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# ATTENTION BIAS FOR POSITIVELY AND NEGATIVELY VALENCED SLEEP-RELATED PICTORIAL STIMULI IN PSYCHOPHYSIOLOGICAL INSOMNIA, DELAYED SLEEP PHASE SYNDROME AND NORMAL SLEEP: AN INVESTIGATION USING THE DOT-PROBE PARADIGM

AND

### **CLINICAL RESEARCH PORTFOLIO**

#### **VOLUME I**

## (VOLUME II BOUND SEPARATELY)

# Alison Murie MA (Hons) August 2008

Submitted in partial fulfilment of the requirements of the degree of Doctor of Clinical Psychology (DClinPsy)

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## **CHAPTER 1: SYSTEMATIC LITERATURE REVIEW**

# The Impact of Cognitive-Behavioural Therapy (CBT) on Dysfunctional Beliefs and Attitudes about Sleep – A Systematic Review

Prepared in accordance with submission to Journal of Sleep Research (Appendix 1.1)

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#### SUMMARY

The efficacy of Cognitive-behavioural Therapy (CBT) for insomnia is well-established within the extant literature, where the impact of treatment upon sleep parameters, such as sleep onset latency and sleep efficiency, is generally the primary outcome measure. Several models now highlight the role of dysfunctional beliefs and attitudes about sleep in the development and maintenance of insomnia, and there is evidence to suggest that sleep-related cognitions are themselves amenable to change in response to treatment. A systematic review was conducted on 14 studies (n = 1339) published between 2000 and 2007 inclusively, the aim of which was to investigate the impact of CBT on dysfunctional beliefs and attitudes about sleep in insomnia; the association between treatment-induced cognitive change and improvements on sleep parameters was also examined. Thirteen of the studies report significant change in dysfunctional sleep-related beliefs in response to CBT, as indicated by reduced scores on the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS). Three of the studies analysed the association between cognitive change and improvements in sleep; although positive correlations were observed, the level of association was generally modest. Overall, the findings suggest that CBT is effective in reducing dysfunctional sleep-related cognitions, and that reductions in such cognitions are associated with improvements on both subjective and objective measures of sleep. Furthermore, these improvements are maintained at follow-up assessment. However, a number of important methodological caveats to the evidence base presented herein must be considered when forming conclusions regarding the impact of CBT upon dysfunctional sleep-related cognitions in insomnia, and their association with improvements on an individual's overall sleep condition.

KEYWORDS: sleep, insomnia, beliefs, attitudes, CBT

#### **INTRODUCTION**

Insomnia is a highly prevalent, often debilitating condition, characterised by difficulty initiating sleep, frequent and prolonged nocturnal awakenings, or early morning awakenings with an inability to return to sleep (Research Diagnostic Criteria; RDC; Edinger et al., 2004). Estimated prevalence rates suggest that approximately one third of the adult population experiences insomnia symptoms, and approximately 6% satisfy formal diagnostic criteria for insomnia (Ohayon, 2002). Pre-existing insomnia is the highest attributable, potentially treatable, risk factor for first episode depressive disorder and for recurrence of depression in adults and older adults (Riemann and Volderholzer, 2003; Cole and Dendukuri, 2003). Insomnia can therefore be detrimental at both an individual and societal level, resulting in reduced quality of life, and significant public and healthcare costs.

Research investigating the role of cognitive factors potentially implicated in the development and/or maintenance of insomnia has commonly focused on the impact of pre-sleep or nocturnal unwanted intrusive thoughts (also referred to as worry or cognitive arousal). However, it is not clear whether these situational and automatic thoughts are implicated in the aetiology of insomnia, or whether they are merely an epiphenomenon of poor sleep (Waine et al., in press). However, the importance of underlying unhelpful beliefs and attitudes about sleep (schema) has become increasingly recognised. Morin (1993) conducted the first empirical study to specifically investigate whether people with chronic insomnia held more unhelpful beliefs about sleep in comparison to good sleepers. Older adults with insomnia were found to endorse stronger beliefs about the potential negative consequences of insomnia, were

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less realistic than good sleepers about how much sleep they required, and were more likely to worry about losing control and the unpredictability of sleep (Morin, 1993).

Several theoretical models now highlight the role of dysfunctional beliefs and attitudes about sleep in the development and maintenance of insomnia (e.g. Espie, 2002; Harvey, 2002; Lundh and Broman, 2000) whereby such beliefs are presumed to "underlie and perpetuate sleep-related anxiety and habits that interrupt the sleep process" (Edinger and Wohlgemuth, 2001, Pg., 494). For example, it is hypothesised that the belief that there is little that one can do about poor sleep may maintain sleep-related anxiety. Similarly, the belief that it is necessary to try to 'catch up' for lack of sleep, may lead to what are described as 'self-defeating compensatory practices', such as remaining in bed beyond the usual rising time, or napping during the daytime.

Empirical evidence implicating dysfunctional sleep-related cognitions in insomnia is growing. In a recent cross-sectional study examining the potential role of arousal, distress, and sleeprelated beliefs in the maintenance of insomnia (Jansson and Linton, 2008), beliefs regarding the long-term negative consequences of insomnia were found to be more strongly associated with persistent insomnia than arousal or distress. Reduction in unhelpful beliefs following treatment, as measured by self-report questionnaire measures such as the Dysfunctional beliefs and Attitudes about Sleep Scale (DBAS: Morin, 1993), has been associated with both objective and subjective improvements in other sleep parameters (e.g. Edinger et al., 2001; Morin et al., 2002; Carney and Edinger, 2006). This may suggest that a greater degree of dysfunctional sleep-related cognitions may be "a cognitive pathway for mediating treatment

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effects" (Espie et al., 2001, Pg., 65). These studies suggest therefore that dysfunctional beliefs and attitudes about sleep may be important contributory and perpetuating mechanisms that warrant further investigation.

#### AIMS AND OBJECTIVES

CBT is currently considered to be the treatment of choice for persistent insomnia (Espie, 1999; Jacobs et al., 2004). Literature documenting the efficacy and clinical effectiveness of psychological and behavioural interventions for insomnia tends to emphasise the impact of such treatments on specific sleep parameters such as sleep onset latency (SOL), prolonged wakefulness after sleep onset (WASO) and sleep efficiency (SE) as primary outcome measures (e.g. Smith et al., 2002; Irwin et al., 2006; Morin et al., 2006). However, as outlined previously, growing evidence suggests that dysfunctional sleep-related beliefs and attitudes may be implicated in the development and/or maintenance of insomnia, and that they may be amenable to change in response to cognitive-behavioural interventions. As stated, dysfunctional sleep-related cognitions may reinforce and perpetuate sleep-disruptive practices, such as napping. CBT interventions aim to restructure unhelpful beliefs, and may therefore prove effective in shifting the cognitive processes underlying insomnia, thus promoting improved sleep habits and the subsequent reduction of the sleep complaint. However, the impact of such treatment upon sleep-related dysfunctional beliefs and attitudes has not been systematically investigated. The specific contribution of modifying dysfunctional sleep-related cognitions in the treatment of insomnia therefore requires clarification.

This review sought to systematically investigate the existing literature that provides evidence that dysfunctional sleep-related beliefs and attitudes endorsed by people with insomnia change in response to CBT. The relation of such changes to other key symptoms of insomnia, such as reduced sleep efficiency and prolonged wakefulness after sleep onset, will also be examined.

The following research questions are therefore addressed:

- Do cognitive-behavioural interventions for insomnia alter dysfunctional beliefs and attitudes about sleep?
- 2) Are CBT-induced changes in dysfunctional sleep-related cognitions related to improvements in other indices of clinical improvement (sleep parameters) for insomnia?

#### **METHODS**

#### Search Strategy

Articles for inclusion for this review were primarily identified via a computerised search of the following electronic databases accessed through the OVID gateway:

- Ovid MEDLINE (1996 to 2007)
- CINAHL (1982 to November Week 4 2007)
- EMBASE (1988 to 2007)

- PsychINFO (1985 to November 2007)
- All EBM reviews

#### **Search Terms**

The following terms were used in key word and title searches:

- 1. Sleep OR Insomnia
- 2. Cognitive OR Behavioural Therapy
- 3. Beliefs **OR** Attitudes
- 4. The above search terms were then combined using AND.

Duplicates were removed and the search was limited to studies written in the English language, published between 1982 and December 2007. A visual search of the bibliographies of retrieved studies and related systematic and meta-analytic reviews previously published within the field (Morin et al., 1994; Murtagh and Greenwood, 1995; Morin et al., 1999; Smith et al., 2003; Montgomery and Dennis, 2003; Irwin et al., 2006; Morin et al, 1996), was conducted, in addition to a hand search of key specialist journals, specifically *Sleep, Journal of Sleep Research, Behavioural Research and Therapy*, and *Behavioural Sleep Medicine* from 1997-2007. Key authors within the area of interest who were identified from the initial search were contacted via electronic email. Study eligibility was determined by reading the titles and, where necessary, the abstracts of identified papers using the specified inclusion and exclusion criteria.

#### **Inclusion Criteria**

Only studies that reported data from clinical trials - specifically, randomised controlled trials, controlled trials and open trials that, using standardised measures, investigated change in dysfunctional beliefs and attitudes about sleep (either as a primary or secondary variable) in response to CBT interventions for insomnia, were included. Studies evaluating the psychometric properties of self-report scales designed to assess sleep-related cognitions, such as the DBAS (Morin, 1993), reporting data for CBT-induced pre-to-post treatment cognitive change, were also included. Participants were required to be aged 18 and above; no upper age limit was imposed as it was observed during the screening and selection process that the majority of the studies available used samples comprising older samples. A diagnosis of primary or secondary insomnia was necessary. Studies involving participants with secondary insomnia was their primary complaint.

#### **Exclusion Criteria**

Cross-sectional research designs were excluded. Treatment studies that solely reported outcome with regard to observed change in sleep parameters, with no measure of cognitive change in response to treatment, were also excluded. Studies involving participants with secondary insomnia were excluded if their insomnia was secondary to or co-morbid with, progressive medical conditions known to affect sleep e.g. congestive heart failure, cancer, pulmonary disorders. Studies involving participants presenting with sleep difficulties arising from medical conditions associated with ageing or organic factors (such as the presence of a deteriorating neurological disorder), a major psychiatric disorder (e.g. major depression), or another sleep disorder (such as sleep apnea or periodic limb movements during sleep) were also excluded, as were those in which there was evidence of co-morbid substance misuse (e.g. alcohol dependence or use of alcohol as a sleep aid). Studies of circadian disorders (e.g. Delayed Sleep Phase Syndrome; Advanced Sleep Phase Syndrome) and those using other non-pharmacological therapies that are not psychological or behavioural in content (Light Therapy, Complementary and Alternative Therapies) were not included in this review.

#### **Data Extraction**

A structured checklist comprising criteria for rating the methodological quality of the literature was developed (Appendix 1.2). A total of 35 items were included in the quality rating checklist. Three of these items were specific to randomized, controlled trials only. The author and one other independent (unmasked) rater assessed the quality of selected articles across the following six domains: Objectives; Design and Methodology; Sample; Treatment Quality; Assessment and Outcome; Study Conclusions. Items were based upon checklists devised by Downs and Black, (1998) and Sindhu et al., (1997). Possible total scores ranged from 0–37 for randomized, controlled clinical trials, and 0-34 for the non-experimental trials. Where studies reported secondary analysis of data from previously conducted trials, quality ratings within the Design and Methodology, Sample, and Treatment Quality domains were achieved by reviewing the original trials from which the data had been sourced. Data extracted by each rater were then compared; any disagreements between raters were discussed, and a consensus quality score was reached. Scores were then converted into a quality category (representing the overall percentage of quality criteria satisfied by each individual study) as follows: "Excellent" (80% - 100%; "Good" (60% - 79%); "Adequate" (40% - 59%) and "Poor" (below 40%). The allocation of descriptive categorical ratings is common practice within the

systematic review literature (e.g. Dafters, 2006, unpublished manuscript).

#### RESULTS

#### Search Results

The overall selection/exclusion process is summarised in Appendix 1.3. A total of 159 articles were identified during the initial electronic database search. Study eligibility was determined by reviewing the titles and where necessary the abstracts of identified papers using the specified inclusion and exclusion criteria. When duplicates, those articles which clearly did not fit inclusion criteria from their title, review articles, book chapters and unpublished dissertations were excluded, this left 18 potential articles. Visual search of the bibliographies of retrieved studies and systematic and meta-analytic reviews previously published within the field revealed 2 further potential articles. These 20 articles were then screened according to the full inclusion and exclusion criteria, revealing a total of 14 articles for inclusion. Hand search of key specialist journals, and email correspondence with key authors within the field requesting potentially relevant articles yielded no further studies.

#### **Reliability of Quality Rating**

Agreement on each of the individual item scores between the two raters reached 96%, a high rate of agreement. 100% agreement was reached through discussion between the two raters.

#### **Characteristics of Included Studies**

Table 1 summarises the main features of the studies included in this review. Of the 14 articles,

11 employed a randomised controlled design; three of these, however, reported secondary analysis of data obtained from previous trials not originally designed to investigate dysfunctional sleep-related cognitions directly (Edinger and Wohlgemuth, 2001; Morin et al., 2002; Carney and Edinger., 2006). Two of the studies were open clinical trials (Espie et al., 2000; Harvey et al., 2007). The paper by Espie et al., (2000) also reports secondary analysis of previously obtained data. The remaining study was a controlled clinical trial (Verbeek et al., 2006). Table 1 shows the quality ratings (percentages) of each of the included studies. Three of the studies were rated as "Excellent" (Carney and Edinger, 2006; Edinger et al., 2001; Espie et al., 2000). The remaining studies were rated as "Good".

#### **Characteristics of Participants**

A full breakdown of the participants included in each study is provided in Table 1. In total, 1339 participants were enrolled in the 14 studies; 1021 of these completed treatment, thus producing an overall attrition rate of 24%. The majority of the studies used participants with a diagnosis of primary insomnia alone, with the exception of Espie et al., (2000), Harvey et al., (2007), and Jansson and Linton, (2007), who used participants with primary and secondary insomnia. Generally, there were more females enrolled in the majority of the studies. Ten of the studies had a sample with a mean age of 40-60 years; two of the studies had a mean age of >60 years; the remaining studies had a mean age of 21.2 years (Means et al., 2000) and 38 years (Wu et al., 2006). Mean duration of insomnia ranged from 7.9 months (Jansson and Linton, 2007) to 22.89 years (Harvey et al., 2007).

#### - Insert Table 1 Here -

Details of the interventions delivered across the studies included in this review are provided in Table 1. A description of the treatment components is provided in Appendix 1.4.

#### **Characteristics of Excluded Studies**

Of the 20 studies initially deemed eligible for inclusion in the review, 6 were excluded following full reading of the text. Details of these studies, and reasons for their exclusion, are cited in Appendix 1.5.

#### **Evidence of Improvement in Sleep Parameters**

Details of improvement in sleep parameters in response to CBT for each study are provided in Table 1.

#### Measurement of Dysfunctional Beliefs and Attitudes about Sleep

To address the main aims and objectives of this review, reduction in participants' scores on the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS) from pre-to-post treatment, and, where applicable, at follow-up assessment, was used to examine whether CBT alters dysfunctional sleep-related cognitions in insomnia. Other sleep-related cognitive scales are available, such as the Sleep Disturbance Questionnaire (SDQ; Espie et al., 1989). However, the DBAS was used consistently across all of the studies included in this review, and it is the scale that been subject to the most rigorous statistical appraisal.

#### Description of the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS)

The DBAS (Morin, 1993) was developed within an older adult population, and incorporates 30 items across five cognitive themes considered to be critical to chronic insomnia. These can be described as follows:

Theme 1: Maladaptive beliefs about the effects of insomnia
Theme 2: Beliefs about the unpredictability and uncontrollability of sleep
Theme 3: Unrealistic expectations about sleep needs
Theme 4: Misconceptions about the causes of insomnia
Theme 5: Erroneous beliefs about sleep-promoting strategies.

Respondents are required to rate their level of agreement/disagreement on a 100-mm visual analogue scale ranging from 0 (*strongly disagree*) to 100 (*strongly agree*). Higher total scores on the scale indicate more dysfunctional beliefs and attitudes about sleep. Several studies have shown that people with insomnia produce higher and more pathologic scores on the DBAS than normal sleepers (Morin et al., 1993; Carney and Edinger, 2006).

#### **Main Findings**

Research Question 1: Does cognitive-behavioural therapy for insomnia alter dysfunctional beliefs and attitudes about sleep?

In response to Question 1, findings are presented in the following order:

• Studies reporting the psychometric properties of the dysfunctional beliefs and attitudes

scale.

- Studies that investigate change in dysfunctional sleep-related cognitions in response to CBT as the primary research question.
- Studies that use the DBAS as a primary outcome measure, but do not investigate treatment induced cognitive change as the primary research question.
- Studies that investigate change in dysfunctional sleep-related cognitions as a secondary or process measure.

Quality criteria points awarded and overall quality criteria grading for each article are provided in brackets. Both treatment-induced reduction in overall total DBAS scores and reductions across each of the 5 themes following treatment are reported, as appropriate. Effect sizes (Cohen's d) were computed, where data permitted, using the standardised difference between two means. For example, in the study by Strom et al., (2004), the effect size was computed as the difference between the reported mean values (pre-to-post treatment reductions in DBAS total scores in response to CBT relative to the wait-list control condition) divided by the pooled standard deviation.

Studies reporting the Psychometric properties of the Dysfunctional Beliefs and Attitudes about sleep scale.

**Espie et al., (2000): (Quality Rating = 81 %; Excellent)** presented data from 60 participants drawn from a study investigating the effectiveness of CBT for chronic insomnia delivered within general medical practice. Participants completed the DBAS at baseline, post-treatment

and at 3-month follow-up. The original full-scale 30-item DBAS was found to have reasonable internal consistency (Cronbach's alpha = 0.72). Only one of the five subscales (Theme 2) demonstrated sound internal consistency, and principal components analysis did not confirm the theme structure proposed by Morin et al., (1993). A revised abbreviated version of the form (DBAS-10) was developed from the 10 items that demonstrated significant post-treatment and follow-up changes over baseline. These items were assumed to have validity, depict stable shift in beliefs and attitudes in response to treatment, and were significantly lower at post-treatment (Table 1). The DBAS-10 was found to correlate highly with the original DBAS. Overall therefore, items in the DBAS-10 demonstrated measurement sensitivity to cognitive-behavioural treatment, and may therefore be considered to provide a useful outcome measure of treatment-related cognitive change (Espie et al., 2001).

Edinger and Wohlgemuth (2001): (Quality Rating = 80 %; Excellent) replicated the work of Espie et al., (2000) and evaluated the psychometric properties of the full scale DBAS and the abbreviated DBAS-10 in a volunteer sample of 73 individuals with primary insomnia, drawn from a study comparing the efficacy of CBT against standard progressive muscle relaxation training and a quasi-desensitisation placebo control. DBAS-10 data were extracted from the full DBAS, completed by participants prior to treatment and at multiple posttreatment assessment points. The DBAS-10 was again found to correlate highly with the full DBAS scale and demonstrated respectable internal consistency. The authors analysed the sensitivity of the full DBAS and DBAS-10 for detecting decreases in dysfunctional beliefs about sleep resulting from CBT. Pre-to-post treatment change scores on the DBAS-10 successfully discriminated those who received CBT from those who received other types of behavioural insomnia treatments (Table 1) with the CBT group showing significantly greater reductions in pre-to-post treatment (6-months) scores compared with the other two groups. Trends towards similar group differences on the full DBAS were observed, but these did not reach statistical significance.

#### **Methodological Limitations**

The nature of the participant samples used within these studies should be noted when considering their contribution, both to the evaluation of the DBAS as a measure, and to the evidence that dysfunctional beliefs and attitudes about sleep are amenable to change in response to CBT for insomnia. Espie et al., (2000) used a 'real-world' sample of clinic-presenting participants. Their findings may potentially be perceived to reflect more accurately both the nature of the beliefs held by the 'typical' insomnia patient, and their responsivity to psychological intervention. Conversely, Edinger and Wohlgemuth (2001) used a highly screened sample, comprising only middle-aged and older adults. The generalisability of their findings to younger age groups may therefore be limited.

The treatment studies that are subsequently reviewed use either the original 30-item DBAS scale or the abbreviated DBAS-10. As a high correlation between total scores on the abbreviated DBAS-10 and those on the full-scale DBAS has been observed, the abbreviated version can be assumed to be a representative substitute for the original instrument. As stated, the analysis by Espie et al., (2000) did not confirm the theme structure proposed by Morin et al., (1993). Therefore, where studies have computed cognitive change for each of the five separate themes comprising the full DBAS-30, the limitations of doing so in relation to the key objectives of this review are discussed. All of the included studies calculate change in total

DBAS scores from pre-to-post treatment, and (where applicable) at follow-up assessment, to assess change in sleep-related cognitions in response to treatment.

Studies that investigate change in dysfunctional sleep-related cognitions in response to cognitive-behavioural treatment as the primary research question.

Of the 14 studies included in the review, 4 (Edinger et al., 2001; Morin et al., 2002; Carney and Edinger, 2006; Wu et al., 2006) investigated cognitive change in response to CBT as a key research question.

Edinger et al., (2001): (Overall quality rating = 82%; Excellent) examined the degree to which CBT reduces dysfunctional sleep-related cognitions using a randomised, double-blind, placebo-controlled design. Seventy-five volunteers responding to newspaper advertisements who met strict criteria for persistent primary insomnia were equally divided and assigned to CBT, progressive muscular relaxation training (RT), or a sham behavioural intervention (PC). During the course of the study, a total of 65 participants received CBT or RT either as their initially assigned treatment (CBT = 25; RT = 25) or subsequent to completing the PC condition (CBT = 9; RT = 6).

Participants completed the DBAS-30 at pre-to-post treatment and at 6-month follow-up. Only data from the 46 (24 CBT and 22 RT) of these who completed treatment and returned for the 6-month follow-up assessment were included in the follow-up analyses. Following their analysis of the psychometric properties of the DBAS in the aforementioned study (Edinger

and Wohlgemuth, 2001) items comprising the DBAS-10 were used to compare treatment groups over time. The authors hypothesised that the CBT group would show significantly larger pre-to-post treatment changes in dysfunctional beliefs about sleep than those in the RT or PC group. They also predicted that, compared with the RT group, those who had received CBT would show larger reductions (relative to pre-treatment levels) in dysfunctional beliefs about sleep at follow-up.

Overall, the results of this study suggest that CBT produced significantly larger pre-to-post treatment reductions in dysfunctional sleep-related cognitions compared to RT or PC (effect sizes are reported in Table 1). In addition, in comparison to RT, CBT produced larger reductions in dysfunctional sleep-related cognitions that endured at 6-month follow-up.

Morin et al., (2002): (Overall quality rating = 76%; Good) conducted a randomised controlled trial designed to compare changes in sleep-related cognitions obtained following CBT and pharmacological therapy for insomnia. Data collection was part of a larger study comparing the efficacy of CBT and Pharmacotherapy (PCT) for late life insomnia (Morin et al., 1999a). Seventy-two older adults recruited through newspaper advertisements who met criteria for chronic primary insomnia completed one of the following four treatments: CBT; PCT; combined CBT and PCT (COMB) or medication placebo (PLA). Completed DBAS-30 scores were available for 66 participants at baseline, 69 post-treatment, and 56, 55, and 53 participants at each of the 3-, 12-, and 24-month follow-up assessments, respectively. Missing data were not replaced nor estimated for these analyses. Analysis of DBAS scores from post-treatment to 24-month follow-up was not included because there were too many missing data.

No significant differences between groups on pre-treatment DBAS-30 total scores were found. The CBT and COMB groups had significantly lower DBAS total scores post-treatment compared to PCT and PLA. Effect sizes for each group, based on change scores from baseline to post-treatment, are noted in Table 1. Analysis of variance conducted on post-treatment, 3- and 12-month follow-up scores revealed that all three active treatment conditions endorsed fewer dysfunctional beliefs and attitudes about sleep than the PLA group, but there were no significant differences between them. The authors note that analyses of variance computed for each of the five separate DBAS-30 themes generally yielded the same results as those observed for the total DBAS-30 score and were therefore not reported.

Overall, the results of this study suggest that CBT, either alone or in combination with pharmacotherapy, reduces the dysfunctional beliefs and attitudes held by older adults presenting with chronic insomnia.

Wu et al., (2006): (Overall quality rating = 79 %; Good) conducted a randomised controlled trial comparing the effects of CBT and PCT on sleep parameters, dysfunctional beliefs and attitudes about sleep and daytime functioning. Seventy-seven patients recruited through newspaper advertisements and letters to physicians, were randomly assigned to one of four experimental conditions. Six participants dropped out prior to the mid-treatment phase, therefore the results were based upon 71 participants who received either CBT; PCT; CBT and PCT combined (COMB) or placebo (PLA). The DBAS-30 was administered at pre-and-post treatment, and at 3- and 8-month follow-up assessments. Cognitive change was assessed directly in relation to the 5 specific themes comprising the DBAS-30.

No differences were found between the four conditions for Themes 1 and 4 at pre-to-posttreatment, or at the 3-and 8-month follow-ups. Pre-to-post treatment scores for Themes 2, 3, and 5 of the DBAS were significantly lower in the CBT group compared to the PCT group. Significant differences for these themes between the four groups were found at 3- and 8-month follow-up. Effect sizes (pre-to-post treatment, 3- and 8- month follow-up) for Themes 2, 3, and 5 in response to CBT alone are noted in Table 1. There were no significant differences in dysfunctional beliefs and attitudes about sleep in relation to Themes 2, 3 and 5 for the PCT group, which suggests that the PCT intervention did not effectively change dysfunctional attitudes and beliefs about sleep.

**Carney and Edinger (2006): (Quality rating = 80%; Excellent)** present the results of a randomised parallel-group design, with participants selected from previously conducted trials investigating the use of CBT in the treatment of insomnia. The aim of the study was to identify DBAS items that changed significantly over the course of a CBT programme designed to alter insomnia sufferers' dysfunctional sleep cognitions. Participants (n = 128), largely recruited through newspaper advertisements, were included in the study. Twenty-eight participants received alternative behavioural therapies that did not specifically target sleep-related cognitions, specifically progressive muscular relaxation training or sleep hygiene; the remaining participants received CBT.

This study identified 8 items on the DBAS-30 that declined significantly more in response to CBT compared to RT and sleep hygiene. Six of these items were those previously found to be sensitive to the effects of CBT in the abbreviated 10-item version of the DBAS (Espie et al.,

2000; Edinger and Wohlgemuth, 2001). The themes to which the items found to decline belonged are noted in Table 1.

#### **Methodological Limitations**

The study by Edinger et al., (2001) was a large placebo-controlled and well-blinded, investigation, employing robust randomisation procedures. Specific treatment credibility and purity measurements were administered, indicating that participants perceived the different treatment conditions to be equally credible, and that the therapists maintained strict adherence to the treatment protocol. However, the sample size was fairly moderate, which may have reduced the power for potential differences among conditions to be detected. The sample was well-screened and more highly selected than patients typically presenting in healthcare settings; in addition, the sample was comprised of older adults, aged 40-80 years of age, thus limiting the generalisability of the reported findings. Also the high attrition rate warrants that follow-up data be interpreted cautiously.

There are a number of general methodological flaws in the study by Wu et al., (2006). Specifically, it is not possible to ascertain whether the method of randomisation used in the allocation of participants to the various experimental conditions was robust (e.g. use of computer-generated random number table), as the authors do not explicitly report the procedure applied. Furthermore, no measure of treatment credibility was administered.

Wu et al., (2006) evaluated cognitive change directly in relation to the 5 specific themes comprising the DBAS. However, statistical support for the theme structure proposed by Morin

et al., (1993) is limited (Espie et al., 2000). Carney and Edinger (2006) adopted a similar method, assessing change in dysfunctional sleep-related cognitions in association with each of the 30 individual DBAS items. However, examining changes in individual DBAS items over time may be limited by the potential for measurement error due to the inherent unreliability of single items.

Morin et al., (2002) took no measure of treatment credibility or purity. Although the articles by Morin et al., (2002) and Carney and Edinger (2006) reported that they address change in dysfunctional sleep-related cognitions in response to CBT as a primary research question, they do so using secondary analysis of data drawn from previously conducted trials designed to measure change in sleep parameters as the key outcome measure.

Studies that use the DBAS as a primary outcome measure, but do not investigate treatment induced cognitive change as the primary research question.

