PREDICTORS OF SUBJECTIVE/OBJECTIVE SLEEP DISCREPANCY IN POOR SLEEPERS: EXAMINING DAILY ASSOCIATIONS USING MULTILEVEL MODELLING

A thesis submitted to the University of Manchester for the degree of Doctor of Clinical Psychology (ClinPsyD) in the Faculty of Biology, Medicine and Health.

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This thesis was completed by Vanessa Herbert for the degree of Doctor of Clinical Psychology at the University of Manchester. The thesis title is “Predictors of subjective/objective sleep discrepancy in poor sleepers: examining daily associations using multilevel modelling”. The thesis was submitted on 7th July 2016. It has been prepared in a paper based format.

Paper 1 is a systematic review of the impact of cognitive behavioural therapy for insomnia on cognitive performance. It has been prepared for submission to Sleep Medicine Reviews. A systematic search of the literature was conducted using the following online databases: EMBASE, PsycINFO, MEDLINE, MEDLINE In-Process, All EBM Reviews, CINAHL and the Cochrane Library. Eighteen studies met inclusion criteria. We found preliminary evidence for small to moderate effects of CBT-I on subjective measures of cognitive functioning. Few of the effects were statistically significant, likely due to small sample sizes and limited statistical power. There was a lack of evidence with regards to the impact of CBT-I on objective cognitive performance. We conclude that adequately powered RCTs, utilising both subjective and objective measures of cognitive functioning are required. Implications for future research are discussed.

Paper 2 is an empirical study of predictors of subjective/objective sleep discrepancy in poor sleepers. It has been prepared for submission to the journal *SLEEP*. The study combined seven days of actigraphy with daily assessment of sleep perceptions, self-reported arousal, sleep effort and mood upon awakening. High levels of intra-individual variability in measures of sleep discrepancy were observed. Multilevel modelling revealed that pre-sleep cognitive activity and mood upon awakening were significant and independently predictive of total sleep time misperception. Specifically, higher levels of pre-sleep cognitive activity and lower mood on awakening predicted underestimation of objective total sleep time. Higher levels of sleep effort predicted overestimation of objective sleep onset latency. The results suggest that naturally occurring day-to-day fluctuations in various psychophysiological variables are related to subjective/objective sleep discrepancy in poor sleepers.

Paper 3 is a critical appraisal of the systematic review and the empirical paper. The strengths and limitations of the methodologies used are discussed and areas for future research are suggested.

Declaration

No portion of the work referred to in the thesis has been submitted in support of an application for another degree or qualification of this or any other university or other institute of learning;

Copyright Statement

Paper 1: Systematic Review

The following paper has been prepared for submission to ‘Sleep Medicine Reviews’. The guidelines for authors can be found in Appendix A.

Title: Does cognitive behavioural therapy for insomnia improve cognitive performance? A systematic review and narrative synthesis

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Conflict of interest statement:

The authors report no conflicts of interest relating to material presented in this review. SDK has previously acted as consultant for Sleepio/Big Health Ltd.

Summary

Individuals with insomnia consistently report difficulties pertaining to their cognitive functioning. Perceived impairment in daytime functioning is typically what motivates individuals with insomnia to seek treatment. Cognitive behavioural therapy for insomnia (CBT-I) is a recommended treatment for insomnia and is associated with robust, long-term improvements in sleep parameters. Less is known about the impact of CBT-I on the daytime correlates of the disorder. A systematic review and narrative synthesis was conducted in order to summarise and evaluate the evidence regarding the impact of CBT-I on cognitive functioning. Reference databases were searched and studies were included if they assessed cognitive performance as an outcome of CBT-I, using either self-report questionnaires or cognitive tests. Eighteen studies met inclusion criteria, comprising 923 individuals with insomnia symptoms. There was substantial heterogeneity in study methodology. The standardised mean difference was calculated at post-intervention and follow-up. We found preliminary evidence for small to moderate effects of CBT-I on subjective measures of cognitive functioning. Few of the effects were statistically significant, likely due to small sample sizes and limited statistical power. There was a lack of evidence with regards to the impact of CBT-I on objective cognitive performance. We conclude that adequately powered RCTs, utilising both subjective and objective measures of cognitive functioning are required. Implications for future research are discussed.

Keywords: Insomnia, Cognitive Behavioural Therapy, CBT-I, cognitive functioning, narrative synthesis

Abbreviations

AFI	Attentional Function Index
ANT-I	Attentional network test – interactions task
AT	Autogenic training
BBTI	Brief behavioural treatment for insomnia
BPT	Behavioural placebo treatment
CBT-I	Cognitive behavioural therapy for insomnia
CT	Cognitive therapy
DSM-IV	Diagnostic and statistical manual of mental disorders, fourth edition
IPC	Information pamphlet control
ISI	Insomnia severity index
LM1	Logical memory test immediate recall
LM2	Logical memory test delayed recall
MFI-20	Multidimensional fatigue inventory
MMSE	Mini-mental state examination
NR	Not reported
NRT	Non-randomised trial
PMR	Progressive muscle relaxation
QR	Quality rating
RL	Relaxation
RT	Reaction time
PSQI	Pittsburgh sleep quality index
RCT	Randomised controlled trial
SC	Stimulus control
SH	Sleep hygiene
SHUTi	Sleep healthy using the internet
SMD	Standardised mean difference
SR	Sleep restriction
TONI	Test of nonverbal intelligence
USA	United States of America
UT	Uncontrolled trial

WASO	Wake time after sleep onset
WLC	Wait list control
WMS-CR	Wechsler memory scale – Chinese revision

Introduction

Insomnia is characterised by persistent difficulties with sleep initiation and/or maintenance. Insomnia is recognised as a 24-hour disorder, with impairments in daytime functioning and distress attributed to poor sleep being one of the core diagnostic criteria (1, 2). Traditionally considered as a secondary symptom, there is now evidence that insomnia is involved in the aetiology and maintenance of psychopathology (3) and is an independent risk factor for the development of cardiovascular disease (4) and diabetes (5). This has led to an acknowledgement of the need for attention to and treatment of insomnia, regardless of the co-occurring presence of mental or physical health problems (6).

Impairments in cognitive abilities are among the most commonly reported daytime symptoms of insomnia (7). Individuals with insomnia describe decrements in attention, concentration and memory which impact on decision making, social functioning and performance at work (8, 9) and leave individuals with insomnia feeling as though they are “struggling through the day” (10). Interestingly, subjective reports of cognitive deficits have not been consistently corroborated by objective measurements and a body of research assessing cognitive performance in people with insomnia compared to normal sleepers is marked by discrepant findings (11). A recent meta-analysis which combined the results of studies with small sample sizes concluded that insomnia is associated with mild to moderate impairments on tasks assessing working memory, episodic memory, problem solving, choice reaction time, information processing and selective attention (12). This review also highlighted the need for studies with greater statistical power and the assessment of a wider range of cognitive functions in order to improve our understanding of the nature and extent of cognitive impairment in insomnia. Uncertainty about the impact of insomnia on objective cognitive performance contrasts with research into the effects of experimental sleep loss, where findings have been more robust. Specifically, chronic sleep deprivation has been associated with deleterious effects across several cognitive domains, including memory, attention and executive functioning (13).

Evidence from prospective longitudinal studies indicates that chronic insomnia is an independent risk factor for cognitive decline (14, 15). It has also been suggested that the changes in sleep architecture that tend to occur with advancing age (more sleep fragmentation, earlier awakenings and less slow wave sleep) contribute to normal, age-related cognitive decline (16, 17). Sleep is known to play a role in brain plasticity and memory formation and therefore chronically disturbed

sleep is proposed to lead to impairment in cognition through its impact on brain function and brain health (18, 19). The majority of evidence for this comes from animal models of chronic sleep deprivation, which have demonstrated reductions in hippocampal cell proliferation and generation of new neurons (for review see (18)) . In humans, brain imaging studies have reported smaller hippocampi in those suffering from chronic insomnia (20, 21) and associations between hippocampi size and performance on a range of cognitive tests has also been demonstrated (20, 22), highlighting a potential pathway through which chronically disturbed sleep leads to cognitive deficits. Other studies report cortical atrophy in insomnia that might confer a risk for cognitive impairment (23).

Cognitive Behavioural Therapy for Insomnia (CBT-I) is a psychological treatment that aims to change the patterns of maladaptive thinking and behaviour which are proposed to maintain sleep difficulties (24, 25). Multiple reviews and meta-analyses support the efficacy of CBT-I in creating long lasting improvements in various indices of sleep, including sleep efficiency, sleep onset latency and wake-time after sleep onset (26, 27). When asked to describe the experience of insomnia it is usually the problems encountered during wake and the pervasive impact that sleep difficulties have on quality of life and wellbeing that are emphasised, as opposed to specific difficulties with the sleep initiation/maintenance (9, 10). Indeed, there is evidence that the perceived impact of sleep disturbance on daytime functioning is what prompts individuals with insomnia to pursue treatment (28, 29). It seems that medical advice is sought once a certain level of daytime dysfunction is reached, in the hope that treatment will remedy perceived impairments (30). Accordingly, treatment options for insomnia should aim to improve not only the sleep deficit, but also the daytime symptoms that contribute to the burden of the disorder (31). However, relatively little is known about the effects of CBT-I on daytime symptoms. Recent reviews have concluded that psychological treatment for insomnia is associated with improvements in quality of life (10, 32). However, quality of life is a broad concept, encompassing aspects of physical and psychological wellbeing. Moreover, quality of life is usually assessed using instruments which were not developed specifically for use in insomnia populations. Therefore, the impact of treatment on those aspects of daytime functioning which are known to be of particular concern in insomnia is not clear.

The aim of the current review was to conduct a systematic appraisal of the literature and evaluate the evidence for the effects of CBT-I on cognitive functioning. We chose to focus on cognition because impairments in this area are one of the most commonly endorsed daytime complaints (33,

34) and there is evidence from both subjective reports and objective measures, for cognitive deficits in individuals with insomnia compared to good sleepers. To our knowledge, the impact of CBT-I on cognition has yet to be evaluated systematically. This is in spite of the likely important contribution of this variable to many of the adverse consequences of insomnia, such as lowered work productivity, increased frequency of accidents (35) and declining health (36).

Method

Search strategy and selection

Online databases EMBASE, PsycINFO, MEDLINE, MEDLINE In-Process, All EBM Reviews, CINAHL and the Cochrane Library were searched from inception to 19th March 2016.

The review used a title and abstract word strategy, which included the following terms: (sleeplessness OR insomnia* OR "sleep initiation" OR "sleep maintenance" OR "sleep disorder*" OR dysomnia* OR "poor sleep*" OR "sleep problem*" OR "sleep disturbance*") AND ("cognitive behav* therap*" OR "behav* therap*" OR CBT OR CBT-I OR ICBT OR "self-help" OR "sleep hygiene" OR "stimulus control" OR "sleep restriction" OR relaxation OR "behav* modification" OR "cognitive therap*" OR imagery OR psychotherap*). Appendix B details the search strategy for each database. Reference lists of published meta-analyses and systematic reviews of CBT-I trials were also searched to locate additional papers (26, 37).

Citations were exported to reference management software and duplicates were removed. Titles and abstracts were reviewed. If the abstract indicated that the study evaluated a psychological treatment for insomnia, the full text was acquired in order to identify if cognitive functioning was measured as a treatment outcome. Relevant articles were then assessed against the inclusion and exclusion criteria.

For each of the articles identified, the reference list was searched and the article was entered into the "similar articles" search tool in PUBMED, in order to find additional relevant articles. Reviews, opinions, editorials, grey literature and conference abstracts were not included.

Criteria for inclusion of research articles

Empirical studies were included if they recruited an adult sample (18 years and over) with insomnia at either the disorder or symptom level, as defined by the Diagnostic and Statistical Manual for Mental Disorders (6) , the International Classification of Sleep Disorders (1), Research Criteria for Insomnia (2) or measures of insomnia severity such as the Insomnia Severity Index (ISI) (38) and the Pittsburgh Sleep Quality Index (PSQI) (39). Studies using samples where insomnia was comorbid with, or secondary to, a health or psychiatric condition were eligible for inclusion.

The treatment for insomnia was required to incorporate at least two of the five most widely accepted components of CBT-I; cognitive therapy, stimulus control, sleep restriction, sleep hygiene

and relaxation (40) (see table 1 for a description of the components). There was no requirement for the treatment to combine cognitive and behavioural components and we did not specify inclusion criteria on the basis of delivery modality or duration of treatment. Treatments which were modified for a particular population and/or comorbid condition were eligible. Studies in which the treatment comprised therapies from other treatment modalities (e.g. bright light therapy or hypnotic medication) were not included.

In order to be eligible the study was required to assess cognitive functioning as a treatment outcome. Studies were classified as having assessed cognitive functioning if they administered a measure which probed one of the following domains: attention, concentration, perception, memory, verbal functions, construction, concept formation, reasoning, executive functions or motor performance (41). Cognitive functioning could be assessed subjectively using standardised questionnaires or single items (e.g. visual analogue scales), or objectively using cognitive tests. Studies utilising subjective measures of cognitive functioning were inspected by three of the authors (VH, SK and DP) in order to consider if the subjective measure adequately assessed an aspect of cognitive functioning. A consensus was required in order for the study to be deemed as eligible.

As the literature is limited, all types of treatment evaluation study designs were included. Studies were required to be written in English and published in peer-review journals.

Table 1: CBT-I treatment components

Treatment (level of endorsement) (Morgenthaler et al., 2006).
Stimulus control therapy (standard)
Behavioural recommendations designed to reinforce the association between the bed or the bedroom and sleep, and to strengthen a consistent sleep-wake schedule: (a) go to bed only when sleepy; (b) get out of bed when unable to sleep; (c) use the bed for sleep only (no reading, problem-solving in bed); (d) arise at the same time every morning; (e) avoid napping.
Sleep restriction therapy (guideline)
A method that limits the time spent in bed as close as possible to the actual sleep time, thereby producing a mild sleep deprivation, which results in more consolidated sleep. The sleep window is gradually increased throughout a few days or weeks until optimum sleep duration is achieved.
Relaxation training (standard)
Clinical procedures aimed at reduction of somatic tension (e.g., progressive muscle relaxation, autogenic training) or intrusive thoughts (e.g. imagery training, meditation) interfering with sleep. Most relaxation techniques need professional guidance initially and daily practice for a few weeks.
Cognitive therapy (insufficient evidence as single therapy)
Psychotherapeutic method aimed at alleviating excessive worries and revising misconceptions about sleep, insomnia and daytime consequences. Specific targets include unrealistic sleep expectations, fear of the consequences of insomnia, and misconceptions of the cause of insomnia.
Sleep hygiene education (insufficient evidence as single therapy)
General guidelines about health practices (e.g. diet, exercise, substance use) and environmental factors (e.g. light, noise, temperature) that might promote or interfere with sleep: (a) avoid stimulants (e.g. caffeine, nicotine) for several hours before bedtime; (b) avoid alcohol around bedtime as it fragments sleep during the second half of the night; (c) exercise regularly, it can deepen sleep; (d) do not watch the clock; (e) keep the bedroom environment dark, quiet and comfortable.
Cognitive behavioural therapy (standard)
A combination of any of the above behavioural (e.g. sleep restriction, stimulus control instructions, relaxation) and cognitive procedures.

Reproduced from Morin & Benca, 2012. The Lancet.

Quality Assessment

The Downs and Black checklist (42) was used to assess the methodological quality of included studies. It includes 27 items assessing study reporting, external validity, internal validity and power in relation to randomised and non-randomised studies. It has adequate levels of internal consistency, good inter-rater reliability and high test-retest reliability (42). Item 27 relating to power was omitted due to the fact that cognitive functioning was a secondary outcome within the majority of the included studies and therefore power calculations were not based on this variable. Details of how the quality assessment tool was used to rate the included studies is presented in Appendix C. Quality assessment was performed by two researchers independently, for 17% of the included studies. There was a strong level of agreement between the researchers (Kappa = .90).

Data extraction

Each article was read in full in order to extract and record details of the included studies within a standardised extraction form. This form included the following information: authors and year of published report, methods (study design, assessment time points, blinding, procedure for randomisation), participant characteristics (study population, sample size, age, gender distribution), intervention details (components and mode of delivery), information about the control or comparator intervention and outcomes on a measure of cognitive functioning.

Data synthesis

Findings were combined and summarised in a narrative synthesis. Factors which may explain differences in the direction or size of effects across studies (e.g. study methodology) were considered and discussed, as per guidelines for conducting narrative synthesis (43) .

As part of the synthesis, quantitative data regarding effect sizes are presented. The standardised mean difference (SMD) was used as a measure of effect size and was calculated for studies utilising a randomised controlled trial design, providing the necessary post-treatment and follow-up data. Additional data was sought from authors where required. The SMD was derived by subtracting the mean for the control group from the mean for the treatment group and dividing the result by the pooled standard deviation. The direction of the values for the standardised mean difference was adjusted so that a positive value indicated better cognitive functioning in the treatment group compared to the control group at the post-treatment and follow-up time points.

The SMD is a summary statistic that is recommended for use when studies assess the same construct, but it is measured in a variety of ways across included studies(44). The SMD was calculated for the post-intervention and follow-up time points as opposed to change from baseline scores because the latter requires the studies to have reported the standard deviation of the change and this statistic is rarely reported. There is no advantage of SMD of change scores over SMD of the post-intervention scores in terms of baseline imbalances because both can be affected, however the SMD of post-intervention scores is thought to be a more conservative measure of effect size(45).

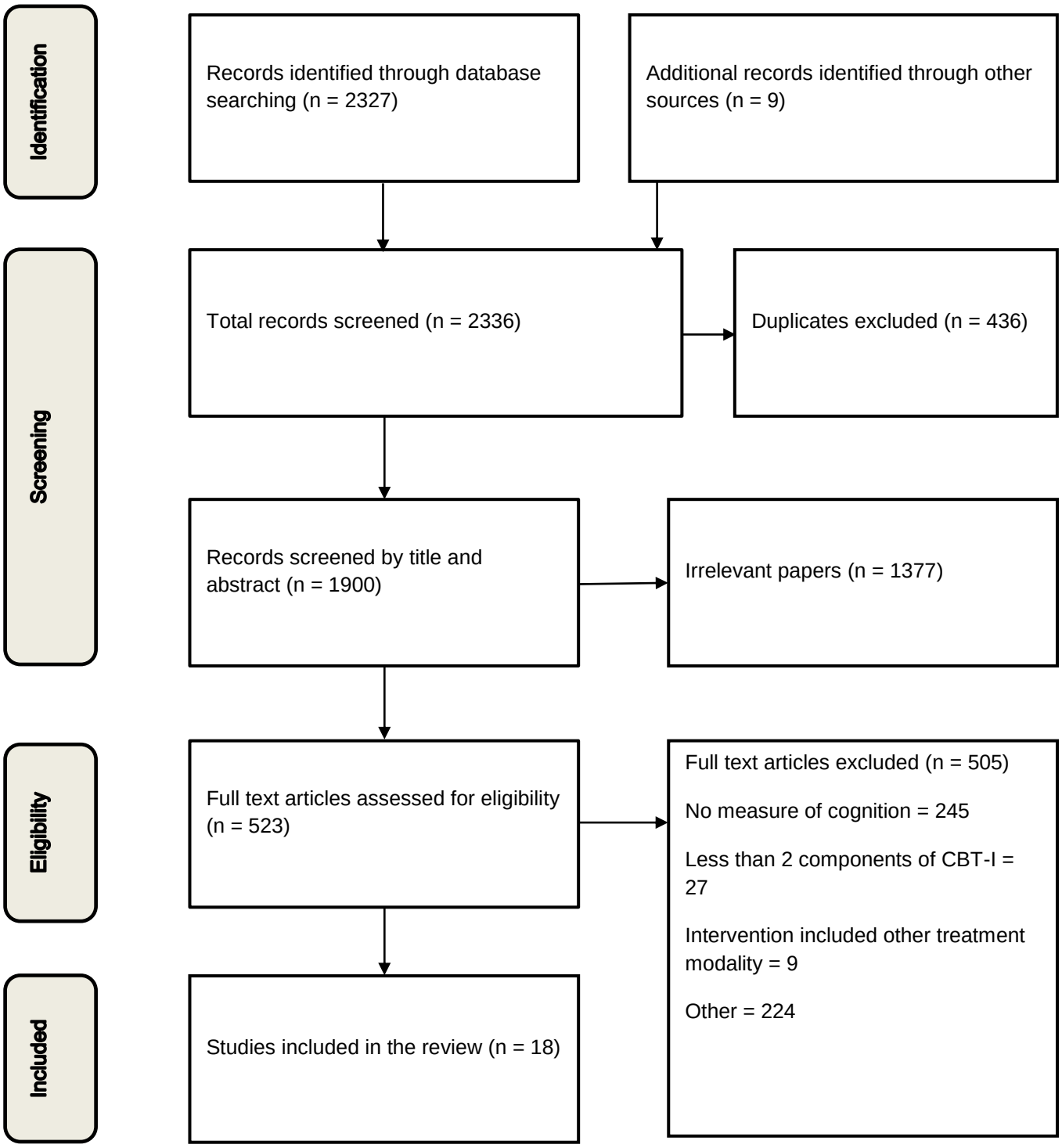
A meta-analysis was not considered appropriate for the current review due to the small number of studies available and the considerable heterogeneity in the methodology and outcomes of included studies.

Results

Study selection

Searches of databases and bibliographies yielded 2336 potentially relevant citations, of which 436 were duplicates and 1377 were deemed ineligible on the basis of title and abstract. Full text was retrieved for 523 studies and these papers were assessed against the inclusion and exclusion criteria. A final sample of 18 studies was included in the review. A flow chart of the study selection process is presented in figure 1.

Figure 1: Search results



Description of studies included within the review

Study design.

