 not at all moderately very							
4. How physically tense were you in bed last night? 0 1 2 3 4 not at all moderately very							

APPENDIX H

Actigraph Device



APPENDIX I

Spielberger State/Trait Anxiety Questionnaire

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

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

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

1. I feel calm	①	②	③	④
2. I feel secure	①	②	③	④
3. I am tense	①	②	③	④
4. I feel strained	①	②	③	④
5. I feel at ease	①	②	③	④
6. I feel upset	①	②	③	④
7. I am presently worrying over possible misfortunes	①	②	③	④
8. I feel satisfied	①	②	③	④
9. I feel frightened	①	②	③	④
10. I feel comfortable	①	②	③	④
11. I feel self-confident	①	②	③	④
12. I feel nervous	①	②	③	④
13. I am jittery	①	②	③	④
14. I feel indecisive	①	②	③	④
15. I am relaxed	①	②	③	④
16. I feel content	①	②	③	④
17. I am worried	①	②	③	④
18. I feel confused	①	②	③	④
19. I feel steady	①	②	③	④
20. I feel pleasant	①	②	③	④

APPENDIX J

Beck Depression Inventory – Fast Screen

1.

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

2.

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

3.

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

4.

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

5.

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

6.

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

7.

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

APPENDIX K

Good Sleep Guide

DURING THE EVENING

1 Put the day to rest Think it through Feeling worried or anxious can be one of the main causes of a sleepless night Tie up "loose ends" in your mind and plan ahead A notebook may help

2 Take some light exercise such as walking or swimming early in the evening. Generally try to keep yourself fit

3 Wind down during the course of the evening Try not to do anything that is mentally demanding within 90 minutes of bedtime Try having a warm bath or listening to some soothing music

4. Do not sleep or doze in the armchair. Keep your sleep for bedtime

5 Do not drink too much coffee or tea and only have a light snack for supper. Do not drink alcohol to aid your sleep - it usually upsets sleep

6 Make sure your bed and bedroom are comfortable not too cold and not too warm

AT BEDTIME

1 Go to bed when you are "sleepy tired" and not before

2 Do not read or watch TV in bed Keep these activities for another room

3 Set the alarm for the same time every day - 7 days a week, or at least until your sleep pattern settles down

4 Put the light out

4. Put the light out when you get into bed and try to ensure your room is as dark as possible

5 Let yourself relax and tell yourself that "sleep will come when it's ready" Enjoy relaxing even if you don't at first fall asleep

6 Do not try to fall asleep Sleep is not something you can switch on deliberately but if you try to switch it on you can switch it off

IF YOU HAVE PROBLEMS GETTING TO SLEEP

1 Remember that sleep problems are quite common and they are not as damaging as you might think Try not to get upset or frustrated

2. If you are awake in bed for more than 20 minutes then get up and go into another room

3 Do something relaxing for a while and don't worry about tomorrow Avoid watching the clock, as this will only make the time pass more slowly People usually cope quite well even after a sleepless night.

4. Go back to bed when you feel "sleepy tired"

5. Remember the tips from the section above and use them again each time you waken up

6. A good sleep pattern may take a number of weeks to establish. Be confident that you will achieve this in the end by working through the "GOOD SLEEP GUIDE"

This guide has been prepared by Professor Colin A Espie, University of Glasgow, Department of Psychological Medicine, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH

APPENDIX L

Sleep questionnaire from Experiment 1

Please list below at least 5 objects that you would associate with bedtime and sleep.

Primary Investigator: Lauren Macphee
Supervisor: Dr Biello/Prof Espie
Email: Lauren@psy.gla.ac.uk

APPENDIX M

Pilot Questionnaire from Experiment 4

Below is a list of words that you have to assign a rating. The rating you have to assign is positive, negative or neutral. Try not to think about your responses too much, just put down the initial response that comes to you. Please mark your response by ticking the corresponding column.

For the last two columns please answer the questions below and mark a rating between 1 and 10 in the correct column.

Question 1: How closely related to sleep would you rate each of these words. 1 being not closely related and 10 being very closely related.

Question 2: Based on your answers in the first columns please add a rating between 1 and 10 for how positive or negative each word is in relation to sleep. 1 being a very negative sleep word and 10 being very positive sleep word.

	Positive	Neutral	Negative	Question 1	Question 2
Dream/ dreaming					
Naps/ napping					
Vigilant					
Drowsiness					
Nightmare					
Bed					
Fatigue					
Comfy/ comfort/ comfortable					
Rested					
Cosy					
Dozing/ doze					
Slumber					
Refreshed					
Snooze/ snoozy					
Restless					
Stress					
Untroubled					
Pillow					
Insomnia					
	Positive	Neutral	Negative	Question 1	Question 2

Watchful					
Asleep					
Rest					
Tossing/ Turning					
Kip					
Yawning					
Motionless					
Tranquillity					
Waking					
Sluggish					
Lethargy					
Contented					
Snug					
Worry					
Lonely/ loneliness					
Aggravated					
Tired					
Exhausted					
Sleepy					
Alert/ alertness					
Snoring					
Arousal					
Siesta					
Awake					
Ritual					
Peaceful					
Relaxed					
Silence					
Wakeful					
Disturbed					
Carefree					
Duvet					
Weary					
Drained					
Whacked					

If there are any other sleep words that are not listed above that you feel should be considered please note them below.