Four studies are relevant to this section (Means et al., 2000; Lichstein et al., 2001; Verbeek et al., 2006; Jansson and Linton, 2007)

Means et al., (2000): (Quality Rating = 72%; Good) used a randomised controlled design to investigate differences in daytime functioning following relaxation therapy. A total of 139 students were recruited from university undergraduate psychology courses. Twenty-one participants either voluntarily dropped out of the study, or were excluded from the analysis, leaving a sample of 118 students (57 with insomnia – SWI; 61 not complaining of insomnia –

SNI). Twenty-eight SWI were randomly assigned to receive progressive relaxation therapy (RT). The DBAS–30 was used, not as a measure of cognitive change in response to treatment per se, but as a measure of daytime functioning. At baseline, SWI demonstrated higher levels of impairment on DBAS-30 measures compared to SNI. Treated SWI did not significantly improve on the DBAS-30 at post-treatment. All students, irrespective of group allocation, demonstrated improved scores on the DBAS at post-treatment.

Lichstein et al., (2001): (Quality Rating = 76 %; Good) conducted a study similar to that by Means et al., (2000), using a randomised, placebo-controlled trial investigating the impact of relaxation and sleep restriction for late-life insomnia. A highly screened sample of 85 older adults with primary insomnia recruited via public service announcements were assigned to relaxation, sleep restriction or placebo desensitisation. Following attrition, a total of 74 participants completed the study. Drop-outs did not differ on clinical or demographic data. The DBAS-30 was completed at baseline, post-treatment and 1-year follow-up. Similar to Means et al., (2000) this study used the DBAS-30 as a measure of daytime functioning. Results showed that DBAS scores changed significantly across time only in the relaxation group. Participants in the relaxation group had significantly lower DBAS-30 scores at followup than at baseline, with a moderate effect size (Table 1) with no other significant comparisons across time. No significant between groups differences on DBAS-30 scores were observed.

Verbeek et al., (2006): (Quality Rating = 74 %; Good) compared the short-and-long term effects of group versus individual CBT. Thirty-two participants with chronic primary insomnia were individually treated. Seventy-four participants with either primary or secondary insomnia

were divided over 12 groups. All participants had been referred to a specialised sleep centre by a physician; those with organic sleep disorders or co-morbid psychopathology were excluded. The primary outcome measures were subjective sleep, quality of life, and psychological wellbeing (as represented by change in dysfunctional sleep-related cognitions). Participants completed the DBAS-30 at baseline, pre-treatment (after a waiting period of 1 to 3 months), post-treatment, and at follow-up (1-, 3- and 6-months). Analyses were only made for the subgroup of participants for whom data at all four assessment points were available (n = 18 individual treatment; n = 40 group treatment).

Dysfunctional beliefs and attitudes about sleep were found to significantly improve in response to treatment, with no significant differences in reduction of DBAS-30 scores between the two conditions. This improvement remained significant at follow-up. Observed effect sizes (baseline to post-treatment; baseline to follow-up) for group and individual treatments are reported in Table 1. Overall, the results indicate that CBT for insomnia effectively improves sleep-related cognitions when delivered both on an individual basis and in a group format.

Jansson and Linton (2007): (Quality rating score = 77 %; Good) conducted a randomised controlled design with assessments at pre-treatment and at 1-year follow-up to compare the effects of cognitive behaviour group therapy and a self-help package for insomnia. Participants (n = 156) were recruited via newspaper advertisements and were randomly assigned to one of the two treatment conditions; 136 participants completed the 1-year follow-up. The primary outcome variables were reduction in dysfunctional beliefs and attitudes about sleep (as measured by the DBAS-10), negative daytime symptoms and sleep parameters. Compared to the self-help control group, the CBT group reported a significantly larger

reduction in dysfunctional beliefs and attitudes about sleep compared to the self-help group (percentage reductions and effect sizes are reported in Table 1). The results of this study demonstrate reduction in dysfunctional beliefs and attitudes about sleep in response to CBT.

#### **Methodological Limitations**

As treated participants received only 3 sessions of RT, the failure to find a significant effect of RT on DBAS-30 scores in the study by Means et al., (2000) might be due to the relatively short duration of the treatment. A general weakness of the Lichstein et al., (2001) study is that all participants received sleep hygiene instructions, including those in the placebo group. Sleep hygiene has been found to have a moderate therapeutic effect (Morin et al., 1994) and therefore cannot be regarded as an innocuous intervention. Both the studies by Means et al., (2000) and Lichstein et al., (2001) use highly selected, and in terms of age, potentially unrepresentative samples. However, they both assess treatment purity, whereas the other studies reviewed within this section do not.

The study by Verbeek et al., (2006) is limited by the fact that there was no randomisation of participants to individual or group treatment and that the criteria for allocation to group or individual treatment differed (i.e. primary versus secondary insomnia). Although there was a waiting-list control, as with the Lichstein et al. (2001) study, these individuals received sleep hygiene advice, and therefore do not represent a pure control group.

The internal validity of the study by Jansson and Linton (2007) may be jeopardised by the high attrition rates. Although no intention to treat analysis was performed, drop-outs did not significantly differ on demographic or clinical data. As the aim of this study was to examine

the effects of early intervention, the mean duration of insomnia was 7.9 months, which in comparison with the other studies included in this review, is relatively short (see Table 1).

Studies that assess change in dysfunctional sleep-related cognitions as a secondary or process measure.

Four of the included studies (Mimeault and Morin, 1999; Bastien et al., 2004; Strom et al., 2004; Harvey et al., 2007) assessed treatment-induced cognitive change as a secondary or process variable.

**Mimeault and Morin (1999): (Quality Rating = 76%; Good)** evaluated the efficacy of selfhelp as a brief, accessible treatment for insomnia. Fifty-eight adults with primary insomnia recruited through media advertisements were randomly assigned to self-help treatment (cognitive-behavioural bibliotherapy; BT), BT with weekly phone consultations (BTPC), or a waiting-list control (WL). Four participants in the BT condition dropped out during the course of treatment, leaving a total sample of 54; dropouts did not differ from the final sample on clinical or demographic data, and were excluded from the data analyses because they did not complete at least 50% of the treatment. Participants completed the French version of the DBAS-30 (BAS; Blais et al., 1997), which has been found to have good internal consistency and adequate test-retest reliability, at pre-and-post treatment, and again at 3-month follow-up. The authors report scores on the DBAS over time as an 'ancillary' measure', with changes in sleep parameters representing the primary outcome variable. Improvements on the BAS were observed post-treatment within both the BT and BTPC groups, with effect sizes reported in Table 1. BT and BTPC were comparable at follow-up and remained more improved than controls.

**Bastien et al., (2004): (Quality Rating = 77 %; Good)** investigated whether CBT for insomnia produced different outcomes when delivered using different formats. Forty-five adults with primary insomnia recruited through newspaper advertisements were randomly assigned to one of three CBT-treatment conditions (individual, group or telephone consultation). The previously mentioned French version of the DBAS-30 (BAS; Blais et al., 1997) was administered at pre-and-post treatment, and again at 3-and 6-month follow-up. Change in BAS scores in response to treatment was reported specifically as an ancillary measure. Dysfunctional beliefs and attitudes were found to improve over time. No statistical differences in BAS scores between the different treatment conditions were observed across the assessment phases. Effect sizes are reported in Table 1. These findings suggest that CBT is effective in reducing sleep-related cognitions using all 3 methods of treatment delivery.

**Strom et al., (2004): (Quality rating = 70 %; Good)** investigated the effects of an internetbased intervention for insomnia. Participants (n = 109) were recruited from newspaper and internet advertisements and were randomly assigned to either cognitive-behavioural self-help treatment or a waiting list control condition. Twenty-eight participants dropped out, leaving a total sample of 81 participants. Drop-outs were found to differ on pre-treatment measures of sleep efficiency, total amount of sleep, and wake time during the night, suggesting that their sleep problems were less pronounced. Data from the DBAS-30 were collected at pre-and posttreatment, and are reported by the authors as an ancillary measure. The CBT group showed significantly greater pre-to-post treatment reductions in dysfunctional sleep-related cognitions compared to the control group, with an effect size of 0.81.

Harvey et al., (2007): (Quality rating = 79 %; Good) report findings from an open trial of a recently developed cognitive therapy intervention for chronic insomnia designed to target the processes suggested to maintain insomnia, including "unhelpful beliefs about sleep". A sample of 19 people meeting diagnostic criteria for primary or secondary insomnia recruited from local psychiatrists, general practitioners, psychologists, and newspaper advertisements, completed the study. Scores on the DBAS-30 across time (pre-treatment, post-treatment, 3-, 6, and 12-month follow-up) were categorised as a 'process measure' rather than a specific outcome measure per se. A significant reduction in dysfunctional beliefs and attitudes about sleep from pre-to-post treatment was observed, with an effect size of d = 2.46. This reduction was maintained at 12-month follow-up.

#### **Methodological Limitations**

General methodological caveats to the studies by Mimeault and Morin (1999) and Bastien et al., (2004) must be considered. Within these studies, the sample sizes were relatively small, highly selected, and no specific measures of treatment credibility were taken. In the absence of a no-treatment control group in the study by Bastien et al., (2004), it is impossible to rule out regression to the mean as an explanation for improvements over time. Although DBAS score data are reported in the results section of the paper, Mimeault and Morin (1999) provide no clear rationale for the administration of this measure. The authors make no reference to the DBAS results in their discussion. Delivery of a treatment intervention via the internet as done by Strom et al., (2004) inherently reduces the methodological rigour of the study. The extent to which the sample is representative is uncertain, as is the validity and reliability of assessing/diagnosing insomnia via the internet, although research in other fields shows that the psychometric properties for self-report scales delivered via the internet are comparable with those delivered using the traditional paper-and-pencil manner (Buchanan, 2002). In addition, the duration of insomnia necessary for inclusion in the study was established at 3 months, whereas 6 months is the norm in other studies. However, only 2 participants had insomnia for less than 6 months. The study is limited by the lack of follow-up assessment. Although the authors note the large effect size for reduced dysfunctional beliefs and attitudes about sleep specifically, they have not discussed this finding, or its potential clinical and research implications.

The absence of a control group in the study by Harvey et al., (2007) means that the improvements observed may be due merely to regression to the mean. The sample is also relatively small, thus limiting the generalisability of the findings. The cognitive therapy protocol proposed in this study may therefore warrant evaluation using a controlled research design.

# Question 2: Are CBT-induced changes in dysfunctional beliefs and attitudes about sleep related to other indices of clinical improvement (sleep parameters) for insomnia?

Three of the previously reviewed studies (Edinger et al., 2001; Morin et al., 2002; Carney and Edinger, 2006) analysed the possible association between treatment-induced change in sleep-related cognitions and improvements in sleep parameters.
Edinger et al., (2001) hypothesised that treatment-related changes in dysfunctional sleeprelated cognitions as measured by the DBAS-10 would be significantly correlated with treatment-related improvements on polysomnography (PSG), sleep diaries, and a global insomnia questionnaire designed to assess the presence or absence of nocturnal and diurnal insomnia symptoms (Insomnia Symptom Questionnaire; ISQ, Spielman et al., 1987). To test this hypothesis, the authors conducted two sets of correlational analyses as outlined below.

# <u>Pre-to-post treatment changes in total DBAS-10 score, ISQ, PSG parameters and Sleep</u> <u>Diary measures</u>

Correlations were computed for all 3 groups (CBT, RT or behavioural placebo control) and for each group separately. Correlation coefficients are reported in Table 1. Across the entire study sample, pre-to-post treatment reductions in DBAS-10 total scores were significantly correlated with pre-to-post treatment reductions in PSG values of wake time after sleep onset (WASO), global insomnia symptoms as measured by the ISQ, and increases in PSG values of sleep efficiency (SE). When the correlational analyses were conducted for each group separately, all three of these correlations were found to be significant only within the CBT group. Thus, short-term reductions in dysfunctional beliefs about sleep were most consistently related to short-term improvements in other outcome measures among those who received CBT.

#### Pre-to-follow-up changes in total DBAS-10 total score, ISQ and Sleep Log measures

Correlations were computed for all CBT and RT treated participants who completed the follow-up assessment and were conducted for both the CBT and RT groups combined and in isolation. Improvements in DBAS-10 scores were only related to improvements in participants' subjective sleep diary ratings of 'restedness' and sleep quality within the CBT

group. In line with the findings pertaining to short-term changes, reductions in dysfunctional beliefs about sleep were most consistently related to improvement in other outcome measures among those who received CBT. However, although significant, the level of association was modest (Table 1).

Morin et al., (2002) correlated pre-to-post-treatment change scores on the DBAS-30 with change scores on SE as measured by daily sleep diaries and PSG. These analyses were computed for all participants in the four conditions (CBT; PCT; COMB; PLA). Reduced DBAS scores were found to significantly correlate with improved SE as measured by daily sleep diaries. However, the level of association was generally low (Table 1). The correlation between DBAS-30 scores and sleep efficiency as measured by PSG was almost significant.

Correlations were also computed between change scores for the 5 separate themes comprising the DBAS-30 and change scores on sleep efficiency from the pre-to-post treatment period. Improvements for Themes 1 and 3 were correlated with improved SE as measured by daily sleep diaries alone; improvements for Theme 2 were correlated with improved SE as measured by both daily sleep diaries and PSG; improvements for Theme 4 were correlated with improved SE as measured by PSG alone. Changes for Theme 5 were not significantly correlated with SE.

Post-treatment DBAS-30 changes were significantly correlated with SE as measured by daily sleep diaries at each of the 3 follow-up assessment points. However, the level of association observed varied from low to modest (Table 1). Analyses for each separate theme of the DBAS indicated that post-treatment scores for Themes 1, 2, 4 and 5 were significantly correlated with

SE at 12- and 24-month follow-up assessments; post-treatment scores for theme 3 were significantly correlated with SE at 24-month follow-up. Therefore, these results indicate that more adaptive beliefs and attitudes about sleep were associated with better maintenance of sleep improvements at follow-up.

**Carney and Edinger (2006)** examined the relationship between pre-to-post treatment change scores on each of the DBAS-30 items and WASO obtained from daily sleep diaries, in addition to change scores for the ISQ. Intention-to-treat analysis (last observation carried forward) was used when follow-up (3 month) data were missing. The sample was divided into "improved" and "unimproved" groups on the basis of the degree of change participants demonstrated on the selected outcome measures over the course of the study (the 50% showing greater decline in ISQ scores and greater reduction in WASO were categorised as "improved").

Analysis of variance revealed that ISQ "improved" participants had significantly greater preto-post treatment reductions on 13 of the 30 DBAS items than did those defined as "unimproved" on the ISQ. All 5 subscales of the DBAS were represented among these 13 items. WASO-improved patients had significantly greater DBAS change scores than those defined as unimproved on the WASO for three items; one item related to Theme 1, and the remaining two items related to Theme 2. Between improved versus unimproved participants, only 2 items from the DBAS were found to be significant across both variables; these pertained to Themes 1 and 2. Five items, pertaining to Themes 1, 3 and 5, were found to be non-significant across the three variables. Overall, reduced scores on 15 of the 30 DBAS items in response to CBT were related to improvements in sleep variables.

## **Methodological Limitations**

As stated previously, the high attrition rate in the study by Edinger et al., (2001) warrants that follow-up data be interpreted cautiously. A limitation of the study by Morin et al., (2002) is that the correlational analyses were conducted for all participants across all four treatment conditions combined. The failure to compute further correlational analyses for each of the treatment conditions separately as done in the studies by Edinger et al., (2001) and Carney and Edinger (2006) means that it is not possible to specify differential effects between the treatment conditions. The data presented by Morin et al., (2002) therefore tell us only that change in dysfunctional sleep cognitions are associated with other indices of clinical improvement at a global level, but do not reveal for which of the treatments this association was most consistent.

In addition to analysing change in total DBAS scores across time, Morin et al., (2002) evaluate the association between improvements in sleep parameters and reduced scores for each of the five DBAS themes. Similarly, Carney and Edinger (2006) evaluated the association between improved sleep and each of the 30 individual DBAS items. As stated previously, statistical support for the theme structure is limited (Espie et al., 2000), and analysing change in response to specific items may be compromised by potential measurement error resulting from the unreliability of single items. Unlike the other two studies, Carney and Edinger (2006) take no objective measures of sleep improvement (such as PSG), and as the authors highlight, the reliance upon 'paper-and-pencil' self report measures may exaggerate the relationship between cognitive change and sleep improvement following treatment for insomnia.

# DISCUSSION

Dysfunctional sleep-related cognitions have become increasingly recognised as potential contributory and maintaining factors in insomnia. CBT, an intervention developed specifically to restructure the unhelpful cognitions that underlie psychological conditions, has proven efficacy and effectiveness in the management of insomnia. This review aimed to systematically evaluate the current evidence pertaining to the impact of CBT on dysfunctional beliefs and attitudes about sleep in the treatment of insomnia. Specifically, two key questions were addressed. *1) Does CBT for insomnia alter dysfunctional beliefs and attitudes about sleep? 2) Are CBT-induced changes in dysfunctional sleep-related cognitions related to improvements in other indices of clinical improvement (sleep parameters) for insomnia?* The evidence relating to each question is discussed separately. The limitations of the evidence base are described. Finally, the clinical implications of the presented findings and recommendations for future research are outlined.

# 1) Does CBT for insomnia alter dysfunctional beliefs and attitudes about sleep?

Of the 14 studies included in this review, 4 were assigned an overall quality rating of "excellent". The remaining studies were all rated as "good". Thirteen studies can be considered to provide evidence of significant change in dysfunctional sleep-related beliefs in response to cognitive-behavioural interventions, as indicated by reduced scores on the Dysfunctional Beliefs and Attitudes about Sleep Scale, both the original 30-item scale (DBAS-30; Morin, 1993) and the abbreviated 10-item scale (DBAS-10; Espie et al., 2000).

Only one study (Means et al., 2001) failed to yield positive findings for the therapeutic impact of cognitive behavioural interventions upon dysfunctional sleep-related cognitions.

Of the 13 studies that provide evidence that insomnia sufferers' dysfunctional sleep-related cognitions decline over the course of CBT, two investigate the psychometric properties of the DBAS (Espie et al., 2000; Edinger and Wohlgemuth, 2001). Four studies investigated change in dysfunctional sleep-related cognitions in response to CBT as the primary research question (Edinger et al., 2001; Morin et al., 2002; Wu et al., 2006; Carney and Edinger, 2006). However, two of these studies (Morin et al, 2002; Carney and Edinger, 2006) report secondary analysis of previously obtained data gathered from trials not originally designed to address this question specifically. Of the remaining 7 studies, 3 use cognitive change as a primary outcome measure, but do not investigate treatment-induced cognitive change as the primary research question (Lichstein et al., 2001; Verbeek et al., 2006; Jansson and Linton, 2007). The remaining 4 studies investigate change in dysfunctional sleep-related cognitions as a secondary or process measure.

Both studies reporting the psychometric properties of the DBAS scale were assigned an overall quality rating of "Excellent", and suggest that CBT produces significant pre-to-post treatment reductions in dysfunctional sleep-related cognitions. Of the four treatment studies that investigated change in dysfunctional sleep-related cognitions in response to CBT as the primary research question, two were assigned an overall quality rating of "Excellent" (Edinger et al., 2001; Carney and Edinger, 2006). All four studies demonstrate that CBT is effective in inducing pre-to-post treatment cognitive change compared with alternative behavioural therapies (for example, relaxation and sleep hygiene education) and both sham psychological

and medication placebo; these gains are sustained through to follow-up assessments, ranging from 6 to 12 months.

Morin et al., (2002) demonstrate that CBT, either alone or in combination with pharmacotherapy, effectively reduces dysfunctional sleep-related cognitions. These cognitive changes may be attributed to the specific effects of CBT, as no such improvements were obtained among those individuals who received drug therapy in isolation. The fact that participants in all three active treatments, including those receiving PCT alone, endorsed fewer dysfunctional sleep cognitions at the follow-up assessments compared with the placebo group, suggest that changes in beliefs and attitudes achieved at post treatment with CBT, either alone or in combination with medication, are maintained over time. Although the results also suggest that PCT alone improved sleep cognitions, participants within this condition were found to have consistently higher DBAS-30 scores at each follow-up assessment compared to the two CBT treatments. Furthermore, no significant difference in change in DBAS-30 scores over time was observed within the PCT condition alone. CBT was also found to be more effective in improving sleep variables than PCT. Wu et al., (2006) report similar findings which demonstrate that, compared to pharmacotherapy, CBT produced significantly greater reductions in DBAS scores (for particular themes) pre-to-post treatment. Compared to the CBT group, the PCT group reverted back to their pre-treatment condition, reporting somatic and cognitive symptoms associated with chronic insomnia. The absence of substantial change in sleep-related psychological activity in response to PCT may indicate that the long-term therapeutic effects of pharmacological interventions for insomnia are limited. Overall these

findings may add credence to the theory that dysfunctional sleep cognitions underlie and maintain sleep disruption.

Of the four studies that used the DBAS as a primary outcome measure, but were not designed to investigate the impact of CBT on sleep-cognitions as a primary research question, only three provide evidence of change in sleep cognitions in response to treatment. In the study by Means et al., (2000), all participants, with and without insomnia, and regardless of whether they had received relaxation therapy, showed reduced levels of dysfunctional sleep cognitions at post-treatment. However, despite demonstrating more dysfunctional sleep cognitions at baseline entry to therapy, those with insomnia who received treatment did not significantly improve at post-treatment. As stated previously, the duration of treatment falls short of the recommended number of relaxation sessions (5 to 12), and the degree of improvement in response to relaxation has been linked to the number of sessions delivered. For example, Espie et al., (1989) reported improved sleep and daytime functioning in insomnia following 8 weeks of RT intervention. However, it could also be argued that the lack of significant change in dysfunctional sleep-related cognitions following relaxation is not surprising given that this is essentially a behavioural intervention, and therefore not specifically aimed at targeting an individual's beliefs about sleep. It has been suggested that behavioural interventions for insomnia such as relaxation might ameliorate not only physiological arousal but cognitive arousal as well, hence the inclusion of such studies in this review. However, the nonsignificant effect observed in the study by Means et al., (2000) may demonstrate the need for psychological interventions that directly target sleep-related cognitions for any change in these to be achieved. Although Lichstein et al., (2001) conducted a similar study and found significant reductions in dysfunctional sleep-cognitions in response to relaxation, no

differences were observed in comparison with the placebo control. Furthermore, the magnitude of the effect was relatively modest in comparison to that observed in the majority of the other studies. Lichstein et al., (2001) also comment on the association between the number of sessions and the impact of relaxation, and proposed to increase the number of sessions from 6 to 8 in subsequent studies. However, the lack of a significant group difference and the comparatively small effect size observed may also highlight the need for interventions directly targeting cognitions, particularly when considered in light of the findings of Edinger et al (2001) and Carney and Edinger (2006), who found that CBT, comprising of a specific cognitive element, produced significantly greater improvement on dysfunctional beliefs and attitudes about sleep than relaxation.

Studies that assess change in dysfunctional sleep-related cognitions as a secondary or process measure also provide evidence for cognitive change in response to CBT. The findings of Bastien et al., (2004) and Mimeault and Morin (1999) highlight that CBT is effective in reducing dysfunctional sleep-related cognitions regardless of the mode of treatment delivery (e.g. group versus individual) used. Although Harvey et al., (2007) assess cognitive change as a process measure rather than a key outcome variable per se, this study expands upon the extant literature in the sense that it is the first to fully evaluate a treatment intervention designed specifically to target the cognitive processes, including dysfunctional sleep-related cognitions, presumed to underlie the development and maintenance of insomnia. The findings by Strom et al., (2004) are also interesting. Overall, between-groups effect sizes for the primary sleep variables were low. Although results showed statistically significant improvement in primary outcome measures, improvements upon these were observed in the control group also. However, although taken as a secondary measure, greater pre-to-post treatment reduction in dysfunctional sleep-related cognitions was observed for CBT relative to controls, with a large effect size. This finding highlights the potential importance of using such change as a primary outcome measure. However, it should be noted that this study was assigned the lowest overall quality rating score (70%) across all of the studies reviewed, therefore this result should perhaps be interpreted cautiously.

# 2. Are CBT-induced changes in dysfunctional sleep-related cognitions related to improvements in other indices of clinical improvement (sleep parameters) for insomnia?

The evidence for an association between cognitive change and improvements in sleep is scarce as only three of the included studies (Edinger et al., 2001; Morin et al., 2002; Carney and Edinger, 2006) have specifically analysed this. Overall, the findings from these studies suggest that not only is CBT particularly effective in reducing dysfunctional beliefs about sleep, reductions in such beliefs are significantly associated with other clinical indices of positive insomnia treatment outcome, particularly sleep efficiency and wake after sleep onset. Improvements on these variables have been observed both pre-to-post treatment and at followup, and via objective PSG and subjective sleep diary and questionnaire data. However, as with all correlational analyses, results do not necessarily prove causation and, these associations, although significant, were generally modest. The approach by Morin et al., (2002) and Carney and Edinger (2002) in assessing cognitive change and its association with improvements in sleep in direct relation to the specific themes and items on the DBAS is limited by the psychometric properties of the scale. As stated, the thematic structure proposed by Morin (1993) is not confirmed statistically (Espie et al., 2000), and analysis of single items introduces measurement error due to the inherent unreliability of single items. Nevertheless, this method of analysis may be considered useful on a descriptive level, and may help identify potentially important targets for maximising sleep outcomes.

### Limitations of the Evidence Base

When considering the evidence presented herein, there are a number of methodological caveats that must be considered. Generally, when rating the overall quality of the studies, many failed to satisfy those criteria likely to influence the overall internal and external validity of the studies. For example, the majority of the participants were well-screened and highly selected research volunteers recruited via advertisement, therefore reducing the extent to which the positive findings can be generalised to the clinical pole. Only three studies (Espie et al., 2000; Verbeek et al., 2006; Harvey et al., 2007) use a clinically referred sample, thus providing evidence (with greater generalisability) for the clinical *effectiveness* of CBT both generally, and in terms of reducing dysfunctional sleep-related cognitions in insomnia. Typically, with the exception of the studies by Means et al., (2000) and Wu et al., (2006) the samples largely comprised older adults, aged 40-80 years of age, thus limiting the generalisability of the findings to younger age groups.

The sample size of many of the studies was small to moderate (e.g. Harvey et al., 2007; Bastien et al., 2004; Edinger et al., 2001), and a high attrition rate was observed. Although drop-outs did not generally differ from those completing treatment, few studies replaced or estimated these data. Small sample sizes may have reduced the power for potential differences among conditions to be detected, and where studies have a high attrition rate, follow-up data should be interpreted cautiously.

Many of the studies are weakened by the absence of a non-treatment control group (e.g. Bastien et al., 2004; Carney and Edinger, 2006; Harvey et al., 2006) or have used a control group that has been corrupted by the introduction of an active therapeutic component, specifically, sleep hygiene (Lichstein et al., 2001; Verbeek et al., 2006). This dictates that the possibility that the improvements observed were due merely to regression to the mean, or as a result of non-specific therapy effects, such as participants expectations of improvement, rather than the direct therapeutic effects of the treatment, cannot be ruled out.

The majority of the studies reported herein stem from the same research groups. For example, three of the studies have been conducted by Edinger and co-workers, and two have come from the Morin research group. Although there is no overlap in the participants included in this review, difficulties with this include recruiting from the same geographic population, and using the same treatment protocols and models. The possibility that different findings would have been obtained with different versions of the treatment cannot be ruled out. This is important as it again reduces the external validity of the findings, as, inevitably, in clinical practice, treatment protocols will differ across different sites. Furthermore, two of the studies that investigate change in dysfunctional sleep-related cognitions in response to CBT as the primary research question rely upon secondary analysis of data obtained from previous trials that were not designed to investigate this question specifically. This highlights the need for independent research trials that are specifically designed to investigate the impact of CBT on dysfunctional sleep-related cognitions, and their association with improvements in sleep parameters.

Although the findings presented here suggest that CBT positively impacts upon dysfunctional beliefs and attitudes about sleep, the evidence is limited by the fact that only one instrument has been used to assess this. The utility of the DBAS to measure treatment induced cognitive change in insomnia may be questioned. Prior to the study by Espie et al (2000) the component structure, reliability and validity of the DBAS scale had not been formally evaluated. Although total DBAS-30 and DBAS-10 scores correlate highly, Espie et al., (2000) identified that only 10 of the items from the full 30-item scale were valid and sensitive to change over time. It is also possible that other beliefs that are critical to insomnia are absent from the existing DBAS scales. Particularly, there are only a few items that assess beliefs relating to unrealistic expectations about sleep needs and misconceptions regarding the causes of insomnia (Themes 3 and 4).

The original DBAS scale was prepared for publication in the English language (although developed within a French-Canadian population). However, the majority of the studies reviewed here use samples of French-, Dutch-, Swedish-, and Chinese- speaking nationals. In studies using French speaking participants (e.g. Bastien et al., 2004) the French version of the DBAS (the BAS; Blais, 1997) has been used. This measure has been reported to have good internal consistency (Cronbach's alpha = 0.90) and adequate test-retest reliability (r = 0.72). However, other studies have used translated versions of the scale, which have not been validated. The authors of these studies do not address or highlight this in their reports, but this factor should be considered when interpreting the results of these studies in relation to the aims and objectives of this review.