There were a range of study designs within the included studies; 11 RCTs (46-56), five uncontrolled trials (57-61), one non-randomised controlled trial (62) and one case series (63). Study characteristics are presented in table 2.

Table 2: Characteristics of studies

Author (year)	**Design**	**Population**	**Intervention: components of CBT-I (No. of participants) Mode of delivery No of sessions Duration**	**Comparator (No. of participants) Mode of delivery No of sessions Duration**	**Mean age % female**	**Length of follow up**	**Cognitive outcome**	**Summary of cognitive outcomes at follow up**	**QR score out of 26**
Arnedt et al. (2007) (57)	UT	Insomnia comorbid with alcohol dependence	CBTI-A: SR, SC, SH, CT, RP (7) Individual 4 face-to-face and 4 telephone 8 weeks	None	38.6 42.9%	4 and 8 weeks after baseline	MFI-20 Mental Fatigue	No significant improvement between baseline and mid-intervention or post intervention	12
Arnedt et al. (2011) (46)	RCT	Insomnia comorbid with alcohol dependence	CBTI-AD: SR, SC, SH, CT (9) Individual 4 face-to-face and 4 telephone	BPT (8) Individualised 4 face-to-face 4 telephone 8 weeks	CBTI-AD: 46.2 33.3% BPT:	8 weeks after baseline	MFI-20 Mental Fatigue	Scores were reduced at post-intervention for both groups.	19

			8 weeks		46.1 37.5%			At post-intervention the BPT group reported greater levels of mental fatigue than the CBTI-AD group, however differences were non-significant.	
Arnedt et al. (2013) (47)	RCT	Chronic insomnia	CBTI-Phone: SR, SC, SH, CT, RP (15) Individual 4-8 telephone 4- 8 weeks	IPC (15) Participants instructed to read and follow recommendations in a CBT-I pamphlet	CBTI-Phone: 38.1 100% IPC: 40.0 80%	8 and 12 weeks after baseline	MFI-20 Mental Fatigue	Scores were reduced at post-intervention for both groups and this remained at the 12-week	17

								follow-up. No significant difference between the groups at post-intervention or follow-up.	
Casault et al. (2015) (48)	RCT	Insomnia comorbid with cancer	mCBT-I: SC, SR, SH, CT, RP (20) Individual Self-help + 3 telephone consultations 6 weeks	No treatment control (18)	mCBT-I: 56.9 95% Control: 57.0 88.9%	6, 12 and 24 weeks after baseline	QLQ-C30 Cognitive Functioning	Significantly greater improvement in the mCBT-I group from pre-to post-intervention compared to the control group.	20
Davidson et al. (2001) (58)	UT	Insomnia comorbid with cancer	Sleep therapy: SC, SH, CT, RL, RP (14) Group	None	54.7 91.7%	4 and 8 weeks after baseline	QLQ-C30 Cognitive Functioning	Trend for an improvement in cognitive functioning.	11

			6 sessions 9 weeks						
Jansson & Linton (2005) (49)	RCT	Chronic insomnia	CBT: SC, SR, SH, CT, RP (64) Group 6 sessions 6 weeks	Self-help information containing modules on SC, SR and RL (72). Participants instructed to read and apply the material	CBT: 50.0 84% Control: 49.0 71% b	158 weeks after baseline	Concentration Difficulties (0-5)	Significantly greater reduction in concentration difficulties in the CBT group compared to the control group.	17
Lami et al. (2016) (59)	UT	Insomnia comorbid with fibromyalgia	CBT-I: SC, SR, SH, CT, RL, RP (39) Group 9 sessions 9 weeks	None	46.29 53.6%	9 and 21 weeks after baseline	MFI-20 Mental Fatigue	Significant improvement in mental fatigue across the three time points.	11
Matthews et al.	RCT	Insomnia in women	CBTI: SR, SC, SH, CT, RP (32)	BPT (28) Individual	CBTI: 52.1	6, 12 and 24 weeks	Attentional Function	Trend for a significantly	18

(2014) (50)		following treatment for breast cancer	Individual 4 face-to-face and 2 telephone 6 weeks	4 face-to-face and 2 telephone 6 weeks	100% BPT: 52.85 100% b	after baseline	Index	greater improvement from baseline to post-intervention and follow-up in the CBTI group relative to the BPT group.	
Miró et al. (2011) (51)	RCT	Insomnia comorbid with fibromyalgia	CBT: SC, SR, SH, CT, RP (22) Group 6 sessions 6 weeks	SH (22) Group 6 sessions 6 weeks	CBT: 43.9 100% SH: 50.2 100% b	6/7 weeks after baseline	ANT-I task	Significantly greater improvement in attention-alerting and attention-control in the CBT group compared to the SH group. No significant	17

								difference between the groups in change in overall RT or attention-orienting.	
Omvik et al. (2008) (52)	RCT	Older adults with chronic insomnia	CBT: SC, SR, SH, CT, RL (23) Individual 6-8 sessions 6-8 weeks	Zopliclone 7.5 mg (22) 6 weeks	CBT: 59.7 NR Zopiclone: 62.0 NR	6-8 and 30-32 weeks after baseline	Vigilance Test	Change in performance on this test was not significantly different between the groups.	15
Quesnel et al. (2003) (63)	Case series	Insomnia secondary to metastatic breast cancer	CBT: SC, SR, SH, CT, RP (10) Group 8 sessions 8 weeks	None	54.3 100%	8-10, 20-22, and 32-34 weeks after baseline	QLQ-C30 Cognitive Functioning	Significant improvement from pre to post intervention and no	10

								significant change from post-treatment to follow-up.	
Ritterband et al. (2012) (53)	RCT	Insomnia secondary to cancer	CBT-I: SC, SR, CT, RP (14) Internet 6 modules 6-9 weeks	Wait list control (14)	SHUTi: 53.7 100% WLC: 59.6 71.4%	9 weeks after baseline	MFSI-SF Mental Fatigue	Greater reduction in mental fatigue in the SHUTi group compared to WLC.	17
Simeit et al. (2004) (62)	NRT	Cancer patients with insomnia undergoing rehabilitation	Sleep management programme: SH, SC, CT, RL, RP. RL: PMR (80) or AT (71) Group 3 sessions 3-4 weeks	Standard rehabilitation (78) Group 3-4 weeks	PMR: 60.2 70% AT: 57.6 80.3 Control: 57.6	3/4, 9/10 and 27/28 weeks after baseline	QLQ-C30 Cognitive Functioning	Significant improvement between baseline and follow-up for all the groups. Change in scores was not significantly different	12

					75.6%			between the groups.	
Sun et al. (2013) (54)	RCT	Older adult poor sleepers	SH, RL (40) Group 4 sessions 4 weeks	SH (40) Brochure	Experimental: 68.6 70.3% Control: 70.8 73.3% b	12, 24 and 52 weeks after baseline	MMSE, WMS-CR	Scores on the MMSE and four subtests from the WMS-CR significantly increased in the experimental group whereas they decreased in the control group.	14
Swanson et al. (2013) (60)	UT	Insomnia comorbid with post-partum depression.	CBTI: SC, SR, SH, RL, CT, RP (16) Individual 5 sessions 5 weeks	None	30.0 100%	4 weeks after baseline	MFI-20 Mental Fatigue	Significant improvement	10

Taylor et al. (2014) (55)	RCT	Chronic insomnia in college students	CBT-I: SC, SR, SH, RL, CT (17) Individual 6 sessions 6 weeks	Wait list control (17)	CBT-I: 19.5 23.5% WLC: 19.9 58.8%	6 and 18 weeks after baseline	MFI-20 Mental Fatigue	No significant difference between the groups in change in mental fatigue.	19
Tomfohr-Madsen et al. (2016) (61)	UT	Insomnia in pregnancy	CBT-I: SC, SR, CT, RP (14) Group 5 sessions 5 weeks	None	31.0 100%	7 weeks after baseline	MFSI-SF Mental Fatigue	Significant improvement	12
Wilckens et al. (2016) (56)	RCT	Chronic comorbid insomnia in older adults	BBTI: SR, SC (42) Individual 2 face-to face sessions and 2 telephone 4 weeks	Information-only Control (40) Participants were instructed to read 3 publications containing information about behavioural treatment for	BBTI: 72.6 65.8% IC: 70.5 71.8% [b]	4 weeks after baseline	Logical Memory Test, TONI, Letter-number sequencing	No significant difference between treatment groups in change in cognitive performance between	12

				insomnia and received a telephone call after two weeks.				baseline and follow-up.	

Note. AT = autogenic training, BBTI = brief behavioural treatment of insomnia, BPT = behavioural placebo treatment, CT = cognitive therapy , IPC = information pamphlet control, NR = not reported, NRT = non-randomised trial, PMR = progressive muscle relaxation, QR = quality rating, RCT = randomised controlled trial, RL = relaxation, RP = relapse prevention, RT = reaction time, SC = stimulus control, SH = sleep hygiene, SR = sleep restriction, UT = uncontrolled trial, WLC = wait list control

[b] = data is for participants who completed the follow-up only.

Study quality.

The methodological quality of studies varied, with scores ranging between 10 and 20 out of a possible 26 (mean score = 15). The uncontrolled trials and the case series scored poorly due to the high risk of bias. There were only two studies that attempted to blind participants to the intervention. Few studies used intent-to-treat analysis in order to limit bias from attrition. In the studies that administered cognitive tests, there was no blinding of outcome assessment. When weighing up the evidence, more weight was given to the data from RCTs and higher quality studies. Full quality ratings for each of the included studies are shown in table S1.

Participants.

The sample size of the included studies ranged from seven to 229. There were a total of 923 individuals with insomnia symptoms in the eighteen included studies. Across the studies, the mean age of participants was 50.0 years and 71.0% of participants were female. This is comparable to findings from another meta-analyses of outcomes following CBT-I (26) and fits with research showing that insomnia symptoms are more common in women and with advancing age (64).

The criteria used to verify insomnia varied across studies. Half of the studies used the diagnostic and statistical manual of mental disorders fourth edition (65), fifth edition (6) or research diagnostic criteria (2) (47, 51-53, 55, 56, 59, 62, 63). Six studies used scores on the insomnia severity scale combined with self-report difficulties with sleep onset/maintenance (46, 48, 50, 57, 60, 61). One study used scores on the PSQI (54). Two studies developed their own criteria based on DSM-IV (49, 58).

The included studies sampled individuals from a range of populations. There were two studies of individuals with fibromyalgia (51, 59), six studies of cancer patients (48, 50, 53, 58, 62, 63), two studies of adults recovering from alcohol dependence (46, 57), one study of college students (55), three studies of older adults (52, 54, 56), one study of women with post-partum depression (60), one study of pregnant women (61) and two studies of adults without any psychiatric or physical health comorbidities (47, 49). In the majority of included studies (13/18), insomnia was considered “secondary” to, or comorbid with, a physical or mental health condition (46, 48, 50, 51, 53, 56-63). Of those studies that reported the duration of insomnia symptoms (10/18), mean values for the sample ranged from 3.9-174 months, with an average duration across studies of 75.83 months.

Interventions.

The majority of studies (16/18) delivered an intervention that combined cognitive, behavioural and educational components. There were two exceptions; the study by Wilckens et al. (56) which incorporated only the educational and behavioural components of CBT-I (sleep hygiene, stimulus control and sleep restriction) and the study by Sun et al. (54) which combined sleep hygiene with relaxation techniques. Many of the included studies failed to provide sufficient details of the intervention components to enable replication, particularly for sleep restriction.

A range of methods were used to deliver the intervention. Eight studies delivered the intervention in a group setting (49, 51, 54, 58, 59, 61-63). In three studies, the intervention was delivered by a clinician, in individual face-to-face sessions (52, 55, 60). Four studies combined individual face-to-face sessions with telephone consultations (46, 50, 56, 57). One study delivered the intervention via weekly telephone calls aided by written treatment modules (47). One study combined a self-help manual with three brief telephone consultations (48). One study utilised an online format (53). The length of treatment ranged from 3 to 9 weeks (mean length of treatment = 6 weeks). All of the studies indicated that a structured treatment protocol was used and eleven studies made explicit reference to the treatment being manualised (46-49, 51, 53, 55, 59, 61-63). Eight studies described efforts to ensure treatment fidelity (e.g. use of checklists, meetings between researchers, evaluation of recordings of treatment delivery) (46, 47, 50, 55, 56, 59, 61, 63).

It was common for the studies included in this review to adapt the intervention to address the links between comorbid or secondary conditions and sleep. The two studies of adults recovering from alcohol dependence adapted the intervention to include psychoeducation about the effects of alcohol use and withdrawal on sleep (46, 57). One of these studies also included cognitive restructuring of unhelpful thoughts and beliefs about relapse (46). A study in cancer patients provided psychoeducation about how to cope with fatigue using physical activity (63). Both of the studies in fibromyalgia patients provided psychoeducation about the link between fibromyalgia, pain and sleep (51, 59).

Control and comparative conditions.

Both passive and active control conditions were used. Two RCTs compared CBT-I to behavioural placebo treatment (46, 50). One RCT compared CBT-I to Zopiclone (52). Four studies used self-help information as a comparator (47, 49, 54, 56). One study compared CBT-I delivered in a group format to sleep hygiene delivered in a group format (51). One non-randomised controlled trial

compared sleep management training delivered within a standard cancer rehabilitation treatment to the rehabilitation treatment alone (62). Three RCTs compared CBT-I to no treatment (48, 53, 55).

Subjective cognitive outcome measures.

A summary of subjective outcome measures can be found in table 3. Eight studies used the mental fatigue subscale from the Multidimensional Fatigue Inventory (MFI-20) (46, 47, 53, 55, 57, 59-61). This subscale contains four items which assess cognitive symptoms of fatigue, such as difficulties concentrating ("When I am doing something, I can keep my thoughts on it", "I can concentrate well", "My thoughts easily wander" and "It takes a lot of effort to concentrate on things") (66). Four studies used the cognitive subscale from the European Organisation for Research and Treatment of Cancer QLQ-C30, which is an instrument that evaluates quality of life in cancer patients (48, 58, 62, 63). This subscale contains two items probing concentration and memory ("Have you had difficulties concentrating on things, like reading a newspaper or watching television" and "Have you had difficulty remembering things?") (67). One study used the Attentional Function Index (AFI) (50), which is a measure of perceived effectiveness in common activities requiring attention and working memory. The total score on this instrument is thought to provide an overall index of attentional functioning (68). One study used a Likert scale measure of difficulties with concentration (49).

Table 3: Summary of subjective measures of cognitive functioning

Measure	Author (Year)	Description/Subscales	Items/Scoring	Sample Items (cognition)	Psychometric properties
Multidimensional Fatigue Inventory	Smets et al. 1995	Designed to measure fatigue in cancer patients. Assessed general fatigue, physical fatigue, reduced motivation, reduced activity and mental fatigue.	24 item self-report questionnaire. Items are rated on a 7-point Likert scale. Participants are required to rate to what extent a particular item applies to them.	"When I am doing something, I can keep my thoughts on it", "I can concentrate well", "My thoughts easily wander", "It takes a lot of effort to concentrate on things"	Sample consisted of cancer patients receiving radiotherapy, patients with chronic fatigue syndrome, psychology students, medical students, army recruits and junior physicians (The Netherlands; Total *N* = 1469 (66)). Adequate internal consistency (Cronbach's α for mental fatigue subscale ranged from 0.77-0.93). Small convergent validity correlation with a VAS measure of fatigue (*r* = .23, p<0.001). Five factor

					model supported by the data.
European Organisation for Research and Treatment of Cancer QLQ-C30	Aaronson et al. 1993	Second generation questionnaire designed to assess health-related quality of life in cancer patients. Comprises five functional subscales (physical functioning, role functioning, emotional functioning, social functioning and cognitive functioning) and a global health and quality of life scale.	30 item self-report questionnaire. Items rated on a 4-point Likert scale. Participants are required to rate to what extent the item applies to them ("not at all", "a little", "quite a bit", "very much"). A scoring algorithm is applied, so that subscale scores range from 0 to 100, with higher scores indicating superior functioning.	Cognitive functioning subscale includes two items: "Have you had difficulties concentrating on things, like reading a newspaper or watching television" and "Have you had difficulty remembering things".	Sample consisted of lung cancer patients (From 13 countries, *N* = 305 (67)). Internal consistency was inadequate (Cronbach's α for cognitive functioning subscale was 0.56). Convergent validity correlation not reported.
Attentional Function Index	Cimprich et al. 2011	Designed to assess perceived effectiveness in daily activities supported by attention and working memory.	14 item self-report questionnaire. VAS format. Each item consists of a 100mm horizontal line anchored with "not at all" (0mm) and "extremely	"Remembering to do all the things you started out to do", "How hard you find it to concentrate on details", "How often you make mistakes on what you are	Sample consisted of women with breast cancer (USA; *N* = 32, (68)). Internal consistency was adequate (Cronbach's α ranged from 0.89 to 0.94

			well/great deal" (100mm). Participants are required to place a mark on a line to indicate their functioning. Total score is obtained by computing an average of the 16 items.	doing", "Getting easily annoyed or irritated".	when assessed over four different time points). Other forms of validity and reliability have not been assessed for this version of the questionnaire. Has demonstrated sensitivity to change in response to an intervention to improve attention in cancer patients (68) .

Objective cognitive outcome measures.

A summary of objective outcome measures can be found in table 4. The Attentional Network Test - Interactions task (ANT-I) was utilised by Miro et al (51). This is a computerised cognitive test which assesses three aspects of attentional functioning (orienting, alerting and executive functioning), as well as the interactions between these three networks (69). One study (52) used the VIGIL test which is a computerised test of sustained attention (70). Two of the included studies used neuropsychological tests to assess cognitive performance (54, 56). The study by Wilckens et al. (56) administered the Logical Memory Test to assess episodic memory (71), the Test of Nonverbal Intelligence III (TONI) to assess abstract reasoning and figural problem solving (72) and the Letter-Number Sequencing test to assess working memory (73). The study by Sun et al. (54) administered the Mini Mental State Examination (MMSE) (74) to assess global cognitive functioning and the Wechsler Memory Scale (WMS-CR) (75) to examine memory function.

Table 4: Summary of objective measures of cognitive functioning

Measure	Author/ Year	Description	Scoring	Psychometric properties
Attentional Networks Test – Interactions Task	Fan et al. 2002	Computer based task. Developed to assess the three attention networks (alerting, orienting and executive control). Participants are required to determine whether a central arrow (the target) points left or right. Initially a fixation cross is presented and is followed by an alerting signal (2000 Hz sound) on half of trials, an orienting cue (asterisk presented above or below the fixation cross) on two-thirds of the trials. The target and flankers are then displayed, either at the same location as the previous orienting cue or in the opposite location. The target and the flanker are congruent in half	The alerting effect is calculated by subtracting the mean RT of trials with an alerting cue from the mean RT of trials with no alerting cue. The orienting effect is calculated by subtracting the mean RT of trials with an orienting cue from the mean RT of trials with no orienting cue. Executive control was assessed by subtracting the mean RT of all congruent flanking trials from the mean RT of all incongruent flanking trials.	Test-retest reliability is adequate for each of the attentional networks (executive control r =. 77, orienting r =.61, alerting *r* =.52) (69). The authors did not uncover any research relating to the convergent validity.

		of trials.		
Vigil Test	Schuhfried, 2004	Computer based task. Developed to assess sustained attention. A dot moves along a circular path in small increments. When the increment is increased, the participant is required to press a button.	Mean RT in seconds and number of correct responses are calculated.	No published information available.
Logical Memory Test	Wechsler 1997b	Paper and pencil, neuropsychological task, taken from the Wechsler Memory Scale, version 3 (WMS-III). Participants are read two stories, each four sentences long and are required to recall the story immediately (LM1) and also after a 30 minute delay (LM2).	The number of key phrases accurately recalled is recorded. Normative data available for conversion to standardised scores.	Test-retest reliability is high (LM1 *r* = .74, LM2, *r* = .76) (76). Evidence for large practice effects, particularly in young adults (77). Demonstrated sensitivity to change to early cognitive changes in older adults (78).

Test of Non-Verbal Intelligence	Brown et al. 1997	Paper and pencil neuropsychological task. Language free measure of abstract reasoning. There are 50 items arranged in order of difficulty. Participants are presented with a set of symbols where an item is missing. They are required to identify the relationship between symbols and select which one fits the empty cell accordingly.	Each correct item is given a point. Normative data available for conversion to standardised scores.	Authors report evidence for reliability and convergent validity. Test-retest reliability has been demonstrated in school children (79). Studies of undergraduate students have provided evidence for concurrent validity (80).
Letter-Number Sequencing	Wechsler 1997a	Paper and pencil neuropsychological task which assesses auditory working memory. A series of letters and numbers are read to participants. They are then required to recall the digits in numerical order and the letters in alphabetical order.	Longest letter-number sequence. Normative data available for conversion to standardised scores.	Adequate test-retest reliability sample (r = . 70-.79) and internal consistency in the normative sample (Split half reliability coefficient = .80-.89) (81).

Wechsler Memory Scale – Chinese Revision	Gong et al. 1989	Paper and pencil neuropsychological task. Designed to evaluate various memory and working memory functions. Original version in English, with a normative sample from the American population. Adapted for the Chinese language.	Number of correct responses.	Test-retest reliability is adequate (*r* = .82).
Mini Mental State Examination	Folstein et al. 1975	Paper and pencil neuropsychological task. Brief screening measure of global cognitive functioning designed to evaluate the severity of cognitive impairment and assess cognitive change.	Number of correct responses out of 30.	Several studies have examined internal consistency and alpha levels range from 0.68 to 0.96. Test-retest reliability coefficient for healthy samples and cognitively impaired samples range from .80 to .95 (82).

Effect sizes for cognitive outcomes

The SMD could not be calculated for five studies due to lack of a control or comparator group (57-61). For one study, SMD at post-treatment and follow-up was not calculated due to non-random assignment to groups (62). It was not possible to calculate the SMD for the study conducted by Matthews et al. (50) due to insufficient data. Two studies reported follow-up data only (49, 54). SMD values at post-treatment and follow-up for subjective measures are presented in table S2 and SMD values for objective measures are presented table S3.