# CONCLUSIONS

From the 14 studies included in this review, only one failed to provide evidence of the positive impact of CBT in reducing dysfunctional beliefs and attitudes about sleep in insomnia. Reductions in dysfunctional sleep-related cognitions were related to improvements in sleep parameters, such as sleep efficiency and wake time after sleep onset. The significant correlations obtained from the studies analysing the association between the magnitude of change on sleep-related cognitions and the degree of sleep improvements at post-treatment may be assumed to suggest that cognitive changes that occur during the course of treatment are crucial for the long-term maintenance of therapeutic gains. However, few studies specifically investigate this and the associations observed are generally modest.

Overall, the findings suggest that treatment that directly targets cognitions is essential, as CBT was consistently observed to be better than alternative behavioural strategies such as relaxation in both reducing dysfunctional sleep-related cognitions and inducing improvements in sleep parameters over the course of treatment. Furthermore, these changes were maintained at follow-up assessment. Similar benefits of CBT over pharmacotherapy have also been observed. The comparative benefits of CBT in improving sleep parameters also indicates that the observed reductions in dysfunctional beliefs and attitudes about sleep reported by the majority of the studies are indeed produced by CBT per se, and are not merely a by-product of sleep improvements, regardless of the type of treatment that produced those changes. Overall, the findings may suggest that the inclusion of cognitive restructuring strategies may be essential to alter sleep-related cognitions, and it may be possible that similar changes would not have been observed with behavioural or pharmacological alternatives.

However, a number of methodological caveats relating to the internal and external validity of the findings, and to the use of the DBAS scale in the measurement of cognitive change in response to treatment, have been identified, and must be considered when making conclusions regarding the impact of CBT upon dysfunctional sleep-related cognitions in insomnia, and their association with improvements on an individual's overall sleep condition. Nevertheless, the evidence presented herein may be considered to provide further evidence to implicate dysfunctional beliefs and attitudes about sleep in the development and maintenance of insomnia, and highlight the potential therapeutic benefit of using CBT strategies to specifically target such cognitions in its management.

## **RECOMMENDATIONS FOR FUTURE RESEARCH**

Further studies addressing the methodological limitations highlighted by this review, particularly those using clinical samples and conducted by other, independent research groups are required. Furthermore, although the DBAS appears, at the present time, to be the best existing tool for the systematic assessment of dysfunctional sleep-related cognitions, further work is required to expand and refine this instrument, and the development of further cognitive scales is warranted. Refinement of the DBAS may not only improve measurement sensitivity for the detection of cognitive change in response to CBT over time, it may also provide greater insight into the specific beliefs that are most critical to the development and maintenance of insomnia, and therefore benefit from targeted intervention.

Few studies specifically investigate the association between cognitive change and improvements in sleep, albeit with positive results. However, future studies evaluating CBT

for insomnia should examine this issue further in order to clarify the role of dysfunctional beliefs and attitudes in the development and maintenance of insomnia, and determine whether changes in such cognitions are essential to effectively treat insomnia. Morin (2003) recommended that outcome assessment in insomnia should be extended beyond the reduction of insomnia symptoms as represented by sleep parameters. Similarly, Verbeek et al., (2006) imply that for treatment to be wholly effective it should also produce meaningful change in other domains. Although there are methodological issues that require further attention, it is hoped that this review will alert both practitioners and researchers within the insomnia field to the potential benefits, both theoretically and pragmatically, of using interventions that directly target dysfunctional beliefs and attitudes about sleep in the successful treatment of insomnia.

# References

\*Included Studies; \*\*Excluded Studies

\*\*Arnedt, J.T., Conroy, D., Rutt, J., Aloia, M.S., Brower, K.J. and Armitage, R. An open trial of cognitive behavioural treatment for insomnia co-morbid with alcohol dependence. *Sleep Med*, 2007, 8: 176-180.

\*Bastien, C.H., Morin, C.M., Quellet, M.C., Blais, F.C. and Bouchard, S. Cognitivebehavioural therapy for insomnia: comparison of individual therapy, group therapy and telephone consultations. *J. Consult. Clin. Psychol*, 2004, 72: 653-59.

Blais, F.C., Gendron, L., Mimeault, V. and Morin, C.M. Evaluation de l'insomnie: Validation de trois questionnaires [Evaluation of insomnia: Validation of three questionnaires]. *L'Encephale*, 1997, XIII, 447-453.

Buchanan, T. Online assessment: Desirable or dangerous? *Prof Psychol: Res and Practice*, 2002, 33: 148-154.

\*Carney, C.E. and Edinger, J.D. Identifying critical beliefs about sleep in primary insomnia. *Sleep*, 2006, 29(3): 342-350.

\*\*Carney, C.E., Edinger, J.D., Manber, R., Garson, C. and Segal, Z.V. Beliefs about sleep in disorders characterized by sleep and mood disturbance. *J. Psychosom. Res*, 2007,62:179-188

Cole, M.G. and Dendukuri, N. (2003). Risk factors for depression among elderly community subjects: A systematic review and meta-analysis. *American J. of Psychiatry*, 2003, 160: 1147-1156.

Downs, S.H. and Black, N. The feasibility of creating a checklist for the assessment of the methodological quality of both randomised and non-randomised studies of health care interventions. *J. Epidemiol. Community Health*, 1998, 52: 377-384.

\*Edinger, J.D., Wohlgemuth, W.K., Radtke, R.A., Marsh, G. and Quillian, R. Does cognitivebehavioural insomnia therapy alter dysfunctional beliefs about sleep? *Sleep*, 2001, 24(5): 591-599.

\*\*Edinger, J.D. and Wohlgemuth, W.K. A primary care "friendly" cognitive behavioural insomnia therapy. *Sleep*, 2003, 26: 177-12.

Edinger, J.D., Bonnet, M.H., Bootzin, R.R., Doghramji, K., Dorsey, C.M. and Espie, C.A. Derivation of research diagnostic criteria for insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep*, 2004, 27: 1567–96.

\*\*Ellis, J., Hampson, S.E. and Cropley, M. The role of dysfunctional beliefs and attitudes in late-life insomnia. *Psychosom. Res*, 2007, 62: 81-4.

Espie, C.A., Brooks, D.N., and Lindsay, W.R. An evaluation of tailored psychological treatment of insomnia. J. Behav. Ther. Exper. Psychiatry, 1989, 20: 143-155.

Espie, C.A. Cognitive behaviour therapy as the treatment of choice for the treatment of insomnia. Wiley, Chichester, England, 1999.

\* Espie, C.A., Inglis, S.J., Harvey, L., and Tessier, S. Insomniacs' attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep scale and the Sleep Disturbance Questionnaire. *J. Psychosom. Res*, 2000, 48: 141-148.

Espie, C.A. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Ann. Rev. Psychol*, 2002, 53: 215–43.

Harvey A.G. A cognitive model of insomnia. Behav. Res. Therapy, 2002, 40: 869-93.

\*Harvey, A.G., Sharply, A.L., Rae, M.J., Stinson, K. and Clark, D.M. An open trial of cognitive therapy for chronic insomnia. *Behav. Res. Ther*, 2007, 45: 2491-2501.

Irwin, M.R., Cole, J.C., and Nicassio, P.M. Comparative Meta-Analysis of Behavioral Interventions for Insomnia and Their Efficacy in Middle-Aged Adults and in Older Adults 55+ Years of Age. *Health Psychology*, 2006, 25: 3-14.

Jacobs, G.D., Edward, F., Stickgold, R. and Michael, W. Cognitive behaviour therapy and

pharmacotherapy for insomnia: a randomised controlled trial and direct comparison. Arch. Intern. Med, 2004; 164: 1888-1896.

\*Jansson, M. and Linton, S.J. Cognitive-behavioural group therapy as an early intervention for insomnia: A randomised controlled trial. *J. Occ. Rehab*, 2005, 15(2): 177-190.

\*Lichstein, K.L., Riedel, B.W., Wilson, N.M., Lester, K.W. and Aguillard, R.N. Relaxation and sleep compression for late-life insomnia: a placebo controlled trial. *J. Consult. Clin. Psychol*, 2001, 69: 227-39.

Lundh, L. G., & Broman, J. E. (2000). Insomnia as an Interaction Between Sleep-Interfering and Sleep-Interrupting Processes. *J. Psychosom. Res*, 49: 299–310.

\*Means, M.K., Lichstein, K.L., Epperson, M.T. and Johnson, C.T. Relaxation therapy for insomnia: Nighttime and daytime effects. *Behav. Res, Ther*, 2000, 38: 665-678.

\*Mimeault, V. and Morin, C.M. Self-help treatment for insomnia: Bibliotherapy with and without professional guidance. *J. Consult. Clin. Psychol*, 1999, 67(4): 511-519.

Montgomery, P. and Dennis, J. Cognitive behavioral interventions for sleep problems in adults aged 60. *Cochrane Database Syst Rev*, 2003, (1): CD003161.

Morin, C.M., Stone, J., Trinkle, D., Mercer J. and Remsberg, S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol. Aging*, 1993, 8: 463-7.

Morin, C.M., Culbert, J.P. and Schwartz, M.S. Non-pharmacological interventions for

insomnia: a meta-analysis of treatment efficacy. *American J. of Psychiatry*, 1994, 151: 1172-1180.

Morin, C.M., Hauri, P.J., Espie, C.A., Spielman, A., Buysse, D.J. and Bootzin, R.R. Nonpharmacologic treatment of insomnia: an American Academy of Sleep Medicine Review. *Sleep*, 1999, 22: 1134-56.

\*Morin, C.M., Blais, F.C. and Savard, J. Are changes in beliefs and attitudes about sleep related to sleep improvements in the treatment of insomnia? *Behav. Res. Ther*, 2002, 47: 741-752.

Morin, C.M. Measuring outcomes in randomised clinical trials of insomnia treatments. *Sleep Med Reviews*, 2003, 7(3): 263-269.

Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A. and Lichstein, K.L. Psychological and behavioural treatment of insomnia. Update of the recent evidence (1998-2004) prepared by a Task Force of the American Academy of Sleep Medicine. *Sleep*, 2006, 29: 1398-1414.

Murtagh, D.R. and Greenwood, K.M. Identifying effective psychological treatments for insomnia: a meta-analysis. *J. Consul. Clin. Psychology*, 1995, 63: 79-89.

Ohayon, M.M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med. Rev, 2002, 6: 97-111.

Riemann, D. and Volderholzer, U. Primary insomnia: A risk factor to develop depression. J.

\*\*Rybarczyk, B., Lopex, M., Schelbe, K. and Stepanski, E. Home-based video CBT for comorbid geriatric insomnia: A pilot study using secondary data analyses. *Beh Sleep Med*, 2005, 3(3): 158-175.

Sindhu, F., Carpenter, L. and Seers, K. Development of a tool to rate the quality assessment of RCT's using a Delphi Technique. *J. Advanced Nursing*, 1997, 25: 1262-1268.

Smith M.T., Perlis, M.L., Park, A., Giles. D.E., Pennington, J.A. and Buysse, D.J. Behavioral treatment vs pharmacotherapy for Insomnia - A comparative meta-analyses. *American J. Psychiatry*, 2002, 159: 5-11.

\*Strom, L., Pettersson, R. and Anderson, G. Internet-based treatment for insomnia: A controlled evaluation. J. Consult. Clin. Psychol, 2004, 72: 113-120.

\*Verbeek, I.H., Konings, G.M., Aldenkamp, A.P., Declerck, A.C. and Klip, E.C. Cognitive behavioural treatment in clinically referred chronic insomniacs: group versus individual treatment. *Beh Sleep Med*, 2006, 4(3): 135-51.

\*\*Vincent, N., Penner, S. and Lewycky, S. What predicts patients' perceptions of improvement in insomnia? *J. Sleep Res*, 2006, 15: 301-308.

Waine, J., Broomfield, N.B. and Espie, C.A. Development and validation of the sleep metacognitive beliefs scale. *J. of Behav. Ther. & Exper Psychiatry* (in press).

\* Wu, R., Bao, J., Zhang, C., Deng, J. and Long, C. Comparison of sleep condition and sleeprelated psychological activity after cognitive-behaviour and pharmacological therapy for chronic insomnia. *Psychother & Psychosom*, 2006, 75: 220-228.

Author (s) (Year) Quality Rating (%)	Design	Sample Size (enrolled/completed) % female; Age (mean); diagnosis; duration of complaint (mean in years, unless otherwise stated)	Treatment/control conditions	Treatment Duration (number of weeks/lengths of sessions); Follow-up (FU) assessments	Outcome Measures/time of assessment	Principal Findings, including observed Effect Sizes (ES – Cohen's d)
Bastien et al., (2004) Quality Rating Score % = (77)	RCT	45/34; 64%; 41.8; Primary Insomnia; 15.25	Individual CBT (n = 15); Group CBT (n = 16); Self-help CBT and telephone support (n = 14)	8 weeks; 6 months	Sleep Diaries; ISI; French version of the DBAS-30 BDI BDI	ISI and DBAS scores decreased pre-post treatment in all 3 conditions (p<0.01). There were no between-group differences on DBAS scores. ES for ISI reductions were: individual ( $d = 3.08$ ); Group ( $d = 2.65$ ); Telephone ( $d = 1.35$ ). ES for DBAS reductions were: Individual ( $d = 2.65$ ); Group ( $d = 2.43$ ); Telephone ( $d = 2.65$ ); Group ( $d = 2.43$ ); Telephone ( $d = 3.06$ ). Improvements were maintained at FU. CBT produced significant changes in SE, WASO, and SQ. Pre-post treatment ES for these variables respectively were as follows: Individual ( $d = 1.08$ , 1.37, 0.66); Group ( $d = 1.19$ , 1.34, 0.76); Telephone ( $d = 1.00$ , 1.63, 0.33). Sleep improvements were maintained at FU. There were no group differences on these sleep variables.
Carney & Edinger (2006) Studies 2 & 3 Quality Rating Score = % (80)	Secondary analysis of data drawn from previous RCT's	128; 42.2%; 54.3 Primary Insomnia; 13.4	Individual CBT ( $n = 100$ ); RT ( $n = 20$ ); SHE ( $n = 8$ )	CBT (6-8 weeks); RT (6 weeks); Sleep Hygiene (2 Weeks); 3 month FU	DBAS, ISQ, Sleep diaries	8 DBAS-30 items showed significantly greater change in response to CBT than alternate therapies. Items found to reduce were related to the DBAS-30 themes as follows: theme 1 = 2 items; theme 2 = 2 items; theme 4 = 0; theme 5 = 3 items. Declining scores on 15 DBAS-30 items were related to 1 or more indices of sleep improvement. DBAS was significantly correlated with the ISQ ( $r$ = 0.49, $R^2$ = 24% p<0.001). Those improved

Table 1: Summary of included articles (for key see end of table)

on the ISQ showed greater pre-post treatment reductions on 13/30 DBAS items. WAS0 improved had greater DBAS reductions in 3 items (theme $1 = 1$ item; them $2 = 2$ items). CBT showed greater reduction in DBAS scores (pre-post treatment), p<0.025. CBT produced larger long-term reductions in DBAS scores at FU than did RT, p<0.01.	CBT had greater pre-preduction in DBAS than RT (0.86; $p<0.01$ ). CBT signific DBAS-10 scores pre-post t 0.86). ES for the RT and PC (0.27 and $d = 0.20$ , respectivel DBAS-10 reductions were CBT versus RT at follow Across all participants, pre-DBAS-10 total scores were correlated with reductions in ISQ and SE. All three of the were significant only for th WAS0 ( $r = 0.45$ ; $R^2 = 21\%$ ), $R^2 = 21\%$ ) and SE ( $r = -4$ Pre-to-follow-up improvem DBAS were only related to in subjective (diary) ratings of $r$ 0.45; $R^2 = 21\%$ ) and sleep quarts of $R^2 = 21.2\%$ within the CBT ( $R^2 = 21.2\%$ ) withe core $R^2 = 21.2\%$	Internal consistency of DBAS-30 = Cronbach's alpha of 0.72. DBAS-10 correlated highly with the DBAS-30 ( $r = 0.826$ ). DBAS-10 demonstrated treatment-related measurement sensitivity; DBAS values were significantly lower at post-
DBAS; Sleep diaries, PSG	PSG; Sleep diaries; ISQ; DBAS	DBAS-30 (DBAS-10 scores extracted from full 30 item scale)
6 weeks; 6 months	6 weeks; 6 months	6 weeks; 3 months
Individual CBT; RT; PLA (quasi- desensitisation)	Individual CBT; (RT); PLA (sham behavioural intervention)	CBT
73; 46.6%; 54.3; Primary Insomnia; 13.8	75/46; 46.7%; 55.3; Primary Insomnia; 13.6	178/60; 68%; 49.8; Primary and Secondary Insomnia; 60% >5 years
Secondary analysis of data from previous RCT	RCT	Secondary analysis of data drawn from previous
Edinger & Wohlgemuth, 2001 Quality Rating Score % = (80)	Edinger et al. (2001) Quality Rating Score % = (83)	Espie et al., (2000) Quality Rating Score % = (84)

	Open Clinical Trial					treatment (p values ranging from 0.009 to 0.0001) $df = 90$ , and at follow-up (p values ranging from 0.007 to 0.0001), $df = 59$ .
Harvey et al. (2007)	Open clinical trial	19; 52.6%; 48.5; Primary and Secondary Insomnia; 22.89	Cognitive Therapy (CT)	6-22 (average = 14); 12 month	ISI, sleep diary, DBAS, BDI, PSG	Significant pre-to-post treatment reductions on DBAS-30 scores were observed ( $d =$ 2.46) - also retained at FU. No participant
Quality Rating Score % = (79)						met criteria for primary insomnia at post- treatment and 3-, 6-, and 12-month FU. ISI total score reduced significantly at post-
						treatment $(d = 2.20)$ ; this reduction was retained a 12- month FU. Decrease in SOL and WASO, and increase in TST at post- treatment were also retained at FU (SOL, $d$ = 0.48; WASO, $d = 0.45$ ; TST, $d = 0.64$ ).
Jansson & Linton (2005)	RCT	165/136; 77%; 49.8 Primary and Secondary Insomnia; 7.9 months	CBT (Group); Self- help information package	6 weeks; 12 months	Sleep Diaries; DBAS; HADS	At FU, the CBT-group intervention produced significantly larger reductions on DBAS-10 total scores, with a 23% reduction
Quality Rating Score $\% = (77)$						help group from baseline to FU, $p < 0.001$ ( $d$ = 0.89 and $d$ = 0.29) respectively. Group CBT produced significantly greater improvement from baseline to FU on TST, SQ, and SE ( $d$ = 1, 1.32, 1.03).
Lichstein et al., (2001)	RCT	89/72; 74%; 68.03; Primary Insomnia; 8.93	RT (n = 27), SR (n = 24; Placebo (n = 23)	6 weeks; 12 months	Sleep Diaries; PSG; Epworth Sleepiness Scale:	RT group had significantly lower DBAS-30 total scores at FU ( $d = 0.57$ ). No group differences on DBAS-30 scores were observed. SR and relaxation more effective
Quality Rating Score % = (76)					DBAS; Insomnia Impact Scale, Fatigue Severity Scale	that Placebo on sleep continuity variables at post-treatment and FU. Subjective (sleep diary) WASO reduced from 67 min (baseline) to 43 (post) to 38 (follow-up) in SR, $d = 0.70$ , compared to 67 min (baseline), 43 (post), and 52 (FU) for relaxation, $d = 0.16$ . No significant change
Means et al.	RCT	139/118; 68.5%; 21.2	RT; Wait list control	3 weeks; 5 weeks	Sleep Diaries;	on PSG measures. SR produced best outcomes at FU. No group differences on DBAS-30 scores

(2000)		Primary insomnia; 3.5			DBAS; Fatigue	were observed. RT produced more
Quality Rating					Epworth Sleepiness	but not SOL. The magnitude of
Score $\% = (72)$						improvement was small: WASO for the RT
					State Worry Ouestionnaire	group reduced from 22 min (baseline) to 13 min (nost-treatment) $d = 0.57$ . SF reduced
					Insomnia Impact	from 84.8% (baseline) to 88.4% (post-
						treatment), $d = 0.51$ ).
Mimeault &	RCT	58/54; 59%; 50.8	CB	6 weeks;	Sleep Diares;	Improvements at post-treatment were
Morin (1999)		Primary Insomnia; 14.4		3 months	PSQI; ISI; Sleep	obtained on the DBAS-30 ( $d = 0.81$ ; BT)
-			e		mer	and $d = 1.31$ (BTPC). Participants in both
Ouslity Rating			Wait-list control		undex; Frencn	rearment conditions improved significantly
Score $\% = (76)$						on 1 W 1 and 32 at post-ucambur, Lincu sizes for TWT and SF respectively were $d =$
						0.77 and $d = 0.68$ for the BT, and $d = 1.22$
						and $d = 1.14$ for BTPC. BT and BTPC were
						comparable at follow-up and remained more
						improved than controls.
Morin, Blais &	Secondary		CBT $(n = 18);$	8 weeks;	Sleep diary,	CBT and COMB produced greater
Savard (2002)	analysis if	Primary Insomnia; 17.0	PCT (temazepam) n	3, 12 & 24-months	PSG, DBAS	improvements on the DBAS-30 at post-
	data		= 20; CBT+PCT			treatment $(d = 1.84 \text{ and } 1.89 \text{ respectively})$
	drawn		(COMB) n= 20; PLA			than PCT ( $d = 0.31$ ) and PLA (p<0.05). All
Quality Rating	from		(n = 20)			three active conditions showed reduced
Score $\% = (76)$	previous					DBAS scores at 3 and 8 months follow-up
	RCT					than PLA but there were no significant
						group differences. Reductions of DBAS-30
						scores were significantly correlated with
						improvements in diary measured SE ( $r = -$
						.28; $R^{\prime} = 7.8\%$ , p<0.05. The correlation
						between DBAS-30 scores and SE (PSG
						measured) was approaching significance (r
						= -0.24; $R'$ = 5.8%, p<0.065). More
						adaptive beliefs and attitudes about sleep at
						post-treatment were associated with better
						maintenance of sleep improvements at each
						follow-up assessment: 3 months ( $r = -0.22$ ;

J	r												·	r						1
$\begin{array}{l l} R^2 = 4.8\%, \ p<0.05); \ 12 \ months \ (r =36; \ R^2 \\ = 13\%, \ p<0.01); \ 24 \ months \ (r = -0.51; \ R^2 = 26\%, \ p<0.0005). \end{array}$	Greater pre-to-post treatment reduction of DBAS-30 scores in CBT ( $d = 0.81$ ) relative to controls ( $d = 0.01$ ) Greater improvements	observed on 1 W 1, 151, and 5E sleep variables in treated relative to controls $(d = 0.78; d = 0.45; d = 0.7)$ respectively. However, improvements were also found in the control group $(d = 0.27; d = 0.11; d = 0.39)$ .	DBAS-30 scores reduced significantly in		to post-treatment) were $d = 1.79$ and $d =$	1.97 for group and individual treatment respectively. This improvement remained	significant at follow-up $(d = 1.77 \text{ and } d =$	1.43), for group and individual treatments respectively CRT produced significant	changes in SOL, TST, and WASO: Group d	= 0.6, 0.3 and 0.5, respectively; Individual d	= 0.85; 0.68, and 0.8 respectively. For TST	and SE, improvements were maintained at	Individual $(d = 0.62 \text{ and } 0.8)$ , respectively).	Pre-Post treatment scores on themes 2,3, and		the CBT group versus the PCT group.	between the four proups were found at each	follow-up assessment. Effect sizes for	themes $2, 3$ , and 5 in response to CBT alone	were as follows: Pre-post treatment $(d = 1.82, d = 1.25, d = 1.75)$ ; 3-month FU $(d = 1.82, d = 1.25, d = 1.75)$ ; 3-month fu $(d = 1.82, d = 1.75)$ ; 3-month fu $(d = 1.82, d = 1.75)$ ; 3-month fu $(d = 1.82, d = 1.75)$ ; 3-month fu $(d = 1.82, d = 1.75)$ ; 3-month fu $(d = 1.82, d = 1.82)$ ; 3-month fu $(d = 1.82, d = 1.82)$ ; 3-month fu $(d = 1.82, d = 1.82)$ ; 3-month fu $(d = 1.82, d = 1.82)$ ; 3-month fu $(d = 1.82, d = 1.82)$ ; 3-month fu $(d = 1.82, d = 1.82)$ ; 3-month fu $(d = 1.82)$ ; 3-mon
	Sleep Diaries; DBAS; Medication	Index; HAUS;	Sleep diaries,	Profile; Sleep Evaluation Form	DBAS; Utrecht	Coping List								PSG, Sleep	diaries, PSAS,	DBAS, PSQI				
	5 weeks; no FU		6 weeks; 1-3-and	sdn-m		· · · · ·									3 and 8 months					
	Internet CBT (with RT; n = 30); Wait list control (n =	(00	CBT (Group V	List Control										CBT (n= 19)	PCT (temazepam; n=	(70); CBT + PCT	$(\text{CUMD}; II^{-} 10);$ PLA (n= 17)			
	109/80; 65%; 44.1; Primary Insomnia; 10.6		106/58; 61.3%; 44.4;	Insomnia; 14.9 years											Primary Insomnia;NS					
	RCT		Controlled	Trial										RCT						
	Strom et al. (2004)	Quality Rating Score % = (70)	Verbeek et al.	(0007)	Quality Rating	Score $\% = (74)$								Wu et al. (2006)		Outlite: Dation	Score $\% = (79)$			

2.32, d = 1.27, d = 3.52; 8 month FU ( $d =$	2.28, d = 0.64, d = 3.40). No differences in	themes 2, 3, and 5 were observed in the PCT	group. Subjective SOL, SE, and TST were	better in the PCT group than in the CBT	group at end of treatment. At 3 month	follow up, subjective and objective SOL,	SE, TST were better in the CBT group:	Subjective: $(d = 1.17, d = 1.58; d = 0.83);$	Objective: $(d = 1.38, d = 1.15, d = 1.19)$	than in both the PCT and the combined	group. Treatment gains were sustained at 8	month FU for the CBT group, but the PCT	group had returned to their baseline level of	sleeping difficulties. COMB showed a	variable long-term effect.

Attitudes about Sleep Scale; BAI: Beck Anxiety Inventory; BDI: Beck Depression Inventory; ES: Effect Size; FU: Follow-Up; SE: Sleep Insomnia Severity Questionnaire; PLA: Placebo; PSG: Polysomnography; CT: Cognitive Therapy; TST: Total Sleep Time; SOL: Sleep RCT: Randomised Controlled Trial; CBT: Cognitive-behavioural Therapy; ISI: Insomnia Severity; DBAS: Dysfunctional Beliefs and Onset Latency; TWT: Total Wake Time; PSAS: Pre-Sleep Arousal Scale; PSQI: Pittsburgh Sleep Quality Index; ISI: Insomnia Severity Efficiency; WASO: Wake After Sleep Onset; SQ: Sleep Quality; RT: Relaxation Training; SHE: Sleep Hygiene Education; ISQ: Index; PCT: pharmacotherapy; COMB: CBT + PCT. Note:

# **CHAPTER 2: MAJOR RESEARCH PROJECT**

# Attention Bias for Positively and Negatively Valenced Sleeprelated Pictorial Stimuli in Psychophysiological Insomnia, Delayed Sleep Phase Syndrome and Normal Sleep: An Investigation using the Dot-Probe Paradigm.

Prepared in accordance with submission to Journal of Sleep Research (Appendix 1.1)

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#### SUMMARY

Studies using computerised reaction time tasks demonstrate attention bias for sleep-related stimuli relative to neutral stimuli in Psychophysiological Insomnia (PI). The mechanisms that 'drive' this attention bias remain unclear. Cognitive models of PI posit both threat and craving as two candidate processes. However, few studies have specifically investigated this. This study used the 'dot-probe' task to examine the attention bias effect generated by sleep pictures which were intrinsically negative in valence (threat) and intrinsically positive in valence (craving). A between-groups design - PI, Delayed Sleep Phase Disorder (DSPS) and Normal Sleepers (NS) - was employed. It was hypothesised that PI would show greater attention bias to all sleep stimuli (negative and positive combined) relative to neutral stimuli compared with DSPS and NS. This effect was predicted to occur both at the level of threat (negative sleep pictures) and craving (positive sleep pictures). No a priori prediction regarding any differential attention bias effect generated by positive compared to negative sleep pictures was made. Fifty-eight individuals completed the study (PI = 18; DSPS = 19; NS = 21). Group allocation was determined by combined DSM-IV and ICSD-2 diagnostic criteria, confirmed by questionnaire, sleep diary and actigraphy data. Orthogonal planned contrasts on mean attention bias scores did not support any of the specified hypotheses. Observed effect sizes and post-hoc power calculations highlighted that the present sample size lacked sufficient power to detect differences in the degree of attention bias shown towards negative sleep stimuli and all sleep stimuli combined, across the PI, DSPS, and NS groups. In summary, no clear conclusions regarding the role of threat and craving in driving attention bias in PI can be drawn from the present findings. However, methodological limitations may explain this. Replication of this study using a larger sample size, further refinement of the experimental stimuli and use of psychophysiologic measures of attention, such as facial EMG, is recommended.