Narrative synthesis

Current evidence for the effect of CBT-I on subjective measures of cognitive functioning.

Seven RCTs administered a subjective measure of cognitive functioning. A significant improvement following the intervention compared to control was reported in three RCTs (48, 49, 53). The SMD at the post-intervention time-point was calculated for two of these studies (48, 53) (see figure 2). The study by Casault et al. (48) in cancer patients, demonstrated an improvement in scores on the cognitive functioning subscale of the QLQ-C30 at post-intervention, in the group that received minimal CBT-I compared to a no treatment control group. This difference was not maintained at the 12 or 24-week follow-up, due to improvement in scores in the control group. The study by Ritterband et al. (53) in cancer survivors reported a greater improvement in mental fatigue in the group that received internet delivered CBT-I (SHUTi) compared to control. Our calculations of SMD revealed no significant difference between the groups in mental fatigue at the post-intervention time-point. This discrepancy is likely due to the fact that post-intervention SMD does not take baseline scores into account. In this RCT, in spite of random allocation, there were significant differences between the groups at baseline in the variables sex and type of cancer, which were not adjusted for in the analysis. Moreover, participants in the SHUTi group displayed higher levels of mental fatigue than participants in the control group at baseline and therefore the significant improvement in the intervention group could be explained by regression towards the mean. Thus, the results from this study should be interpreted with caution.

The study by Jansson and Linton (49) in individuals with chronic insomnia, evaluated the impact of CBT-I on difficulties with concentration, one-year post intervention. They reported significantly greater

improvements in concentration between baseline and follow-up in the CBT-I group compared to control, however our calculations of SMD revealed that the groups were not significantly different at follow-up (see figure 3). Similarly to the study by Ritterband et al. (53), this discrepancy is likely due to the fact that post-intervention SMD does not take baseline scores into account. In this study there was a high level of attrition between randomisation and follow-up (17.6%), which may have introduced bias. The researchers reported that individuals who dropped out were not significantly different from those who completed the study in terms of demographic or clinical data (including concentration difficulties) and there was no differential loss of participants across the groups. However, an intention-to-treat analysis of the data was not performed and therefore potential bias from attrition is possible. The lack of statistically significant differences between the groups at follow-up may be explained by low sensitivity of the measure utilised and the long period of time between intervention and follow-up, during which the impact of the intervention may have diminished.

Four RCTs reported a non-significant difference between groups in change in subjective cognitive outcomes between baseline and post-intervention (46, 47, 50, 55). The study by Matthews et al. (50) in breast cancer survivors, demonstrated a trend towards a significant group x time interaction for scores on the AFI. The reported effect size was moderate (Cohens d = 0.56) and therefore the lack of statistical significance is likely due to insufficient power. Two studies using active controls (behavioural placebo treatment (BPT) and a CBT-I self-help information pamphlet), one in alcohol dependent patients and the other in individuals with chronic insomnia, showed significant improvements in mental fatigue between baseline and post-intervention for both the intervention group and the control group (46, 47). For both of these studies, improvements were larger in the intervention group however differences were not statistically significant. Finally, the study by Taylor et al (55) showed no effect of CBT-I on mental fatigue in College Students. The authors suggest that this may be due to possible floor effects. Mean score on the MFI-20 in this sample is comparable to the scores reported by other studies included in this review, therefore floor effects seem unlikely to explain the lack of differences.

There were five uncontrolled trials that administered a subjective measure of cognitive functioning (57-61). Three of these studies, one in Fibromyalgia patients, one in women with post-partum depression and one in pregnant women, reported significant improvements between baseline and post-intervention (59-61). A study in cancer survivors reported a trend for improved cognitive functioning

(58) and a study in alcohol dependent patients reported no significant change in mental fatigue following CBT-I. The studies that did not find a statistically significant improvement had small samples (*N* = 14 and *N* = 7). The case series conducted by Quesnel et al. (63) in women who had been treated for non-metastatic breast cancer, reported a significant improvement in score on the QLQ-C30 between baseline and post-intervention, which was maintained at follow-up. The non-randomised controlled trial reported by Simeit et al. (62) in cancer patients undergoing an oncological rehabilitation treatment programme, reported that all groups showed significant improvements in self-report cognitive functioning between baseline and follow-up. There were no significant group x time interactions in this study.

The weight of evidence from the non-randomised control trial, case series and uncontrolled trials point to positive effects of CBT-I on subjective measures of cognitive functioning. However the risk of bias in these studies is high. Evidence from the RCTs included in this review is mixed and there was only one RCT (48) that demonstrated a significant difference between groups at the post-intervention time point. There is a great deal of heterogeneity within the included studies and factors that differentiate between those RCTs that report significant improvements in cognitive functioning following the intervention, and those RCTs that report non-significant findings, are not clearly discernible. A high proportion of the included studies that administered a subjective measure of cognitive functioning were conducted in individuals who had recovered from, or were in the process of recovering from, cancer. The majority of these studies reported significant improvements in subjective cognitive functioning following CBT-I or a trend for significance. It is possible that enhancements in perceived cognitive functioning following CBT-I are more likely to be observed in cancer patients or survivors of cancer. It is known that cancer and the treatments for cancer can lead to cognitive impairment (83) and this group may be more susceptible to cognitive deficits following disturbed sleep as a result of an already existing level of cognitive impairment related to cancer and/or cancer treatment, In addition, this group may display greater levels of concern about their cognitive performance and therefore improvements in subjective cognitive functioning may be more easily identified following an intervention.

Our SMD calculations suggest that on the whole, CBT-I is associated with larger improvements in subjective measures of cognitive functioning compared to control, however confidence intervals are large, reflecting the small sample size and low statistical power (eight out of the eleven RCTs had *N* ≤

60 which means that studies would only have the ability to detect an effect size *f* of 0.37), which limit the conclusions that can be drawn.

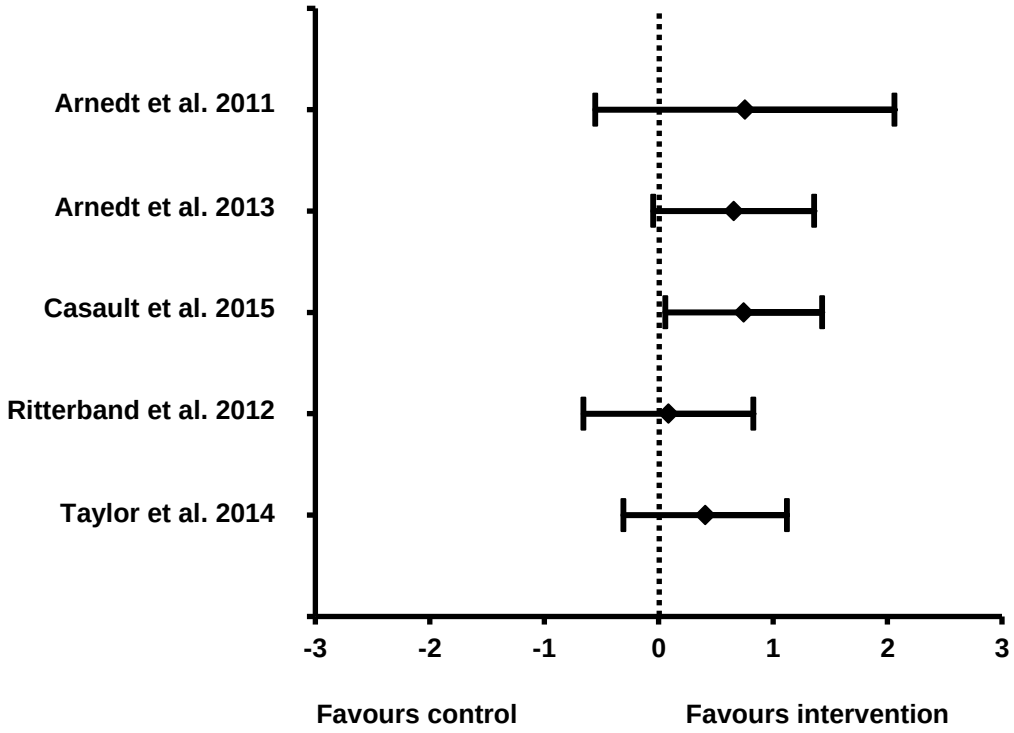


Figure 2: Post-intervention SMD and confidence intervals for subjective measures of cognitive functioning

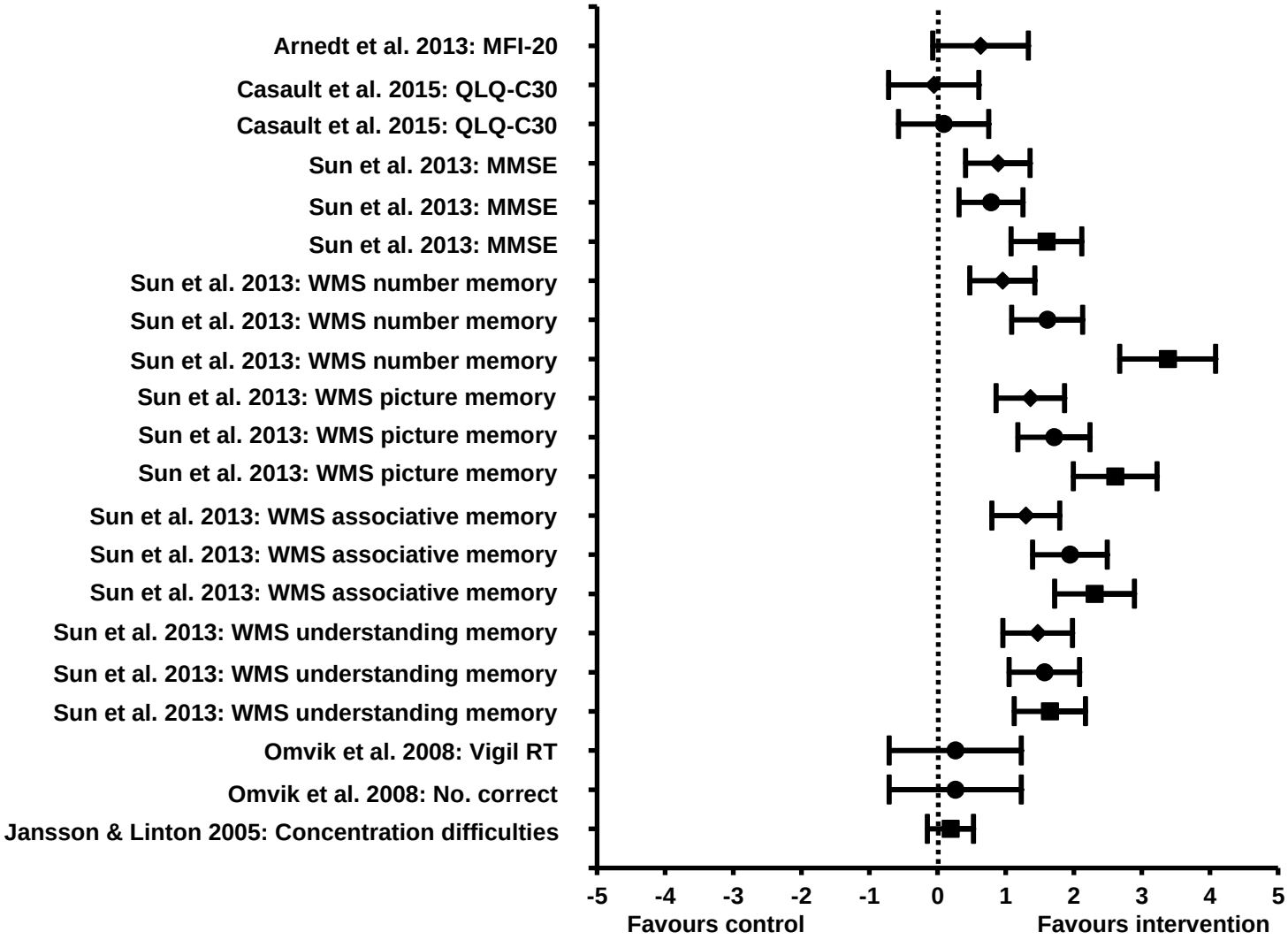


Figure 3: Follow-up SMD and confidence intervals for cognitive outcomes. Diamonds represent 12-week follow-up, circles represent 24-32 week follow-up and squares represent 52-58 week follow-up.

Current evidence for the effect of CBT-I on objective measures of cognitive functioning.

Four RCTs evaluated the impact of CBT-I on objective measures of cognitive functioning (51, 52, 54, 56). Two of these studies reported positive findings (51, 54) and two studies reported null findings (52, 56). SMD for objective measures at post-intervention are presented in figure 3. The study by Miro et al. (51) in individuals with Fibromyalgia found greater improvements in two aspects of attention (alerting and executive control), in the intervention group compared to the control group. Our calculation of SMD revealed no significant difference between the groups on any aspect of performance on the ANT-I task at the post-intervention time-point. This discrepancy may be due to baseline differences between the groups in age, which were adjusted for in the statistical analysis performed by Miro et al. The study by Sun et al. (54) in older adults, reported improvements in scores on the MMSE and the WMS-CR following the intervention, which became larger at subsequent follow-ups. These findings were corroborated by our calculations of SMD, which showed statistically significant differences between groups on scores on the MMSE and WMS-CR at each of the follow-up time-points (12, 24 and 52 weeks) (see figure 3). In this study, participants were provided with sleep hygiene information and were taught relaxation skills which they were encouraged to practice both individually and within groups, for a period of one year. Performance on cognitive tests was compared to a group that received sleep hygiene information only. The large effects observed in this study may be explained by non-specific effects of the intervention such as participant expectations of improvement and the increased social contact provided by group relaxation practice. The Researchers administering the neuropsychological tests were not blind to group assignment, which may have led to an exaggeration of treatment effects through ascertainment bias. In addition, the control group displayed worsening performance on cognitive tests over the course of the study, which may be due to age-related cognitive decline. The impact of CBT-I on objective cognitive performance may be greater in populations with declining cognition.

The study by Wilckens et al. (56) examined the impact of brief behavioural treatment for insomnia (BBTI) on cognitive functioning in older adults. BBTI focuses on the behavioural aspects of CBT-I and utilises stimulus control and sleep restriction. In this study they found no significant difference between the groups on change in performance on the cognitive tests. One possible reason for null findings is the short duration between baseline and post-intervention (4 weeks). A decrease in TST across groups

was reported which in the intervention group, is likely a result of sleep restriction. Sleep restriction therapy has been associated with increases in daytime sleepiness and impairments in vigilance during the acute phase (84). Therefore, the side-effects of sleep restriction may have impacted on performance on cognitive tests, contributing to null findings. In addition the time of day at which the cognitive tests were administered was not controlled for, and this may have introduced additional variance, possibly obscuring the effects of the intervention (85).

The study by Omvik et al. (52) compared the effects of CBT-I and Zopiclone on sustained attention, in older adults. They found the number of correct responses on the Vigilance Task significantly worsened in the CBT-I group between baseline and post-intervention. Again, this may be related to the short-term effects of sleep restriction therapy (84). There were no significant group x time interactions for any index of performance on the Vigilance Task, either at post-intervention or follow-up. This study had a very small sample size (*N*=45) and an active control. Therefore, the lack of group x time interactions may reflect insufficient statistical power to detect small effects. The authors did not account for participants who dropped out of the study and therefore attrition bias may also be a factor.

We identified few studies which evaluated the impact of CBT-I on objective measures of cognitive functioning. The evidence from the studies included in this review is mixed. Heterogeneity in the nature of the tasks used to probe cognitive functioning may contribute to the disparate findings. Specifically, there is evidence that individuals with insomnia show impairments in specific cognitive domains (12) and it is possible that those studies which found significant effects, examined cognitive functions that were more likely to be impaired at baseline. Confidence intervals for SMD at post-intervention and follow-up tend to be smaller for objective measures of cognitive functioning compared to the subjective measures of cognitive functioning, reflecting more precise measurement of constructs.

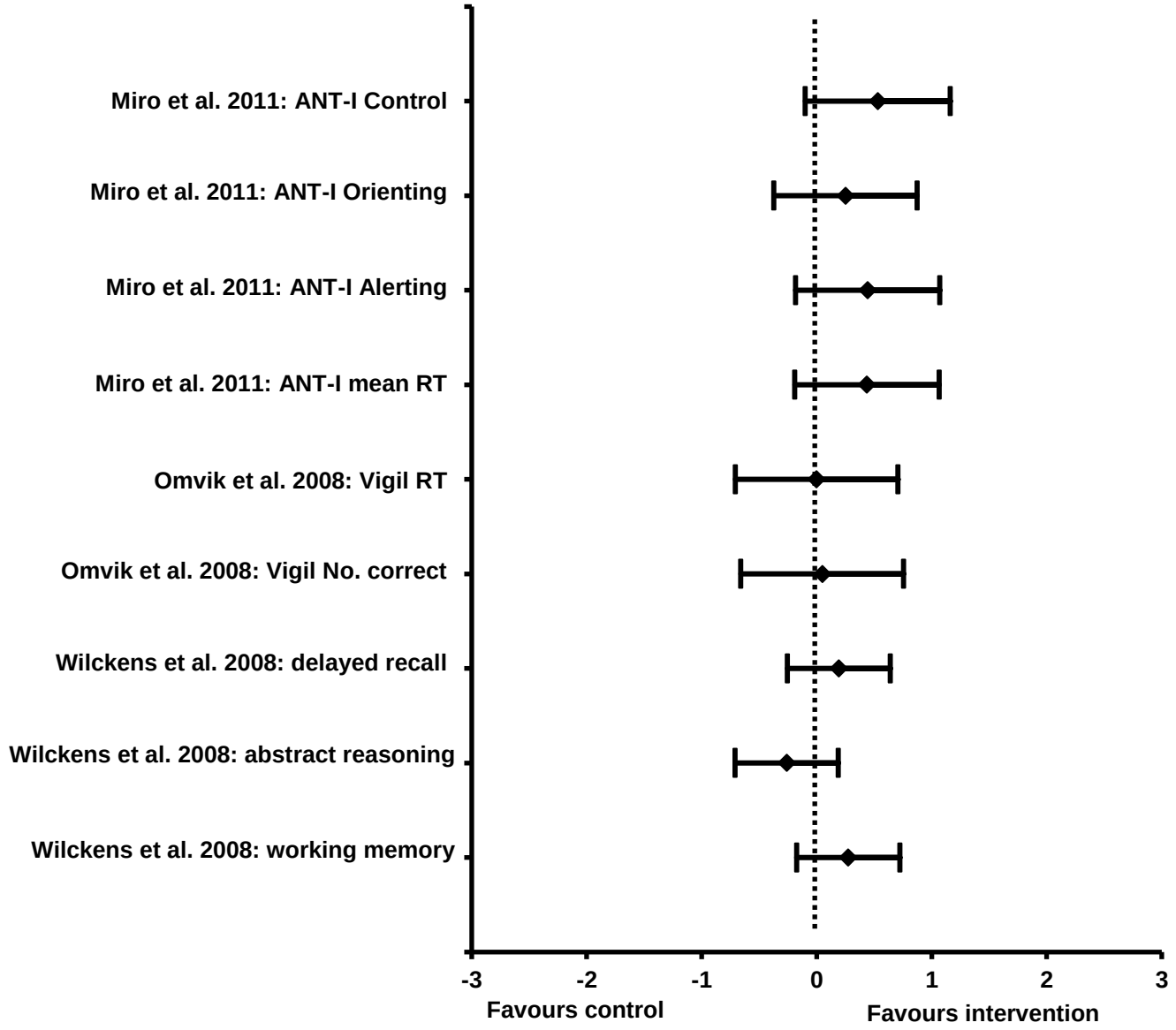


Figure 4: Post-intervention SMD and confidence intervals for objective measures of cognitive functioning

Relationship between sleep and cognitive functioning.

There were three studies that examined the relationship between changes in sleep and changes in cognitive functioning, from pre to post-intervention (51, 52, 56). The study by Omvik et al. (52) reported no significant associations between change in sleep efficiency or slow wave sleep and change in performance on the vigilance task. The study by Wilckens et al. (56) calculated correlations between change in performance on cognitive tests and change in WASO, delta power (absolute and relative) and sigma power (absolute and relative). They found a significant positive association between increased absolute delta power and improved abstract reasoning. In addition, decreased relative sigma power was significantly associated with improvements in abstract reasoning. Neither of these associations was moderated by treatment group. There were no significant associations between change in sleep and change in performance on delayed recall or working memory tasks. On the basis of significant associations between delta power and abstract reasoning, Wilckens et al. concluded that more condensed and continuous sleep is associated with better performance on tasks tapping executive functioning in older adults however BBTI does not appear to do this differentially to control.

Miro et al. (51) calculated the association between change in sleep quality and change in performance on the ANT-I task. They found a significant positive association between sleep quality and performance on the attention control index from the ANT-I, which is proposed to be a measure of executive functioning. There were no significant associations between change in sleep quality and any other index of the ANT-I. On the basis of these results, Miro et al. concluded that improvements in sleep quality are associated with better executive functioning in individuals with fibromyalgia.

Associations between changes in sleep and changes in cognitive functioning were exploratory outcomes in these trials and therefore there was limited correction for multiple testing, which increases the chances that some of the significant associations uncovered are due to type-one error. There were no studies that conducted mediation analysis. The associations reported do not provide evidence with regards to the direction of the effect and experimental manipulations are required in order to determine whether there are specific aspects of sleep architecture that are causally related to cognitive performance. In spite of these limitations, these findings do highlight possible variables to target in future treatments seeking to deliver sleep-related cognitive improvement.