# **INTRODUCTION**

Psychophysiological Insomnia (PI) is characterised by conditioned sleep difficulty and/or heightened arousal as indicated by inability to initiate sleep at the desired time, increased focus on and anxiety about sleep, mental arousal, and heightened somatic tension (Research Diagnostic Criteria; RDC, Edinger et al., 2004). The exact mechanisms underlying the development and maintenance of PI remain unclear. One mechanism posited by two recent models (Harvey, 2002; Espie et al., 2006) is selective attention or 'attention bias', whereby stimuli that are salient to an individual are likely to attract attention, so that certain aspects of processing are prioritised, possibly at the expense of other aspects.

Harvey's *Cognitive Model of Insomnia* (2002) emphasises the role of excessive negative cognitive activity, whereby worry about sleeping and the consequences of not sleeping precipitates the **preferential monitoring** of internal and external sleep related cues, such as bodily sensations consistent with falling asleep or not falling asleep, and features of the bedroom environment. The Attention-Intention-Effort (AIE) model developed by Espie et al., (2006) is more specifically grounded in **information processing** theory. This model proposes that normal sleep is an automatic and involuntary process. In PI, this automaticity is inhibited by a developmental pathway beginning with an initial selective attention to sleep-related internal and external cues, and culminating in the explicit, conscious intention and subsequent effort to initiate sleep.

# **Evidence for Attention Bias in PI**

Evidence of attention bias in PI can be drawn from several sources. Support for Harvey's

model stems from phenomenological/descriptive studies (Neitsert-Semler and Harvey, 2004) and 'real world' experiments (Neitzert-Semler and Harvey, 2006; Tang et al, 2006), which demonstrate that insomnia is, in terms of self-report, associated with sleep-related selective attention (Espie et al., 2006).

Support for biased information processing within the A-I-E framework comes from several studies that measure attention bias in insomnia objectively via computerised tasks, in which reaction time towards stimuli presented on the computer screen is used as an index of cognitive arousal. These include the Emotional Stroop Task (Taylor et al., 2003) and the Inducing Change Blindess (ICB) and Flicker paradigm (Jones et al., 2005; Marchetti et al., 2006). These studies provide substantial evidence that people with insomnia have an attention bias for sleep-related stimuli, where stimulus salience is thought to influence response time because of their 'grabbiness' (defined as uncontrolled prolonged attention capture or focusing) of sleep-related word or pictorial stimuli relative to neutral stimuli (see Espie et al., 2006, for a detailed review).

This effect has been observed in insomnia relative to normal sleepers and those with other forms of sleep disorder, such as Delayed Sleep Phase Syndrome (DSPS), a circadian timing disorder, which, like insomnia, is characterised by difficulties in initiating sleep (Pavlova et al., 2001). For example, MacMahon et al (2006) used another computerised paradigm, the Dot-Probe task, to explore whether systematic processing differences in attention bias existed between Good Sleepers (GS) and those with PI and DSPS. In this task, sleep-related and neutral word pairs are presented simultaneously on a computer screen. Attention is measured by recording the speed of manual responses to a visual probe that could appear in the spatial

location of either word, immediately after the display of that word has ended (Marchetti et al., 2006). Measuring the impact of salient, sleep-related words on the probe reaction times in the two spatial areas indicates whether visual attention has shifted towards or away from such stimuli. In this study, individuals with PI showed significantly greater attention bias toward sleep-related words (in comparison to neutral words) when compared to GS and DSPS.

Although the replication of attention bias data across the experimental paradigms noted above highlights the stability of the attention bias phenomenon in insomnia, few studies have investigated the processes that underlie or 'drive' sleep-related attention bias. Spiegelhalder et al., (2008) used a newly developed 'mixed modality' (visual auditory) task in addition to the Stroop task to examine attention bias between primary insomnia, sleep experts and normal sleeper controls. The sleep expert control group was used to investigate the impact of *frequency of concept usage (FOCU)*. FOCU relates to the "expert hypothesis" of attention bias proposed by Ryan (2002), who found that alcohol-related attention bias was observed not only in problem drinkers, but also in staff working within substance abuse clinics. Speigelhalder et al (2008) observed that the insomnia group showed increased sleep-related attention bias on the Stroop task compared with the sleep expert group. The authors of this study conclude that this finding refutes the possibility that FOCU underlies sleep-related attention bias, and propose that people with insomnia are perhaps more emotionally and cognitively affected by sleep-related stimuli than sleep experts.

#### What, then, does drive attention bias in PI?

Two possible psychological processes – threat and craving – have been proposed to underlie the attention bias that has been detected in PI (Espie et al., 2006; Harvey et al., 2002).

# i) Threat

Attention bias has been implicated in the maintenance of several anxiety-related psychological disorders (e.g. Cooper and Fairburn, 1992; Bryant and Harvey, 1996; Salkovskis, 1999). Beck's cognitive model of emotional disorders posits that attention biases are motivated by negative beliefs or 'schema' stored in long-term memory (Beck, 1967). When activated, these schema direct information processing, including attention, towards stimuli congruent with them. This increases an individual's belief in negative automatic thoughts, resulting in hypervigilance and pre-occupation with danger and threat (Beck and Clark, 1997). Evidence suggests that anxious individuals are indeed characterised by selective attention bias toward threat (e.g. Mogg and Bradley, 1999), and it has been argued that this may have a causal role in anxiety disorders (Matthews and McLeod, 2002).

Harvey's Cognitive Model of Insomnia (2002) fits well with this 'threat-monitoring' model, and posits that selective attention to internal and external sleep related cues culminates in the overestimation of perceived deficit in sleep and daytime performance. The individual then believes that they have had less sleep than they actually have, and that their daytime performance is worse than it actually is. This leads to further excessive, negative cognitive activity, such that true deficit in sleep and daytime performance occurs and a vicious cycle of insomnia begins (Harvey, 2002). Perceived inability to sleep may therefore be conceptualised and experienced as a significant threat, possibly as a result of the associated reduced quality of life, poor work performance, and physical and psychological ill–health.

As stated, the AIE pathway defines normal sleep as passive and effortless, whereby good

sleepers are assumed to fall asleep without consciously thinking about how they manage to do so. As Espie et al., (2006) suggest, if an individual with PI was once able to sleep normally, without being aware of how they did so, it is possible that sudden inability to sleep might be experienced as threatening. This is then thought to direct an individual's attention focus onto sleep and sleeplessness and give rise to actions aimed at reducing wakefulness.

# ii) Craving

Attention bias is not only observed in relation to threat. It has been implicated in the maintenance of alcohol, heroin, and nicotine addiction, where the stimuli are appetitive or reinforcing rather than threatening (Espie et al., 2006). For example, selective attention bias to relevant word or picture stimuli has been found in alcoholics and problem drinkers, but not social drinkers (e.g. Lusher et al., 2004). This suggests that problem drinkers are more likely to detect alcohol-related stimuli in the environment, which might then evoke memories of drinking and maintain their addiction by producing a 'craving' sensation (Lusher et al, 2004). Given that sleep is a basic human need, it may be that the less sleep a person has, the more valuable it becomes – sleep-related attention bias may then result from a person's 'craving' (i.e. preoccupation and longing) for good sleep when experiencing chronic sleep difficulties (Espie et al., 2006).

Similarly, attention bias for addiction-related stimuli amongst smokers and alcoholics has been explained by the *Motivational Theory of Strong Current Concerns* (Cox et al., 2006). A current concern is defined as a "time-binding motivational state" of an individual between two particular points in time, whereby "individuals first become committed to pursuing a specific goal and subsequently to either attaining the goal or abandoning its pursuit". Current concerns
are therefore manifest as the urge or 'craving' to use an addictive substance at a particular point in time, which can be influenced by cues and stimuli related to that substance. It is presumed that the greater an individual's current concern about an addictive substance, the greater the attentional bias for stimuli related to that substance will be – that is, the value an individual attributes to a goal and their experiences associated with it influences their attentional sensitivity to cues and stimuli related to that goal. Parallels may be drawn between this theory and the AIE pathway, where explicit intention to sleep generated by attention bias may reflect what Espie et al., (2006) refer to as an 'attention-for-action' mechanism, which subsequently results in individuals with PI engaging in efforts to initiate sleep.

Recently, Speigelhalder et al., (in press) examined the role of threat (conceptualised as 'sleeplessness' or poor sleep quality as measured by participants scores on the PSQI), and craving ('sleepiness' as measured by the SSS), in 105 non-clinical participants again using the Stroop task and the aforementioned mixed modality task. The Stroop task revealed significant positive linear relationships between sleep-related attentional bias and both poor sleep quality and sleepiness. A significant negative interaction effect between these two variables on the cognitive bias was also observed, suggesting that attentional bias scores reduce when poor sleep quality is associated with high sleepiness and high sleep quality is associated with low sleepiness. The mixed modality task did not yield any significant findings. On the basis of these findings, the authors conclude that sleep-related attention bias can be generated by both threat (sleeplessness) and craving (sleepiness). Both the role of threat (of sleeplessness) and craving (for sleep) appear therefore to require further experimental investigation (Espie et al., 2006).

# Manipulating the emotional valence of sleep-related stimuli

In the sleep-related attention bias studies conducted to date, the experimental stimuli used have not been intrinsically commanding of attention, nor were they particularly emotive or threatening (Espie et al., 2006). This differs from the stimuli used to measure attention bias in other disorders. For example, within the anxiety literature, the stimuli used vary considerably in content and emotional valence, and have been shown to consistently evoke discrete subjective and physiological reactions (Yiend and Matthews, 2001), thus increasing the possibility of demonstrating processing biases by maximising the salience of the stimuli. Manipulating the emotional valence of sleep-related stimuli used in attention bias paradigms may prove an effective way to investigate what underlies or 'drives' the selective attention bias seen in insomnia.

Furthermore, there are limitations to using word stimuli in attention bias tasks. Research demonstrates that verbal attention paradigms do not generate the same attention bias data as pictorial versions, as pictorial stimuli are more likely to induce responses that mimic those experienced in real life situations (Yiend and Matthews, 2001). Word stimuli may be clearly negative in valence, but it is unclear if they encapsulate a severe or highly salient threat. For this reason, attention bias studies investigating state and trait anxiety have used emotionally valenced pictorial stimuli (e.g. weapons, corpses, dangerous animals; Mogg et al., 2000; Yiend and Matthews, 2001; Fox et al., 2002). In the context of insomnia, research specifically attempting to differentiate sleep-related pictures into those that are intrinsically negative and those that are intrinsically positive in valence is necessary.

## Aims and Hypotheses

This study was part of an ongoing programme of research within the University of Glasgow Sleep Center (UGSC), in collaboration with the Department of Psychology. The aim was to investigate the role of the two candidate processes posited thus far - threat and craving – in modulating the attention bias detected in insomnia, by manipulating the emotional valence of pictorial sleep-related stimuli. Specifically, the attention bias effect generated by pictures which were intrinsically negative in valence (threat) and intrinsically positive in valence (craving) amongst individuals with PI, Delayed Sleep Phase Disorder (DSPS) and Normal Sleepers (NS), was investigated using a dot-probe paradigm.

## Hypothesis 1

It was anticipated that this study would initially replicate the findings of previous studies which implicate attention bias in PI. That is, participants with PI would demonstrate attention bias towards sleep-related stimuli relative to neutral stimuli, as measured by faster dot-probe reaction times, whereas those with DSPS and NS would not.

# Hypotheses 2 and 3

Having demonstrated this, it was predicted that this effect would be observed both at the level of threat and craving stimuli as follows:

a) Participants with PI would show greater attentional bias than those with DSPS and NS towards negative (threatening) sleep-related pictorial stimuli, as measured by faster dot-probe reaction time, in comparison to neutral pictures.

b) Participants with PI would also show greater attention bias than those with DSPS and NS towards positive (craving) sleep-related pictorial stimuli, as measured by faster dotprobe reaction time, in comparison to neutral pictures.

As both threat and craving are posited to drive attention bias in PI, no a priori hypothesis regarding variation in the magnitude of the attention bias effect generated by negative relative to positive sleep pictures was specified.

# Ethics

Ethical approval for the study was granted by the Medical Faculty of the University of Glasgow, and by NHS Greater Glasgow and Clyde, Community Mental Health Division (Appendices 2.3 and 2.4).

#### **METHOD**

# Design

A between-groups design (PI, DSPS, NS) was employed. The control group of individuals reporting no sleep difficulties are referred to as 'Normal Sleepers' (NS) in line with current Research Diagnostic Criteria (RDC; Edinger et al., 2004) for insomnia. It is not yet clear that attention bias has a primary role in the aetiology of insomnia, as information processing bias of this kind could occur because sleep is continually disturbed, making selective attention to sleep a secondary characteristic of chronic insomnia (Marchetti et al., 2006). Previous attention bias studies therefore recommend the inclusion of not only control samples of Normal Sleepers, but also comparison groups with other forms of sleep disorder such as

DSPS. As stated previously, both DSPS and PI are characterised by difficulties initiating sleep (Alvarez et al., 1992). The cause of this difficulty in PI is attributed to psychological factors. As DSPS is thought to result from a circadian timing disorder, cognitive factors are not believed to influence the development or maintenance of DSPS; individuals who present with DSPS are therefore not expected to show attention bias to sleep-related stimuli as observed in PI (MacMahon et al., 2006).

# Selection of the Computerised Attention Bias Task

Studies using complex pictorial scenes within both the fields of anxiety and addiction typically use the dot-probe paradigm (e.g. Bradley et al., 1997; Mogg et al., 1995; Mogg and Bradley, 2000). Two versions of the dot-probe task are available. *Probe-position* tasks require participants to identify the position of the probe on the screen (e.g. left or right). However, such tasks might induce preferential monitoring of one region of the computer screen over the other (Mogg and Bradley, 1999). *Probe-classification* tasks require participants to identify the type of probe displayed e.g. an upwards or a downwards arrow. This task promotes monitoring of both sides of the display, but the relationship between the stimulus and response is more difficult to learn, resulting in reaction times that are more likely to be variable, and higher error rates. Mogg and Bradley (1999) observed that, in non-clinical samples, the probe-position task yielded faster reaction times, fewer errors and similar effect sizes, compared with the probe-classification task. The primary dependent variable was therefore reaction time to a dot-probe-detection response; latencies to detect this probe were used to index the extent to which these groups selectively attend to either positive or negative sleep-related stimuli, or neutral stimuli.

# Stimulus Exposure Duration

A stimulus exposure duration of 500-ms was chosen in line with standard practice within the dot-probe literature (e.g. Yiend & Matthews, 2001; Ehrman et al., 2002; Bradley et al., 2003). Literature suggests that the attentional system is not unitary, but comprises discrete elements, specifically, '*engagement*' (i.e. the orienting of attention necessary for the initial detection of stimuli), and '*disengagement*' (i.e. the prolonged holding of attention in that particular location once stimuli have been detected, Posner, 1980). In this study the engagement component is investigated, for which stimulus exposure duration of 500-ms is thought to be optimum. This is supported by evidence of concordance between the attentional bias index derived from probe reaction time data (using 500-ms stimulus displays) and the direction of initial shift in eye gaze to emotional stimuli in dot-probe tasks (Bradley et al., 2000).

# Justification of Sample Size

Mogg and Bradley (1999) investigated effect sizes obtained when measuring attention bias amongst anxious individuals for emotionally valenced pictorial stimuli using a probe-position, dot-probe paradigm. They observed that estimated effect sizes varied from +0.61 to +0.81, with a mean of +0.71. Therefore, a power calculation, assuming an effect size of +0.71, estimated that 21 participants would be required in each of the 3 groups (PI, DSPS, and NS) to detect significant differences at an alpha level of 0.5, with a power of 0.8 (one-tailed).

## PARTICIPANTS

#### **Inclusion and Exclusion Criteria**

For the PI group, participants met Research Diagnostic Criteria (RDC; Edinger et al., 2004)

for Psychophysiological Insomnia (of the sleep-onset type). These criteria have been derived from, and combine, existing DSM-IV and ICSD-2 criteria. Those with PI were classified as having *at least* "sub-threshold insomnia" as measured by the Insomnia Severity Index (ISI), scored >5 on the Pittsburgh Sleep Quality Index (PSQI), were defined as "Neither Type" on the Morning-Eveningness Questionnaire (MEQ), and experienced sleep disruption for longer than 6 months. Sleep-onset difficulties typical of PI were confirmed by sleep diary data.

For the *DSPS* group, participants satisfied combined DSM-IV and ICSD-R criteria for DSPS, as RDC criteria for this condition have not been defined. They were classified as "Definite Evening Type" on completion of the MEQ. Circadian phase delay was confirmed by sleep diary data and objective actigraphy data.

Normal sleepers were required to score <5 on the PSQI, report themselves as being 'normal' sleepers, and meet no criteria for a sleep disorder at the present time or in the past, and were classified as "Neither Type" on completion of the MEQ.

For all 3 groups, exclusion criteria included: active psychological or drug interventions for sleep problems; sleep disturbance due to a combination of PI and DSPS (or as a consequence of substance misuse or physical health, as assessed during interview following completion of the experimental procedure); meeting RDC criteria for any other sleep disorder, and scoring above the cut off for depression (11) on the HADS. As anxiety is a common feature associated with insomnia, scoring above the cut off for anxiety on the HADS did not result in exclusion. All participants had normal vision, or corrected to normal vision.

# Sample

Participants were primarily students at the University of Glasgow. A small proportion (n = 8) were staff working within NHS Greater Glasgow & Clyde. Students were recruited through a mass email sent round the university system. Following the procedure used by Marchetti et al., (2006), this email asked some brief questions designed to help the researcher appraise individual sleeping patterns (Appendix 2.5). NHS staff were recruited through a similar electronic notice placed on the local staff intranet site.

In total, 152 individuals responded. Of these, 109 reported symptoms similar to PI and DSPS and were emailed a second time for further information regarding their sleep patterns. Eightyeight individuals responded to this second e-mail; seventy-five were assessed as potentially suitable and therefore completed the experimental procedure. Following application of rigorous criteria and prospective diary and (for the DSPS group) actigraphic measurement, 17 participants were excluded from the data analyses. Reasons for exclusion were: failure to return all questionnaire data (3); failure to wear the Actiwatch as instructed (1); having an error rate above the 5% threshold (2); sleep difficulties resulting from sleep maintenance rather than onset (3) and recreational drug use (2). One participant disclosed that they were using light-box therapy for Seasonal Affective Disorder. A further participant was excluded due to complaints of chronic back pain, which appeared to have contributed to the onset of their sleep complaint.

Finally, an additional three participants were excluded from the study as their sleep difficulties appeared to result from a combination of PI and DSPS; another participant appeared to present

with Non-24-h Sleep-Wake Syndrome (Free-Running pattern). For these 4 participants, all interview, questionnaire, sleep diary and actigraphy data were reviewed collaboratively by the researcher and an expert within the field to confirm sleep status and ineligibility for the study.

# MATERIALS

A total of 72 colour photograph picture pairs were developed for this study. 12 neutral practice photographs (of non-sleep related objects e.g. basket/spoon) were divided into 6 practice pairs. 40 sleep-related photographs were differentiated equally into 2 photo sets, so that there were 20 photographs associated with 'poor' sleep (photographs that were intrinsically negative in content and therefore represented 'threat'), and 20 which were associated with 'good' sleep (i.e. photographs that were intrinsically positive in content and therefore represented 'craving').

Sleep-related photographs were taken within rooms at the University of Glasgow Sleep Centre by the researcher using a Nikon 'Coolpix' 8-megapixel digital camera. As far as possible, photographs were counterbalanced in terms of content, number of objects, and gender of models. Photograph pairs were prepared using Adobe Photoshop and Microsoft PowerPoint, and image lightening and contrasting settings were matched as far as possible for all photographs. Attempts were made to standardise the position of models within the photographs in order to control for any biased attention towards faces that may have appeared closer on the screen and therefore commanded greater attention.

The content of the sleep-related photographic stimuli was generated from scales derived from research identifying the 'live' cognitions that people with Insomnia have while lying awake in

bed unable to sleep. Specifically, items from the Glasgow Content of Thoughts Inventory (GCTI: Harvey and Espie, 2004), the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS; Morin, 1993), the Sleep Disturbance Questionnaire (SDQ; Espie et al., 1989) and the Self-Statements Test (Fichten et al., 1998) were used. Transcripts of audio-diaries, completed immediately before bedtime and upon wakening as part of two qualitative studies investigating intrusive thoughts and the impact of insomnia upon quality of life and daytime functioning, respectively (Wicklow and Espie, 2000; Kyle et al., 2008), were also used to derive the content of the sleep pictures. Photographs depicted sleep quality associated with both night and daytime effects. Once developed, photographs were initially screened by the experimenter and researchers at the UGSC for valence and clarity of composition. Remaining pictures were then presented to a panel of 10 independent student raters, who were asked to rate the degree to which pictures were associated with negative and positive sleep using a 9-point scale, where 0 =not at all positive/negative and (-/+) 8 =extremely negative/positive. This procedure was adapted from similar attention bias studies conducted within the anxiety field (e.g. Mogg et al., 2000; Yiend and Matthews, 2001). Only those pictures that were rated with a mean score of +/-6 were selected for inclusion. Mean ratings (with standard deviations) of picture valence (positive and negative) were then calculated and are presented in Table 1.

## - Insert Table 1 Here -

Each sleep photo was then paired with a neutral, non-sleep related photograph, matched as closely as possible in terms of overall composition and degree of visual complexity (brightness, orientation, size, gender and number of people/objects in each picture), as judged by eye. A further 40 neutral, non-sleep photographs were divided into 20 picture pairs, and

used as filler material (see Yiend and Matthews, 2001). The neutral photographs used in critical (sleep-neutral) trials in this study were selected from the International Affective Picture System (IAPS; Lang et al., 1999), a standardised set of photographic material with normative ratings for valence and arousal. Neutral photographs used for the filler trials (not included in the overall analyses) were taken from a selection of photographs used in a previous study investigating the role of emotional arousal as measured by facial EMG recordings in insomnia (Baglioni et al., unpublished manuscript).

## **Measures and Equipment**

## Measures to test Hypotheses

Attention bias was measured by speed of manual responses to the dot-probe. The dot-probe task was programmed by the researcher using the experiment-generating package SuperLab Pro 4.0 (Cedrus Corporation), and was implemented using a Dell Latitude (Series D531) laptop computer. The size of the monitor was approximately 33-cm (diagonally) and the viewing distance was approximately 100-cm. Each photo measured 13-cm long and 8-cm wide when displayed on the screen. Attention bias was measured by speed of manual responses to the dot-probe. Millisecond timing was determined through SuperLab Pro and an external response box (Model RB-830; Cedrus Corporation) was used to collect response latencies in order to avoid timing errors associated with standard keyboard or mouse input.

# Sleep Quality Measures

The following measures were administered to assess each participant's sleep quality in order to aid the differential diagnosis of PI, DSPS, and NS, and confirm group allocation:

- The *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al., 1989) – measures retrospective sleep quality and disturbance over a one month period, distinguishing between 'good' and 'poor' sleepers. The scale has proven reliability, with an overall Cronbach's alpha coefficient of 0.83, and demonstrated validity, with observed sensitivity and specificity of 85.6% and 86.5% respectively (Smith & Wegener, 2003). A PSQI global score of >5 indicates severe difficulty in at least two areas, or moderate difficulty in more than three areas, of sleep quality.

- The *Insomnia Severity Index* (ISI; Morin et al., 1993) was administered to qualify the severity of participants' subjective symptoms and consequences of insomnia as well as the degree of concerns or distress caused by these difficulties. There are 4 categories: (No Clinical Significant Insomnia; Subthreshold Insomnia; Clinical Insomnia - Moderate Severity, and Clinical Insomnia – Severe). The ISI has been validated against both polysomnographic (PSG) and prospective sleep diary measures, and demonstrates convergence with clinical interview criteria. It has proven reliability and validity for distinguishing individuals diagnosed with primary insomnia from normal sleeper controls, with observed Cronbach's alpha co-efficients ranging from 0.74 to 0.78, and sensitivity and specificity values of 94% (Smith & Wegener, 2003).

- The *Morningness-Evening Questionnaire* - *Reduced Form* (MEQ; Horne & Ostberg, 1976) was used to evaluate individual differences in circadian rhythms. It classifies individuals into one of five different chronotypes as follows: Definitely Morning Type; Moderate Morning Type; Neither Type; Moderately Evening Type; Definitely Evening Type. The MEQ has been found to have adequate internal consistency with full-scale Cronbach's alpha coefficients

ranging from 0.78 to 0.83 (Chelminski et al., 2000).

- The *Stanford Sleepiness Scale* (SSS; Hoddes et al., 1973) was administered after completion of the dot-probe task to assess participant's level of alertness at the moment of testing in order to determine whether, should any positive results be observed, these were likely to be due to an actual attention bias effect, or due to poor task performance as a result of sleepiness.

- A brief structured interview schedule targeting essential DSM-IV and ICSD-R diagnostic criteria for PI and DSPS was administered consistently across all participants (Espie, 2002; Morin & Espie, 2003).

- Participants completed a Standard *Sleep Diary* (Espie, 1991), upon wakening for seven days following completion of the dot-probe task. This asks participants for qualitative estimates of sleep e.g. "how long did it take for you to fall asleep last night", in order to determine the participants' subjective Sleep Onset Latency (SOL - length of time, in minutes, taken to fall asleep at night) and Total Sleep Time (TST - total amount of time, in minutes, slept). The usefulness of subjective sleep diary measurement is recognised in the assessment of general insomnia disorder (RDC; Edinger et al., 2004).

- Actiwatch AW4 (Cambridge Neurotechnology) - small, non-intrusive wrist-worn devices – were used to record objective rest/activity periods based on wearer movements in order to determine the sleep/wake (circadian) patterns of participants thought to be presenting with DSPS. Actigraphy was also used to assist with the differential diagnosis of PI and DSPS, confirming group allocation where there was uncertainty regarding an individual's status.

Actigraphy is suggested to provide highly reliable data on an individual's sleep and wake periods (Sadeh et al., 1989; Sadeh et al., 1995). One week of actigraphic and sleep diary monitoring, in addition to clinical interview, has been highlighted as the best method to diagnose circadian sleep phase disorders (Dagon, 2002). Data were downloaded using Actiwatch Sleep Analysis 2002, version 4.04. Van Someren et al's (1999) non-parametric circadian rhythm analysis (NPCRA) was used 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).

# Descriptive and Clinical Measures

The *Hospital Anxiety and Depression Scale* (HADS; Zigmond and Snaith, 1983) – a selfreport questionnaire consisting of 14 four point Likert-scaled items, 7 for anxiety (HADS-A) and 7 for depression (HADS-D) - was administered to aid sample description and the application of inclusion/exclusion criteria. The HADS has been shown to have good internal consistency and test-retest reliability, be sensitive to change, and provide a valid assessment. Factor analytic studies support the 2 factors (anxiety and depression). Cronbach's alpha has been found to range from 0.78 to 0.93 for the HADS-A, and 0.82 to 0.90 for the HADS-D (Bjelland et al., 2002).

## **Procedure and Experimental Protocol**

As stated previously, participants initially contacted the researcher in response to a mass email, providing details of their sleeping patterns based upon a brief set of questions. The researcher then screened and selected the responses that best resembled the traits of the three sleep groups involved in this study (Appendix 2.6). Suitable candidates were then sent an electronic copy of the participant information sheet (Appendix 2.7), outlining the purpose of the study and what participation would involve, and were invited to complete the experimental procedure. On the day of testing, participants were provided with a paper copy of the participant information sheet, and given the opportunity to discuss any questions pertaining to the study. Informed consent was then obtained (Appendix 2.8).

The experimental procedure was conducted in assessment rooms at the Department of Psychology, University of Glasgow, and at the UGSC based at the Southern General Hospital, Glasgow. Efforts were made to standardise the procedure, and sound and illumination levels were controlled. After signing the consent form, participants were asked to sit in a comfortable and relaxed position. All participants were then given the same set of instructions which appeared on the computer screen, and the experimenter gave a verbal description of the task to ensure that it was fully understood. They were informed that pairs of pictures would briefly appear on the screen followed by an asterisk (the dot-probe) in the position formerly occupied by one of the pictures. Participants were instructed to strike the left or right response key as quickly and as accurately as possible to indicate the position of the target.