Discussion

Summary of evidence

The aim of this review was to summarise the literature and evaluate evidence for the effects of CBT-I on cognitive functioning. Using broad inclusion criteria, we identified 18 relevant studies. For the majority of these studies, cognition was a secondary outcome and assessed within a general measure of self-reported daytime functioning. Only four of the included studies administered an objective measure of cognitive functioning and across these studies, the range of cognitive domains examined was limited. The lack of research into the impact of CBT-I on cognitive functioning is surprising, given that impairments in concentration and memory are among the most commonly reported and most troubling daytime symptoms of insomnia (34).

On the basis of post-intervention and follow-up SMD, the majority of studies showed small to moderate effects of CBT-I on cognition. Three of the included studies reported statistically significant improvements, which our calculation of SMD at post-intervention/follow-up did not corroborate. Baseline imbalances resulting from small sample sizes or failure in randomisation may have contributed to the reported significant improvements in the intervention group compared to the control group. The discrepancy in findings may also reflect limitations of utilising the SMD of post-intervention/follow-up scores, as opposed to change in score from baseline.

The included studies generally had small samples with limited statistical power to detect less than large effects. There was one high quality RCT, which incorporated an active control condition, participant blinding and utilised intent to treat analysis to minimise bias from attrition (50). This study found a trend for a significant effect of CBT-I on self-report attentional function, with a moderate effect size. On the basis of this study and other lower quality RCTs and uncontrolled trials, there appears to be preliminary evidence for a small to moderate size effect of CBT-I on subjective measures of cognitive functioning. The impact of CBT-I on objective measures of cognitive functioning is more equivocal, predominantly due to lack of research. It is clear that further adequately powered RCTs, utilising both subjective and objective measures of cognitive performance are required, before firm conclusions can be drawn.

In terms of maintenance of the effects, there were nine studies that conducted a follow-up assessment of cognitive functioning, three to six months following the intervention. The majority of these studies

found no significant difference between post-intervention and follow-up scores, indicating that where there were initial gains, these tended to be maintained. In one study, performance on the vigilance test worsened between baseline and post-intervention but improved between post-intervention and follow-up (52). This may be related to the sleep restriction component of the intervention, which has been associated with increased daytime sleepiness and impairments in vigilance during the acute phase of implementation (84, 86). There is evidence that improvements in sleep parameters following CBT-I evolve over time (87) and therefore it is possible that improvements in cognitive performance also take time to be manifest. Thus the timing of assessments is an important consideration for future studies, especially where sleep restriction is part of the treatment.

Study quality and sources of bias

The risk of bias from inadequate control and lack of blinding in the included studies is high. Although double-blinded placebo controlled studies are difficult to implement when evaluating psychological interventions, a sham insomnia treatment (known as behavioural placebo treatment) has been developed (88). Indeed, behavioural placebo treatment was utilised as a control in two of the included studies (46, 50). However, the majority of studies in this review used inactive control conditions and participants were not blind to group assignment. Therefore, the specific effects of CBT-I cannot be separated from non-specific effects, such as therapist attention and participant expectation of benefit. The issue of expectancy may be particularly relevant when assessing cognitive functioning, since expectancy effects have been shown to exert a strong influence on objective measures (89) and subjective perceptions of cognitive performance (90).

There were five uncontrolled trials and one case-series included within this review. Three of these studies reported effect sizes, all of which were large effects. Meta-analyses of outcomes following psychological interventions commonly observe that uncontrolled studies report larger effects than RCTs (91, 92). This is likely due to biases that result from issues such as regression to the mean. Due to the high risk of bias, we gave less weight to results from the uncontrolled trials and case-series, when considering the evidence in this review. Nevertheless, the evidence from uncontrolled trials largely supports data from the RCTs, indicating positive effects of CBT-I on self-report measures of cognitive functioning.

Mechanisms

There were three studies that investigated relationships between changes in sleep and changes in cognitive functioning. Omvik et al. (52) found no significant association between change in any sleep parameter and change in performance on the Vigilance Test. Wilckens et al. (56) concluded that more consolidated and continuous sleep (as indicated by lower PSG defined WASO and increased absolute delta power) is associated with better executive functioning in older adults, although this was not moderated by the BBTI intervention. Miro et al. (51) reported that improvements in self-reported sleep quality following CBT-I were associated with better performance on a task assessing executive functioning. Together, these results suggest that enhanced sleep continuity, experienced subjectively as better sleep quality (93), is related to superior executive functioning.

These findings support research demonstrating that individuals with insomnia show impairments on complex rather than simple tasks. This is thought to result from greater dependence of these tasks on functioning of the prefrontal cortex (12). There is evidence for hypo-activation of the prefrontal cortex during performance of a category fluency task in individuals with primary insomnia compared to controls. Intriguingly, this hypo-activation was attenuated following sleep therapy (which comprised CBT-I, bright light therapy and body temperature manipulations), with concomitant improvements in phonemic fluency (94). Clearly if CBT-I does lead to improvements in cognition, it will be important to discover the mechanism through which this occurs. One hypothesis which deserves further investigation, is that improved sleep continuity and normalisation of sleep architecture leads to enhanced functioning of the prefrontal cortex and subsequent improvements in those cognitive domains that recruit this brain region (95).

Fatigue, anxiety and low mood, which are often present in insomnia (particularly comorbid insomnia), have also been associated with cognitive impairment (41). There is evidence that CBT-I leads to improvements in these variables (96-99) and therefore, in addition to investigating the potential mediating role of various sleep parameters, future studies should also examine the contribution of changes in secondary psychological variables.

Measures of cognitive functioning

Discordance between self-report measures of cognitive functioning and performance on neuropsychological and cognitive tests has been demonstrated in individuals with primary insomnia (100). Specifically, self-report measures tend to show higher levels of impairment than is found using objective assessment. This discordance is thought to be driven by selective attention to the daytime consequences of insomnia, a phenomenon which is proposed to contribute to the maintenance of the disorder (101). It is possible that CBT-I impacts on subjective and objective measures differentially, for example it may alter perceptions of impairment without impacting on objective performance. In addition, it is possible that there are different mechanisms through which CBT-I impacts on subjective and objective cognition. We were not able to examine this in the current review because there were no studies that investigated cognitive functioning using both subjective and objective measures. It was not possible to compare the effects of CBT-I on subjective and objective measures across studies, due to heterogeneity. Future studies should consider the inclusion of both subjective and objective assessments of cognitive function, in order to enable such a comparison.

In spite of guidelines espousing the importance of investigating the daytime correlates of insomnia, there are no specific cognitive performance measures or self-report questionnaires that are recommended for routine use in insomnia samples (102). A meta-analysis (12) concluded that insomnia is associated with impairments on tasks assessing episodic memory, problem-solving, working memory and several aspects of attention (choice reaction time, information processing and selective attention). Performance of those with insomnia was comparable to normal sleepers for other aspects of attention (alertness, divided attention, sustained attention, vigilance), perceptual and psychomotor processes, verbal functions, procedural memory, some aspects of executive functioning (verbal fluency, cognitive flexibility) and general cognitive functioning. A caveat to these findings is that for some of the cognitive domains examined, there were few studies available and therefore low statistical power may explain lack of group differences. Accordingly, assessment of a broad range of cognitive domains is encouraged. However, where this is not feasible, researchers should focus on the assessment of those cognitive domains where impairments in insomnia samples has been demonstrated most robustly. In addition, future studies should consider the impact of time of day on cognitive performance and seek to standardise this variable across participants and test sessions (85).

There are a number of factors which require consideration with regards to the selection of tools for assessing changes in cognitive performance, in individuals with insomnia. Clearly the test needs to demonstrate sufficient sensitivity to enable the detection of potentially subtle changes over time. There appears to have been little consideration of this issue by the studies included within this review. Individuals with insomnia may compensate for deficits through increased cognitive effort and the recruitment of additional cerebral resources (100). Such compensation may obscure both objective deficits and improvements over time. Accordingly, it seems important to include cognitive tests which are sufficiently difficult to overwhelm compensatory processes. Alternatively, future studies could combine manipulations of task difficulty with neuroimaging, so that the role of compensatory mechanisms can be elucidated (103).

The subjective outcome measures utilised in the included studies were developed for use in clinical samples, predominantly cancer patients. The validity of using these measures to assess cognitive functioning following an insomnia intervention is questionable. The experience of cognitive impairment related to insomnia may be qualitatively different to that associated with a physical health problem such as cancer. There are no subjective measures of cognitive functioning that have been developed for individuals with insomnia. This should be addressed in future research.

Heterogeneity

Meta-analyses have highlighted various factors which moderate sleep outcomes following CBT-I. These include rate of attrition, length of treatment, level of personal support, method of treatment delivery and duration of insomnia symptoms (104, 105). Due to the preliminary nature of the literature and the large number of ways in which the included studies varied, it was not possible to identify specific moderators of improvements in cognitive performance. A large proportion of the included studies were conducted in cancer patients and the majority of these studies reported a significant improvement in subjective measures of cognitive functioning following CBT-I, or a trend for statistically significant improvement. However, it was not possible to draw any strong conclusions on the basis of study population because there was insufficient variability across the included studies to enable valid subgroup comparisons. It is possible that particular characteristics of an individual with insomnia promote greater improvements in cognitive functioning following treatment. One characteristic that should be examined is cognitive reserve. There is evidence that older adults with less cognitive reserve

(as indicated by lower levels of education) are more vulnerable to the negative effects of poor sleep on some aspects of cognitive functioning (106). This is thought to result from less capacity for compensation. Improvements in cognitive functioning may therefore be more easily identified in this group. The nature of the insomnia complaint may also moderate outcomes. Specifically, there is evidence that those with objective short sleep duration show greater deficits in cognitive performance (107, 108) and therefore we might expect to see greater improvements in cognitive functioning in this group, following an intervention.

Limitations of the current review

This review has a number of limitations. First, a high proportion of the included studies were from cancer patients, which limits the generalisability of our synthesis. Second, the inclusion criteria used were broad, which resulted in studies that differed on a range of factors, including methodology, population studied, aspects of the intervention and outcomes measures. Such high levels of clinical and methodological diversity precluded the use of meta-analysis and also meant that we were not able to clearly identify factors that contributed to the heterogeneity in outcomes. Third, we included uncontrolled trials and studies with low methodological quality, but resisted drawing conclusions on the basis of their findings, due to high risk of bias. It could be argued that there was little benefit to including these studies however we reasoned that it was necessary to summarise all of the available evidence, given that this is an area of very limited research. Fourth, we combined studies that were very different in terms of content of intervention and method of delivery. For example, we included studies where the intervention combined just two components of CBT-I (relaxation and sleep hygiene), with studies that delivered all of the components. The validity of this is questionable. We did not conduct a meta-analysis and therefore the results from heterogeneous studies were not statistically combined. We suggest that high levels of variability in terms of the interventions delivered is reflective of the current state of the field, whereby there is a lack of clarity concerning the necessary and sufficient components of a CBT-I intervention.

Conclusions

There is preliminary evidence for small-to-moderate effects of CBT-I on self-report cognitive functioning. Included studies tended to have small sample sizes and many failed to find statistically

significant results, therefore adequately powered RCTs are required before firm conclusions can be drawn. There is a distinct lack of research with regards to the effects of CBT-I on objective cognitive performance, which is surprising given the importance of cognition for everyday functioning. A consensus with regards to specific cognitive domains that should be assessed in insomnia samples should be a priority for researchers. There was substantial heterogeneity in the included studies, reflecting the broad inclusion criteria used and the preliminary nature of this research area.

Practice Points

- Impairments in cognitive functioning are among the most commonly reported and troubling daytime symptoms of insomnia, however little is known about whether insomnia treatments lead to improvements in cognitive performance.
- There is preliminary evidence for small to moderate positive effects of CBT-I on self-report cognitive functioning however adequately powered RCTs are required before firm conclusions can be drawn.
- There is a lack of evidence with regards to the impact of CBT-I on objective cognitive performance.

Research Agenda

Further research should aim to:

- Establish a battery of cognitive tests to be used in insomnia samples.
- Evaluate the effects of CBT-I on cognitive functioning, using a well powered and high quality study design, including both subjective and objective measures.
- Compare the effects of CBT-I on subjective and objective measures of cognitive functioning.
- Investigate the mechanisms through which CBT-I leads to improvements in cognitive functioning and establish whether this is different for subjective and objective outcomes.
- Identify variables that enhance improvements in cognitive functioning

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Table S1: Quality ratings

Criteria	Arnedt et al. 2007	Arnedt et al. 2011	Arnedt et al. 2013	Cassault et al. 2015	Davidson et al. 2001	Jansson & Linton, 2005	Lami et al. 2016	Matthews et al. 2014	Miro et al. 2011	Omvik et al. 2008	Quesnel et al. 2003	Ritterband et al. 2012	Simeit et al. 2004	Sun et al. 2013	Swanson et al. 2012	Taylor et al. 2014	Tomfohr-Madsen et al. 2016	Wilkens et al. 2016
1.Hypothesis/aim clear?	1	1	1	1	1	1	1	1	1	0	1	1	0	1	1	1	1	1
2. Main outcomes clearly described?	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3. Participants clearly described?	1	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1	1	1

4. Interventions clearly described?	1	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1	1	0
5. Distribution of principal confounders clearly described? Age, severity (0,1,2)	0	2	2	2	0	1	0	2	2	2	0	2	2	1	0	2	0	0
6. Main findings clearly described?	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

7. Estimates of random variability provided?	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1
8. Adverse events reported?	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
9. Characteristics of participants lost to follow up reported?	1	1	0	0	0	1	0	1	0	0	0	1	0	0	0	1	1	0	

10. Actual probability values reported?	1	0	0	1	1	1	1	1	1	1	1	1	0	1	0	1	0	1
11. Sample approached representative?	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
12. Participants representative?	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
13. Treatments representative?	0	0	1	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0

14. Attempted blinding – participants?	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
15. Attempted blinding – assessors?	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0
16. Unplanned analyses?	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1	1	1	0	
17. Time period same for cases and controls?	0	1	0	1	0	0	0	1	1	1	0	0	1	1	0	1	0	1	

18. Statistical tests appropriate?	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
19. Was compliance with the intervention reliable?	0	1	1	0	0	1	0	0	0	1	0	1	0	0	0	1	1	1	
20. Main outcomes valid & reliable?	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1

21. Intervention and control from same population?	0	1	1	1	0	1	0	1	1	1	0	1	1	1	0	1	0	1
22. Recruited over same time period?	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0
23. Randomisation?	0	1	1	1	0	1	0	1	1	1	0	1	0	1	0	1	0	1
24. Randomisation concealed?	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0

25. Adequate adjustment for confounding?	0	1	1	1	0	1	0	1	1	0	0	0	1	0	0	1	0	0
26. Numbers lost to follow up accounted for?	1	1	1	1	0	1	1	1	0	0	0	0	0	0	1	0	1	0
27. Sufficient power?																		
Total Score	12	19	17	20	11	17	11	18	17	15	10	17	12	14	10	19	12	12

Table S2: Post-intervention and follow-up data used to calculate SMD for subjective measures of cognitive functioning

Author	Intervention	Cognitive outcome	Assessment point	Intervention Group			Control Group			SMD (95% CI)
				N	Mean	SD	N	Mean	SD	
Arnedt et al. (2011) (44)	CBTI-AD	MFI-20 Mental Fatigue[a]	7 weeks	6	-7.30	2.80	4	-9.50	3.10	0.75 -0.55, 2.06
Arnedt et al. (2013) (45)	CBTI-Phone	MFI-20 Mental Fatigue[a]	8 weeks	18	-8.90	2.40	15	-10.6	2.8	0.66 -0.05, 1.36
Arnedt et al. (2013) (45)	CBTI-Phone	MFI-20 Mental Fatigue[a]	12 weeks	18	-8.6	2.4	15	-10.5	3.6	0.63 -0.07, 1.34
Casault et al. (2015) (46)	Minimal CBT-I	QLQ-C30 Cognitive Functioning	6 weeks	17	86.57	13.32	18	72.18	23.67	0.74 0.06, 1.43
Casault et al.	Minimal CBT-I	QLQ-C30	12 weeks	17	82.35	15.63	18	83.33	20.79	-0.05

(2015) (46)		Cognitive Functioning								-0.72, 0.61
Casault et al. (2015) (46)	Minimal CBT-I	QLQ-C30 Cognitive Functioning	24 weeks	17	83.82	15.26	18	82.41	16.16	0.09 -0.57, 0.75
Jansson & Linton (2005) (47)	CBT-I	Concentration Difficulties (0-5)[a]	58 weeks	64	-2.5	1.1	72	-2.7	1.0	0.19 -0.15, 0.53
Ritterband et al. (2012) (51)	SHUTi	MFSI-SF Mental Fatigue[a]	9 weeks	14	-5.29	4.20	14	-5.64	3.93	0.09 -0.66, 0.83
Taylor et al. (2014) (53)	CBT-I	MFI-20 Mental Fatigue[a]	6 weeks	17	-9.88	4.10	14	-11.38	3.11	0.41 -0.31, 1.12

Note. [a] = mean values were multiplied by -1 to adjust for differences in the direction of measurement.

Table S3 : Post-intervention and follow-up data used to calculate SMD for objective measures of cognitive functioning

Author	Intervention	Cognitive outcome	Assessment point	Intervention Group			Control Group			SMD (95% CI)
				N	Mean	SD	N	Mean	SD	
Miro et al. (2011) (49)	CBT-I	ANT-I Control[a]	7 weeks	20	-87.92	29.13	20	-104.70	33.84	0.53 -0.119 1.16
Miro et al. (2011) (49)	CBT-I	ANT-I Orienting	7 weeks	20	66.17	24.26	20	58.15	38.16	0.25 -0.37, 0.87
Miro et al. (2011) (49)	CBT-I	ANT-I Alerting[a]	7 weeks	20	-51.85	31.33	20	-68.98	44.81	0.44 -0.18, 1.07
Miro et al. (2011) (49)	CBT-I	ANT-I Mean RT[a]	7 weeks	20	-617.00	78.39	20	-653.60	88.57	0.44 -0.19, 1.06
Omvik et al. (2008) (50)	CBT-I	Vigil RT	6-8 weeks	17	-0.54	0.07	14	-0.54	0.09	0.000 -0. 71, 0.71
Omvik et al. (2008) (50)	CBT-I	Vigil No. correct	6-8 weeks	17	98.59	1.54	14	98.50	2.21	0.05 -0.66, 0.76
Omvik et al. (2008) (50)	CBT-I	Vigil RT[a]	30-32 weeks	7	-0.55	0.07	10	-0.57	0.08	0.26 -0.71, 1.23
Omvik et al. (2008) (50)	CBT-I	Vigil No. correct	30-32 weeks	7	98.86	1.35	10	99.40	1.78	-0.33 -1.31, 0.64

Sun et al. (2013) (52)	SH + RL	MMSE	12 weeks	38	26.30	2.71	37	23.39	3.78	0.89 0.41, 1.36
Sun et al. (2013) (52)	SH + RL	Number memory	12 weeks	38	10.59	1.77	37	8.97	1.62	0.95 0.48, 1.43
Sun et al. (2013) (52)	SH + RL	Picture memory	12 weeks	38	10.95	2.3	37	7.08	3.33	1.36 0.86, 1.87
Sun et al. (2013) (52)	SH + RL	Associative memory	12 weeks	38	3.65	1.15	37	1.99	1.40	1.30 0.80, 1.80
Sun et al. (2013) (52)	SH + RL	Understanding memory	12 weeks	38	9.86	3.43	37	5.16	2.93	1.47 0.96, 1.98
Sun et al. (2013) (52)	SH + RL	MMSE	24 weeks	38	26.46	5.07	37	22.89	3.93	0.79 0.32, 1.26
Sun et al. (2013) (52)	SH + RL	Number memory	24 weeks	38	11.41	2.65	37	7.84	1.65	1.61 1.09, 2.13
Sun et al. (2013) (52)	SH + RL	Picture memory	24 weeks	38	12.32	3.42	37	6.74	3.09	1.71 1.18, 2.24
Sun et al. (2013) (52)	SH + RL	Associative memory	24 weeks	38	4.38	1.45	37	1.83	1.15	1.95 1.40, 2.50
Sun et al. (2013) (52)	SH + RL	Understanding memory	24 weeks	38	11.03	3.51	37	6.05	2.79	1.57 1.05, 2.09

Sun et al. (2013) (52)	SH + RL	MMSE	52 weeks	38	27.57	2.26	37	22.71	3.67	1.60 1.08, 2.12
Sun et al. (2013) (52)	SH + RL	Number memory	52 weeks	38	11.62	1.80	37	7.53	1.33	3.38 2.68, 4.09
Sun et al. (2013) (52)	SH + RL	Picture memory	52 weeks	38	12.68	2.38	37	6.39	2.44	2.61 1.99, 3.23
Sun et al. (2013) (52)	SH + RL	Associative memory	52 weeks	38	4.78	1.56	37	1.74	1.01	2.31 1.72, 2.89
Sun et al. (2013) (52)	SH + RL	Understanding memory	52 weeks	38	11.41	3.26	37	6.50	2.65	1.65 1.12, 2.18
Wilckens et al. (2016) (54)	BBTI	Delayed recall	4 weeks	38	20.4	6.8	39	19.0	7.7	0.19 -0.26, 0.64
Wilckens et al. (2016) (54)	BBTI	Abstract reasoning	4 weeks	38	25.8	6.3	39	27.4	6.0	-0.26 -0.718, 0.19
Wilckens et al. (2016) (54)	BBTI	Working memory	4 weeks	38	11.1	2.6	39	10.4	2.5	0.27 -0.17, 0.72

Note. [a] = mean values were multiplied by -1 to adjust for differences in the direction of measurement.

Paper 2: Empirical Paper

The following paper has been prepared for submission to ‘SLEEP. The guidelines for authors can be found in Appendix D.

Title: Predictors of subjective/objective sleep discrepancy in poor sleepers: examining daily associations using multilevel modelling

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DISCLOSURE STATEMENT

This was not an industry supported study. Dr Kyle has consulted for Sleepio Ltd. The other authors have indicated no financial conflict of interests.

CORRESPONDENCE INFORMATION

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Abstract

Study Objectives: This study sought to examine predictors of subjective/objective sleep discrepancy in poor sleepers using a daily process design.

Methods: Forty-two individuals with insomnia symptoms (mean age = 36.2 years, 81% female) were recruited to take part in a prospective study which combined seven days of actigraphy with daily assessment of sleep perceptions, self-reported arousal, sleep effort and mood upon awakening.

Results: A high level of intra-individual variability in measures of sleep discrepancy was observed. Multilevel modelling revealed that higher levels of pre-sleep cognitive activity and lower mood upon awakening were significant and independently predictive of underestimation of total sleep time. Greater levels of sleep effort predicted overestimation of sleep onset latency.

Conclusions: Naturally occurring day-to-day fluctuations in various psychophysiological variables are related to subjective/objective sleep discrepancy.

Key Words: subjective/ objective sleep discrepancy, misperception, insomnia

Significance

This is the first study to use a daily process approach to examine the relationship between psychophysiological variables and subjective/objective sleep discrepancy. We find that measures of cognitive arousal, sleep effort and mood upon awakening predict sleep discrepancy outcomes. Our findings could have important clinical implications, by highlighting variables to target in interventions seeking to improve the accuracy of sleep perceptions in individuals with insomnia

Introduction

Insomnia disorder is characterised by persistent difficulties with the initiation and/or maintenance of sleep, leading to daytime impairment.[1, 2] Epidemiological studies have shown that approximately one in ten people meet criteria for insomnia disorder.[3, 4] Insomnia can have severe consequences for many aspects of life, including work performance, social functioning and health.[5-10]

A diagnosis of insomnia disorder is typically based on self-reported symptoms alone. However, numerous studies have demonstrated a mismatch between subjective reports and objective estimates of sleep in people with insomnia.[11] Specifically, many individuals with a diagnosis of insomnia disorder do not demonstrate sleep abnormalities according to objective assessments such as Polysomnography (PSG). Where objective measures do corroborate abnormalities in sleep, it is often not to the extent that the subjective report suggests.[12-16] There is debate in the field as to whether the subjective/objective mismatch represents a distinct subtype of insomnia (sleep state misperception, subjective insomnia, paradoxical insomnia), or is a more general feature of the disorder.[11, 14, 15, 17, 18] Whilst there is considerable variation in the accuracy of sleep perceptions within insomnia samples,[14, 15, 19] on average, individuals with insomnia have a tendency to underestimate actual sleep[15, 18]. This is in contrast to good sleepers, who tend to overestimate sleep[20-23] or estimate objective sleep parameters accurately.[18, 24, 25] These findings have implications for the assessment and diagnosis of insomnia and highlight uncertainties that exist around whether insomnia is best captured by subjective or objective methods. They also expose the potential importance of perceptions of sleep, for our understanding insomnia.

The discrepancy between subjective reports and objective estimates has been demonstrated in a variety of indices of sleep, including total sleep time (TST),[12-14, 26] sleep onset latency (SOL),[13, 24, 27-29] wake-time after sleep onset (WASO)[24, 28, 29] and sleep efficiency (SE).[26, 30] It occurs in primary insomnia and also in patient groups where insomnia is comorbid with other health or psychiatric conditions.[31-36] There is evidence that discrepancy increases with advancing age and may play a role in the higher rates of self-reported insomnia in later life.[29]

The tendency for people with insomnia to underestimate objective sleep has been conceptualised in several ways; (i) as an exaggeration of sleep difficulties, perhaps due to more general psychological characteristics and personality traits,[19, 27] (ii) as a meaningful phenomenon which

reflects a localised sleep disturbance with yet to be elucidated physiological markers[20, 28] and (iii) as a cognitive distortion which contributes to the maintenance and escalation of insomnia.[37]

According to Harvey's Cognitive Model (2002), those who underestimate their sleep may be more at risk of developing objective sleep deficits due to increased preoccupation with sleep and increased sleep related anxiety and arousal, which is antithetical to optimal sleep onset and maintenance.[37] In line with this model, studies which have sought to correct sleep discrepancy have demonstrated changes in insomnia related anxiety and distress and concomitant reductions in self-reported insomnia symptoms.[38-40] There is emerging evidence that Cognitive Behavioural Therapy for Insomnia (CBT-I) improves the accuracy of sleep perceptions[30], a finding which highlighted the possibility that correction of discrepancy could account for some of the efficacy of treatments for insomnia.

A variety of psychological and physiological mechanisms have been proposed to underlie sleep discrepancy (for a review see Harvey and Tang [11]). These include cognitive arousal,[41, 42] physiological arousal,[41-43] cortical arousal,[20, 44] selective attention,[45] memory bias,[11] and sleep fragmentation.[46, 47] A recent study by Takano and colleagues (2016)[48] reported that higher levels of pre-sleep cognitive arousal were associated with underestimations of TST and overestimations of SOL, in a community sample comprising individuals with and without insomnia symptoms. This work is consistent with correlational and experimental studies that have reported associations between pre-sleep cognitive activity and sleep discrepancy in insomnia samples.[19, 41]

The studies conducted thus far have assessed sleep discrepancy over a single night or averaged data from multiple nights. Therefore, little is known about intra-individual variability in sleep discrepancy and whether it is affected by natural, day-to-day variations in psychological factors. There is some evidence for high-levels of night-to-night variability in sleep discrepancy in older adults, where the discrepancy between self-report and actigraphy based estimates of sleep onset latency was found to be 150% more variable within the same individual across nights, compared to between individuals. [29] In addition, there is emerging evidence for intra-individual variability in some of those factors which are proposed to underlie discrepancy, such as arousal[49, 50] and sleep fragmentation[51]. The importance of examining intra-individual variability in sleep/wake patterns is increasingly being recognised.[52] Although daily and average values of sleep parameters tend to be highly correlated, information may be concealed when only average or single values are considered. It is possible that daily values and averaged values have overlapping but distinctive

aetiology. For example, daily values may be more highly associated with state like psychophysiological variables than average values. In this study, we utilised multilevel modelling to investigate whether arousal (cognitive, physiological), sleep effort, sleep fragmentation and mood upon awakening predicted sleep discrepancy across seven nights, in a group of poor sleepers. We chose to investigate multiple constructs in order to tease out the most important contributors. Multilevel modelling allows for the analysis of within-person changes in variables across nights whilst accounting for the influence of between-subject variations in the relationships of interest. By using this approach, we sought to examine intra-individual variability in sleep discrepancy and assess the relationship between sleep discrepancy and psychophysiological variables over multiple nights, without the requirement to aggregate data.[53] We chose to focus on cognitive arousal and sleep fragmentation because these constructs have the strongest evidence as predictors of subjective/objective sleep discrepancy.[11] We included two measures of cognitive arousal; one that assesses general cognitive arousal and another that assesses the content and frequency of thoughts during the pre-sleep period, due to preliminary evidence that certain aspects of cognitive arousal may be more closely associated with sleep disturbance.[54] We included a measure of self-reported physiological arousal because an experimental study has shown that increases in physiological arousal lead to increases in TST and SOL discrepancy. [41] The inclusion of a measure of sleep effort was based on research demonstrating that sleep effort is strongly associated with subjective reports of sleep disturbance but not objective sleep parameters (PSG).[55] Mood upon awakening was assessed due to evidence that low mood and general feeling state at the time of reporting of subjective sleep, may mediate underestimations of objective sleep parameters.[35, 56] It was hypothesised that higher levels of arousal (cognitive and physiological), sleep effort, sleep fragmentation and worse mood on awakening would be associated with greater levels of discrepancy in TST. It was also hypothesised that higher levels of arousal (cognitive and physiological), sleep effort and worse mood on awakening would be associated with greater levels of discrepancy in SOL. We focused on discrepancies in TST and SOL because the subjective/objective discrepancy has been demonstrated most robustly in these indices.

Method

Procedure

Participants were recruited from the staff and student population of the University of Manchester and the local community, through advertisement. Ethical approval for the study was obtained from the University of Manchester Research Ethics Committee (see Appendix E).

Inclusion criteria were: 1) aged between 18 and 65 years and 2) score on the Sleep Condition Indicator (SCI) ≤ 16 indicating probable insomnia disorder. Exclusion criteria were: 1) sleep disorder other than insomnia, 2) current treatment for any medical or psychiatric disorder, 3) current use of hypnotic medication or medication that is known to cause drowsiness.

People who were interested in taking part in the study were directed to a webpage which provided information about the study and screening questionnaires. The researcher contacted those participants who were deemed eligible according to their responses on the screening questionnaires.

Each eligible participant attended the University for one face-to-face session with the lead researcher (VH), where they provided informed consent to take part in the study (Appendix F) and completed a battery of baseline questionnaires.

Participants were then provided with a Motionwatch 8 actigraph watch (CamNTech Ltd.; Cambridge, UK) in order to collect objective estimates of their sleep. Participants wore the watch continuously from the initial meeting with the researcher for a seven-day period. At the same time, participants recorded their subjective experiences of sleep by completing the Consensus Sleep Diary – Morning administration[57] each morning upon awakening.

Alongside the sleep diary, participants completed retrospective assessments of their subjective experiences of general cognitive arousal, physiological arousal, sleep effort and specific pre-sleep cognitive activity, for the preceding night. Finally, participants provided a rating for their current mood state.

Screening Measures

All participants checked a box to endorse the inclusion and exclusion criteria.

The Sleep Condition Indicator[58] (Appendix G) was administered to assess insomnia symptoms. It was presented on the study webpage. It is an eight item scale which evaluates SOL, WASO, sleep quality, impact of sleep problems on daytime functioning, duration of sleep problems, nights per week where a sleep problem is present, the extent to which the individual is troubled by the sleep problem and the history of the sleep problem.

Participants are asked to provide ratings with reference to a typical night in the past month. The SCI utilises a five-point scale (0-4), with lower scores indicating greater difficulties. Total score on the SCI ranges between 0 and 32, with scores ≤16 indicating probable insomnia [58]. The SCI has satisfactory internal consistency (Cronbach α = 0.86) and correlates strongly with measures of clinical insomnia such as the Pittsburgh Sleep Quality Index (PSQI)[59] and the Insomnia Severity Index (ISI)[60]. In this sample, internal consistency of the SCI was not demonstrated (Cronbach α = 0.28). This suggests that the questions in this scale may be assessing different constructs.

A screening tool for the identification of sleep disorders other than insomnia, which was developed by experts in the field of sleep disorders[61] (Appendix H), was presented on the study webpage. Those scoring positive for potential narcolepsy, sleep breathing disorder, restless leg syndrome, circadian rhythm sleep disorder or parasomnia were deemed ineligible to take part in this study.

Baseline Measures

Assessments of dysfunctional beliefs and attitudes about sleep, depression, anxiety, stress and sleep-related attentional bias were administered in order to characterise the sample.

The Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16)[62] (Appendix I) was administered to evaluate participants' beliefs about sleep, specifically their views on the causes, consequences and treatment of sleep problems. The scale is a brief form of the original 30 item DBAS and contains 16 items which are rated on a 10-point Likert scale ranging from 0 (strongly disagree) to 10 (strongly agree). Total scores are averaged to create a mean item score, with higher mean scores indicating stronger endorsement of maladaptive beliefs about sleep. The DBAS-16 has adequate internal consistency (Cronbach α = 0.77) and good test-retest reliability (*r* = 0.83).[62] In this sample internal consistency was good (Cronbach α = 0.84).

Participants also completed the Depression, Anxiety and Stress Scale (DASS-21)[63] (Appendix J) in order to provide a measure of depression, anxiety and stress over the past week. Each item is

scored from 0 (did not apply to me at all over the last week) to 3 (applied to be very much or most of the time over the last week). This scale has been validated for non-clinical samples, where it demonstrated adequate internal consistency (Cronbach's α = 0.88 for depression, 0.82 for anxiety, 0.90 for stress and 0.93 for the total scale) and good convergent and discriminant validity when compared with other measures of anxiety and depression. [64] In this sample, internal consistency was satisfactory (Cronbach α = 0.93 for depression, 0.83 for anxiety, 0.87 for stress and 0.94 for the total scale).

Daily Measures

Actigraphy

Participants wore a MotionWatch 8 actigraphy watch, which is a tri-axial, wrist-worn accelerometer. The digital accelerometer enables the differentiation of probable sleep and wake states for each 30 second period of recording using a sleep/wake algorithm. Actigraphy has been validated against PSG, where it has shown high levels of sensitivity for detecting sleep.[65]

Participants were asked to press the event marker when they got into bed at night and again when they got out of bed the following morning. Data were downloaded and analysed using Motionware 1.1.20 (CamNTech) software. Responses from the sleep diary were used to set the sleep period analysis window (i.e. between the "got into bed" and "got out of bed" times). In cases where these data were missing from the sleep diary, we used the event markers to mark the analysis window. This occurred for less than 1% of data points.

The following sleep variables were extracted: TST, SOL, SE and sleep fragmentation index. TST is the total time spent in sleep according to the epoch-by-epoch wake/sleep categorisation. SOL is the time which elapsed between the participant getting into bed and the participant falling asleep. SE is the total sleep time, expressed as a percentage of the total time spent in bed. The sleep fragmentation index is a percentage of the total time categorised as mobile in the epoch-by-epoch mobile/immobile categorisation and the number of immobile bouts which were less than or equal to one minute in length. The sleep fragmentation index is a measure of the degree of sleep discontinuity.

Sleep Diary

This diary of subjective sleep experiences is widely used in sleep research and was developed through collaboration between a panel of experts in the field of insomnia and potential users of the

Diary[57] (Appendix K). Items directly assess or permit the calculation of total time in bed, total sleep time, sleep onset latency, wake-time after sleep onset and sleep quality.

Subjective TST was taken from participants' estimates of total sleep time. Subjective SOL was calculated from the time that the participant got into bed using the following formula: (Time at which the individual attempted to fall asleep + sleep onset latency) – Time at which the individual got into bed.

Predictors of Discrepancy

Each morning participants completed four visual analogue scales (0-100mm) (Appendix L), assessing general cognitive arousal ("Last night, as you were attempting to fall asleep or return to sleep, did thoughts keep running through your mind?") and physiological arousal ("Last night, as you were attempting to fall asleep or return to sleep, did you experience a jittery nervous feeling in your body?"). These questions were adapted from items on the Pre-Sleep Arousal Scale[66] and the same or similar single item questions have been employed in previous daily diary designs.[67, 68] Sleep effort was assessed using an item adapted from the Glasgow Sleep Effort Scale, [69] ("How much effort did you put into sleeping last night?", with anchors "No effort" at 0mm and "A lot of effort" at 100mm). Current mood state was assessed by asking participants, "How would you describe your mood right now?" with anchors "very bad mood" (0mm) and "very good mood" (100mm).

Participants completed the Glasgow Content of Thoughts Inventory (GCTI) (Appendix M) in order to provide a retrospective measure of the content and frequency of pre-sleep cognitive activity. The GCTI was completed in the morning with reference to cognitive activity during the preceding night. The Inventory consists of 25 items which were developed on the basis of live audio recordings of pre-sleep thought content in people with insomnia. Participants were requested to indicate on a 4-point scale (1 = Not at all, 2 = A little, 3 = A fair amount, 4 = A great deal) to what extent specific thoughts kept them awake the previous night. Scores were summed and higher scores indicate a greater level of pre-sleep cognitive activity. The inventory has demonstrated good test-retest reliability (ICC=.88) and satisfactory internal consistency (Cronbach α = 0.87). In a validation study, scores on this scale successfully discriminated between individuals with insomnia and good sleepers, with a score of 42 yielding a sensitivity of 100% and a specificity of 83%.[70] In the current sample, internal consistency for the total score was good (Cronbach α = 0.91 for day 1, 0.91 for day 2, 0.89 for day 3, 0.90 for day 4, 0.92 for day 5, 0.91 for day 6 and 0.92 for day 7).

Data Analysis

Outcome Variables

A TST discrepancy score was calculated using the formula outlined by Manconi and colleagues (known as the misperception index, or MI):[18] (objective TST – subjective TST)/objective TST. MI values range between +1 and -1. Perfect correspondence between subjective and objective estimates of TST results in an MI equal to 0. Positive MI values indicate underestimation of objective sleep and negative MI values indicate overestimation of objective sleep.

The discrepancy score for SOL (SOL_d) was calculated using the following formula: SOL_d = objective SOL – subjective SOL. SOL_d is positive when SOL is underestimated compared to objective values. SOL_d is negative when SOL is overestimated compared to objective values.

Descriptive statistics were computed in order to examine the distribution of the outcome variables (Table 2).

Multilevel Modelling

All analyses were performed in Stata, version 14 (Statacorp, 2015, Texas, USA). All outcome and predictor variables were assessed for normality via histogram inspection and analysis of skewness and kurtosis statistics. SOL_d was negatively skewed and scores for the GCTI, general cognitive arousal, physiological arousal, sleep effort and sleep fragmentation were positively skewed. Therefore subsequent analyses used nonparametric bootstrapping with 200 replications to account for non-normality of the data.

Due to the hierarchical nature of the data (observations are nested within participants) the assumption of independence of observations is violated and therefore multilevel modelling was implemented. Linear mixed effects models (“xtmixed”) were estimated using maximum likelihood estimation. For each analysis, participant number was included as a random effect. Two sources of variance were partitioned in the dataset; differences between participants on average levels of daily variables and difference within participants on their daily reporting of variables over time. Initially univariate models were estimated for each predictor variable. Significant univariate predictors were then entered into a multivariate model in order to assess their independent contribution. Separate models were estimated for MI and SOL_d.

Results

Participants

One-hundred and fifty-one self-reported poor sleepers completed the screening questionnaires. Of these, 45 were excluded because their score on the SCI did not indicate probable insomnia. Thirty-eight people were excluded due to a possible sleep disorder (other than insomnia), three due to a psychiatric disorder, one person was excluded due to use of hypnotic medication and one person was excluded due to use of a medication which causes drowsiness.

Of the 63 participants who were eligible to take part, 42 were enrolled in the actigraphy phase of the study. The most commonly cited reason for eligible participants not taking part was scheduling difficulties. Objective sleep data was not available for one participant due to malfunctioning of the actigraphy watch. Therefore the final sample comprises data from 41 participants. Of a possible 287 assessment points for subjective and objective sleep, a total of 280 (98%) were completed.

Demographic and baseline characteristics of the sample are displayed in Table 1. Mean score on the SCI was 10.95, which is well below the cut-off for probable insomnia. Eighty-three per cent of participants reported enduring problems with sleep (> 1 year). Mean score on the DBAS is similar to that seen in insomnia samples [62]. Mean scores on the three subscales of the DASS-21 indicate high levels of depression, anxiety and stress in this sample compared to normative data from the general population (depression = 91st percentile, anxiety = 92nd percentile, stress = 96th percentile). The proportion of participants whose scores were in the severe or extremely severe range, differed according to the subscale (depression subscale = 26.8%, anxiety subscale = 26.3% and stress subscale = 55%). Scores on the DASS in this sample are higher than those reported for an insomnia disorder sample. [71]

Table 1: Demographic and clinical characteristics

Variable	Mean	SD
Age (years)	36.15	14.01
Sex (% female)	81	-
SCI	10.95	3.07
DBAS	4.86	1.29
DASS - Depression	7.80	7.15
DASS - Anxiety	5.98	5.22
DASS - Stress	13.73	7.05

Mean estimates of sleep parameters are shown in Table 2. Actigraphy and sleep diary values for SOL and SE are similar to those reported for insomnia disorder samples in other published work. Estimates of TST appear to be slightly higher in this sample. [72-74]

Table 2: Diary and actigraphy sleep parameters across study nights

	Data Type	n	Mean	Min	Max	SD
TST	Subjective	281	391.27	135	880	89.34
	Actigraphy	281	425.13	142	759	73.12
SOL	Subjective	280	59.49	0	480	57.96
	Actigraphy	281	27.69	0	311	36.44
SE	Subjective	278	74.92	30.34	100	15.15
	Actigraphy	281	81.13	51.20	96.50	7.36
MI	-	280	0.07	-0.48	0.64	0.18
SOL_d	-	279	-31.86	-445	73	51.18

TST = total sleep time, SOL = sleep onset latency, SE = sleep efficiency, MI = misperception index, SOL_d = sleep onset latency discrepancy. TST and SOL were measured in minutes. SE represents time asleep as a % of total time in bed.

Misperception Index

Descriptive statistics for MI are presented in Table 2. The mean value for MI was 0.07, indicating that participants estimated on average, 7% of objective sleep as wake. The distribution of MI approximated a normal distribution. Differences between days in the same participant accounted for 54.1% of the variation in MI, suggesting high levels of intra-individual variability.

Daily Predictors of Misperception Index

Univariate analyses showed that total score on the GCTI was a significant predictor of MI, with higher levels of pre-sleep cognitive activity predicting more positive values for MI (underestimation of sleep time).

General cognitive arousal, sleep effort and sleep fragmentation index were also significant predictors of MI, with higher values on these variables predicting more positive values for MI (underestimation of sleep time). Self-reported physiological arousal was not a significant predictor. Mood on awakening was a significant predictor of MI, with better mood on awakening predicting more negative values for MI (overestimation of sleep time). The coefficients, standard errors, confidence intervals and significance values for each of the predictors in the univariate analysis are displayed in Table 3.