Following the instructions, participants completed the dot-probe task, which took no longer than 15-20 minutes. First, the participants completed 6 practice trials. Practice pairs contained pictures of non-sleep related objects and were not used in the main body of the task. A rest period followed that terminated when the participant pressed any of the keys on the response pad. Participants then completed the remaining 180 experimental trials and 80 filler trials (comprising neutral picture pairs which were not included in the analyses, see Yiend & Matthews, 2001). Each trial consisted of the following sequence of events (as summarised in

Figure 1): a fixation cross (+) appeared in the centre of the computer screen for 500-ms. Immediately following the offset of the fixation cross, a pair of photographs was presented for 500-ms. After 500-ms, the pictures disappeared and a single-asterisk dot-probe appeared immediately in the location of either the sleep-related or neutral picture. Participants were asked to press one of two keys as quickly and as accurately as possible to indicate the location of the probe (left or right). The probe remained on the screen until the correct response was made. An inter-trial interval of 1000-ms followed each trial.

Stimuli were presented in two blocks of 120 trials each. Blocks were separated with a rest period, which again ended when the participant pressed any key on the response pad. Each of the picture pairs was repeated 4 times during the study - the relative positions (left or right) of each pair of pictures were counterbalanced within the experiment. Repeated stimuli pairs have previously been used in this paradigm without any evidence of priming effects (Mathews, Ridgeway & Williamson, 1996). There was an equal probability that the probe would replace either the positive sleep-related, negative sleep-related or neutral picture. The two blocks were presented in a fixed order to all participants. Trial order within a block was randomly determined for each participant by Superlab Pro to ensure that order effects did not confound results. An illustration of a single trial sequence in the dot-probe task is presented in Appendix 2.9.

After completing the task, each participant was interviewed to evaluate his/her general sleep patterns, using a structured interview format (Espie, 2002; Morin & Espie, 2003). This interview took no longer than 10 minutes. Participants then completed all self-report measures, each taking no more than 5 minutes to complete. All participants then completed a standard

sleep diary for seven nights following the experiment. Participants who resembled the traits of DSPS, or whose status (PI or DSPS) was not clear, were asked to wear an actiwatch for 7 days following the experiment, and their subjective diary responses were later compared with their recorded sleep data from the actigraph. Participants were contacted via email one week later, at which time they were fully debriefed about the purpose of the study and the hypotheses. All participants were emailed a copy of The Good Sleep Guide (Espie, 1994), recommended by the British Sleep Society, and those complaining of either PI or DSPS were given some general advice, based on the Good Sleep Guide, regarding managing their sleep difficulties. Participants within the PI group were subsequently invited to participate in a cognitive-behavioural treatment study (granted full ethical approval) being conducted within the UGSC.

# RESULTS

All analysis was conducted using the Statistical Package for the Social Sciences (SPSS).

# **Participant Characteristics**

## Demographic and Clinical Data

A total of 58 participants (37 females and 21 males), with a mean age of 30.1 years (SD = 8.96) across each of the three groups were included in the overall analyses. ANOVA and posthoc testing with Tukey's Honestly Significant Differences (HSD) test demonstrated that the PI group was significantly older than the DSPS group (Table 2). Subsequent analysis of covariance (ANCOVA) and post hoc testing with Tukey's HSD, with age as a covariate, showed that age significantly predicted participants' scores on the SSS. Age was not, however, found to have a significant effect on any of the other measures. Furthermore, as the study was conducted across two different sites, ANCOVA indicated that test location did not significantly predict participants' scores on any of the demographic or clinical measures. Therefore, ANCOVA for the SSS is reported here. For all other demographic and clinical measures, uncorrected ANOVA are reported.

# Levels of anxiety and depression

ANOVA and Tukey's HSD across the 3 groups revealed significantly higher mean levels of anxiety and depression, as measured by the HADS, in the PI group (9.94 and 5.78 respectively) compared with the DSPS (6.21 and 2.42) and NS (4.0 and 1.05) groups. No statistical difference was observed between the NS and DSPS group on these measures. Fifty per cent (n = 9) of the participants within the PI group presented with a level of anxiety that could potentially warrant clinical intervention (i.e. they satisfied criteria for clinical 'caseness', as indicated by a score of 11 or above on the HADS-A scale). This compares to 15.7% (n = 3) of the DSPS group. None of the NS control group met caseness for anxiety. Given the hypothesised role of psychological factors in the development and maintenance of PI, elevated levels of anxiety within PI relative to normal sleep controls and those whose sleep complaints derive from a circadian timing disorder, is to be expected. These data can therefore be assumed to indicate reliable assessment and diagnosis of participants' sleep status, and subsequent group allocation.

#### Sleep Related Measures

ANOVA and Tukey's HSD revealed that the PI and DSPS groups scored significantly higher than the NS group on both the ISI and the PSQI (Table 2). Individuals within the DSPS group were rated as significantly more 'evening' type than the other 2 groups (lower scores on the MEQ indicate a greater tendency towards 'eveningness'). This finding confirms that the participants within the DSPS group presented with circadian phase delay characteristic of delayed sleep phase syndrome (ICSD-2, *American Academy of Sleep Medicine*, 2005). ANCOVA, with age as a covariate, and Tukey's HSD revealed that individuals with PI were significantly less alert following completion of the dot-probe task, as measured by mean scores on the SSS, than those within the NS group. There were no significant differences in the level of alertness observed between the PI and DSPS, or between the DSPS and NS, groups.

ANOVA for subjective sleep onset latency (SOL) revealed significant differences across the three groups (P < 0.05). Post-hoc testing with Tukey's HSD test confirmed that the PI group reported longer SOL than both the NS and DSPS group; individuals within the DSPS group reported longer SOL than the NS group. ANOVA and Tukey's HSD revealed that the total sleep time (TST) was statistically significantly lower within the PI group compared to the DSPS and NS groups. No difference in TST was observed between the NS and DSPS groups. Subjective sleep diary data therefore confirm participants sleep status and subsequent group allocation.

Actigraphy aided the differential diagnosis between PI and DSPS in individual cases. Analysis of mean L5 (time of onset of lowest five hours of activity) and M10 (time of onset of highest ten hours of activity) was  $3:58 \pm (60.8)$  and  $13:45 \pm (145.8)$  respectively (data are presented as mean 24-h clock times with SD in minutes). These data are indicative of circadian phase delay within individuals within the DSPS group.

#### **Dot-probe Data Analysis**

#### Preparation of Reaction Time Data

Reaction times from filler trials, and trials with errors, were excluded from the analysis. To eliminate outliers, reaction times were excluded if they were less than 200-ms, greater than 2000-ms, or more than 2 S.D. above the mean. (Ratcliff, 1993). As noted earlier, two participants had an error rate that was above a 5% threshold and were therefore excluded from all analyses. As the study was conducted across two different sites, ANCOVA was used to assess the effect of test location on the dot-probe data. ANCOVA indicated that age, mean levels of alertness as measured by the SSS, and test location did not predict dot-probe data. Uncorrected ANOVA confirmed that the 3 groups did not differ significantly [F(2, 56) = .159, p = .853] on error rate (1.02 % of the total data). Figure 1 illustrates that no significant difference was observed between groups on mean overall reaction times to all stimuli combined [F(2, 56) = 1.072, p = .349].

## - Insert Figure 2 Here –

## Analysis to test Hypotheses

Attention bias scores were calculated for each stimulus type relative to neutral stimuli in order to clarify the net effect of the different stimuli types on attention, using the following equation:

#### [(SleepLProbeR + SleepRProbeL) - (SleepLProbeL + SleepRProbeR)]/2

where Sleep = Sleep Related Picture; L = Left and R = Right (e.g. SleepLProbeR represents the mean reaction time when the sleep related picture appears on the left hand side of the computer screen and the probe appears on the right hand side). This equation was first devised by Matthew and MacLeod (1988), and its use is now standard practice within the dot-probe literature (e.g. MacMahon et al., 2006; Bradley et al., 2007).

The attention bias score summarises the interaction between sleep-related picture position and probe position on reaction time, thus providing a measure of the relative speeding of reaction time towards probes that appear in the same location as the sleep-related picture (congruent) compared to response times for a dot-probe that is in the different position to the sleep picture (incongruent). There are four potential combinations of dot-probe and sleep picture as denoted by the equation. Consequently, it is necessary to take account of all four potential combinations, whilst calculating a difference in reaction time towards a dot-probe that is in the same position as a sleep-picture and a dot-probe that is in a different position to the sleep picture (i.e. the difference between incongruent and congruent presentations). This difference is obtained by dividing the above equation by two. Positive values represent vigilance towards sleep-pictures relative to neutral pictures; negative values represent avoidance (MacMahon et al., 2006).

#### Hypothesis 1

It was predicted, a priori, that participants within the PI group would demonstrate a greater degree of attention bias towards *all* sleep-related stimuli (negative and positive combined) relative to neutral stimuli, as measured by faster dot-probe reaction times, whereas those with DSPS and NS would not. Mean and SD attention bias scores for sleep relative to neutral stimuli were  $+2.1 \pm (9.5)$  for the PI group;  $-2.3 \pm (10.1)$  for the DSPS group; and  $-4.3 \pm (12.3)$  for the NS group (Figure 2). Orthogonal planned contrasts on mean attention bias scores, with

an assumption of equal variances, did not support this prediction (Table 3).

Visual inspection of mean attention bias scores (Figure 2) suggests a degree of differential vigilance towards sleep relative to neutral stimuli by the PI in comparison to the NS group. Furthermore, the p value observed from the orthogonal planned contrast between these two groups was .08. A calculation based on observed means and standard deviations yielded an effect size (*Cohen's d*) of .58 (Table 3). A retrospective power calculation (based on the significance level obtained, the actual differences observed, and the original sample size) was therefore performed to investigate the extent to which the present sample size may have influenced this result. This analysis indicated that an additional 11 participants across the two (PI and NS) groups would be required for a statistically significant difference in attention bias towards sleep relative to neutral stimuli to be observed.

Given that 50% of the PI group presented with a level of anxiety that met clinical caseness, a Pearson product-moment correlation was calculated between attention bias scores for negative and positive sleep stimuli combined and HADS-A scores to establish whether there was an underlying association across all participants. No correlation was observed (r = 0.10; p = .449).

# - Insert Figure 2 Here –

## Insert Table 3 Here –

# Hypothesis 2

It was also predicted, a priori, that participants within the PI group would show greater

attentional bias than those with DSPS and NS towards negative (threatening) sleep-related pictorial stimuli, as measured by faster dot-probe reaction time, in comparison to neutral pictures. Mean and SD attention bias scores for negative sleep photos within the PI group were  $+ 3.2 \pm (8.8)$ ;  $-0.9 \pm (7.7)$  for the DSPS group; and  $-1.2 \pm (7.3)$  for the NS group (Figure 5). Orthogonal planned contrasts on mean attention bias scores, with an assumption of equal variances, did not support this prediction (Table 3). For differences between mean attention bias scores for negative sleep stimuli between the PI and NS groups, an effect size of .54 was observed. A retrospective power calculation indicated that, for a statistical difference in the degree of attention bias towards negative sleep relative to neutral stimuli between the PI and NS groups to be observed, an additional 7 people across the two groups would be required.

## -Insert Figure 5 Here -

# Hypothesis 3

It was also predicted that participants with PI would show greater attention bias than those with DSPS and NS towards positive (craving) sleep-related pictorial stimuli in comparison to neutral pictures. Mean and SD attention bias scores for the positive sleep pictures were  $-.02 \pm (5.5)$  for the PI group;  $-1.4 \pm (4.8)$  for the DSPS group and  $-0.6 \pm (4.33)$  for the NS group (see Figure 5). Orthogonal planned contrasts on mean attention bias scores, with an assumption of equal variances, did not support this prediction (Table 3). Given the low p value and *Cohen's d* effect sizes observed (Table 3) it was not thought appropriate to perform further retrospective power calculations for these data.

# Attention bias for negative compared to positive sleep stimuli

To investigate whether there was any difference in the magnitude of attention bias shown

towards negative and positive sleep stimuli, a paired sample t-test, using the mean attention bias scores observed for the PI group, was performed. The difference observed was non-significant [t(-1.201) df 17, p = .246, ns]. Given this non-significant result within the experimental group, no further analysis between stimuli valence within the other sleep groups was conducted.

## DISCUSSION

Recent cognitive models (Harvey, 2002; Espie, 2006) implicate selective attention or 'attention bias' towards sleep-related stimuli in the development and/or maintenance of PI, although the precise psychological mechanisms that underlie this attention bias remain unclear. This study employed the dot-probe paradigm to investigate the role of two processes threat and craving - which have been proposed to modulate sleep-related attention bias in PI. In line with findings from previous studies (e.g. Marchetti et al., 2006; MacMahon et al., 2006), it was hypothesised that, compared with NS and DSPS, people with PI would demonstrate greater attention bias towards sleep-related (regardless of emotional valence) relative to neutral pictorial stimuli. Visual inspection of mean attention bias scores for all sleep stimuli (negative and positive combined) revealed an overall trend towards greater sleeprelated attention bias in PI relative to NS controls, with a moderate effect size. However, orthogonal planned contrasts on mean attention bias scores did not confirm this trend statistically. Therefore, no statistically significant difference in attention bias for sleep-related relative to neutral pictorial stimuli in PI compared with NS (or DSPS) was observed in this study.

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It was also predicted that PI would demonstrate sleep-related attention bias at the level of both threat (negative sleep stimuli) and craving (positive sleep stimuli). As both threat and craving are proposed to drive attention bias in PI, no a priori hypothesis regarding any difference in the magnitude of the attention bias effect generated by negative relative to positive sleep pictures was made. Visual inspection of mean attention bias scores revealed a possible trend toward greater attention bias for negative sleep stimuli relative to neutral stimuli within PI compared with both the NS and DSPS groups, with moderate effect sizes. However, orthogonal planned contrasts on mean attention bias scores across groups for negative sleep stimuli did not confirm this trend statistically. Therefore, there was no statistically significant difference in attention bias for negative sleep stimuli relative to neutral stimuli within PI compared to either NS or DSPS.

With regard to positive sleep stimuli, visual inspection of the data, analysis of the effect size observed, and orthogonal planned contrasts, revealed no evidence to support the hypothesis that PI would show greater sleep-related attention bias towards positive relative to neutral sleep stimuli in comparison to the NS or DSPS group. In addition, no statistically significant differences in attention bias towards negative relative to positive sleep stimuli within PI compared to DSPS and NS were observed.

Statistically, therefore, the results of the present study fail to replicate the findings of previous studies that provide evidence for the presence of sleep-related attentional bias in PI relative to NS and DSPS, using similar computerised tasks adapted from cognitive experimental psychology, (e.g. Jones et al., 2005; Marchetti et al., 2006; MacMahon et al., 2006;

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Speigelhalder et al., in press). Subsequently, no definitive conclusions regarding the potential role of threat and craving in driving attention bias proposed by recent cognitive models of PI (Harvey, 2002; Espie et al., 2006) may be drawn from the findings presented here.

However, there are certain limitations to the study that might explain the present findings. Specifically, there is evidence to suggest that the current sample size might lack sufficient power for the hypothesised attention bias effects to be detected. As stated, visual inspection of the data suggested a possible trend towards increased attention bias for sleep-related stimuli (regardless of valence) in PI relative to NS. Post-hoc power analysis confirmed that a relatively small number of additional participants across the two groups (n = 11) would have been required for a statistically significant sleep-related attention bias effect to have been observed. Similarly, visual inspection of the data suggested a possible trend for attention bias towards negative sleep stimuli in PI compared to both the NS and the DSPS groups. Post hoc power analysis again indicated that a relatively small increase in the number of participants across the groups (n = 7) would be required to achieve a statistically significant attention bias effect.

The findings of the post-hoc power analysis reported here fit well with the a priori power estimate specified during the initial design and conceptualisation phase of the study, which indicated that, using a pictorial dot-probe paradigm, 21 participants in each of the three groups would be required to detect a statistically significant effect at an alpha level of 0.5, with a power of 0.8 (1-tailed) (Mogg and Bradley, 1999). Further evidence that the present study is perhaps underpowered may be derived from the previous dot-probe study conducted by

MacMahon et al., (2006), who observed a significant attention bias effect for sleep relative to neutral stimuli (Effect Size = 0.67) with 21 and 20 participants within the PI and NS groups respectively. Furthermore, visual inspection of the mean attention bias scores obtained from the MacMahon et al., (2006) study, as presented in Figure 4, reveals an overall pattern of results similar to those obtained in the present study (Figure 2). These data therefore suggest that the present study should be replicated using a larger sample size.

## - Insert Figure 4 Here -

The overall power of the study to uncover the predicted attention bias effects in PI relative to NS and DSPS may also be limited with regard to the sensitivity of the dot-probe task, specifically in relation to the nature of the stimuli employed. MacMahon et al., (2006) proposed that subsequent sleep-related attention bias studies using the dot-probe task employ pictorial rather than word stimuli. This recommendation was borne from evidence within the anxiety literature that suggests that, although considered a reasonably pure measure of attention (Mogg and Bradley, 1999), the dot-probe task may also be regarded 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). Within the attention bias literature, it is generally thought that the use of pictorial stimuli in attention bias tasks may maximise the saliency of the stimuli, thereby increasing the likelihood of detecting processing biases, whilst simultaneously maintaining the degree of experimental control offered by the dot-probe (Mogg and Bradley, 1999).

This is therefore the first sleep-related attention bias study to use complex pictorial stimuli, with contrasting emotional and cognitive content. The sleep-related stimuli used in the task are

novel, and therefore not previously proven to be of sufficient salience to elicit attention bias in individuals with PI. Furthermore, it was highlighted from the outset that it may be difficult to represent sleep and sleeplessness, and its associated cognitive-emotional factors, pictorially. However, the content of the pictures was carefully generated from themes within cognitive scales and audio diary transcripts stemming from research that investigated the 'live' cognitions, both in relation to good and poor quality sleep, reported by individuals with insomnia (e.g. Kyle et al., 2008; Wicklow and Espie, 2000). In addition, the final photographs used in the dot-probe task were selected on the basis of ratings provided by multiple independent raters for emotional valence and association with sleep. The matching of sleep stimuli with neutral stimuli into picture pairs to be presented within the dot-probe task was also stringently conducted, as described in the methods section. Therefore, a robust, prospectively planned procedure was adhered to when developing the stimuli.

An important observation is that, compared with negative sleep stimuli, the potential to encapsulate positive or 'good quality' sleep pictorially in a variety of novel ways was somewhat restricted. As stated previously, because the daytime consequences associated with poor and good sleep are prominent in the self-report of people presenting with insomnia, each sleep-stimuli set comprised photographs representing sleep quality associated with both night and day-time effects. For example, many of the positive sleep 'day' photos depicted models sitting at a desk, looking alert and functioning to a high capacity. The converse was depicted within the negative, poor sleep photograph set. It is possible, therefore, that the stimuli representing the association between good sleep and subsequent daytime functioning may not have been salient enough to be processed as such by people with PI. Stimuli intended to depict daytime functioning associated with good sleep may potentially have been perceived as neutral in content and valence, and therefore not sufficiently representative of 'craving'. Taken as a whole therefore, it might be possible that the positive stimuli set may not have been perceived to be as strongly associated with sleep and sleep quality as the negative sleep pictures. However, as the sleep stimuli were generated from research identifying the actual cognitions of people with insomnia, it was anticipated that they would be selectively processed by individuals with attention bias to sleep-related stimuli.

If relevant, this potential difficulty in representing the cognitive and emotional markers associated with good quality sleep may, at least partially, explain why the present findings did not support the predicted attention bias for positive sleep stimuli relative to neutral stimuli, and the lack of any trend towards this effect being evident within the data. However, as reported, all sleep photos were selected on the basis of valence ratings provided by independent raters. Consideration had been given to conducting a secondary analysis to compare the attention bias effect generated by 'sleep-day' versus 'sleep-night' stimuli, which may have perhaps facilitated further investigation into the possibility of the inclusion of sleep pictures that may not have sufficiently represented craving. However, given the non-significant attention bias effect within PI for all sleep-stimuli combined, this analysis was not thought appropriate.

A final point regarding the nature of the stimuli used relates to the fact that all sleep-related photographs were developed by the researcher using the same group of models and locations throughout, and were therefore fairly rigidly standardised. A potential limitation therefore might be that, although the neutral stimuli were certainly non-sleep related and neutral in emotional valence, and were matched in terms of content, image composition and other relevant factors as closely as possible, they may, as a whole, represent a comparatively heterogeneous stimuli set.

A strength of the study identified from the demographic and clinical data is that the groups are well defined, that is, the specified inclusion/exclusion criteria have been both appropriate and adhered to, thus helping to ensure good discrimination on the independent variable, and reducing the possibility that the non-significant results observed in this study are due to any overlap between groups. Although the PI group was significantly older than the DSPS group, and significantly less alert, as measured by scores on the SSS, these potentially confounding variables were appropriately controlled for in the statistical analyses, with neither age nor level of alertness found to predict the dot-probe data.

A further potential strength is that there was no significant difference in overall mean speed of reaction time to all stimuli combined across the three groups. This differs from the results from MacMahon et al., (2006) who observed that the PI group responded significantly faster towards all stimuli compared to the NS group, who in turn responded more quickly to all stimuli than the DSPS group. This finding was attributed to potentially elevated levels of arousal in the PI group, a feature that has been found in other studies examining the physiological features of PI (e.g., Bonnet and Arand, 2003). This finding therefore again raises the issue regarding the potential role of attention bias in the development and maintenance of

PI. Although the replication of attention bias data across the studies conducted to date highlights the stability of the phenomenon in PI, it remains unclear whether it has an aetiological role in PI, or whether it is merely an associative factor. It might therefore be argued that this study controls for cognitive hyperarousal more effectively than the MacMahon et al., (2006) study, in which it was proposed that both increased cortical arousal and attention bias may both be implicated in the development and maintenance of PI, and may even work in tandem. The absence of hyperarousal within the PI group employed in this study could potentially be explained by their increased scores on the SSS, indicating a general reduction in alertness. However, the SSS did not predict the dot-probe data. In addition, the PI group in the MacMahon et al., (2006) study was found to be less alert as measured by the SSS, yet, as stated, their overall faster reaction times towards all stimuli combined, compared to the other two groups, were indicative of hyperarousal. It therefore seems that it would be prudent for future studies to specifically control for any bias associated with hyperarousal when investigating selective attention for sleep-related stimuli in PI.

Although this study failed to support the predicted attention bias effects, contradictory findings have been observed in other fields of interest. For example, Mogg et al., (2002), using the dot-probe task, failed to find evidence of attention bias towards exam-related stimuli within individuals with high-trait anxiety when testing was conducted outwith the exam period. However, an attention bias effect was again observed when the exam period began. This finding raises the possibility that the predicted significant attention bias effects might have been observed in the present study had the participants been tested at a time when sleep loss was more salient, for example, during the early hours of the morning, or prior to important events where performance was crucial (MacMahon et al., 2006). Instead,

participants were tested at various times of the day; there was no cost attached to poor performance on the dot-probe task, which may have reduced the saliency of their sleep difficulties (MacMahon et al., 2006). Future studies might therefore give consideration to testing participants at a time when sleep loss is likely to be more salient. To this end, MacMahon et al., (2006) suggest that future studies schedule the time of testing to concur with individuals circadian rhythms/sleep-wake patterns. However, this would necessitate actigraphic assessment of the sleep-wake schedule of all participants prior to their completion of the dot-probe task, which could increase the chance of priming participants to sleep-related stimuli, and it was not possible to incorporate this into the design of the present study due to practical and theoretical reasons. Nevertheless, this approach should be considered when developing future sleep-related attention bias studies.

Testing participants when the saliency of their sleep difficulties is most pertinent makes conceptual sense when considered within the context of the Motivational Theory of Current Concerns (Cox et al., 2006). As highlighted in the introduction, an individual's current concerns will make him/her particularly sensitive to cues associated with valued goals, or with the means of obtaining these goals. Research has shown a close relationship between current concerns and greater responsivity towards salient cues (Klinger, 1996). This is relevant to the investigation of both threat and craving. This study used a sample drawn mainly from the university student population. Students arguably have greater flexibility to adjust their daily schedules to accommodate a disrupted sleep pattern than do those in full-time employment. Difficulty sleeping at night and subsequent impairment in daytime functioning may therefore be experienced as less threatening - i.e. less of a current concern - and attract less attention

within both the PI and DSPS groups, than those in regular employment (MacMahon et al., 2006). Future studies investigating the role of threat in mediating sleep-related attention bias might therefore include a measure of concern about sleep, such as the Anxiety and Preoccupation about Sleep Questionnaire (ASPQ; Tang, 2003) to investigate any possible associations between level of concern and performance on the dot-probe task.

As stated before, one of the strengths of the present study is that the groups are well defined, thus ensuring the discrimination of the independent variable; participants met diagnostic criteria for PI, and the mean ISI and PSQI scores were well above the mean for the DSPS and NS groups. With regard to the theory of current concerns, it might be argued that the predicted attention bias effects would have been obtained had the sample been drawn from a clinical or 'treatment seeking' population where threat (of sleeplessness) and craving (for sleep – 'sleepiness') might have been more salient. However, as stated, previous sleep-related attention bias studies have repeatedly used similar recruited samples and successfully found a positive attention bias effect.

Similarly, the concept of current concerns may be relevant to the study conducted by Field et al., (2004), which failed to find an overall attention bias effect for cannabis related stimuli amongst cannabis users. However, there was wide variation in the frequency of cannabis use within their sample, ranging from 'very occasional' to 'very heavy'. A significant positive correlation between 'very heavy' cannabis use and attention bias was observed. The authors argued therefore that attention bias would have been detected in the cannabis user group as a whole if only heavy cannabis users had been used. As stated previously, further work to illustrate a clear and direct link between PI and attention bias is still required. It is

recommended therefore that future sleep-related attention bias studies investigate whether there are any associations between the severity of insomnia and sleep-related attention bias. This approach might foster greater clarity as to whether attention bias is intrinsic to the development or maintenance of PI, as proposed by the AIE Pathway (Espie et al., 2006), and not merely an associative factor, and perhaps shed light on the psychological predictors and correlates that influence the transition between acute and chronic insomnia.

# Summary of main conclusions and recommendations for future research.

In summary therefore, the present study failed to yield statistically significant results necessary to conclusively confirm the hypotheses generated and replicate the previous findings cited. As highlighted, this may be attributable to limitations in the power of the study to detect the anticipated attention bias effects. As a consequence, the role of threat and craving in driving attention bias in PI requires further investigation.

Replication of this study, using an increased sample size (perhaps drawn from a clinical, treatment-seeking population), in addition to further work to refine the pictorial stimuli used, is recommended. Consideration should be given to exploring the effects of time of day at which attention bias tasks are administrated, and how this concurs with individuals specific sleep-wake patterns, in order to increase the saliency of threat/craving associated with sleep and sleeplessness.

As PI comprises cognitive and physiological components, and given that reaction time differentials on attention bias tasks are considered to be an indirect measure of attention bias, the use of psychophysiologic measures of attention, such as heart rate (Broomfield and Turpin,

2005) or eye-tracking (Field et al., 2004) might provide further insight into the role of threat and craving in the development and maintenance of PI. In particular, research on facial EMG activity in response to different emotional stimuli has demonstrated increased corrugator (frown) muscle activity in response to negatively valenced stimuli. Similarly, increased zygomatic (cheek) muscle activity has been observed in response to positively valenced pictures (Hubert, de Jong-Meyer, 1991). A future study might investigate whether emotional arousal, as measured by facial EMG activity in response to positive and negative sleep-related pictures, produces a similar pattern of results observed in the attention bias data, and whether a relationship between these two variables could be detected. This would be achieved by simultaneously recording facial EMG activity whilst participants complete the dot-probe task.

The implication of attention bias in treatment has been explored in relation to anxiety and substance disorders. Studies using the emotional Stroop task demonstrate that individuals with emotional disorders show reduced Stroop interference by disorder-related words following successful treatment (e.g. Mattia et al., 1993; Mathews et al., 1995). By analogy, decreased attention bias towards sleep-related stimuli might be observed following successful treatment of insomnia. Furthermore, there may be potential for using attention bias in the treatment of insomnia itself, through desensitisation (Spiegalhalder et al., 2008). This 'cue exposure' to threatening stimuli is well established in the treatment of anxiety disorders, and there is growing interest in its efficacy for reducing craving and relapse in substance misuse (Loeber et al., 2006). Research to further understand the attention bias phenomena in PI may therefore be crucial for the development of targeted therapeutic interventions.