Table 3: Results from the univariate mixed model analysis predicting MI

Predictors	C	95% CI	SE	P	n
GCTI	0.0059	0.0039, 0.0080	0.0010	**<0.001**	267
Cognitive Arousal	0.0012	0.0004, 0.0020	0.0004	**0.003**	277
Physiological Arousal	0.0004	-0.0005, 0.0012	0.0004	0.38	278
Sleep Effort	0.0017	0.0009, 0.0026	0.0004	**<0.001**	278
Sleep Fragmentation	0.0027	0.0010, 0.0044	0.0009	**0.002**	280
Mood	-0.0023	-0.0033, -0.0013	0.0005	**<0.001**	278

C = coefficient of the predictor, 95% CI = 95% confidence intervals, SE = standard error, P = significance level, n = number of observations, GCTI = Glasgow content of thoughts inventory, Mood = mood upon awakening

A multivariate analysis of the univariate predictors revealed that scores on the GCTI and mood on awakening were significant independent predictors of MI. Specifically, worse mood on awakening

and higher levels of pre-sleep cognitive activity predicted more positive values for MI (underestimation of sleep time) (Table 4).

Table 4: Results from the multivariate mixed model analysis predicting MI

Predictors	C	95% CI	SE	P	n
GCTI	0.0038	0.0010, 0.0066	0.0014	**0.007**	265
Cognitive Arousal	-0.0004	-0.0015, 0.0006	0.0005	0.448	265
Sleep Effort	0.0008	-0.0003, 0.0020	0.0006	0.160	265
Sleep Fragmentation	0.0016	0.0002, 0.0035	0.0009	0.076	265
Mood	-0.0014	-0.0026, -0.0003	0.0006	**0.024**	265

C = coefficient of the predictor, 95% CI = 95% confidence intervals, SE = standard error, P = significance level, n = number of observations, GCTI = Glasgow Content of Thoughts Inventory, Mood = mood on awakening

Sleep Onset Latency Discrepancy

Descriptive statistics for SOL_d are presented in Table 2. The distribution of SOL_d was negatively skewed. Differences between days in the same participant accounted for 82.9% of the variation in SOL_d.

Daily Predictors of Sleep Onset Latency Discrepancy

The univariate analysis showed that sleep effort was the only statistically significant predictor of SOL_d. Higher values of sleep effort were associated with more negative values for SOL_d (Table 5), indicating that greater sleep effort predicts overestimation of the time taken to get to sleep

Table 5: Results from the univariate mixed model analysis predicting SOL_d

Predictors	C	95% CI	SE	P	n
GCTI	-0.5549	-1.2741, 0.1642	0.3669	0.130	266
Cognitive Arousal	0.0088	-0.2596, 0.2772	0.1369	0.949	276
Physiological Arousal	0.0427	-0.2288, 0.3141	0.1385	0.758	277
Sleep Effort	-0.2974	-0.5470, -0.0477	0.1274	**0.020**	277
Mood	0.3877	-0.0239, 0.7992	0.2100	0.065	277

C = coefficient of the predictor, 95% CI = 95% confidence intervals, SE = standard error, P = significance level, n = number of observations, GCTI = Glasgow Content of Thoughts Inventory, Mood = mood upon awakening

Discussion

A mismatch between subjective and objective estimates of sleep parameters is commonly observed in people with insomnia, however little is known about the mechanisms underlying this phenomenon. This study sought to determine predictors of subjective/objective sleep discrepancy in individuals with insomnia symptoms. Using actigraphy and sleep diaries, we conducted repeated longitudinal assessment of sleep discrepancy, pre-sleep and next-day psychophysiological factors, across seven days and nights. Our results highlight roles for arousal, sleep effort, mood upon awakening and sleep fragmentation.

Magnitude of subjective/objective sleep discrepancy

In line with previous investigations of sleep discrepancy, this study demonstrated a tendency for individuals with insomnia symptoms to underestimate their sleep. When subjective and objective measures of sleep were estimated across seven days, on average participants estimated 7% of total sleep time as wake.

The average MI in this study is less than has been documented in two previous studies.[18, 26] A study by Manconi and colleagues (2010) found two distributions of MI in their sample of insomnia patients, one that had an average MI value of 0.365 (termed moderately misperceptive) and a second that had an average MI value of 0.989 (termed highly misperceptive). A study by Dittoni and colleagues (2013) reported average MI of 0.247. There are several possible explanations for the lower average MI value obtained in this study. Firstly, in the current study participants were self-reported poor sleepers, recruited from the general population and may have been experiencing less severe symptoms of insomnia. In line with this, the average subjective TST was 207.8 minutes in the study by Manconi et al. (2010) and 271.2 minutes in the study by Dittoni et al. (2013), values which are considerably lower than the average subjective TST in this sample (391.3 minutes). Secondly, in the current study objective sleep was assessed using actigraphy over multiple nights, whereas in the studies by Manconi et al. (2010) and Dittoni et al. (2013), objective data was based on one night of laboratory PSG. Actigraphy tends to overestimate total sleep time compared to PSG[65] and there is evidence for systematic differences in observed sleep parameters depending on whether measurement of sleep is performed in the home or a laboratory

environment. [75, 76] Average MI in the current study is similar to the value estimated from a study utilising a more similar methodology (multiple nights of actigraphy combined with sleep diary).[77]

In this study, examination of subjective and objective sleep over multiple nights enabled us to identify high levels of intra-individual variability for MI in our sample. Overall, 54.1% of the variation in MI was due to differences between days within the same participant, suggesting that single measurements of MI may provide an incomplete picture. Both overestimation and underestimation of sleep was evident. Of the 41 poor-sleepers who took part in the study, 30 (73%) thirty displayed a mixture of over and underestimation of TST. Our findings do not support the proposal that underestimation of TST is a consistent and trait like feature of people with insomnia.

Average discrepancy in SOL was -31.86 minutes, which is larger than values reported in two studies utilising similar multiple time-point, actigraphy assessment.[29, 78] In the study by Williams and colleagues (2013), average discrepancy in SOL for those with insomnia symptoms was -16.84 minutes. In the study by Kay et al. average SOL_d in an insomnia complaint group was -6.76 minutes. For both of these studies, sleep was assessed over a longer period of time than in the current study (14 nights) and therefore more reliable average values for SOL_d may have been obtained. In addition, these studies were conducted in older adults and differences in the magnitude of SOL_d may be related to the observation that insomnia in older adults tends to involve difficulties with the maintenance sleep rather than the initiation of sleep.[79] The average SOL_d obtained in the current study is similar to that estimated from baseline data reported in a group of "sleep-onset insomniacs", undergoing paradoxical intention. [80]

In this sample, 17.1% of the variation in SOL_d was due to differences between participants and the remainder (82.9%) was due to differences between days within the same participant. Thus, there is considerable intra-individual variability in SOL_d. Both overestimation and underestimation of SOL was evident, however the frequency of underestimation of SOL was relatively small, with just 14.7% of subjective SOL values representing an underestimation.

Predictors of subjective/objective sleep discrepancy

Univariate analyses revealed that cognitive arousal (general cognitive arousal and specific pre-sleep cognitive activity measured using the GCTI), sleep effort, sleep fragmentation and mood upon awakening were all significant predictors of MI. Counter to our hypothesis, physiological arousal was not a significant predictor of MI. Multivariate analysis revealed that pre-sleep cognitive

activity and mood upon awakening provided statistically significant, independent contributions to MI. With regards to SOL_d, univariate analyses identified sleep effort as the only statistically significant predictor.

These findings suggest that day-to-day, naturally occurring fluctuations in cognitive arousal are associated with subjective/objective discrepancy in TST. These data corroborate evidence from an experimental study in which provoking an increase in cognitive arousal led to increases in TST sleep discrepancy.[41] They also support the work of Takano and colleagues (2016),[48] who found that cognitive arousal was uniquely associated with TST and SOL discrepancy in a community sample. Mechanisms through which cognitive arousal contributes to sleep discrepancy have been proposed, however the evidence is limited. One suggestion is that cognitive arousal distorts the perception of time because a unit of time is perceived as longer when more information is processed (through greater levels of mentation under high arousal conditions).[41] Another proposal is that cognitive arousal maintains an enhanced level of sensory and memory processing during sleep onset, which obscures the distinction between sleep and wakefulness.[20] In line with this, an association between high-frequency EEG activity during non-rapid eye movement (NREM) sleep and subjective/objective sleep discrepancy, has been observed.[20, 81] High frequency EEG activity is thought to be a marker of sensory processing and memory formation. Further research implementing fine grained measurement of sleep such using techniques such as high density EEG is required, in order to advance our understanding of the possible neurophysiological processes underlying associations between cognitive arousal and sleep discrepancy.

In this study, cognitive arousal was assessed using two measures; the GCTI which evaluates the content and frequency of pre-sleep cognitions and a visual analogue scale rating the extent to which participants experienced thoughts running through their minds (general cognitive arousal) during the pre-sleep period. In the univariate analyses, both measures of cognitive arousal were significant predictors of MI, however in the multivariate analysis, only the GCTI was a significant predictor. Clearly there is substantial overlap between these two measures, as indicated by the moderate strength correlation between responses ($r(266) = .63$, $p < .001$). Shared variance may explain why only the GCTI was a significant predictor of MI in the multivariate analysis, however there is little change in the predictive value of general cognitive arousal when score on the GCTI is omitted from the multivariate analysis. The GCTI probes a wide variety of intrusive thoughts which are known to be commonly experienced by individuals with insomnia in the pre-sleep period and

contains items such as “How frustrated/upset I am feeling” and “How nervous/anxious I am feeling”, which may capture the emotional and physiological sequelae of intrusive thoughts in a way that a single question about a racing mind does not. Although the GCTI is predominantly a measure of cognitive arousal, it appears to tap into hyperarousal more broadly and this may be the reason that it is the strongest predictor of MI in this study.

The findings from this study suggest that physiological arousal is not related to subjective/objective discrepancy in TST or SOL. This is contrary to reports from a previous study in which manipulations of physiological arousal using caffeine have led to changes in sleep discrepancy[41] and a study in which physiological arousal was shown to predict discrepancy in SOL.[48] Our findings do however lend support to research which has demonstrated that cognitive arousal plays a more important role than physiological arousal in subjective sleep complaints.[66, 82-84] There is debate around the validity of separating the different aspects of arousal, given evidence that they interact. For example, inducing cognitive arousal can provoke increases in physiological arousal.[85] The null findings in this study may be due to lack of sensitivity in our measure of physiological arousal as opposed to lack of role for physiological arousal in sleep discrepancy. We implemented a single item, self-report assessment of physiological arousal, which demonstrated sufficient sensitivity to detect associations between physiological arousal and various subjective and objective sleep parameters in a previous study of chronic pain patients.[68] However, self-report may be less sensitive in the domain of physiological arousal. Future work should administer validated subjective and objective measures of physiological arousal in order to fully assess its contribution to sleep discrepancy.

Mood on awakening was a significant predictor of MI in both the univariate and multivariate analyses. Previous work has revealed associations between sleep discrepancy and depressive symptoms assessed at baseline. [78, 86] The findings from this study extend that work by showing that daily fluctuations in morning mood are associated with subjective/objective discrepancy in TST. The relationship between sleep discrepancy and mood upon awakening may be explained by mood congruent memory bias, in which an individual remembers or selectively processes information that is consistent with current mood. This is a phenomenon which has been documented in individuals with clinical depression[87] and during depressed mood induction in non-clinical populations.[88] Specifically, when an individual is making a judgement about how well they slept the previous night, current feeling state may distort memory such that low mood or dysphoria

at the time of reporting leads to negatively biased judgements of sleep quantity and/or quality. It has long been established that memory is a reconstructive process affected by bias and error. [89, 90] Moreover, mood congruent memory biases have been demonstrated in a variety of contexts, including symptom reporting.[91, 92]

Another possible explanation for the association between sleep discrepancy and mood on awakening is that greater sleep discrepancy leads to worse mood on awakening or that these variables are related by means of a third factor, such as sleep quality. An association between sleep quality and next-day affect is well established[93, 94] and a number of studies have suggested a link between poor sleep quality and increased subjective/objective sleep discrepancy. [28, 30] Due to the nature of the study design, causal inferences with regards to the relationships uncovered cannot be made. Experimental investigations are required in order to determine the direction of the effect and potential mediators of the relationship. For example, future studies could use a mood induction paradigm to examine the impact of mood upon awakening on subsequent subjective reports of sleep quantity and quality.

Consistent with our hypothesis, sleep effort was a significant predictor of both MI and SOL_d. These findings lend support to the attention-intention-effort model of insomnia[95] which proposes that explicit intention to sleep inhibits normal de-arousal and subsequently hinders sleep. Sleep effort appears to play a particularly important role in SOL discrepancy, where it was the only significant predictor. In line with our findings, a previous study reported that reductions in sleep effort mediated the improved accuracy of sleep perceptions following paradoxical intention.[80] One possibility is that sleep effort maintains and exacerbate sleep difficulties through distorting perceptions of sleep.

Sleep fragmentation index is a measure of interruption of sleep due to physical movement and is utilised as an assay of sleep continuity. The results from this study revealed a significant association between sleep fragmentation and MI in the univariate analysis, whereby higher levels of sleep fragmentation were associated with underestimation of TST. These findings concur with reports from an experimental study, in which inducing brief awakenings led to overestimates of sleep onset latency in normal sleepers.[46] More frequent awakenings may lead to shallower forms of sleep and greater levels of cortical activity, resulting in difficulties distinguishing wake from sleep.[96]

Strengths and Limitations

This study has several limitations. Firstly, we examined linear associations between variables and therefore no causal inferences can be made. Secondly, potentially overlapping constructs were assessed, as indicated by moderate/strong correlations between many of the predictor variables. This complicates the interpretation of the results from the multivariate analysis. Thirdly, the use of actigraphy enabled assessment of sleep across multiple days in the home environment which increases the ecological validity of findings, however actigraphy is known to overestimate sleep time in individuals with insomnia.[97] This has implications for the reliability of the sleep discrepancy outcome variables. Fourthly, our sample consisted predominantly of females, which limits the generalisability of results. Fifthly, we did not assess sleep microstructure and therefore the contribution of EEG parameters that are proposed to play a role in sleep discrepancy were not examined. Sleep discrepancy has been associated with heightened brain activity during PSG defined sleep.[44] Brief arousals from REM sleep and time spent in REM sleep have also been shown to correlate with degree of discrepancy [98]. It will be important for future studies to include measures such as high density EEG, in order to understand how the relationships uncovered in the current study are expressed across different levels of explanation (i.e. underlying physiological processes). Sixthly, there are two ways in which subjective estimates of total sleep time can be calculated. In the current study, we chose to use the participants' global estimates of total sleep time, as opposed to calculating their total sleep time on the basis of reports of the time that they went to bed and the time that they got up in the morning. We reasoned that concern about sleep is more likely based on global estimates of sleep time. Interestingly, in many cases there was not complete concordance between these two methods of calculating subjective total sleep time. The implications of this are that similar studies that calculate subjective total sleep time using a different method from the one utilised in the current study, may report different findings. Finally, we did not conduct rigorous screening for sleep, physical health or psychiatric comorbidities and we did not assess whether participants were taking substances that might induce sleeplessness (e.g. medications, caffeine, alcohol, illicit drugs). Therefore it is possible that these factors influenced our findings.

The main strength of this study is that multilevel modelling was used in order to investigate sleep discrepancy over seven days. By using this statistical approach, we were able to avoid aggregating that data collected, instead using all of the available information to generate a more accurate measure of sleep discrepancy and its association with various psychophysiological variables. We found high levels of intra-individual variability in both MI and SOL_d, which supports the assertion

that single measurements or aggregated measures of sleep discrepancy are likely to provide an incomplete picture.

Clinical implications

The results from this study highlight potential target variables for interventions seeking to improve the accuracy of sleep perceptions. Manipulations which lead to more positive perceptions of sleep are associated with reductions in levels of self-reported sleep impairments and sleep-related anxiety and distress.[40] Demonstrations of the discrepancy between subjective and objective estimates of sleep using verbal feedback or behavioural experiments are effective methods for improving the accuracy of sleep perceptions in individuals with insomnia. The results from this study indicate that interventions which lead to reductions in cognitive arousal, sleep effort, sleep fragmentation and improvements in mood upon awakening, may also result in more accurate perceptions of sleep.

Insomnia treatments may benefit from a greater focus on strategies which seek to enhance cognitive and emotional flexibility, in order to alleviate the high levels of cognitive arousal and sleep effort that are observed in individuals with insomnia. A study in which CBT-I was combined with mindfulness practice reported improvements in various indices of subjective sleep, which occurred alongside reductions in pre-sleep arousal and sleep effort.[99] Interestingly, a relationship between frequency of mindfulness meditation practice and reductions in arousal was present, indicating that mindfulness may attenuate cognitive hyperarousal. Objective measures of sleep were not collected in this study and therefore it is not known if the reductions in arousal and sleep effort that occurred during treatment, lead to reductions in sleep discrepancy. In future treatment trials, it will be important to evaluate sleep discrepancy as an outcome and examine whether changes in psychophysiological variables such cognitive arousal and sleep effort, predict more accurate perceptions of sleep.

Conclusions

This is the first study to examine intra-individual variability in sleep discrepancy and explore associations between sleep discrepancy and various psychophysiological factors using a repeated, longitudinal assessment in individuals with insomnia symptoms. High levels of intra-individual variability in estimates of sleep discrepancy were demonstrated, validating the daily process design utilised. Associations between arousal, sleep effort, mood upon awakening, sleep fragmentation

and sleep discrepancy were identified. In a multivariate analysis, cognitive arousal and mood upon awakening were independently predictive of total sleep time misperception index. Discrepancy in sleep onset latency was predicted by sleep effort. Further research is required in order to model possible additive and interactive effects among predictor variables.

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Paper 3: Critical Reflection

Word count: 4968

Overview

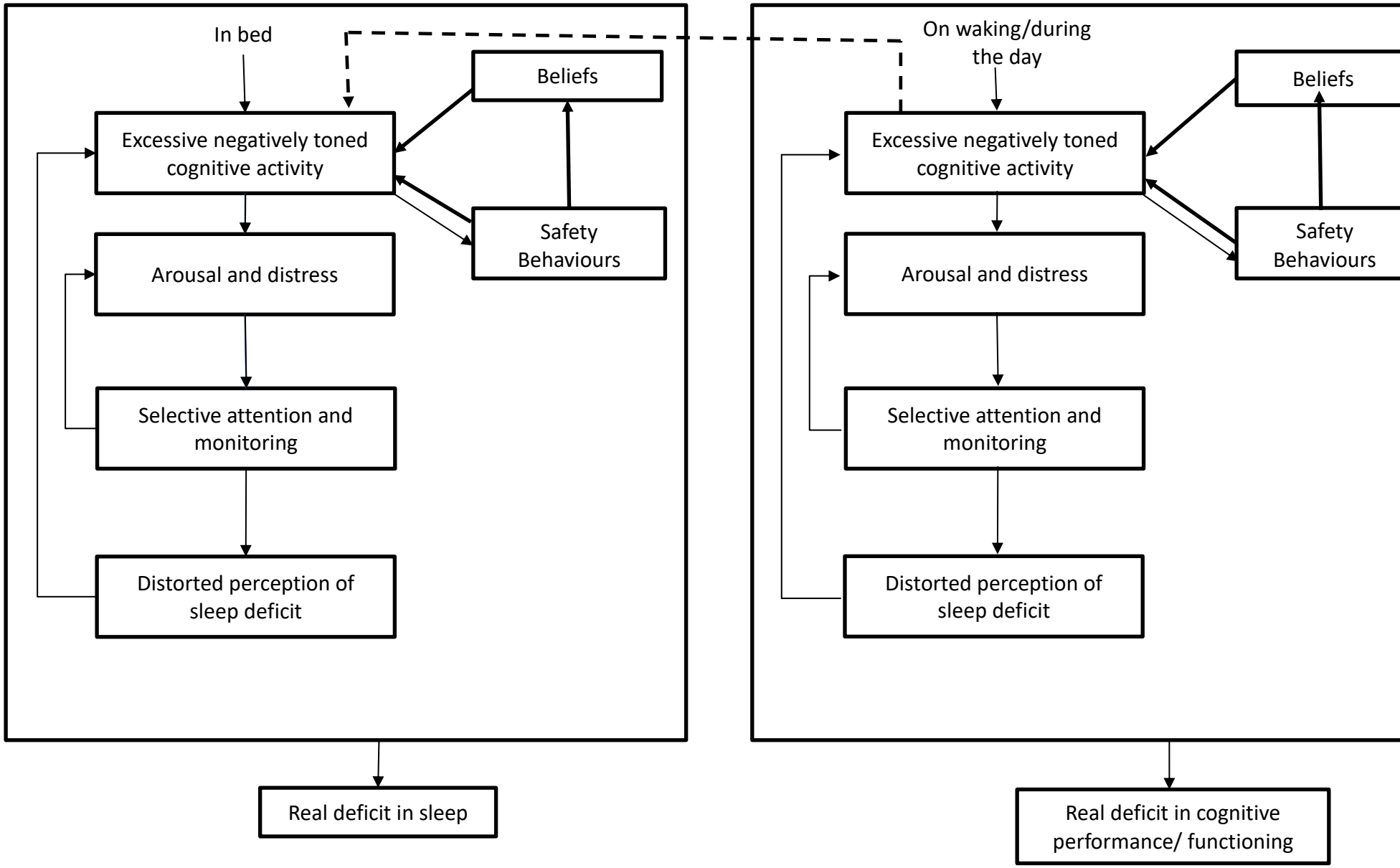
Problems with the initiation/maintenance of sleep are phenomenon that everybody will likely experience at one time or another. Insomnia researchers have sought to understand why some individuals recover after a period of poor sleep, whilst others go on to develop a chronic problem (Harvey, 2002). There is now a wealth of evidence to support the assertion that irrespective of the initial cause of insomnia, psychological processes are important in its maintenance. On this basis, cognitive models of insomnia have been developed. In Harvey's cognitive model, "misperception" of sleep is proposed to be a key mechanism through which insomnia is maintained (Harvey, 2002) (see Figure 1). "Misperception" relates to the observation that a mismatch between objective measures of sleep and self-reported sleep often exists in those with insomnia. Specifically, individuals with insomnia tend to underestimate total sleep time and overestimate sleep latency, relative to both Polysomnography (PSG) and actigraphy (Harvey & Tang, 2012). Harvey suggests that misperception is a cognitive distortion, similar to the type of cognitive distortions observed in conditions such as social anxiety disorder and panic disorder. The model proposes that excessive pre-sleep cognitive activity, related to concerns about getting enough sleep and the impact of poor sleep on daytime functioning/health, leads to increases in physiological arousal and emotional distress. Such anxiety and distress about sleep promotes a narrowing of attention and a bias towards the detection of sleep-related threat cues. There is then a reciprocal process whereby the detection of sleep related threat leads to an exacerbation of worry and concern about sleep, which promotes attentional bias toward sleep-related threat and leads to further exacerbation of worry and concern about sleep. These processes are proposed to result in the individual overestimating their sleep deficit (misperception). In turn, misperception is thought to perpetuate problems with sleep by feeding into concerns that the individual has about not getting enough sleep and the impact of this on daytime functioning/health.

The view that sleep discrepancy reflects a cognitive distortion is not held by all researchers in the field. Others have argued that individuals with insomnia perceive their sleep accurately, however lack of sensitivity in the currently utilised objective methods for assessing sleep have resulted in researchers being unable to observe what it is that individuals with insomnia are actually experiencing (Kay, Buysse, Germain, Hall, & Monk, 2015; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). For example, using a higher resolution measure of cortical activity during sleep

(power spectral analysis), decreased cortical activity in non-rapid eye movement (NREM) sleep and increased relative cortical activation in rapid eye movement (REM) sleep was found in individuals with paradoxical insomnia compared to good sleepers and those with psychophysiological insomnia (St-Jean, Turcotte, Perusse, & Bastien, 2013). They suggest that these differences in activation underlie worse perceived sleep quality in paradoxical insomnia. Individuals with insomnia display more high frequency Electroencephalogram (EEG) during sleep onset, which has led to the suggestion that cortical arousal is the mechanism underlying sleep discrepancy (Perlis, Merica, Smith, & Giles, 2001). Another proposal is that sleep discrepancy can be explained by individuals with insomnia having pockets of neurons simultaneously awake and sleep (local sleep), a characteristic of sleep which is not picked up by cortical EEG/ PSG (Buysse, Germain, Hall, Monk, & Nofzinger, 2011).

Thus there is a great deal of debate within insomnia research regarding the mechanisms underlying subjective/objective sleep discrepancy. We sought to conduct a systematic review of the literature examining the mechanisms underlying sleep discrepancy in order to synthesis the findings and evaluate the evidence for a range of proposed mechanisms. A comprehensive review that takes a broad look at the literature and identifies potential mechanisms already exists (Harvey & Tang, 2012). Therefore we sought to add to the pre-existing review by including more recently published studies and by focusing on studies with methodologies that allow for stronger conclusions in terms of causality and the direction of effects (experiments, treatment studies with mediation analysis). A systematic search was conducted and an insufficient number of studies were retrieved to warrant a meaningful synthesis of findings. Therefore Paper 1 does not focus on subjective/objective sleep discrepancy. Instead we examine the impact of cognitive behaviour therapy for insomnia (CBT-I) on cognitive functioning. Paper 2 reports on an empirical study in which predictors of subjective/objective sleep discrepancy were examined using a daily process approach.

Figure 1: Harvey's cognitive model of the maintenance of insomnia



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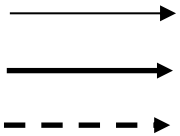

leads to

exacerbates

relationship between night time and daytime processes

Paper 1: Does Cognitive Behavioural Therapy for Insomnia improve cognitive functioning? A systematic review and narrative synthesis

Selection of the review question

Insomnia is associated with impairments in cognitive functioning (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012) and there is evidence that poor sleep contributes to cognitive decline (Altena, Ramautar, Van Der Werf, & Van Someren, 2010). This is thought to be due to the important role of sleep in maintaining brain health and functioning (Kreutzmann, Havekes, Abel, & Meerlo, 2015). Interventions which improve sleep may also lead to improvements in cognitive performance however this has not been systematically examined. The review aimed to evaluate the evidence for improvements in cognitive functioning following CBT-I. Factors that might explain heterogeneity in findings across studies are discussed and a comment on the strengths and weaknesses of the included studies is provided. Finally, a research agenda based on the findings is outlined.

Limitations of the methodology

The review did not include grey literature and this may have biased the findings. It is well known that statistically significant results are more likely to be published than non-significant results (Balshem et al., 2008). The fact that cognitive functioning was a secondary outcome in many of the included studies may have limited this bias to some degree.

The screening of study titles and abstracts was conducted by one researcher independently (VH) and it is possible that potentially relevant articles were missed. Ideally a second researcher would have screened a proportion of the titles and abstracts however this was not possible due to limited resources.

A quality appraisal tool was utilised (Downs & Black, 1998), however the tool has several limitations. The main limitation is that there was no weighting according to methodological importance. For example, one-point was available for “attempted blinding of assessors” and “randomisation” and the same number of points was available for “actual probability values reported” , even though the bias that could result from lack of blinding and randomisation far outweigh the potential bias from inadequacies in reporting. As a result, many of the included studies received low quality ratings on the basis of reporting.

The inclusion criteria for studies specified that the treatment should include any two of the recognised components of CBT-I. There is precedence for this in the literature (Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015). This criteria lead to the inclusion of studies that some may argue should not be classified as having delivered CBT-I, because the treatment did not incorporate cognitive, behavioural and educational components. Due to the preliminary nature of the literature, broad inclusion criteria were specified. In addition, the necessary and sufficient components of CBT-I are yet to be determined and therefore restricting the review to only those studies that utilised cognitive, behavioural and educational components may have missed important findings.

Paper 2: Predictors of subjective/objective sleep discrepancy in poor sleepers: examining daily associations using multilevel modelling

Choice of research question and methodology

Individuals who underestimate their sleep may be at greater risk of developing insomnia, due to the potential impact of misperception on the generation of concern and anxiety about sleep, which can inhibit normal sleep regulatory processes (Harvey & Tang, 2012). It has been suggested that discrepancy between subjective and objective estimates of sleep may represent a prodromal phase that eventually culminates in the development of an objective sleep deficit (Harvey & Tang, 2012). If this is the case, sleep misperception is an important target for intervention.

This study focused on the issue of sleep discrepancy because in spite of its obvious importance for our understanding of insomnia, there is relatively little research or consensus among researchers, concerning its causes and consequences. Initially the aim of this study was to investigate the impact of sleep discrepancy on sleep-related anxiety and subsequent sleep, in order to gather evidence for a proposed pathway through which sleep discrepancy contributes to the maintenance of insomnia. A study using an experimental paradigm was designed, in which we sought to manipulate perceptions of sleep using false feedback. We planned to recruit poor sleepers and deliver predetermined positive or negative feedback about sleep, using an actigraphy watch which allows for the display of pre-programmed written messages. The study protocol specified that sleep-related attentional bias would be assessed using a modified dot probe task (MacMahon, Broomfield, & Espie, 2006). We also planned to evaluate the impact of feedback on daytime functioning using objective and subjective measures of sustained attention and a questionnaire assessing the daytime symptoms of insomnia (Buysse et al., 2007). We hypothesised that those participants who received negative feedback about their sleep would display enhanced attentional bias to sleep-related threat and worse daytime symptoms of insomnia.

Due to concerns about the ethics of deceiving participants by providing them with false feedback about their sleep and the potential implications that this might have in terms of creating greater anxiety about sleep in already poor sleepers, this study was not carried out. Concerns about the ethics of using false feedback were raised by one of my supervisors in October 2014. In January 2015, it was decided that we would complete a study of naturally occurring, day-to-day variations in sleep discrepancy using a daily process approach to investigate the extent to which sleep

discrepancy co-varies with various psychophysiological factors. It was hoped that this would enhance our understanding of the potential mechanisms underlying sleep discrepancy. The advantage of this approach is that it enables the assessment of sleep discrepancy over multiple time-points and captures naturally occurring variations in outcome and predictor variables, providing more ecologically valid information.

Little is known about intra-individual variability in sleep discrepancy, however given evidence that sleep is highly variable in individuals with insomnia (Bei, Wiley, Trinder, & Manber, 2016; Sanchez-Ortuno, Carney, Edinger, Wyatt, & Harris, 2011; Sanchez-Ortuno & Edinger, 2012), we reasoned that high levels of variability in sleep discrepancy would also be present. There has been one study in older adults showing that discrepancy in sleep onset latency is 150% more variable within individuals, than it is between individuals (Kay, Dzierzewski, Rowe, & McCrae, 2013). We supposed that quantifying the level of intra-individual variability in sleep discrepancy in a sample of poor sleepers would make an important contribution to the field.

Limitations of the methodology

Due to the study design, the data cannot speak to causality or the direction of the associations uncovered. Specifically, whilst the findings from this study suggest that sleep discrepancy is associated with cognitive arousal, this may be due to biases that result from retrospective assessment (e.g. hindsight bias) rather than because higher levels of cognitive arousal lead to greater sleep discrepancy. A study in which the measurement of predictor variables precedes the measurement of the outcome variable would allow for a lagged analysis, which could provide information about the direction of the effects. This was not possible in the current study, due to the challenges of assessing experiences during the pre-sleep period, without interfering with actual sleep.

In this study we used actigraphy to provide an objective measure of sleep. As discussed in paper one, actigraphy is not the gold standard for distinguishing sleep from wake. Specifically, there is evidence that actigraphy overestimates sleep time in individuals with insomnia (Sadaka et al., 2014). This is problematic for our calculation of sleep discrepancy, particularly for sleep onset latency (SOL) discrepancy, where the inaccuracies of actigraphy are more apparent. Indeed, measurement error related to the use of actigraphy may have contributed to the high level of intra-individual variability in SOL discrepancy that is evident in this sample. It is possible that different

results would be found with the use of different actigraphy devices/algorithms or with PSG. Therefore, it is important that future studies seek to replicate findings from the current study, using alternative objective methods for assessing sleep. The advantages of using actigraphy in the current study are that it enabled the assessment of objective sleep over multiple nights, in the participants' home environment. The controlled environment under which PSG is carried out precludes the examination of habitual sleep/wake patterns and can also introduce biases (for example the first night effect) (Edinger et al., 1997; Edinger et al., 2001).

This study focused on the contribution of state variables to sleep discrepancy and the influence of trait characteristics was not addressed. It is possible that trait variables moderate the associations uncovered, for example, the relationship between cognitive arousal and sleep discrepancy may be stronger in those with more maladaptive beliefs about sleep. The current study was not adequately powered to investigate this hypothesis however this may be an interesting avenue to explore in future research.

Limitations of the measures

It is possible that the measures utilised in this study assess overlapping constructs. Evidence for this comes from the high correlations between the majority of predictor variables (Table 1). This is problematic for the multivariate analysis, due to potential issues with multicollinearity. Multicollinearity can lead to imprecise estimates of regression coefficients and reduced power to detect significant effects (Yoo et al., 2014). Multicollinearity was assessed by computing tolerance and variance inflation factor statistics (Table 2). Tolerance values were less than the recommended threshold of 10, for all of the predictor variables. Therefore, the inclusion of variables in the multivariate analysis appears justified.

There may be a common factor or factors, which explain the high correlations among variables. Shared variance between predictor variables should be examined and modelled in future studies. For example, using structural equation modelling, a study of 224 healthy individuals who completed questionnaires assessing depressive symptoms, trait anxiety, trait anger, trait positive affect, trait rumination and sleep quality found that the shared variance among the negative emotional factors (a latent variable which they termed negative affect) predicted sleep quality, whereas the individual measures were not predictive (Stewart, Rand, Hawkins, & Stines, 2011). There are a large number

Table 1: Correlation coefficients for daily predictor variables

Predictors	GCTI	Cognitive Arousal	Physiological Arousal	Sleep Effort	Mood	Sleep Fragmentation
GCTI	1.00					
Cognitive Arousal	0.63***	1.00				
Physiological Arousal	0.62***	0.57***	1.00			
Sleep Effort	0.52***	0.50***	0.39***	1.00		
Mood	-0.29***	-0.20**	-0.16**	-0.14*	1.00	
Sleep Fragmentation	0.10	0.08	0.01	0.08	-0.18*	1.00

GCTI = Glasgow content of thoughts inventory, Mood = mood upon awakening
* P < 0.05
** P < 0.01
*** P < 0.001

Table 2: Tolerance and variance inflation factor values for variables predicting MI

Predictors	VIF	1/VIF
GCTI	2.25	0.4451
Cognitive Arousal	2.03	0.4935
Physiological Arousal	1.89	0.5292
Sleep Effort	1.49	0.6702
Mood	1.12	0.8964
Sleep Fragmentation	1.03	0.9682

GCTI = Glasgow content of thoughts inventory, Mood = mood upon awakening

of state and trait psychological factors that are hypothesised to be important in sleep discrepancy. Simultaneously examining potentially overlapping constructs and separating out the influence of their common and unique features may be a way of honing in on the important factors and gaining conceptual clarity. The difficulty with performing these kinds of analyses is that large samples are required.

The choice of which psychological processes to assess was based on evidence from the literature. A limitation of this study is that the contribution of EEG parameters that are proposed to play a role in sleep discrepancy, were not examined. Sleep discrepancy has been associated with heightened brain activity during PSG defined sleep (Perlis et al., 2001). Brief arousals from REM sleep and time spent in REM sleep have also been shown to correlate with degree of discrepancy (Feige et al., 2008). The potential importance of these variables for understanding sleep discrepancy is recognised and the absence of their measurement in this study is related to difficulties in incorporating these techniques into a study methodology which was feasible, given the constraints of time and resources. It will be important for future studies to include measures such as high density EEG, in order to understand how relevant processes are expressed across different levels of explanation (e.g. cognitive arousal and cortical arousal). We found two psychological variables that appear to be independently predictive of total sleep time (TST) misperception (cognitive arousal and mood upon awakening). So far, the literature seems to have focused on single explanations. It seems likely that both bottom-up and top-down cognitive processes contribute. We suggest that the role of multiple psychological and biological processes and their interactions will need to be modelled, in order to fully understand this phenomenon.

This study utilised single question items, taken from validated scales to measure cognitive arousal, physiological arousal and sleep effort. The use of single items has not been validated. The fact that some variables were significant predictors and others were not may be related to the ability of the item, or lack thereof, to reliably assess the underlying construct. The advantage of using single item questions to assess cognitive arousal, physiological arousal and sleep effort was that all of these variables were measured in the same way (using one question, answered on a 100mm VAS), which better enables a comparison between predictors. The use of full scale questionnaires was considered however participant burden was a concern. Moreover, there is evidence from Experience Sampling Method (ESM) studies that excessive diary length can reduce compliance (Morren, van Dulmen, Ouwerkerk, & Bensing, 2009).

The use of sleep diaries likely prompted participants to pay closer attention to their sleep, which may have led to more accurate perceptions of sleep compared to usual. It is also possible that monitoring increased sleep-related anxiety and distress, which according to the cognitive model of the maintenance of insomnia has the potential to result in less accurate perceptions of sleep (Harvey, 2002). There has been little research into the impact of sleep monitoring on subjective/objective sleep discrepancy and this should be an area for future research.

Limitations of the statistical analysis

This study did not employ a correction for multiple comparisons and therefore it is possible that a proportion of the statistically significant results that are reported are due to type 1 error. With regards to TST misperception index, all of the significant predictors from the univariate analyses would remain statistically significant if a Bonferroni correction was applied. However, the significant association between sleep effort and sleep onset latency discrepancy would not survive a correction for multiple comparisons. Bonferroni adjustment has been criticised for increasing the chance of type 2 errors and there is guidance which states that adjustment is required only where the same tests are repeated in subsamples or when there is sequential testing of the data. Given that an association between sleep effort and sleep onset latency discrepancy was hypothesised a priori, a Bonferroni correction was not deemed necessary (Perneger, 1998).

Limitations of the sample

We did not exclude participants from this study on the basis of high levels of symptoms of anxiety or depression and therefore, it is possible that our sample comprises individuals whose insomnia is secondary to an undiagnosed mental health problem. Individuals who are diagnosed with primary insomnia often have symptoms of depression and anxiety and similarly, those whose sleep difficulties are thought to be comorbid with a mental health problem often display the sleep-related anxiety and distress that is present in primary insomnia (Edinger et al., 2011). For these reasons, the division between primary and secondary insomnia has been questioned. Moreover, there is evidence that subjective/objective sleep discrepancy occurs across insomnia diagnoses (Harvey & Tang, 2012). However, it is possible that different results would be found in a sample where more stringent criteria are applied (e.g. well phenotyped psychophysiological insomnia).

A further limitation is that although we attempted to exclude participants with a suspected sleep disorder other than insomnia, our screening was based on self-report as opposed to PSG, which is

likely to be less reliable. The decision to use a self-report assessment was based on the lack of availability of PSG.

Clinical implications of the findings

A common finding from trials evaluating the efficacy of CBT-I is that subjective measures of sleep show more robust improvements than objective measures (Mitchell, Gehrman, Perlis, & Umscheid, 2012). It is possible that CBT-I works (at least in part) by correcting subjective/objective sleep discrepancy. There is evidence that CBT-I leads to reductions in pre-sleep cognitive arousal (Vincent & Lewycky, 2009). Given the results from the current study, one possibility that warrants investigation is that CBT-I reduces subjective/objective sleep discrepancy by attenuating cognitive hyperarousal.

Given that insomnia is a subjective complaint, interventions for insomnia may benefit from a greater focus on treatments which lead to changes in perceptions of sleep. Further research is needed in order to determine whether the addition of interventions which target cognitive arousal, sleep effort and mood (for example, paradoxical intention to alleviate sleep effort (Broomfield & Espie, 2003) and mindfulness meditation practice to reduce cognitive arousal (Ong, Shapiro, & Manber, 2008)) enhance the effectiveness of standard CBT-I.

The high levels of intra-individual variability observed for subjective/objective sleep discrepancy variables indicate that assessment of sleep should be conducted over multiple time points in order to get an accurate picture of the extent to which an individual with insomnia may be underestimating objective sleep.

The findings from this study could also be used to help individuals with insomnia understand the factors which contribute to perceptions of poor sleep. Specifically, assessment of these variables could be included in enhanced sleep diaries. In addition, these factors could be incorporated into behavioural experiments which seek to reduce sleep discrepancy.

Theoretical implications of the findings

The results from this study add to the evidence for an association between cognitive arousal and sleep discrepancy. They also highlight other psychological variables that may contribute to sleep discrepancy, specifically sleep effort and morning mood. Importantly, we identified two factors that

were independently predictive of TST misperception. This suggests that multiple processes are likely to contribute to subjective/objective sleep discrepancy.

The potential impact of memory biases and the role of expectations in subjective/objective sleep discrepancy are relatively understudied. It is well known from research in other fields, such as medically unexplained symptoms and pain, that the recall of perceptual experiences can be biased by a number of factors, including heuristics, situational cues and semantic knowledge (Houtveen & Oei, 2007). Greater levels of subjective/objective discrepancy are apparent when global retrospective evaluations of sleep (e.g. participants are asked to make judgements about their sleep over the past 7 nights) are gathered, compared to single night evaluations (Takano, Boddez, & Raes, 2016). This may be related to the observation that longer intervals between symptom and reporting promote greater reliance on semantic memory. A role for memory biases in subjective/objective sleep discrepancy does not preclude explanations based on an underlying sleep disturbance and these factors may interact. However more research is needed in order to assess the influence of memory biases in subjective/objective sleep discrepancy.

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Appendices

Appendix A: Author Guidelines for Sleep Medicine Reviews

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The aim of this journal is to review all aspects of sleep medicine. It will provide in-depth and up-to-date Clinical Reviews of sleep disorders, including their aetiology, diagnosis, treatment and implications for related conditions at an individual and a public health level, as well as Physiological Reviews, Theoretical Reviews and Historical Notes.

Clinical and (patho)physiological information about sleep disorders published in peer-reviewed journals devoted to the many disciplines involved in sleep medicine are reviewed. These disciplines include cardiology, dentistry, endocrinology, general medicine, geriatrics, neurology, ORL, paediatrics, pharmacology, physiology, psychiatry, psychology and pulmonology.

The journal intends to be an international forum for opinion within the field of sleep medicine, covering areas of controversy and debate as well as areas of future research. It publishes narrative reviews, systematic reviews and editorials primarily for the clinician. Submission of systematic or meta-analytic reviews following validated guidelines is encouraged.

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Please avoid footnotes where possible.

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Practice Points. Present the important points for readers to remember in clearly indicated box(es), e.g.:

Practice Points

Sleep apnea clinical prediction rules may be useful to:

1. exclude the diagnosis when the probability is low and the patient has insignificant symptoms;

2. establish an *a priori* probability before considering the utilization of a non-PSG diagnostic method;

3. prioritize patients for polysomnography according to the probability that they will have a positive result.

Research Agenda. Please indicate points which you feel would repay further research in box(es), e.g.:

Research Agenda

In the future we need to be able to not only predict those with sleep apnea, but also which patients:

1. are at highest risk of morbidity and mortality and whether this risk can be modified by treatment;

2. obtain the most significant improvement in their quality of life as a result of treatment;

3. are most likely to be compliant with therapy.

It is recommended not to use acronyms or abbreviations in the Research Agenda and the Practice Points.

Please note that *Practice Points, Research Agenda* and Asterisked Key References are standard features of reviews published in *Sleep Medicine Reviews* and we ask that authors to pay particular attention to incorporating these into their reviews before submitting the article.

Acknowledgements for personal and technical assistance should be indicated on the title page. Financial support and any conflict of interest are also to be stated in the acknowledgements (see above). The source of equipment and drugs may be included here as well.