# References

Alvarez, B., Dahlitz, M.J., Vignau, J., and Parkes, J.D. The delayed phase sleep phase syndrome: clinical and investigative findings in 14 subjects. *J. Neurol. Neurosurg. Psychiatry*, 1992, 55: 665-70.

Beck, A.T. Depression: Causes and Treatment. University of Pennsylvania Press, Philadelphia, 1967.

Beck, A.T. and Clark, D.A. An information processing model of anxiety: automatic and strategic processes. *Behav. Res. Ther*, 1997, 35: 49–58.

Bjelland, I., Dahl, A.A., Haug, T.T. and Neckelmann, D. The validity of the hospital anxiety and depression scale. An updated literature review. *J. Psychosom. Res*, 2002, 52(2): 69-77.

Bonnet, M.H. and Arand, D.L. Insomnia, metabolic rate and sleep restoration. J. Intern. Med, 2003, 254: 23-31.

Bradley, B.P., Mogg, K. and Lee, S. Attentional biases for negative information in induced and naturally occurring dysphoria. *Behav. Res. Ther*, 1997, 35: 911–27.

Bradley, B.P., Mogg, K. and Millar, N. H. Covert and overt orienting of attention to emotional faces in anxiety. *Cog. and Emotion*, 2000, 14 (6): 789-808.
Bradley, B.P., Mogg, K., Wright, T. and Field, M. Attentional bias in drug dependence: vigilance for cigarette-related cues in smokers. *Psychol. Addict. Behav*, 2003, 17: 66–72.

Broomfield, N.M. and Turpin, G. Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. *Biol. Psychol*, 2005, 68: 179-200.

Bryant, R.A. and Harvey, A.G. (1995). Posttraumatic stress in volunteer fire fighters: Predictors of distress. J. Nervous and Mental Disease, 1995, 183: 267–271.

Buysse, D.J., Reynolds, C.F., Monk, T.H., Berman, S.R, and Kupfer, D.J. The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Res*, 1989, 28: 193-2 I3.

Cedrus Corporation, SuperLab 4. (Stimulus Presentation Software), San Pedro, CA (2006).

Chelminski, T.V., Petros, J.J. and Ferraro, F.R. Psychometric properties of the reduced Horne and Östberg questionnaire. *Personality and Indiv. Difference*, 2000, 29 (3): 469–478.

Cooper, M.J. and Fairburn C.G. Thoughts about eating, weight and shape in anorexia nervosa and bulimia nervosa. *Behav. Res. Ther*, 1992, 30(5): 501-11.

Cox, W.M., Fadardi, J.S. and Pothos, E.M. The Addiction-Stroop test: theoretical considerations and procedural recommendations. *Psychol. Bulletin*, 2006, 132: 443-476.

Dagan, Y. Circadian rhythm sleep disorders (CRSD). *Sleep Med. Reviews*, 2002, 6 (1): 45–54.

Edinger, J.D., Bonnet, M.H., Bootzin, R.R., Doghramji, K., Dorsey, C.M. and Espie, C.A. Derivation of research diagnostic criteria for insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep*, 2004, 27: 1567–96.

Ehrman, R.N., Robbins, S.J., Bromwell, M.A., Lankford, M.E., Monterosso, J.R. and O'Brien, C.P. Comparing attentional bias to smoking cues in current smokers, former smokers, and non-smokers using a dot-probe task. *Drug Alcohol Depend*, 2002, 67: 185–191

Espie, C.A., Brooks, D.N. and Lindsay, W.R. An evaluation of tailored psychological treatment of insomnia. *J. Behav. Ther. Exper. Psychiatry*, 1989, 20: 143-467.

Espie, C.A. The Psychological Treatment of Insomnia. Wiley, Chichester, 1991.

Espie, C.A. *The Good Sleep Guide*. Guidance Report for the National Medical Advisory Committee, *1994*.

Espie, C.A. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Ann. Rev. Psychol*, 2002, 53: 215–43.

Espie, C.A., Broomfield, N.M., MacMahon, K.M.A., MacPhee, L.M. and Taylor, L.M. The Attention-Intention-Pathway in the development of psychophysiologic insomnia: A theoretical

Fichten, C.S., Libman, E., Creti, L., Amsel, R. and Tagalakis, V. (1998). Thoughts during awake times in older good and poor sleepers: the self-statement test: *Cognit Ther. Res*, 1998, 22: 1–20.

Field, M., Mogg, K. and Bradley, B.P. Cognitive bias and drug craving in recreational cannabis users. *Drug and Alcohol Depend*, 2004, 74: 105-111

Field, M., Mogg, K. and Bradley, B.P. Eye movements to smoking related cues: effects of nicotine deprivation. *Psychopharmacol*, 2004; 173: 116-23

Fox, E., Russo, R. and Dutton, K. Attentional bias for threat: evidence for slowed disengagement from emotional faces. *Cogni. Emotion*, 2002, 16: 355–79.

Harvey A.G. A cognitive model of insomnia. Behav. Res. Therapy, 2002, 40: 869-93.

Harvey K.J. and Espie C.A. Development and preliminary validation of the Glasgow content of thoughts inventory (GCTI): A new measure for the assessment of pre-sleep cognitive activity. *Br. J. Clin. Psychol*, 2004, 43: 409–20

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R. and Dement, W.C. Quantification of sleepiness: a new approach. *Psychophysiology*, 1973, 10: 431-436.

Horne J.A. and Ostberg O. A self-assessment questionnaire to determine morningness and eveningness in human circadian rhythms. *Internat. J. Chronobiol*, 1976, 4(2): 97-110.

Hubert W, de Jong-Meyer R. Psychophysiological response patterns to positive and negative film stimuli. *Biol. Psychol*, 1991, 31(1): 73-93.

Jones B.T., MacPhee, L.M., Broomfield, N.M., Jones, B.C. and Espie, C.A. Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. *J. Abnorm. Psychol*, 2005, 114: 249–58.

Klinger, E. Emotional influences of cognitive processing, with implications for theories of both. In: P.M. Gollwitzer and J.A. Burgh (Eds), *The Psychology of Action: Linking Cognition and Motivation to Behaviour*. Guilford Press, New York, 1996.

Kyle, S.D., Espie, C.A. and Morgan, K. A qualitative analysis of daytime functioning and quality of life in persistent insomnia using focus groups and audio-diaries. *Sleep*, 2008, Abstract suppl., 31, 243-244.

Lang, P.J., Bradley, M.M. and Cuthbert, B.N. *The International Affective Picture System* (*IAPS*) *Digitized Photographs*, The Center for Research in Psychophysiology, University of Florida, Gainesville, FL (1999).

Loeber., S, Croissant, B., Heinz, A., Mann, K. and Flor, H. Cue exposure in the treatment of alcohol dependence: Effects on drinking outcome, craving and self-efficacy. *Brit. J. of Clin. Psychol*, 2006, 45 (4): 515-529.

Lundh L.G., Froding, A., Gyllenhammer, L., Broman, J.K. and Hetta, J. Cognitive bias and memory performance in patients with persistent insomnia. *Scand. J. Behav. Ther*, 1997 26: 27–35.

Lusher, J., Chandler, C. and Ball, D. Alcohol dependence and the alcohol Stroop paradigm: evidence and issues. *Drug Alcohol Depend*, 2004, 75: 225–31.

MacLeod, C.M., Mathews, A. and Tata, P. Attentional bias in emotional disorders. *J. Abnorm. Psychol*, 1986, 95: 15–20.

MacMahon, K.M.A., Broomfield, N.M. and Espie, C.A. Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome. *Sleep*, 2006, 29(11): 1420-1427.

Marchetti, L.M., Biello, S.M., Broomfield. N.M., MacMahon, K.M.A., and Espie, C.A. Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiologic insomnia, delayed sleep phase syndrome and good sleepers using the Induced Change Blindness paradigm. *J. Sleep. Res*, 2006, 15: 1–10.

Matthews, A. and MacLeod, C. Cognitive approaches to emotion and emotional disorders. *Annu. Rev. Psychol.* 1994, 45: 25–50.

Matthews, A., Mogg, K., Kentish, J. and Eysenck, M. Effect of psychological treatment on cognitive bias in generalized anxiety disorder. *Behav. Resear. Ther*, 1995, 33: 293-303.

Matthews, A., Ridgeway, V. and Williamson, D. Evidence for attention to threatening stimuli in depression. *Behav. Res. Ther*, 1996, 34: 695-705.

Mattia J.I, Heimberg, R.G. and Hope D.A. The revised Stroop color-naming task in social phobics. *Behav. Res. Ther*, 1993, 31(3): 305-13.

Mogg, K., Bradley, B.P., Millar, N. and White, J. Cognitive bias in generalised anxiety disorder: A follow-up study. *Behav. Res. Ther*, 1995, 33: 927–35.

Mogg, K. and Bradley, B.P. Some methodological issues in assessing attentional biases for threatening faces in anxiety: a replication study using a modified version of the probe detection task. *Behav. Res. Ther*, 1999, 37(6): 595-604.

Mogg, K. and Bradley, B.P. Biases in eye movements to threatening facial expressions in generalized anxiety disorder. *J. Abnorm. Psychol*, 2000, 109(4): 695-704.

Mogg, K., McNamara, J., Powys, M., Rawlinson, H., Seiffer, A. and Bradley, B.P. Selective

attention to threat: A test of two cognitive models of anxiety. *Cog. and Emotion*, 2000, 14 (3): 375-399.

Mogg, K., Bradley, B.P., Field, M. and De Houwer, J. Eye movements to smoking-related pictures in smokers: relationship between attentional biases and implicit and explicit measures of stimulus valence. *Addiction*, 2003, 98: 825–836.

Mogg, K., Phillipot, P. and Bradley, B.P. Selective attention to angry faces in clinical social phobia. *J. Abnorm. Psychol*, 2004, 113(1): 160-165.

Mogg, K., Field, M. and Bradley, B.P. Attentional and approach biases for smoking: an investigation of competing the theoretical views of addiction. *Psychopharmacol*, 2005, 180(2): 333-41.

Morin CM. Insomnia: Psychological Assessment and Management. Guilford Press, New York, 1993.

Morin, C.M., Stone, J., Trinkle, D., Mercer J. and Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol. Aging*, 1993, 8: 463-7.

Morin, C. M. and Espie, C. A. A Clinical Guide to Assessment and Treatment. Kluwer Academic/Plenum Publishers, New York, 2003.

Neitzert-Semler, C. and Harvey, A.G. An Investigation of monitoring for sleep-related threat in primary insomnia. *Behav. Res. Ther*, 2004, 42: 1403–20.

Neitzert-Semler, C. and Harvey, A.G. Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? *Behav. Sleep Med.* 2006, 4: 85–103.

Pavlova, M., Berg, O., Gleason, R., Walzer, F., Roberts, S. and Regestein, Q. Self-reported hyperarousal traits among insomnia patients. *J. Psychosom. Res*, 2001, 51: 435-41.

Posner, M.I. Orienting of attention. Q. J. Exp. Psychol, 1980, 32: 3-25.

Ratcliff, R. Methods for dealing with outliers. Psychol. Review, 1993, 114: 510-532.

Ryan, F. Attentional bias and alcohol dependence: a controlled study using the modified Stroop paradigm. *Addict. Behav*, 2002, 27: 471–482.

Sadeh A., Alster J., Urbach D. and Lavie P. Actigraphically based automatic bedtime sleepwake scoring: validity and clinical applications. *J. Ambulat. Monit*, 1989, 2: 209–216.

Sadeh A., Hauri P.J., Kripke D.F. and Lavie P. The role of actigraphy in the evaluation of sleep disorders. *Sleep*, 1995, 18: 288–302.

Salkovskis, P.M. Understanding and treating obsessive-compulsive disorder. *Behav. Res. Ther*, 1999, suppl 37: 29-52.

Smith, M.T. and Wegener, S.T. Measures of sleep: The insomnia severity index, medical outcomes study (MOS) sleep scale, Pittsburgh sleep diary (PSD), and Pittsburgh Sleep quality index (PSQI). *Arthritis Care and Res*, 2003, 49(5): 184-196.

Spiegalhalder, K., Espie, C.A., Nissen, C. and Riemann, D. Sleep-related attentional bias in patients with primary insomnia compared with sleep experts and healthy controls. *J. Sleep. Res*, 2008, 17: 191-196.

Spiegelhalder K, Espie, C.A. and Riemann, D. Is sleep-related attentional bias due to sleepiness or sleeplessness? *Cog. and Emotion* (in press).

Tang, N.K.J. The Anxiety and Preoccupation about Sleep Questionnaire. In Distorted perception of sleep in insomnia: phenomenology, mechanisms and interventions. *Unpublished Doctoral Dissertation*, 2003.

Tang, N.K.J., Harvey, A.G., and Schmidt, D.A. Sleeping with the enemy: clock monitoring in the maintenance of insomnia. *J. Behav. Ther. Exp. Psychiatry*, 2007, 38: 40-53.

Taylor, L.M, Espie, C.A. and White C.A. A comparative experimental investigation of Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behav. Sleep Med*, 2003, 1: 200–12.

Van Someren, E.J.W., Swabb, D.F., Colenda, C.C., Cohen, W., McCall, W.V. and Rosenquist, B. Bright light therapy: improved sensitivity to its effects on rest-activity rhythms in alzheimer patients by application of non-parametric methods. *Chronobiol. Int*, 1999, 16: 505–518.

Wicklow, A. and Espie, C.A. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav. Res. Ther*, 2000, 38, 679-693.

Williams, J.M.G., Watts. F.N., MacLeod. C. and Mathews, A. Cognitive Psychology and Emotional Disorders. Wiley, New York, 1997 (second edition).

Yiend, J. and Mathews, A. Anxiety and attention to threatening pictures. Q. J. Exp. Psychol. Hum, 2001, 54: 665-81.

Zigmond A.S. and Snaith, R.P. The hospital anxiety and depression scale. *Acta Psychiatry Scand*, 1983, 67: 361-70.



Figure 1: Overall mean reaction times for the PI, DSPS and NS groups for all stimuli.

Figure 2: Mean attention bias scores for PI, DSPS and NS groups for all sleep stimuli relative to neutral stimuli



Figure 3: Mean attention bias scores for PI, DSPS and NS groups for both positive and negative sleep stimuli.







**Table 1**—Valence ratings for Negative (Threat) and Positive (Craving)

 Sleep Stimuli

Valence	Ratings
Negative Stimuli	$6.72 \pm 0.68$
Positive Stimuli	$6.51 \pm 0.43$
Data are presented as mean	± SD.

**Table 2**—Demographic, clinical and sleep characteristics for participants within the PI, DSPS and GS groups

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clock

Data are presented as mean  $\pm$  SD. SOL and TST are presented as minutes.

PI refers to Psychophysiological Insomnia; DSPS, delayed sleep-phase syndrome; NS, normal sleeper; HADS-A, Hospital Anxiety and Depression Scale, Anxiety subscale; HADS-D. Hospital Anxiety and Depression Scale, Depression subscale, PSQI, Pittsburgh Sleep Quality Index; ISI, Insomnia Severity Index, MEQ, Horne and Ostberg Morningness-Eveningness Questionnaire; SSS, Stanford Sleepiness Scale; SOL, sleep onset latency; TST, total sleep time.

\*Indicates significant (p < .05) difference between PI and DSPS with Tukey posthoc Test ‡Indicates significant (p < .05) difference between PI and NS with Tukey posthoc Test †Indicates significant (p < .05) difference between DSPS and NS with Tukey posthoc Test

Table 3—Orthogonal Planned Contrasts, per hypothesis, on mean attention bias scores			
<u>Hypothesis 1</u> All Sleep relative to Neutral Stimuli	t <sub>(2,55)</sub>	p Value (1-tailed)	Effect Size (ES) (Cohen's d)
PI contrasted with NS	1.41	.08	0.58
PI contrasted with DSPS	.94	.18	0.44
DSPS contrasted with NS	.45	.33	0.17
<u><b>Hypothesis 2</b></u> Negative Sleep relative to Neutral Stimuli			
PI contrasted with NS	1.48	.07	0.54
PI contrasted with DSPS	1.34	.09	0.49
DSPS contrasted with NS	.11	.46	-0.04
<u><b>Hypothesis 3</b></u> Positive Sleep relative to Neutral Stimuli			
PI contrasted with NS	.19	.43	0.23
PI contrasted with DSPS	.44	.33	0.08
DSPS contrasted with NS	27	.39	-0.18
Data are presented as actual difference obse overall Effect Size (Cohen's d).			
PI refers to psychophysiological insomnia; l sleepers	USPS, delay	ed sieep-phase syndro	ome, and NS, normal

# **Chapter 3: Advanced Practice 1: Reflective Critical Account**

# Empowerment - A Two-way Street: Reflections on an Initial Assessment with a Client with a Severe and Enduring Mental Illness

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Submitted in partial fulfilment of the requirements of the degree of Doctorate in Clinical Psychology (DClinPsy)

### ABSTRACT

The promotion of *Empowerment*, defined as "gaining the ability to achieve the highest personal aspirations and goals" (Robbins, Chatterjee, & Canda, 1998), can be viewed as the very essence of clinical psychology practice, particularly with vulnerable client groups. This account outlines reflections upon my clinical practice, based upon an initial assessment with a client presenting with a severe and enduring mental illness. My reflections on this initial session have continued to influence my subsequent practice, not just with this particular client, but also with others. This experience has both enhanced my confidence in my skill and competency as a practitioner, and altered my sense of self on a personal level. In this regard therefore, I feel that my work with this client has fostered empowerment within me. When selecting a model of reflective practice within which to frame this account, it was difficult to identify one that I felt fully encapsulated my learning and development. I have chosen therefore not to adhere to any one specific model. Rather, I have tried to extract the key principles underlying reflective practice and have adopted a more integrative approach. This account draws primarily upon elements of Gibbs' (1998) Reflective Cycle, Johns' (1994) Model of Structured Reflection, and Schon's (1983) "Reflective Practitioner". The initial situation upon which this account is based is described, as are my thoughts and feelings associated with it. An analysis of the positive aspects of my practice, as well as those that could have been improved is provided. Key learning points, which I aim to carry forward in future, are also outlined.

### **APPENDICES**

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# **APPENDIX 1**

# SYSTEMATIC LITERATURE REVIEW

# Appendix 1.1 – Guidelines for submission to Journal of Sleep Research

### **Manuscript Submission**

The Journal of Sleep Research receives all manuscript submissions electronically via its submission and review website. To submit a manuscript, please follow the instructions below.

### **Getting Started**

- 1. Launch your web browser (Internet Explorer 5 or higher or Netscape 7 or higher) and go to the journal's Manuscript Central homepage (http://mc.manuscriptcentral.com/josr please note the acronym).
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- 4. Enter your institution and address information as appropriate, and then click "Next."
- 5. Enter a user ID and password of your choice (we recommend using your e-mail address as your user ID), and then select your area of expertise.
- 6. Click "Finish".

Log-in and select "Author Center."

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- 1. After you have logged in, click the "Submit a Manuscript" link in the menu bar.
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- Select the designation of each file in the drop down next to the Browse button.
- When you have selected all files you wish to upload (in groups of 3), click the "Upload Files" button.

Review your submission (in both PDF and HTML formats) before sending to the Journal. Click the "Submit" button when you are finished reviewing.

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receive a confirmation e-mail. You can also access Manuscript Central any time to check the status of your manuscript. The Journal will

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There are several categories of material:

### Commentaries and editorials-

The Editor may invite editorials and commentaries. The Journal will not consider unsolicited editorials or commentaries. Editorials should be approximately 800-1000 words and contain no more than 16 references. The title for an editorial should not exceed 85 characters.

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These should be approximately 2000 words in length, with a maximum of four figures or tables. Fast-track papers are rapidly reviewed and published.

# Regular Research Papers

These are of a more usual length (3000-5000 words), and will preferably be oriented towards basic clinical and non-clinical findings.

### **Review Papers**

These are intended to be well argued, preferably controversial reviews of topical subjects which, it is hoped, will generate debate.

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The Editor welcomes succinct correspondence relating to articles published in the journal, and of an academic and interesting nature.

### **Title Page**

This should contain a concise title of the article, a shortened version (no more than 50 characters including spaces) for the running head, names of the authors, their affiliations, and the full postal and e-mail address, fax and telephone number of an author to whom

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This should be on a separate page, and less than 250 words. It should be followed by up to six key words.

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This should start on a separate page, and include an introduction, methods, results and discussion. The suggested points of insertion of figures and tables, etc., should be indicated. Authors should avoid abbreviations (except for those commonly understood), long sentences, and many juxtaposed numbers in sentences.

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These should be in the Harvard style, i.e. using the names of the authors in alphabetical order (where there are more than two authors, use the first author only, followed by et al.), followed by the year of publication. Unpublished work should only be cited in the text.

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Examples of References:

Loomis, A. L., Harvey, E. N. and Hobart, G. Cerebral states during sleep as studied by human brain potentials. *J. Exp. Psychol.*, 1937, 21: 127144.

Kleitman, N. *Sleep and Wakefulness*. University of Chicago Press, Chicago, 1963 (second edition).

Webb, W. B. Theories about sleep and some clinical implications. In: R. Drucker Colin, M. Shkurovich and M. B. Sterman (Eds), *The* 

Functions of Sleep. Academic Press, New York, 1979: 1936.

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### Appendix 1.2 – Methodology Quality Rating Checklist

#### The Impact of Cognitive-Behavioural Therapy (CBT) on Dysfunctional Beliefs and Attitudes about Sleep: **A Systematic Review**

Quality rating	scale for included studies

Reviewer ID	Article Number
Study Design (please tick): Randomised Controlled Trial (RCT Controlled Clinical Trial Open Clinical Trial	
1. <u>Objectives (Max Score = 1)</u>	
1.1 Are the hypotheses/aims/objectives of the study clearly describ	ed? Y = 1 N = 0
2. Design and Methodology (Max Score = 6)	
2.1 Is the study design appropriate to test the hypotheses?	Y = 1 N = 0
2.2 Was participant allocation to intervention groups randomised?	Y = 1 N = 0 N/A
2.3 Was the method of randomisation described?	Y = 1 N = 0 N/A
2.4 Was the randomisation procedure robust (e.g. use compute independent researcher, blinding)?	r-generated random number table, conducted by an Y = 1 N = 0 N/A
2.5 Was an attempt made to blind participants or the rapists to the s $\ensuremath{N/A}$	tudy hypotheses? Y = 1 N = 0
2.6 Was a well-matched control group employed, or, in the absence	e of a control group, were there attempts to control the

he ICE OF a niioi group, v key variables/potential confounding variables, (e.g. taking medicine to induce sleep, or chronic pain/medical conditions known to affect sleep) in the design (via exclusion) or in the analysis (e.g. ANCOVA) from which the main findings were drawn?

Y = 1 N = 0

#### 3. Sample (Max Score = 11)

3.1 Is the population, and how it was identified/recruited, clear	ly stated?	Y = 1	1 1	N =	0
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3.2 Are the characteristics of the participants included in the study clearly described to allow adequate comparisons to be made? Y = 1 N = 0

3.3 Are the participants comparable at baseline with respect to age and gender and other relevant descriptive Y = 1 N = 0 characteristics?

3.4 Are the inclusion/exclusion criteria clearly specified? Y = 1 N = 0

3.5 Are the inclusion/exclusion criteria appropriate to test hypotheses?	Y = 1 N = 0
<ul> <li>3.6 Are the participants drawn from a:</li> <li>a) Clinically referred/treatment seeking sample</li> <li>b) Highly selected/well screened recruited sample of volunteers</li> </ul>	= 2 = 1
3.7 Was a generally accepted diagnostic criteria used to confirm insomnia diagnosis (e.g. DSM, ICS	SD-2)? Y = 1 N = 0
3.8 Was the reported duration of insomnia reported?	Y = 1 N = 0
3.9 Were potential co-morbid sleep disorders assessed using the IDI, ICSD-2, DSM-IV or a s	creening instrument? Y = 1 N = 0
3.10 Were potential co-morbid psychological disorders/symptoms measured using a reliable and BAI, SCID)?	valid tool (e.g. BDI-II,
Yes (minimum anxiety	and depression) = 1 N = 0
4. <u>Treatment Quality (Max Score = 6)</u>	
4.1 Has a clear rationale for the treatment been given?	Y = 1 N = 0
4.2 Was the treatment protocol/regimen adequately described?	Y = 1 N = 0
4.3 Was the total duration of treatment reported?	Y = 1 N = 0
4.4 Was there a manual that describes the active components of treatment?	Y = 1 N = 0
4.5 Have the therapists been adequately trained in the relevant procedures for this study?	Y = 1 N = 0
4.6 Has treatment credibility been assessed?	Y = 1 N = 0

### 5. Assessment and Outcome (Max Score = 9)

5.1 Are changes in sleep-related dysfunctional beliefs and attitudes a primary dependent variable?

Y = 2

Partly = 1 (e.g. studies which use cognitive change as a primary dependent variable, but to represent change in a different construct e.g. daytime functioning, rather than to assess cognitive N = 0

5.2. Have measurement tools been used at appropriate time points in relation to the design and focus of the	e study? Y = 1 N = 0
5.3 Is there adequate reporting of summary statistics (i.e. means, standard deviations)?	Y = 1 N = 0

5.4 Were the statistical analyses used to assess the main outcomes appropriate and clearly related to questions and hypotheses?	o the study aims, Y = 1 N = 0
5.5 Were drop-out/attrition rates adequately described?	Y = 1 N = 0
5.6 Did dropouts differ on clinical and descriptive data compared to those who completed the study?	Y = 0 N = 1

5.7 Has an intention-to-treat analysis been performed, or for non-experimental studies, were losses of participants at follow up taken into account? Y = 1 N = 0

5.8 Was the follow-up period long enough (at least 3 months) for any sustainable change in outcome resulting from treatment to be measured? Y = 1 N = 0

#### 6. <u>Conclusions (Max Score = 4)</u>

6.1 Does the study relate the results directly to the original aim(s), research question(s) and hypothes		es? Y = 1 N = 0	
	6.2 Do the conclusions drawn directly link to the results achieved?	Y = 1 N = 0	
	6.3 Are recommendations for clinical practice or future research discussed in relation to the findings?	Y = 1 N = 0	
	6.4 Are the limitations of the study clearly expressed?	Y = 1 N = 0	

**TOTAL SCORE** Randomised/Controlled/Open Clinical Trials /37 = % Non-Experimental trials /34 = %

#### **OVERALL RATING (please tick)**

Excellent (80% - 100%) Good (60% - 79%) Adequate (40% - 59%) Poor (below 40%)



\* See Appendix 1.5

# Appendix 1.4 - Psychological and Behavioural Treatments for Insomnia \*

Therapy	Description
Sleep Restriction Therapy	A method designed to curtail time in bed to the actual amount of sleep time. E.g. if a patient reports sleeping an average of 6 hours per night, out of 8 hours spent in bed, the initial recommended sleep window (from lights out to final arising time) would be restricted to 6 hours. Periodic adjustments to this sleep window are made depending upon sleep efficiency, until optimum sleep duration for the patient is obtained.
Relaxation Training	Clinical procedures aimed at reducing somatic tensions (e.g. progressive muscular relaxation) or intrusive thoughts at bedtime that interfere with sleep.
Cognitive Therapy	Psychological methods aimed at challenging and changing misconceptions about sleep and dysfunctional beliefs about insomnia and its perceived daytime consequences.
Sleep Hygiene Education	General guidelines about health practices (e.g., diet, exercise) and environmental factors (e.g., light, noise, temperature) that may aid or disrupt sleep. This may also include psycho- education regarding normal sleep and changes in sleep patterns associated with ageing.
Cognitive- Behavioural Therapy	A combination of any of the above behavioural and cognitive procedures.

\* Adapted from Morin et al., (2006). Psychological and Behavioural Treatment of Insomnia: Update of the Recent Evidence (1998-2004); SLEEP; 29 (11): 1398-1414.

# Appendix 1.5 - Summary of Excluded Articles

Author (s)	Design	Reason for Exclusion*
(Year)		
Arnedt et al., (2007)	Open Clinical Trial	Study assesses reduction in scores on the Dysfunctional Beliefs and Attitudes Scale (DBAS) in response to CBT for insomnia co-morbid with <b>alcohol dependence</b>
Carney et al., (2007)	Cross-sectional Research Design/No intervention	Study investigated dysfunctional sleep- related cognitions in primary insomnia, good sleepers, and insomnia co-morbid with fibromyalgia, major depressive disorder and mood disturbance
Edinger & Sampson (2003)	Randomised Controlled Trial	Participants presented with co-morbid medical conditions known to affect sleep (e.g. chronic fatigue; tinnitus) and current psychological conditions (major mood disorder; PTSD)
Ellis et al., (2007)	Cross-sectional Research Design/No intervention	Study investigates the critical dysfunctional beliefs and attitudes about sleep that distinguish those with late life insomnia from normal sleepers.
Rybarczyk et al., (2005).	Randomised Controlled Trial.	Examined CBT for geriatric insomnia co- morbid with progressive medical conditions known to affect sleep e.g. hypertension, coronary artery disease, congestive heart failure and chronic obstructive pulmonary disease.
Vincent, Penner, & Lewycky (2006)	Open Clinical Trial	Participants presented with co-morbid medical conditions known to affect sleep (e.g. neurological disorders; heart disease), other sleep disorders (sleep apnea) and current psychological conditions (major mood disorder; PTSD)

\*specific exclusion criteria are highlighted in bold text

# **APPENDIX 2**

# MAJOR RESEARCH PROJECT

### **Appendix 2.1 - Amendments to Original Project Proposal**

The following amendments to the originally proposed protocol have been made.

### Amendment 1

It had been proposed that an experienced photographic art student from the University of Glasgow School of Art would produce the sleep-related pictorial stimuli to be used in the dot-probe task. However, this was not possible due to practical reasons. Therefore, the researcher produced all sleep-related photographs.

#### Amendment 2

Initially, it had been proposed to gather actigraphy data from all participants across each of the 3 experimental groups (PI, DSPS, and NS) in order to aid diagnosis and confirm group allocation. However, after further discussion, it was decided that actigraphy data was not required from all participants. Actigraphy data therefore were gathered to confirm circadian phase delay within the DSPS group, and to differentiate between PI and DSPS when uncertainty regarding a participant's status arose. Decisions regarding the necessity to wear an Actigraph was determined both on the basis of information regarding participants sleeping patterns obtained during the initial screening/recruitment process, and on the content of their responses to the questionnaires and structured interview administered following completion of the dot-probe task.

#### Amendment 3

It was originally proposed that the researcher would again meet with all participants one week after completion of the dot-probe task in order to obtain sleep diary and actigraphy data, and to fully debrief each participant regarding the purpose of the study and the hypotheses behind it. Those complaining of either PI or DSPS would also be given general advice regarding their sleeping patterns based on the Good Sleep Guide (Espie, 1994). However, in order to reduce the demands placed upon participants, the researcher paid for the mailing of sleep diaries and actigraphs in lieu of returning for a second appointment. Participants were fully debriefed and, where necessary, provided with general sleep management advice based on the Good Sleep Guide via email approximately 1 week after the completion of the dot-probe task.

Appendix 2.2 – Major Research Project Proposal

# **Major Research Project**

Proposal

Attention Bias for Positively and Negatively Valenced Sleep-related Pictorial Stimuli in Psychophysiological Insomnia, Delayed Sleep Phase Syndrome and Normal Sleep: An Investigation using the Dot-Probe Paradigm.

> Alison Murie March 2007

#### SUMMARY OF PROJECT

Studies using computerised probe tasks adapted from cognitive experimental psychology demonstrate selective attention or 'attention bias' for sleep-related stimuli relative to neutral stimuli, as measured by reaction time differentials, in Psychophysiological Insomnia (PI). However, the mechanisms that 'drive' this attention bias in PI remain unclear. Recent models of PI (Harvey, 2002; Espie et al., 2006) posit both threat and craving as two candidate processes. However, few studies have specifically investigated this. In the attention bias tasks conducted thus far, sleep-stimuli have typically been innocuous, that is, they have been neither intrinsically aversive or appetitive in their valence. In addition, stimuli have been primarily word based. However, there are certain limitations to the use of word stimuli to investigate attention bias effects. The proposed study investigates whether both threat and craving account for the attention bias detected in insomnia, by manipulating the emotional valence of pictorial sleep-related stimuli. The study aims to examine the attention bias effect generated by pictures which are intrinsically negative in valence (threat) and intrinsically positive in valence (craving) amongst individuals with PI, Delayed Sleep Phase Disorder (DSPS) and Normal Sleepers (NS) using a Dot-Probe Paradigm.
#### **INTRODUCTION**

Psychophysiological Insomnia (PI) is characterised by conditioned sleep difficulty and/or heightened arousal as indicated by inability to initiate sleep at the desired time, increased focus on and anxiety about sleep, mental arousal, and heightened somatic tension (Research Diagnostic Criteria; RDC, Edinger et al., 2004). The exact mechanisms underlying the development and maintenance of PI remain unclear. One mechanism posited by two recent models (Harvey, 2002; Espie et al., 2006) is selective attention or 'attention bias', whereby stimuli that are salient to an individual are likely to attract attention, so that certain aspects of processing are prioritised, possibly at the expense of other aspects.

Harvey's Cognitive Model of Insomnia (2002) emphasises the role of excessive negative cognitive activity, whereby worry about sleeping and the consequences of not sleeping precipitates the preferential monitoring of internal and external sleep related cues, such as bodily sensations consistent with falling asleep or not falling asleep, and features of the bedroom environment. The Attention-Intention-Effort (AIE) Pathway model developed by Espie et al., (2006) is more specifically grounded in information processing theory. This model proposes that normal sleep is an automatic and involuntary process. In PI, this automaticity is inhibited by a developmental pathway beginning with an initial selective attention to sleep-related internal and external cues, and culminating in the explicit, conscious intention and subsequent effort to initiate sleep.

#### **Evidence for Attention Bias in PI**

Evidence of attention bias in PI can be drawn from several sources. Support for Harvey's (2002) model stems from phenomenological/descriptive studies (Neitsert-Semler and

Harvey, 2004) and 'real world' experiments (Neitzert-Semler and Harvey, 2006; Tang et al, 2006), which demonstrate that insomnia is, in terms of self-report, associated with sleep-related selective attention (Espie et al., 2006).

Support for biased information processing within the A-I-E framework comes from several studies that measure attention bias in insomnia objectively via computerised tasks in which reaction time towards stimuli presented on the computer screen is used as an index of cognitive arousal. These include the Emotional Stroop Task (Taylor et al., 2003), the Inducing Change Blindess (ICB) and Flicker paradigm (Jones et al., 2005; Marchetti et al., 2006). These studies provide substantial evidence that people with insomnia have an attention bias for sleep-related stimuli, where stimulus salience is thought to influence response time because of their 'grabbiness' (defined as uncontrolled prolonged attention capture or focusing) of sleep-related word or pictorial stimuli relative to neutral stimuli (see Espie et al., 2006, for a detailed review).

This effect has been observed in insomnia relative to normal sleepers and in those with other forms of sleep disorder, such as Delayed Sleep Phase Syndrome (DSPS), a circadian timing disorder, which, like insomnia, is characterised by difficulties initiating sleep (Pavlova et al., 2001). For example, MacMahon et al (2006) used another computerised paradigm, the Dot-Probe task, to explore whether systematic processing differences in attention bias existed between Good Sleepers (GS) and those with PI and DSPS. In this task, sleep-related and neutral word pairs are presented simultaneously on a computer screen. Attention is measured by recording the speed of manual responses for a visual probe that could appear in the spatial location of either word, immediately after the display of that word has ended (Marchetti et al., 2006). Measuring the impact of salient, sleep-related words on the probe reaction times in the two spatial areas indicates whether visual

attention has shifted towards or away from such stimuli. In this study, individuals with PI showed significantly greater attention bias toward sleep-related words (in comparison to neutral words) when compared to GS and DSPS.

Although the replication of attention bias data across the experimental paradigms noted above highlights the stability of the attention bias phenomenon in insomnia, few studies have investigated the processes that underlie or 'drive' sleep-related attention bias.

### What drives attention bias in PI?

Two possible psychological processes - threat and craving - have been proposed to underlie the attention bias that has been detected in PI (Harvey et al., 2002; Espie et al., 2006).

#### i) Threat

Attention bias has been implicated in the maintenance of several anxiety-related psychological disorders (e.g. Cooper and Fairburn, 1992; Salkovskis, 1999; Bryant and Harvey, 1996). Beck's cognitive model of emotional disorders posits that attention biases are motivated by negative beliefs or 'schema' stored in long-term memory (Beck, 1967). When activated, these schema direct information processing, including attention, towards stimuli congruent with them. This increases an individual's belief in negative automatic thoughts, resulting in hypervigilance and pre-occupation with danger and threat (Beck and Clark, 1997). Evidence suggests that anxious individuals are indeed characterised by selective attention bias toward threat (e.g. Mogg and Bradley, 1999), and it has been argued that this may have a causal role in anxiety disorders (Matthews and McLeod, 2002).

Harvey's Cognitive Model of Insomnia (2002) fits well with this 'threat-monitoring' model, and posits that selective attention to internal and external sleep related cues culminates in the overestimation of perceived deficit in sleep and daytime performance. The individual then believes that they have had less sleep than they actually have, and that their daytime performance is worse than it actually is. This leads to further excessive, negative cognitive activity, such that true deficit in sleep and daytime performance occurs and a vicious cycle of insomnia begins (Harvey, 2002). Perceived inability to sleep may therefore be conceptualised and experienced as a significant threat, possibly as a result of the associated reduced quality of life, poor work performance, and physical and psychological ill-health.

As stated, the AIE pathway defines normal sleep as passive and effortless, whereby good sleepers are assumed to fall asleep without consciously thinking about how they manage to do so. As Espie et al., (2006) suggest, if an individual with PI was once able to sleep normally, without being aware of how they did so, it is possible that sudden inability to sleep might be experienced as threatening. This is then thought to direct an individual's attention onto sleep and sleeplessness and give rise to actions aimed at reducing wakefulness.

# ii) Craving

Attention bias is not only observed in relation to threat. It has been implicated in the maintenance of alcohol, heroin, and nicotine addiction, where the stimuli are appetitive or reinforcing rather than threatening (Espie et al., 2006). For example, selective attention bias to relevant word or picture stimuli has been found in alcoholics and problem drinkers, but not social drinkers (e.g. Lusher et al., 2004). This suggests that problem drinkers are more likely to detect alcohol-related stimuli in the environment, which might then evoke

memories of drinking and maintain their addiction by producing a 'craving' sensation within an individual (Lusher et al, 2004). Given that sleep is a basic human need, it may be that the less sleep a person has, the more valuable it becomes - sleep-related attention bias may then result from a person's 'craving' (i.e. preoccupation and longing) for good sleep when experiencing chronic sleep difficulties (Espie et al., 2006).

Similarly, attention bias for addiction-related stimuli amongst smokers and alcoholics has been explained by the Motivational Theory of Strong Current Concerns (Cox et al., 2006). A current concern is defined as a "time-binding motivational state" of an individual between two particular points in time, whereby "individuals first become committed to pursuing a specific goal and subsequently to either attaining the goal or abandoning its pursuit". Current concerns are therefore manifest as the urge or 'craving' to use an addictive substance at a particular point in time, which can be influenced by cues and stimuli related to that substance. It is presumed that the greater an individual's current concern about an addictive substance, the greater their attention bias for stimuli related to that substance will be - that is, the value an individual attributes to a goal and their experiences associated with it, influences their attentional sensitivity to cues and stimuli related to that goal. Parallels may be drawn between this theory and the AIE pathway, where explicit intention to sleep, generated by attention bias, may reflect what Espie et al., (2006) refer to as an 'attention-for-action' mechanism, which subsequently results in individuals with PI engaging in efforts to initiate sleep.

### Manipulating the emotional valence of sleep-related stimuli

In the sleep-related attention bias studies conducted to date, the experimental stimuli used have not been intrinsically commanding of attention, nor were they particularly emotive or threatening (Espie et al., 2006). This differs from the stimuli used to measure attention bias in other disorders. For example, within the anxiety literature, the stimuli used vary considerably in content and emotional valence, and have been shown to consistently evoke discrete subjective and physiological reactions (Yiend and Matthews, 2001), thus increasing the possibility of demonstrating processing biases by maximising the salience of the stimuli. Manipulating the emotional valence of sleep-related stimuli used in attention bias paradigms may therefore prove an effective way to investigate what underlies or 'drives' the selective attention bias seen in insomnia.

Furthermore, there are limitations to using word stimuli in attention bias tasks. Research demonstrates that verbal attention paradigms do not generate the same attention bias data as pictorial versions, as pictorial stimuli are more likely to induce responses that mimic those experienced in real life situations (Yiend and Matthews, 2001). Word stimuli may be clearly negative in valence, but it is unclear that they encapsulate a severe or highly salient threat. For this reason, attention bias studies investigating state and trait anxiety have used emotionally valenced pictorial stimuli (for example, of weapons, corpses, dangerous animals; Mogg et al., 2000; Yiend and Matthews, 2001; Fox et al., 2002). In the context of insomnia, research specifically attempting to differentiate sleep-related pictures into those that are intrinsically negative and those that are intrinsically positive in valence is necessary.

#### AIMS AND HYPOTHESES

There is reasonable evidence that attention bias for sleep-related stimuli may be a feature of PI. However, the principal mechanism that 'drives' this attention bias remains unclear. The primary aim of this study will be to investigate the role of the two candidate processes posited thus far - threat and craving - in motivating the attention bias detected insomnia. This will be explored by manipulating the emotional valence of pictorial sleep-related stimuli. Specifically, the study aims to examine the attention bias effect generated by pictures which are intrinsically negative in valence (threat) and intrinsically positive in valence (craving) amongst individuals with PI, Delayed Sleep Phase Disorder (DSPS) and Normal Sleepers (NS), using a dot-Probe Paradigm.

It is anticipated that this study will initially replicate the findings of previous studies that indicate that attention bias is implicated in PI. The prediction being that participants with PI will demonstrate attention bias towards sleep-related stimuli relative to neutral stimuli, as measured by faster dot-probe reaction times, whereas those with DSPS and NS will not. Having demonstrated this, it is then predicted that this effect will be observed at the level of both threat and craving stimuli as follows:

- Participants with PI will show greater attention bias than those with DSPS and NS towards negative (threatening) sleep-related pictorial stimuli, as measured by faster dotprobe reaction time, in comparison to neutral pictures.

- Participants with PI will also show greater attention bias than those with DSPS and NS towards positive (craving) sleep-related pictorial stimuli, as measured by faster dot-probe reaction time, in comparison to neutral pictures.

#### METHOD

This study is part of an ongoing programme of research within the University of Glasgow Sleep Centre (UGSC), in collaboration with the Department of Psychology.

### Design

A between-groups design (PI, DSPS, NS) will be employed. The control group of individuals reporting no sleep difficulties are referred to as 'Normal Sleepers' (NS) in line with current Research Diagnostic Criteria (RDC; Edinger et al., 2004) for insomnia. It is not yet clear that attention bias has a primary role in the aetiology of insomnia, as information processing bias of this kind could occur because sleep is continually disturbed, making selective attention to sleep a secondary characteristic of chronic insomnia (Marchetti et al., 2006). Previous attention bias studies therefore recommend the inclusion of not only control samples of Normal Sleepers, but also comparison groups with other forms of sleep disorder such as DSPS. As stated previously, both DSPS and PI are characterised by difficulties initiating sleep (Alvarez et al, 1992). The cause of this difficulty in PI is attributed to psychological factors. As DSPS is thought to result from a circadian timing disorder, cognitive factors are not believed to influence the development or maintenance of DSPS; individuals who present with DSPS are therefore not expected to show attention bias to sleep-related stimuli as observed in PI (MacMahon et al., 2006).

#### Selection of the Computerised Attention Bias Task

Studies using complex pictorial scenes within both the fields of anxiety and addiction typically use the dot-probe paradigm (e.g. Bradley et al., 1997; Mogg et al., 1995; Mogg et al., 2000). Two versions of the dot-probe task are available. *Probe-position* tasks require participants to identify the position of the probe on the screen (e.g. left or right). However, such tasks might induce preferential monitoring of one region of the computer screen over the other (Mogg and Bradley, 1999). *Probe-classification* tasks require participants to identify the type of probe displayed e.g. an upwards or a downwards arrow. This task promotes monitoring of both sides of the display, but the relationship between the stimulus and response is more difficult to learn, resulting in reaction times that are more likely to be

variable, and higher error rates. Mogg and Bradley (1999) observed that, in non-clinical samples, the probe-position task yielded faster reaction times, fewer errors and similar effect sizes, compared with the probe-classification task. The primary dependent variable will therefore be reaction time to a dot-probe-detection response; latencies to detect this probe will be used to index the extent to which these groups selectively attend to either positive or negative sleep-related stimuli, or neutral stimuli.

#### Materials

A set of digitised sleep-related pictorial stimuli, differentiated equally into those that are intrinsically negative and positive in valence, will be developed. The exact number of pictures that will be used cannot be specified at this stage. The number of salient pictures developed in similar studies within anxiety and addiction research ranges from 18 to 24 (E.g. Mogg et al., 2000; 2003). Negative and positive sleep-related pictures will be taken by an experienced photographic art student at the Glasgow School of Art, to ensure quality and consistency in image composition. Neutral, non-sleep related pictures, matched as closely as possible in terms of image complexity and composition (brightness, orientation, size, gender and number of people/objects in each picture), will be selected from the International Affective Picture System (IAPS; Lang et al., 1999), a standardised set of photographic material with normative ratings for valence and arousal.

#### **Measures and Equipment**

## Descriptive and Clinical Measures

The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) is a 14item scale designed to measure depressive and anxious symptoms. Participants will be asked to complete this to aid sample description and the application of inclusion/exclusion criteria.

## Sleep Quality Measures

The following measures will be used to assess each participant's sleep quality and aid the differential diagnosis of PI, DSPS, and NS, and confirm group allocation:

- The *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al., 1989), is a reliable and valid measure of sleep quality, distinguishing between 'good' and 'poor' sleepers. A PSQI global score of >5 indicates that a participant is having severe difficulty in at least two areas, or moderate difficulty in more than three areas, of sleep quality.

- The *Insomnia Severity Index* (ISI; Morin et al., 1993) will be administered to qualify the severity of participants' subjective symptoms and consequences of insomnia, as well as the degree of concerns or distress caused by these difficulties. There are four categories: (No Clinical Significant Insomnia; Sub-threshold Insomnia; Clinical Insomnia - Moderate Severity, and Clinical Insomnia - Severe).

- The Morningness-Evening Questionnaire - Reduced Form (MEQ; Horne & Ostberg, 1976) will be used to evaluate individual differences in circadian rhythms. It classifies individuals into one of five different chronotypes as follows: Definitely Morning Type; Moderate Morning Type; Neither Type; Moderately Evening Type; Definitely Evening Type.

- The *Stanford Sleepiness Scale* (SSS; Hoddes et al., 1973) will be administered after completion of the dot-probe task to assess participants' level of alertness during the testing. This will help determine whether results observed are due to an actual attention bias effect or due to poor task performance as a result of sleepiness.

- Participants will be interviewed using a brief structured interview schedule comprising questions relating to DSM-IV and ICSD-2 criteria for PI and DSPS (Espie, 2002; Morin & Espie, 2003).

- Participants will be asked to complete a Standard *Sleep Diary* (Espie, 1991), upon wakening, for seven days following completion of the dot-probe task. This asks participants for qualitative estimates of sleep e.g. "how long did it take for you to fall asleep last night", in order to determine the participants' subjective Sleep Onset Latency (SOL - length of time, in minutes, taken to fall asleep at night) and Total Sleep Time (TST - total amount of time, in hours, slept).

- Actiwatch AW4 (Cambridge Neurotechnology) - small, non-intrusive wrist-worn devices - will be used to record objective rest/activity periods based on wearer movements. Actigraphy will help determine the sleep/wake (circadian) patterns of each participant (Sadeh et al., 1995). Data will be downloaded using the Actiwatch Sleep Analysis 2002, version 4.04. Van Someren et al's (1999) non-parametric circadian rhythm analysis (NPCRA) will be used 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).

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Attention bias will be measured by speed of manual responses to the dot-probe. The dotprobe task will be implemented using a Dell Optiplex GX270 computer and the experiment-generating package SuperLab Pro 2.0 (Cedrus Corporation). The size of the screen will be 28-cm (diagonally) and the viewing distance will be approximately 50-cm. Millisecond timing will be made through SuperLab Pro and an external response box (Model RB-400; Cedrus Corporation) to avoid timing errors associated with standard keyboard or mouse input. Participants will complete a number of practice trials using pairs of neutral, non-sleep related pictures, followed by the experimental trials where each sleeprelated picture will be paired with a neutral picture. Additional pairs of neutral pictures will be used as filler material so that sleep-related stimuli are not presented in every trial. The number of practice, filler and experimental trials to be administered is dependent on the number of sleep-related pictures eventually developed. Each picture pair will be repeated 4 times - the relative positions (left or right) of each pair of pictures will be counterbalanced within the experiment. Repeated stimuli pairs have been used before in this paradigm without any evidence of priming effects (Mathews et al., 1996). Stimuli pairs will be presented on the computer screen in a random order, with re-randomisation of the order for each participant, to ensure that order effects do not confound the results.

Each trial will consist of the following sequence of events: a fixation cross will appear in the centre of the screen for 1000-ms, followed by the picture pair for 500-ms. The pictures will then disappear and a single-asterisk dot-probe will immediately appear in the location of either the sleep-related or neutral picture. Participants will be asked to press one of two keys as quickly and as accurately as possible to indicate the location of the probe (left or right). The probe will remain on the screen until a response has been made. There will be an equal probability that the probe will replace either the positive sleep-related, negative sleep-related or neutral picture. An inter-trial interval of 1000-ms will follow each trial.

### **Participants**

Participants will be staff and students from the University of Glasgow, staff working within NHS Greater Glasgow & Clyde, and members of the general public, aged 18-60 years. Attempts will be made to ensure that the three groups are matched for age and gender.

## **Inclusion and Exclusion Criteria**

i) *PI*: Participants must satisfy Research Diagnostic Criteria (RDC; Edinger et al., 2004) for Psychophysiological Insomnia (of the sleep-onset type). These criteria have been derived from, and combine, existing DSM-IV and ICSD-2 criteria. Those with PI must have *at least* subthreshold insomnia as measured by the ISI, score >5 on the PSQI, and have experienced sleep disruption for longer than 6 months. Participants must be classified as "Neither Type" on completion of the MEQ. Sleep diary data must confirm sleep-onset difficulties typical of PI, and their self-reported sleep problem must be confirmed by objective actigraphy data.

ii) *DSPS:* Participants must meet combined DSM-IV and ICSD-R criteria for DSPS, as RDC criteria for this condition have not been defined. They must be classified as "Definite Evening Type" on completion of the MEQ. Sleep diary data must confirm circadian phase delay, and their self-reported sleep problems must be confirmed by objective actigraphy data.

iii) *NS:* Participants must score <5 on the PSQI, report themselves as being 'normal' sleepers, and meet no criteria for a sleep disorder at the present time or in the past. They must be classified as "Neither Type" on completion of the MEQ.

For all 3 groups, participants will be excluded as a result of active psychological or drug interventions for sleep problems, or when the sleep disorder is suspected as being the result of substance misuse or physical ill health. This will be assessed during interview following completion of the experimental procedure. Participants must not suffer from depressive symptoms and a score above the cut off for depression (11) on the HADS will result in exclusion from analysis. As anxiety is commonly associated with insomnia, scoring above the cut off for anxiety on the HADS will not result in exclusion. All participants should have normal vision, or corrected to normal vision. Those whose sleep problems are due to a combination of PI and DSPS will be excluded. Individuals that fit RDC for any other type of sleep disorder will also be excluded, as will those above 60 and below 18 years of age.

## **Recruitment and Selection**

University staff and students will be recruited through an information notice sent via the University e-mail system asking them to contact the experimenter with answers to a brief set of questions regarding their sleeping patterns (Appendix 2.5). These questions will facilitate the screening and selection of respondents who best resemble the traits of the three sleep groups. Normal sleepers will be invited to complete the experimental procedure. Those who report the traits of PI and DSPS (Appendix 2.6) will be e-mailed a second time, asking more specific questions regarding their sleep patterns. Those who satisfy diagnostic criteria for PI and DSPS will then be invited to complete the experimental procedure. All potential participants will be sent a participant information sheet via e-mail, outlining the

purpose of the study and what would be required of them if they agree to participate (Appendix 2.7) On the day of testing, participants will be provided with a paper copy of the participant information sheet, and given the opportunity to discuss any questions pertaining to the study they may have. They will then be asked to sign a participant consent form (Appendix 2.8). After completing the experimental procedure, the application of rigorous criteria, and prospective diary and actigraphic assessment, will be used to confirm participants' allocation to the PI, DSPS, and NS groups. It is likely that some participants will be excluded from the study at this stage.

NHS staff will be recruited via e-mail bulletin (Appendix 2.5) posted on the NHS Greater Glasgow & Clyde staff website. They will be asked to email the experimenter if they are interested in participating in the study. Recruitment and selection will then follow the same protocol outlined above.

# Feasibility of Recruitment and Selection Procedure

This protocol has successfully been used in previous attention bias studies conducted within the UGSC (e.g. Marchetti et al., 2006) In this study, 253 individuals responded to the initial mass e-mail advertisement. Of these, 189 reported symptoms similar to PI and DSPS and were emailed a second time for further information regarding their sleep patterns. Of these, 157 responded to this e-mail, and 97 were assessed as potentially suitable and therefore completed the experimental procedure. Following application of rigorous criteria and prospective diary and actigraphic measurement, a total of 90 participants were included in the analyses, where N = 30 in each of the experimental groups (PI, DSPS, and GS).

#### Justification of Sample Size

Mogg and Bradley (1999) investigated effect sizes obtained when measuring attention bias amongst anxious individuals for emotionally valenced pictorial stimuli using a probeposition, dot-probe paradigm. They observed that estimated effect sizes varied from +0.61to +0.81, with a mean of +0.71. A power calculation, assuming an effect size of +0.71, estimates that 21 participants will be required in each of the 3 groups (PI, DSPS, and NS) to detect significant differences at an alpha level of 0.5, with a power of 0.8 (one-tailed). Given that it is intended to use demographically well-matched samples, differentiated on sleep parameters on both subjective and objective measures, it is expected that this sample size will be sufficient to detect differences if they exist.

### Procedure

# Development of Pictorial Sleep-related Stimuli

To develop sleep-related photographic stimuli, scales derived from research identifying the 'live' cognitions that people with insomnia have while lying awake in bed unable to sleep will be used. These will include the Glasgow Content of Thoughts Inventory (GCTI: Harvey and Espie, 2004), the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS; Morin, 1993), and the Self-Statements Test (Fichten et al., 1998). Pictures will initially be screened by the experimenter and members of the UGSC for valence and clarity of composition. Remaining pictures will then be presented to a panel of independent student raters, who will be asked to rate the degree to which pictures are positive and negative in valence using a 9-point scale, where 0 = not at all positive/negative and 8 = extremely negative/positive. This procedure has been adapted from similar attention bias studies conducted within the anxiety field (e.g. Mogg et al., 2000; Yiend and Matthews, 2001). Mean ratings of picture valence (positive and negative) will be calculated.

### **Experimental Procedure**

Participants will be met at the University by the researcher. After signing participant consent forms, they will be asked to sit in a comfortable and relaxed position. Participants will then complete the Dot-probe task. This will take no longer than 15-20 minutes. All participants will be given the same set of instructions. These will appear on the computer screen, and the experimenter will explain the task verbally to ensure that it is fully understood. Immediately after completion of the task, each participant will be interviewed to evaluate his/her general sleep patterns, using a structured interview format comprising questions relating to DSM-IV and ICSD-R criteria for PI and DSPS (Espie, 2002; Morin and Espie, 2003). This interview will take no longer than 10 minutes. Participants will complete self-report measures, each taking no more than 5 minutes to complete. Participants will then be asked to wear an actigraph and complete a standard sleep diary for seven nights following the experiment.

Participants will return 1 week later. For each participant, their subjective diary responses will be compared with their recorded sleep data from the actigraph. Participants will then be fully debriefed regarding the purpose of the study and the hypotheses behind it, and any questions will be discussed. Participants who complain of either PI or DSPS will be given a copy of The Good Sleep Guide (Espie, 1994), recommended by the British Sleep Society, and advice in managing their sleep difficulties. In total, this session should take no more than 30 minutes - 1 hour.

### Settings

The experimental procedure will be conducted at suitable rooms in the Department of Psychology, University of Glasgow, which are available for long-term booking, and at the UGSC based at the Southern General Hospital, Glasgow. Sound and illumination levels will be carefully controlled.

### **Data Analysis**

Analysis will be conducted using the Statistical Package for the Social Sciences (SPSS) for Windows.

## Demographic and Clinical Data

For the purposes of sample description and to check that groups are demographically wellmatched, means and standard deviations for age, gender, and the HADS for PI, DSPS, and NS groups will be calculated, followed by an initial ANOVA, and, where appropriate, by post-hoc comparisons (Tukey's Honestly Significant Differences test). ANCOVA will be conducted to further investigate any systematic differences that emerge.

# Sleep Quality Data

To aid differential diagnosis and confirm participants group allocation, means and standard deviations for PI, DSPS, and NS on PSQI, Diary TST (minutes), Diary SOL (minutes), and Actigraphy L5 (24 hour clock) will be calculated, followed by an initial ANOVA, and, where appropriate, by post-hoc comparisons (Tukey's HSD).

# Dot-probe Data Reduction and Analysis

Analysis of reaction-time data will follow the standard practice in Dot-probe literature. Reaction times from filler trials and trials with errors will be excluded, as will those less than 200-ms and greater than 2000-ms. Reaction times more than 2 standard deviations above the mean for each participant will be excluded (Mogg et al., 2004).

In line with standard practice in the dot-probe literature, mean attention bias scores will be calculated for each sleep valence in order to clarify the effect of differently valenced sleep-related stimuli on attention, and comparisons between each of the three groups will be conducted (Bradley et al., 1997). These scores will be calculated by subtracting the mean

reaction times when probes are in the same location as sleep scenes, from the mean reaction times when they are in the opposite location. The attention bias score summarises the interaction between sleep-related picture position and probe position on reaction time, providing a measure of the relative speeding of reaction time to probes that appear in the same location as the sleep-related pictures. Orthogonal contrasts, with an assumption of equal variances, on attention bias scores, will be calculated to examine the degree to which participants with PI selectively attend to positively and negatively valenced sleep-related stimuli, relative to neutral stimuli.

## HEALTH AND SAFETY ISSUES

There are no significant risks to the health and safety of the experimenter or participants associated with this study. Similar procedures have been used with this participant group in previous research, and do not typically cause significant distress. The study will be conducted primarily at university research settings, where appropriate procedures to minimise risk are in place.

#### ETHICAL ISSUES

No significant ethical issues have to be considered. Participants will be provided with the information necessary for them to provide full informed consent, taking care not to reveal unnecessary information that might prime participants to the research aims and questions. Participants will be informed of their right to withdraw their participation and data at any time. All participant data will be anonymised (e.g. the first participant will be coded as 001) and stored within an SPSS database by the experimenter.

### ETHICAL APPROVAL

Ethical approval will be sought from both the NHS Greater Glasgow and Clyde and University of Glasgow ethics committees.

#### FINANCIAL ISSUES

There are no major financial issues. Equipment and software is available within the UGSC, Department of Psychological Medicine, and Department of Psychology at the University of Glasgow. We have secured voluntary assistance for the development of photographic stimuli.

#### TIMETABLE

Development of pictorial stimuli will begin in June 2007 and is expected to be completed by August 2007. Data collection is expected to begin in October 2007, and is estimated to take 8 months (completed by May 2008). Writing and reporting of results is estimated to take two months (completed by July 2008).

## PRACTICAL APPLICTIONS

#### Clinical

Nine percent of the general population report experiencing insomnia on a regular nightly basis (Ancoli-Israel and Roth, 1999). PI is the most common insomnia sub-type, found in 1-2% of the general population, and in 12-15% of all patients seen at sleep centres (ASDA, 1997; AASM, 2005; APA, 1994); pre-existing insomnia is the highest attributable, potentially treatable, risk factor for first episode depressive disorder and for recurrence of depression in adults and older adults (Riemann and Volderholzer, 2003; Cole and Dendukuri, 2003).

Further understanding of the factors underlying the development and maintenance of PI may facilitate the development of targeted interventions that can be applied early to help reduce sleep disturbance and restore normal sleep, thus preventing the development of severe and persistent insomnia, and subsequently reducing the risk and prevalence of

depressive disorders. The primary goal of insomnia treatment may be directed away from solely attempting to increase the amount of sleep obtained towards reducing selective attention and monitoring that may inhibit the expression of normal sleep (Espie et al, 2006).

## Research

Insomnia research is constrained by the absence of reliable objective markers of the phenomenology of poor sleep. This study could contribute to existing literature, adding credence to use of selective attention bias as a proxy measure for cognitive arousal in insomnia. The stimuli developed for this investigation could potentially be used in subsequent studies to answer a range of research questions pertaining to the development and maintenance of insomnia.

# References

Alvarez, B., Dahlitz, M.J., Vignau, J., and Parkes, J.D. The delayed phase sleep phase syndrome: clinical and investigative findings in 14 subjects. *J. Neurol. Neurosurg. Psychiatry*, 1992, 55: 665-70.

American Academy of Sleep Medicine (AASM). International Classification of Sleep Disorders: Diagnostic and Coding Manual. Westchester, IL, 2005 (second edition).

American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC, 1994 (fourth edition).

American Sleep Disorders Association (ASDA). International Classification of Sleep Disorders: Diagnostic and Coding Manual (Revised). Rochester, MA, 1997.

Ancoli-Israel, S. and Roth, T. Characteristics of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey. *Sleep*, 1999, 22: 347–53.

Beck, A.T. Depression: Causes and Treatment. University of Pennsylvania Press, Philadelphia, 1967.

Beck, A.T. and Clark, D.A. An information processing model of anxiety: automatic and strategic processes. *Behav. Res. Ther*, 1997, 35: 49–58.

Bjelland, I., Dahl, A.A., Haug, T.T. and Neckelmann, D. The validity of the hospital anxiety and depression scale. An updated literature review. *J. Psychosom. Res*, 2002, 52(2): 69-77.

Bonnet, M.H. and Arand, D.L. Insomnia, metabolic rate and sleep restoration. J. Intern. Med, 2003, 254: 23-31.

Bradley, B.P., Mogg, K. and Lee, S. Attentional biases for negative information in induced and naturally occurring dysphoria. *Behav. Res. Ther*, 1997, 35: 911–27.

Bradley, B. P., Mogg, K. and Millar, N. H. Covert and overt orienting of attention to emotional faces in anxiety. *Cog. and Emotion*, 2000, 14 (6): 789-808.

Bradley, B.P., Mogg, K., Wright, T. and Field, M. Attentional bias in drug dependence: vigilance for cigarette-related cues in smokers. *Psychol. Addict. Behav*, 2003, 17: 66–72.

Broomfield, N.M. and Turpin, G. Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. *Biol. Psychol*, 2005, 68: 179-200.

Bryant, R. A. and Harvey, A. G. (1995). Posttraumatic stress in volunteer fire fighters: Predictors of distress. *J. Nervous and Mental Disease*, 1995, 183: 267–271.

Buysse, D.J., Reynolds, C.F., Monk, T.H., Berman, S.R, and Kupfer, D.J. The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Res*, 1989, 28: 193-2 I3.

Cedrus Corporation, SuperLab 2.0 (Stimulus Presentation Software). San Pedro, CA, 1999.

Chelminski, T.V., Petros, J.J. and Ferraro, F.R. Psychometric properties of the reduced Horne and Östberg questionnaire. *Personality and Indiv. Difference*, 2000, 29 (3): 469–478.

Cole, M.G. and Dendukuri, N. (2003). Risk factors for depression among elderly community subjects: A systematic review and meta-analysis. *American J. of Psychiatry*, 2003, 160: 1147-1156.

Cooper, M.J. and Fairburn C.G. Thoughts about eating, weight and shape in anorexia nervosa and bulimia nervosa. *Behav. Res. Ther*, 1992, 30(5): 501-11.

Cox, W.M., Fadardi, J.S. and Pothos, E.M. The Addiction-Stroop test: theoretical considerations and procedural recommendations. *Psychol. Bulletin*, 2006, 132: 443-476.

Dagan, Y. Circadian rhythm sleep disorders (CRSD). Sleep Med. Reviews, 2002, 6 (1): 45-54.

Edinger, J.D., Bonnet, M.H., Bootzin, R.R., Doghramji, K., Dorsey, C.M. and Espie, C.A. Derivation of research diagnostic criteria for insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep*, 2004, 27: 1567–96.

Ehrman, R.N., Robbins, S.J., Bromwell, M.A., Lankford, M.E., Monterosso, J.R. and O'Brien, C.P. Comparing attentional bias to smoking cues in current smokers, former smokers, and non-smokers using a dot-probe task. *Drug Alcohol Depend*, 2002, 67: 185–191

Espie, C.A., Brooks, D.N. and Lindsay, W.R. An evaluation of tailored psychological treatment of insomnia. *J. Behav. Ther. Exper. Psychiatry*, 1989, 20: 143-467.

Espie, C.A. The Psychological Treatment of Insomnia. Wiley, Chichester, 1991.

Espie, C.A. *The Good Sleep Guide*. Guidance Report for the National Medical Advisory Committee, *1994*.

Espie, C.A. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Ann. Rev. Psychol*, 2002, 53: 215–43.

Espie, C.A., Broomfield, N.M., MacMahon, K.M.A., MacPhee, L.M. and Taylor, L.M. The Attention-Intention-Pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep Med. Rev*, 2006, 10: 215-245.

Fichten, C.S., Libman, E., Creti, L., Amsel, R. and Tagalakis, V. (1998). Thoughts during awake times in older good and poor sleepers: the self-statement test: *Cognit Ther. Res*, 1998, 22: 1–20.

Field, M., Mogg, K. and Bradley, B.P. Cognitive bias and drug craving in recreational cannabis users. *Drug and Alcohol Depend*, 2004, 74: 105-111

Field, M., Mogg, K. and Bradley, B.P. Eye movements to smoking related cues: effects of nicotine deprivation. *Psychopharmacol*, 2004; 173: 116-23

Fox, E., Russo, R. and Dutton, K. Attentional bias for threat: evidence for slowed disengagement from emotional faces. *Cogni. Emotion*, 2002, 16: 355–79.

Harvey A.G. A cognitive model of insomnia. Behav. Res. Therapy, 2002, 40: 869-93.

Harvey K.J. and Espie C.A. Development and preliminary validation of the Glasgow content of thoughts inventory (GCTI): A new measure for the assessment of pre-sleep cognitive activity. *Br. J. Clin. Psychol*, 2004, 43: 409–20

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R. and Dement, W.C. Quantification of sleepiness: a new approach. *Psychophysiology*, 1973, 10: 431-436.

Horne J.A. and Ostberg O. A self-assessment questionnaire to determine morningness and eveningness in human circadian rhythms. *Internat. J. Chronobiol*, 1976, 4(2): 97-110.

Hubert W, de Jong-Meyer R. Psychophysiological response patterns to positive and negative film stimuli. *Biol. Psychol*, 1991, 31(1): 73-93.

Jones B.T., MacPhee, L.M., Broomfield, N.M., Jones, B.C. and Espie, C.A. Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. *J. Abnorm. Psychol*, 2005, 114: 249–58.

Klinger, E. Emotional influences of cognitive processing, with implications for theories of both. In: P.M. Gollwitzer and J.A. Burgh (Eds), *The Psychology of Action: Linking Cognition and Motivation to Behaviour*. Guilford Press, New York, 1996.

Kyle, S.D., Espie, C.A. and Morgan, K. A qualitative analysis of daytime functioning and quality of life in persistent insomnia using focus groups and audio-diaries. *Sleep*, 2008, Abstract suppl., 31, 243-244.

Lang, P.J., Bradley, M.M. and Cuthbert, B.N. *The International Affective Picture System* (*IAPS*) *Digitized Photographs*, The Center for Research in Psychophysiology, University of Florida, Gainesville, FL (1999).

Loeber., S, Croissant, B., Heinz, A., Mann, K. and Flor, H. Cue exposure in the treatment of alcohol dependence: Effects on drinking outcome, craving and self-efficacy. *Brit. J. of Clin. Psychol*, 2006, 45 (4): 515-529.

Lundh L.G., Froding, A., Gyllenhammer, L., Broman, J.K. and Hetta, J. Cognitive bias and memory performance in patients with persistent insomnia. *Scand. J. Behav. Ther*, 1997 26: 27–35.

Lusher, J., Chandler, C. and Ball, D. Alcohol dependence and the alcohol Stroop paradigm: evidence and issues. *Drug Alcohol Depend*, 2004, 75: 225–31.

MacLeod, C.M., Mathews, A. and Tata, P. Attentional bias in emotional disorders. J. Abnorm. Psychol, 1986, 95: 15–20.

MacMahon, K.M.A., Broomfield, N.M. and Espie, C.A. Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome. *Sleep*, 2006, 29(11): 1420-1427.

Marchetti, L.M., Biello, S.M., Broomfield. N.M., MacMahon, K.M.A., and Espie, C.A. Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiologic insomnia, delayed sleep phase syndrome and good sleepers using the Induced Change Blindness paradigm. *J. Sleep. Res*, 2006, 15: 1–10.

Matthews, A. and MacLeod, C. Cognitive approaches to emotion and emotional disorders. *Annu. Rev. Psychol.* 1994, 45: 25–50.

Matthews, A., Mogg, K., Kentish, J. and Eysenck, M. Effect of psychological treatment on cognitive bias in generalized anxiety disorder. *Behav. Resear. Ther*, 1995, 33: 293-303.

Matthews, A., Ridgeway, V. and Williamson, D. Evidence for attention to threatening stimuli in depression. *Behav. Res. Ther*, 1996, 34: 695-705.

Mattia J.I, Heimberg, R.G. and Hope D.A. The revised Stroop colour-naming task in social phobics. *Behav. Res. Ther*, 1993, 31(3): 305-13.

Mogg, K., Bradley, B.P., Millar, N. and White, J. Cognitive bias in generalised anxiety disorder: A follow-up study. *Behav. Res. Ther*, 1995, 33: 927–35.

Mogg, K. and Bradley, B.P. Some methodological issues in assessing attentional biases for threatening faces in anxiety: a replication study using a modified version of the probe detection task. *Behav. Res. Ther*, 1999, 37(6): 595-604.

Mogg, K. and Bradley, B.P. Biases in eye movements to threatening facial expressions in generalized anxiety disorder. *J. Abnorm. Psychol*, 2000, 109(4): 695-704.

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

Mogg, K., Bradley, B.P., Field, M. and De Houwer, J. Eye movements to smoking-related pictures in smokers: relationship between attentional biases and implicit and explicit measures of stimulus valence. *Addiction*, 2003, 98: 825–836.

Mogg, K., Phillipot, P. and Bradley, B.P. Selective attention to angry faces in clinical social phobia. *J. Abnorm. Psychol*, 2004, 113(1): 160-165.

Mogg, K., Field, M. and Bradley, B.P. Attentional and approach biases for smoking: an investigation of competing the theoretical views of addiction. *Psychopharmacol*, 2005, 180(2): 333-41.

Morin CM. Insomnia: Psychological Assessment and Management. Guilford Press, New York, 1993.

Morin, C.M., Stone, J., Trinkle, D., Mercer J. and Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol. Aging*, 1993, 8: 463-7.

Morin, C. M. and Espie, C. A. *A Clinical Guide to Assessment and Treatment*. Kluwer Academic/Plenum Publishers, New York, 2003.

Neitzert-Semler, C. and Harvey, A.G. An Investigation of monitoring for sleep-related threat in primary insomnia. *Behav. Res. Ther*, 2004, 42: 1403–20.

Neitzert-Semler, C. and Harvey, A.G. Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? *Behav. Sleep Med.* 2006, 4: 85–103.

Pavlova, M., Berg, O., Gleason, R., Walzer, F., Roberts, S. and Regestein, Q. Self-reported hyperarousal traits among insomnia patients. *J. Psychosom. Res*, 2001, 51: 435-41.

Posner, M.I. Orienting of attention. Q. J. Exp. Psychol, 1980, 32: 3-25.

Ratcliff, R. Methods for dealing with outliers. Psychol. Review, 1993, 114: 510-532.

Riemann, D. and Volderholzer, U. Primary insomnia: A risk factor to develop depression. J. Affective Disorders, 2003, 76: 255-259.

Ryan, F. Attentional bias and alcohol dependence: a controlled study using the modified Stroop paradigm. *Addict. Behav*, 2002, 27: 471–482.

Sadeh A., Alster J., Urbach D. and Lavie P. Actigraphically based automatic bedtime sleep-wake scoring: validity and clinical applications. *J. Ambulat. Monit*, 1989, 2: 209–216.

Sadeh A., Hauri P.J., Kripke D.F. and Lavie P. The role of actigraphy in the evaluation of sleep disorders. *Sleep*, 1995, 18: 288–302.

Salkovskis, P.M. Understanding and treating obsessive-compulsive disorder. *Behav. Res. Ther*, 1999, suppl 37: 29-52.

Smith, M.T. and Wegener, S.T. Measures of sleep: The insomnia severity index, medical outcomes study (MOS) sleep scale, Pittsburgh sleep diary (PSD), and Pittsburgh Sleep quality index (PSQI). *Arthritis Care and Res*, 2003, 49(5): 184-196.

Spiegalhalder, K., Espie, C.A., Nissen, C. and Riemann, D. Sleep-related attentional bias in patients with primary insomnia compared with sleep experts and healthy controls. *J. Sleep. Res*, 2008, 17: 191-196.

Tang, N.K.J., Harvey, A.G., and Schmidt, D.A. Sleeping with the enemy: clock monitoring in the maintenance of insomnia. *J. Behav. Ther. Exp. Psychiatry*, 2007, 38: 40-53.

Taylor, L.M, Espie, C.A. and White C.A. A comparative experimental investigation of Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behav. Sleep Med*, 2003, 1: 200–12.

Van Someren, E. J. W., Swabb, D. F., Colenda, C. C., Cohen, W., McCall, W. V. and Rosenquist, B. Bright light therapy: improved sensitivity to its effects on rest-activity rhythms in alzheimer patients by application of non-parametric methods. *Chronobiol. Int*, 1999, 16: 505–518.

Wicklow, A. and Espie, C.A. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav. Res. Ther*, 2000, 38, 679-693.

Williams, J.M.G., Watts. F.N., MacLeod. C. and Mathews, A. Cognitive Psychology and Emotional Disorders. Wiley, New York, 1997 (second edition).

Yiend, J. and Mathews, A. Anxiety and attention to threatening pictures. Q. J. Exp. Psychol. Hum, 2001, 54: 665-81.

Zigmond A.S. and Snaith, R.P. The hospital anxiety and depression scale. *Acta Psychiatry Scand*, 1983, 67: 361-70.

# Appendix 2.3 – University (Medical Faculty) Ethics Committee Approval Letter

### AMMcN/AMJG

Ms Alison Murie Psychological Medicine Academic Centre Gartnavel Royal Hospital 1055 Great Western Road Glasgow G12 0XH

Dear Ms Murie

# **Medical Faculty Ethics Committee**

**Project Title:** Attention Bias for Positive and Negative Sleep Stimuli in Insomnia Project No.: FM05806

The Faculty Ethics Committee has reviewed your application and has agreed that there is no objection on ethical grounds to the proposed study now that the requested revisions have been incorporated. They are happy therefore to approve the project, subject to the following conditions:

- The research should be carried out only on the sites, and/or with the groups defined in the application.
- Any proposed changes in the protocol should be submitted for reassessment, except when it is necessary to change the protocol to eliminate hazard to the subjects or where the change involves only the administrative aspects of the project. The Ethics Committee should be informed of any such changes.
- If the study does not start within three years of the date of this letter, the project should be resubmitted.
- You should submit a short end of study report to the Ethics Committee within 3 months of completion.

Yours sincerely,

Dr. Anne M McNicol Faculty Ethics Officer

# Appendix 2.4 – NHS Ethics Committee Approval Letter

Research Ethics R&D Directorate Gartnavel Royal Hospital 1055 Great Western Road Glasgow G12 0XH www.nhsggc.org.uk

Ms Alison Murie	Dat
Trainee Clinical Psychologist	Yo
NHS Greater Glasgow & Clyde	Ou
Gartnavel Royal Hospital	Dir
1055 Great Western Road	Fax
Glasgow G12 0XH	E-n

Date08 October 2007Your Ref0141 211 3824Direct line0141 211 3814Fax0141 211 3814E-mailLiz.Jamieson@ggc.scot.nhs.uk

Dear Ms Murie

Full title of study:	Attention Bias for Positively and Negatively Valenced
	Sleep-related Pictorial Stimuli in Psychophysiological
	Insomnia, Delayed Sleep Phase Syndrome, and Normal
	Sleep: An Investigation using the Dot-Probe Paradigm
<b>REC reference number:</b>	07/\$0701/109

The Research Ethics Committee reviewed the above application at the meeting held on 04 October 2007. Thank you for attending to discuss the study but as your application was very comprehensive, the Committee did not require any further information.

# **Ethical opinion**

The members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation. However the Consent Form does not include permission to tape the interviews. If the interviews are to be taped then this should be included in the Consent Form. A revised Consent Form should therefore be submitted.

# Ethical review of research sites

The favourable opinion applies to the research sites listed on the attached form.

# **Conditions of approval**

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

# **Approved documents**

The documents reviewed and approved at the meeting were:

Document	Version	Date
Application		30 August 2007
Investigator CV		30 August 2007
Protocol		30 August 2007
Covering Letter		30 August 2007
Summary/Synopsis		30 August 2007
Interview Schedules/Topic Guides		30 August 2007
Questionnaire		30 August 2007
Questionnaire		30 August 2007
Advertisement		30 August 2007
Participant Information Sheet		30 August 2007
Participant Consent Form		30 August 2007
Supervisor CV		30 August 2007

# **R&D** approval

The study should not commence at any NHS site until the local Principal Investigator has obtained final approval from the R&D office for the relevant NHS care organisation.

# Membership of the Committee

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

# Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

07/S0701/109

Please quote this number on all correspondence

With the Committee's best wishes for the success of this project

Yours sincerely

Liz Jamieson Research Ethics Committee Co-ordinator on behalf of Dr Paul Fleming, Chair Enclosures: List of names and professions of members who were present at the meeting and those who submitted written comments Standard approval conditions Site approval form (SF1)

Copy to: Mr Brian Rae

# Glasgow & Clyde Primary Care, Community & Mental Health

# Attendance at Committee meeting on 04 October 2007

#### **Committee Members:**

Name	Profession	Present	Notes
Winifred McCartney	Assistant Co-ordinator	Yes	
Dr Jacqui Anderson	Consultant Psychiatrist	Yes	
Dr Janet Brennand	Consultant Obstetrician	Yes	
Dr Jim Brooks	Lay Member	No	
Dr Adam Burnel	Consultant Psychiatrist	No	
Dr Susan Carr	Consultant in Family Planning and Reproductive Health	Yes	
Ms Lorna Cuthbertson	Senior Clinical Pharmacist	No	
Mr Paul Davies	Principal Pharmacist	Yes	
Mr Philip Dolan	Lay Member	No	
Dr Paul Fleming	Consultant Clinical Psychologist	No	
Mr Martin Hattie	Clinical Nurse Specialist	Yes	
Ms Liz Jamieson	Co-ordinator	Yes	
Rev Cameron Langlands	Lay Member	No	
Mr Eoin MacGillivray	Lay Member	Yes	
Dr Robert McNeil	General Practitioner	Yes	
Mrs Gillian Notman	Joint Occupational Therapy Lead Advisor	Yes	
Mrs Helen Ross	Lay Member	Yes	
Dr David Watt	Consultant Occupational Health	Yes	

# Appendix 2.5 – Recruitment Email

# E-mail Number 1:

The initial mass e-mail will contain some brief information about the Glasgow Sleep Lab, followed by five specific questions:

- 1) Are you someone who struggles to sleep at night?
- 2) Once asleep do you regularly wake during the night? 3
- 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 will conclude 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.

# Email Number 2:

The second email will ask more specific questions:

- 1) How many minutes does it take you, on average, to fall asleep at night?
- 2) Once asleep do you wake at regular intervals throughout the night?
- 3) How long do you predict you are awake during the night?
- 4) On average how many hours of sleep do you get per night?
- 5) Do you view your sleep as a problem
- 6) What time do you get up in the morning? Do you feel refreshed?

For PI specific traits will include:

- 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 will include:

- 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

Examples of responses assessed as potentially suitable (from previous attention bias

studies) for the PI group are:

- "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"

An example of a response that was assessed to be potentially suitable for the DSPS group is:

• "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".

# **Appendix 2.7 – Participant Information Sheet**





#### PARTICIPANT INFORMATION SHEET

## "Attention Bias for Positive and Negative Sleep Stimuli in Insomnia"

Full Title: Attention Bias for Positively and Negatively Valenced Sleep-related Pictorial Stimuli in Psychophysiological Insomnia (PI), Delayed Sleep Phase Syndrome (DSPS), and Normal Sleep (NS): An Investigation using the Dot-probe Paradigm

You are being invited to take part in a research study being conducted within the Department of Psychological Medicine at the University of Glasgow. The study has been approved by the Faculty of Medicine within the University of Glasgow, and by the NHS Greater Glasgow and Clyde. Before you decide whether to take part, it is important to read the following information so you understand why the research is being done and what it will involve. Please take time to read the information carefully and consider whether or not you wish to take part.

#### What is the study about?

Sleep difficulties are very common - at least one in ten adults experience problems either getting to sleep at the desired time or staying asleep during the night. Not getting enough sleep can be upsetting during the night, but it can also make us feel tired and moody during the day.

This study aims to understand some of the reasons why people have difficulty sleeping. It is hoped that the findings from this study will contribute to more effective treatments for sleep problems being developed in the future.

#### What will I have to do?

If you agree to take part in this study, there will be a number of stages to your involvement. Firstly, you will be asked to complete a short task on a computer. This will involve responding to a number of photographs on the screen. This should take no longer than 15 minutes. The researcher will then ask you some questions about your general sleep patterns, and you will be asked to complete some short questionnaires which will tell us exactly which type of sleep problems you are currently experiencing. This part should take no longer than 20 minutes.

Depending on your answers to the questionnaires, you might then be asked to wear an 'Actiwatch' on your wrist for 7 days so that we can measure your sleep and wake patterns. There is a strong link between movement and being awake, and lack of movement and sleep. An actiwatch looks and feels just like a small wristwatch. However, instead of telling the time, it measures how much you are moving every minute, and stores this information in a small micro-processor inside. Once we have the actiwatch back from you, we will use a computer to 'read' the information to determine your sleeping pattern. You will also be asked to complete a simple, brief 'sleep diary' for each of the 7 nights. We can give you a summary of the information on your sleep if you would like.

Your total involvement in the study will be approximately 60 minutes.

# Do I have to take part?

Participation in this study is completely voluntary. You do not have to take part if you don't want to, and you do not have to give any reasons for why you don't want to take part. If you do decide to take part you, are still free to withdraw from the study at ANY point, and do not have to give any explanation for doing so. Any information that had been collected from you would then be destroyed.

# What are the benefits of taking part?

You will receive a through assessment of your current sleep patterns, and will be provided with some general advice to help improve your sleep. The information from this study may also help to develop more effective treatments for people with sleep difficulties in the future.

# What about confidentiality?

Your decision as to whether to participate in the study or not will remain entirely confidential. No-one other than the researcher will know that you are taking part in the study. Any information that you give throughout the course of the study will be kept in a locked cabinet and will be strictly confidential. All questionnaires and other data relating to you will be identified by number only, and there will be no chance that you will be recognised.

# What will happen to the results of this study?

The results of this study will be used as part of the main researcher's Doctorate in Clinical Psychology, and will be submitted for publication in a scientific journal. This can take around two to three years. You can check the Glasgow University sleep research website (<u>http://www.gla.ac.uk/sleepresearch</u> which contains up to date information of studies as they are completed. If you would like to see the final results, the researcher can provide you with a copy when the study is completed.

# If I decide to take part, what happens next?

You will be asked to arrange a time to come along to either the Department of Psychology at the University of Glasgow, or the Sleep Research Laboratory based at the Southern General Hospital, to complete the computer task. You will be given a copy of this information sheet, and if you decide to participate, a signed consent form to keep.

# Who can I contact if I want more information?

If you wish to discuss any of the information written in this sheet, or ask any other questions about the study, please do not hesitate to contact **Alison Murie** at the address given below, who will be happy to answer any questions you may have.

# Main Researcher:

Ms Alison Murie, Trainee Clinical Psychologist, Department of Psychological Medicine, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow, G12 0XH, Tel: 0141 211 3920

E-mail: <u>a.murie.1@research.gla.ac.uk</u>

### Supervisor:

Professor Colin A. Espie, Professor of Clinical Psychology, Department of Psychological Medicine, University of Glasgow. Director of Glasgow University Sleep Research Laboratory, Sackler Institute of Psychobiological Research, Southern General Hospital, 1345 Govan Road, Glasgow, G51 4TF.

# THANK YOU FOR TAKING THE TIME TO READ THIS

# Appendix 2.8 – Participant Consent Form





### PARTICIPANT CONSENT FORM

Participant Identification Number\_\_\_\_\_

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## **Please Initial Box**

- I confirm that I have read the "Participant Information Sheet" for the above study, and understand the information contained within it.
- I have had sufficient opportunity to ask the researcher any questions I may have about the study.
- I understand that participation is voluntary, and that I am free to withdraw from the study at any point, without giving a reason. All data relating to my participation will then be destroyed.
- I agree to take part in the above study.

Participant (signature)

**Researcher** (signature)

Date









# Appendix 2.9 - Illustration of a Single Dot-Probe Trial Sequence