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An excerpt from a table as illustration:

Authors, reference number	Study type	Type of patients	# SUDEP	# SUDEP during sleep	In Bed	% of SR-SUDEP
Antoniuk SA et al 2001 [41]	Uncontrolled descriptive study	Unselected epileptic patients	20	2/8 witnessed deaths	13	25%
Donner EJ et al 2001 [42]	Uncontrolled descriptive study	Children	27		16	59%
Hitiris N et al 2007 [29]	case-control study §	medically refractory epilepsy	62	-	59	95%

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Appendix B: Search Strategy

Literature Search

EMBASE searched through Ovid Online from inception to 2016 week 12

1.((((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap* or psychotherap*)).ti. or ((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*).ab.) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap*).ab.

Results: 186 (19th March 2016)

2. limit 1 to english language

Results: 150 (19th March 2016)

PsycINFO searched through Ovid Online from inception to March week 3 2016

1.((((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap* or psychotherap*)).ti. or ((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*).ab.) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap*).ab.

Results: 144 (19th March 2016)

2. limit 1 to english language

Results: 127 (19th March 2016)

MEDLINE searched through Ovid Online from inception to March week 2 2016

1.((((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap* or psychotherap*)).ti. or ((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*).ab.) and ((((((((((((((cognitive adj behav* adj therap*) or

behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap*).ab.

Results: 86 (19th March 2016)

2. limit 1 to english language

Results: 71 (19th March 2016)

MEDLINE-In Process searched through Ovid Online from inception to 19th March 2016

1.((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap* or psychotherap*)).ti. or ((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*).ab.) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap*).ab.

Results: 14 (19th March 2016)

2. limit 1 to english language

Results: 13 (19th March 2016)

EBM Reviews searched through Ovid Online from inception to March week 3 2016

1.((((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap* or psychotherap*)).ti. or ((((((((((sleeplessness or insomnia* or sleep) adj initiation) or sleep) adj maintenance) or sleep) adj disorder*) or dysomnia* or poor) adj sleep*) or sleep) adj problem*) or sleep) adj disturbance*).ab.) and ((((((((((((((cognitive adj behav* adj therap*) or behav*) adj therap*) or CBT or CBT-I or ICBT or self) adj help) or sleep) adj hygiene) or stimulus) adj control) or sleep) adj restriction) or relaxation or behav*) adj modification) or cognitive) adj therap*) or imagery or psychotherap*).ab.

Results: 20 (19th March 2016)

2. limit 1 to english language

Results: 20 (19th March 2016)

CINAHL Plus searched through EBSCO Host Research Databases

(TI + sleeplessness + OR + AB + sleeplessness + OR + TI + insomnia* + OR + AB + insomnia* + OR + TI + sleep + initiation + OR + AB + sleep + initiation + OR + TI + sleep + maintenance + OR + AB + sleep + maintenance + OR + TI + sleep + disorder* + OR + AB + sleep + disorder* + OR + TI + dysomnia* + OR + AB + dysomnia* + OR + TI + poor + sleep* + OR + AB + poor + sleep* + OR + TI + sleep + problem* + OR + AB + sleep + problem* + OR + TI + sleep + disturbance* + OR + AB + sleep + disturbance) + AND + (TI + cognitive + behav* + therap* + OR + AB + cognitive + behav* + therap* + OR + TI + behav* + therap* + OR + AB + behav* + therap* + OR + TI + CBT + OR + AB + CBT + OR + TI + CBT-I + OR + AB + CBT-I + OR + TI + ICBT + OR + AB + ICBT + OR + TI + self-help + OR + AB + self-help + OR + TI + sleep + hygiene + OR + AB + sleep + hygiene + OR + TI + stimulus + control + OR + AB + stimulus + control + OR + TI + sleep + restriction + OR + AB + sleep + restriction + OR + TI + relaxation + OR + AB + relaxation + OR + TI + behav* + modification + OR + AB + behav* + modification + OR + TI + cognitive + therap* + OR + AB + cognitive + therap* + OR + TI + imagery + OR + AB + imagery + OR + TI + psychotherap* + OR + AB + psychotherap*)

Results limited to English Language: 850 (19th March 2016)

Cochrane Library

#1: sleeplessness OR insomnia* OR “sleep initiation” OR “sleep maintenance” OR “sleep disorder” OR dysomnia OR “poor sleep” OR “sleep problem*” OR “sleep disturbance* ti,ab,kw

Results: 9572

#2: “cognitive behav* therap*” OR “behav* therap*” OR CBT OR CBT-I OR ICBT OR “self-help” OR “sleep hygiene” OR “stimulus control” OR “sleep restriction” OR “relaxation” OR “behav* modification” OR “cognitive therap*” OR imagery OR psychotherapy* ti,ab,kw

Results: 33191

#1 AND #2

Results: 1096 (19th March 2016)

Appendix C: Downs and Black Quality Rating Scale Scoring Information

Item	How the item was scored
1. Hypotheses/aims clear?	If the aims of the study or hypotheses were stated, then this item scored a 1, otherwise a score of 0 was given.
2. Main outcomes clearly described?	If the main outcomes of the study are stated in the introduction or methods section of the paper, this item scored a 1. If the main outcomes were first mentioned in the results section, this item scored a 0.
3. Participants clearly described?	If inclusion and exclusion criteria were clearly stated, this item was scored a 1. If inclusion and exclusion criteria were not clearly specified, a score of 0 was given.
4. Interventions clearly described?	If the treatment and control conditions were clearly described, this item scored a 1, otherwise a score of 0 was given.
5. Distribution of principle confounders clearly described? Age, severity (0,1,2)	If a full list of confounding variables was provided for the treatment and control group, this item scored a 2. If a partial list was provided (for example, the list included age and sex but did not include a measure of insomnia severity), this scored a 1. If there was no control group in the study, this item scored a 0.
6. Main findings clearly described?	If data were presented for the outcome variable of interest (cognitive functioning), this item scored a 1.
7. Estimates of random variability provided?	If the standard error, standard deviation, confidence interval or interquartile range for the cognitive functioning outcome variable was presented, this scored a 1, otherwise a score of 0 was given.
8. Adverse events reported?	If the paper described an effort to measure adverse events, this was scored a 1, otherwise a score of 0 was given.
9. Characteristics of participants lost to follow-up reported?	Where there were no losses to follow-up the study automatically received a score of 1. If there were losses to follow-up and the characteristics of those lost were reported this scored a 1. If the study did not report numbers of participants lost to follow-up, a score of 0 was given.
10. Actual probability values reported?	If p values were presented in full, e.g. $p = 0.035$ as opposed to $p<0.05$, this scored a 1. Otherwise, a score of 0 was given.
11. Sample approached representative?	Participants approached were considered to be representative if they comprised the entire source population, an unselected sample of consecutive patients or a random sample. If the sample approached was representative, a score of 1 was given, otherwise a score of 0 was given. If it was not possible to determine whether the sample approached was representative on the basis of the details reported, a score of 0 was given.
12. Participants representative?	If the study demonstrated that the distribution of confounding variables (e.g., sex, age, etc.) was the same in the study sample compared to what is known about the population (e.g. typical characteristics of those with chronic insomnia), this scored a 1. Otherwise a score of 0 was given.
13. Treatments representative?	If the staff, setting and facilities where the participants were treated was representative of the treatment that the majority of patients receive, a score of 1 was given. If the treatment was delivered in a specialist centre or research setting, a score of 0 was given. If it was not possible to determine if the treatments were representative, a score of 0 was given.
14. Attempted blinding- participants?	If there was an attempt to blind participants to the intervention, a score of 1 was given. If there was no attempt to blind participants, a score of 0 was given.
15. Attempted blinding- assessors?	If there was an attempt to blind those assessing

	outcomes of the intervention, a score of 1 was given. If there was no attempt to blind assessors or if it was not possible to determine from the report if assessors were blind, a score of 0 was given.
16. Unplanned analyses?	If an analysis which was not clearly planned at the outset was conducted, a score of 0 was given. If there were no retrospective, unplanned analyses, a score of 1 was given.
17. Time period the same for cases and controls?	If the length of follow-up was the same for each participant, or where differences in the length of follow-up were adjusted for, a score of 1 was given. Otherwise a score of 0 was given.
18. Statistical tests appropriate?	If the statistical tests used were appropriate for the aims of the study, sample size and distribution of the data, a score of 1 was given. If details regarding the distribution of the data were not presented, it was assumed that the appropriate statistical analysis was used. If it was not possible to determine whether the statistical tests were appropriate on the basis of the report, a 0 was given.
19. Was compliance with the intervention appropriate?	If data relating to treatment fidelity or number of intervention sessions completed by participants was presented, a score of 1 was given. Otherwise, a score of 0 was given.
20. Main outcomes valid and reliable?	If a validated measure was used to assess cognitive function, a score of 1 was given. If the measure used to assess cognition was not validated, a score of 0 was given. If it was not possible to determine the validity and reliability of the measure used, a score of 0 was given.
21. Intervention and control from the same population?	If the participants in the treatment and control groups came from the same population (e.g. selected from the same hospital) a score of 1 was given. If participants in the treatment and control groups came from different populations a score of 0 was given. If it was not possible, on the basis of the report, to determine whether or not participants in the treatment and control groups came from the same population, a score of 0 was given.
22. Recruited over the same period?	If the study stated that participants in the treatment and control groups were recruited over the same period, a score of 1 was given. If it was not possible to determine this on the basis of the report, a score of 0 was given. If there was no control group, a score of 0 was given.
23. Randomisation	If the study stated that participants were assigned to groups randomly, a score of 1 was given. If it was not possible to determine this on the basis of the report, a score of 0 was given. If participants were assigned on a non-random basis, a score of 0 was given. If there was no control group, a score of 0 was given.
24. Randomisation concealed?	If assignment to condition was concealed from both participants and researches until recruitment was complete, a score of 1 was given. If it was not possible to determine this on the basis of the report, a score of 0 was given. If there was no control group, a score of 0 was given.
25. Adequate adjustment for confounding?	If the distribution of confounding variables differed between groups and was not adjusted for, a score of 0 was given. If there was no control group, a score of 0 was given. If the distribution of confounding variables was considered and found to be equal among the treatment and control group, a score of 1 was given.
26. Numbers lost to follow-up accounted for?	If an intent to treat analysis was performed or there were no participants lost to follow-up, a score of 1 was

	given. Otherwise a score of 0 was given.
27. Item omitted	

Appendix D: Author Guidelines for *SLEEP*

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It is a monthly, peer-reviewed scientific and medical journal that is published online. It features a wide spectrum of sleep-related research, and the primary audiences are clinicians and research professionals specializing in sleep-related disorders.

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5-year Impact Factor: 5.753

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All authors must disclose all potential conflicts of interest and verify authorship by submitting the Conflict of Interest

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Clinical Trials Registry- India (CTRI): http://www.ctri.in/Clinicaltrials/index.jsp
German Clinical Trials Register (DRKS): http://www.germanctr.de
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All papers must be double spaced, have one-inch margins (top, bottom, and sides), and include page numbers.

Manuscript should be structured using the following components:

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The title page must include the following:

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- Full first and last names, highest academic degrees, and institutional affiliations for all authors
- The institution at which the work was performed

- Disclosure of the presence OR absence of financial support
- Disclosure of any off-label or investigational use
- Disclosure of the presence OR absence of any conflicts of interest for each author to match what is indicated on their individual Conflict of Interest form
- Clinical trial name, URL, and registration number (if no clinical trial is indicated, please include a statement to this effect)
- Corresponding author's full address, phone and fax numbers and e-mail address
- The total number of figures
- The total number of tables

No submission will be considered for review without all of the above information.

Abstract
Each original manuscript, rapid publication, and short note must be preceded by a structured abstract. The abstract is limited to 250 words. The components of this format are (start each on a new line): **Study Objectives, Methods, Results, Conclusions** and**Keywords.**

Conclusions should not simply restate results and should include as few abbreviations as possible. Abstracts are not required for letters to the editor, editorials and book reviews. Please provide no fewer than three but no more than ten keywords that reflect the content of your manuscript. For guidance consult the Medical Subject Headings - Annotated Alphabetic List, published each year by the National Library of Medicine.

Statement of Significance
The statement of significance will appear on the first page of the manuscript just below the abstract.

The statement of significance should:

- Be no more than 120 words maximum
- NOT be repetitious with the abstract or the "In summary…" paragraph that is often placed at the end of the discussion
- NOT contain references and should avoid numbers, description of methods and acronyms unless necessary
- Provide a clear statement of the importance and novelty of the research, using language that can be understood by scientists or clinicians without special knowledge of the field
- Include a statement about critical remaining knowledge gaps and/or future directions of the work
- For basic science papers, include a reasonable statement about human disease relevance and/or translational implications

Introduction
State the object of research with reference to previous work.

Methods
Describe methods in sufficient detail so that the work can be duplicated, or cite previous descriptions if they are readily available.

Results
Describe results clearly, concisely, and in logical order. When possible give the range, standard deviation, or mean error, and significance of differences between numerical values.

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Interpret the results and relate them to previous work in the field. Include a paragraph near the end of the discussion that briefly lists the limitations of the study.

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The minimum compatible with the requirements of courtesy should be provided.

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Sample citations within the body of a paper
According to our previous work,[1,3-8,19]
The patients were studied as follows[3,4]:

Sample references

Article:
1. Kapur VK, Baldwin CM, Resnick HE, Gottlieb DJ, Nieto FJ. Sleepiness in patients with moderate to severe sleep-disordered breathing. Sleep 2005;28:472-7.

2. Quan SF, Howard BV, Iber C, et al. The Sleep Heart Health Study: design, rationale, and methods. Sleep 1997;20:1077-85.

Book:
3. Guilleminault C, Lugaresi E, eds. Sleep/wake disorders: natural history, epidemiology, and long-term evolution. New York: Raven Press, 1983.

Chapter of a book:
4. Coleman RM, Bliwise DL, Sajben N, et al. Epidemiology of periodic movements during sleep. In: Guilleminault C, Lugaresi E, eds. Sleep/wake disorders: natural history, epidemiology, and long-term evolution. New York: Raven Press, 1983:217-30.

Website:
5. "Journals," American Academy of Sleep Medicine, accessed August 26, 2015, http://www.aasmnet.org/journals.aspx.

DETAILS OF STYLE

Sleep Medicine Terminology
Follow the terminology usage recommendations in the AASM Style Guide for Sleep Medicine Terminology.

Drug Names
Use generic names in referring to drugs; trade names may be given in parentheses after the first mention, but the generic name should be used thereafter.

Abbreviations
Follow the list of abbreviations given in "Uniform Requirements for Manuscripts Submitted to Biomedical Journals" (see section on References). For additional abbreviations, consult the Council of Biology Editors Style Manual (available from the Council of Biology Editors, Inc., 9650 Rockville Pike, Bethesda, MD 20814) or other standard sources. Please note that journal style for the abbreviation of standard deviation is SD. Please do not use SD as an abbreviation for sleep deprivation.

Please provide on a separate page all abbreviations used with their full definition. Each should be expanded at first mention in the text and listed parenthetically after expansion.

REVIEW PROCESS

Editors first determine if a submitted manuscript is suitable for review and publication. Manuscripts selected are then sent for peer-review to reviewers who are selected based on their expertise related to the particular manuscript. After reviews are in, a recommendation of accept, reject or revise (for further consideration) is made by the Associate Editor to the Editor-in-Chief, who makes the final decision.

Manuscripts are reviewed with due respect for the author's confidentiality. At the same time, reviewers also have rights to confidentiality, which are respected by the editors. The editors ensure both the authors and the reviewers that the manuscripts sent for review are privileged communications and are the private property of the author.

When submitting a manuscript for consideration for publication, authors may suggest the names of potential reviewers to invite and/or exclude.

RESUBMISSIONS

If a manuscript is returned to the author(s) for revisions, all resubmissions must follow the Instructions for Submitting a Manuscript and include the following:

1. Both a clean copy and a redline copy of the revised submission. NOTE: If the redline copy was created using “track changes” mode in Word, please create a PDF file of the redline version and upload the PDF file in Rapid Review. If you are not able to create a PDF file of your redline version, please use alternative font colors or highlighting tools in Word to show the redlined changes – not “track changes” mode.
2. You must also upload a letter (Corresponding Author’s Rebuttal) responding to each of the points made by the reviewers.

The deadline for submission of a revised manuscript needing major revisions is two months from the date of the notice. For Minor revisions, the deadline for resubmission is one month. There is no guarantee that a revised manuscript will be accepted for publication.

PLAGIARISM REVIEW

The editorial office carefully monitors papers submitted to *SLEEP* for plagiarism. We define plagiarism to include: literal copying - reproducing a work word for word, in whole or in part, without permission and acknowledgment of the original source; paraphrasing - reproducing someone else's ideas while not copying word for word, without permission and acknowledgment of the original source; substantial copying - copying images, or data from other sources; text-recycling - reusing text from your own previous publications.

Any text contained in a manuscript that is directly copied from another source must be placed within quotation marks and the original source must be properly cited. If a paper captures the essence of a previously published work, that work should be cited. If any paraphrasing is included, the source must be properly referenced and the meaning intended by the source must not be changed. All works that may have inspired a study’s design or manuscript structure must be properly cited.

If plagiarism is detected during any part of the peer review process, the manuscript may be rejected. For published papers where plagiarism is detected, we reserve the right to issue a correction or retract the paper, whichever is deemed appropriate. We reserve the right to inform authors' institutions about plagiarism detected either before or after publication.

PROOFING

All accepted manuscripts are subject to manuscript editing for conciseness, clarity, grammar, spelling and *SLEEP* style. After acceptance all manuscripts will be copyedited. The copyedited version will be sent to the corresponding author for review and approval and returned to the Managing Editor. Once the manuscript is scheduled for publication, the corresponding author will be notified as to the assignment of the manuscript to an issue and page proofs will be sent to the corresponding author. These proofs will be emailed as a PDF file and authors will be expected to return their corrections or approval of these proofs within the timeframe given in the email. *It is the authors’ responsibility to keep their account in Rapid Review current and to notify the Journal Editorial Office (sleepjournals@aasmnet.org) of any changes in contact information after a paper has been accepted.*

ACCEPTED PAPERS

In order to provide readers with access to new scientific developments as early as possible, all manuscripts accepted by the editor will be available online prior to being published. Accepted manuscripts are posted as received - without editing or formatting by the publisher. The layout and appearance of each article will change when published in *SLEEP*.

AHEAD OF PRINT

All accepted papers are listed in PubMed as “Ahead of Print (AOP).” This listing includes the abstract of the paper that has been accepted for publication and places it on the PubMed website in advance of final publication. The listing information is created using the copyedited and author approved manuscript of the papers. The abstract from the final, published version of papers will be used to replace the AOP listing on PubMed.

The Ahead of Print initiative was begun to answer the many requests from authors to have the abstract of their papers available shortly after acceptance. The process was started in September 2014. Please note that papers listed as AOP are not to be considered published. All embargo restrictions remain in effect until the final publication date.

Appendix E: Ethical Approval

MANCHESTER 1824

The University of Manchester

Ref: ethics15224

Miss Vanessa Herbert
School of Psychological sciences
2nd Floor Zochonis Building
Brunswick Street
University of Manchester
M13 9PL

Research Governance, Ethics and Integrity
The University of Manchester
Oxford Road
Manchester
M13 9PL
Tel: 0161 275 2206/2674

Email: research.ethics@manchester.ac.uk

1st June 2015

Dear Vanessa

Study title: Investigating predictors of sleep quality in poor sleepers Ref 15224
Research Ethics Committee No5

I write to thank you for coming to meet the Committee on the 18th May 2015. I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form and supporting documentation and your email of the 28th May 2015 as submitted and approved by the Committee.

This approval is effective for a period of five years. If the project continues beyond that period an application for amendment must be submitted for review. Likewise, any proposed changes to the way the research is conducted must be approved via the amendment process (see below). Failure to do so could invalidate the insurance and constitute research misconduct.

You are reminded that, in accordance with University policy, any data carrying personal identifiers must be encrypted when not held on a secure university computer or kept securely as a hard copy in a location which is accessible only to those involved with the research.

Reporting Requirements:

You are required to report to us the following:

1. Amendments
2. Breaches and adverse events
3. Notification of Progress/End of the Study

Feedback
It is our aim to provide a timely and efficient service that ensures transparent, professional and proportionate ethical review of research with consistent outcomes. In order to assist us with our aim, we would be grateful if you would give your view of the service that you have received from us by completing a feedback sheet https://survey.manchester.ac.uk/pssweb/index.php/779758/lang-en

We hope the research goes well.

Yours sincerely,

Patricia Gorham
Secretary to University Research Ethics Committee
Cc Simon Kyle, Gillian Haddock

Appendix F: Consent Form

MANCHESTER 1824

The University of Manchester

Investigating predictors of sleep quality in poor sleepers

CONSENT FORM

If you are happy to participate please complete and sign the consent form below

Please initial box

I agree to take part in the above project

1. I confirm that I have read the attached information sheet on the above project and have had the opportunity to consider the information.	
2. I confirm that I have had the opportunity to ask questions and had these answered satisfactorily.	
3. I understand that my participation in the study is entirely voluntary and that I am free to withdraw at any time without giving a reason and without detriment to any treatment/service.	

Name of participant	Date	Signature
Name of person taking consent	Date	Signature

Appendix G: Sleep Condition Indicator

	Score				
Item	**4**	**3**	**2**	**1**	**0**
Thinking about a typical night in the last month....					
1....how long does it take you to fall asleep?	0-15 min	16-30 min	31-45 min	46-60 min	≥61 min
2....if you then wake up...how long are you awake for in total? (add all the wakenings up)	0-15 min	16-30 min	31-45 min	46-60 min	≥61 min
3....how many nights a week do you have a problem with your sleep?	0-1	2	3	4	5-7
4....how would you rate your sleep quality?	Very Good	Good	Average	Poor	Very Poor
Thinking about the past month, to what extent has poor sleep					
5....affected your mood, energy or relationships?	Not at all	A little	Somewhat	Much	Very Much
6....affected your concentration, productivity, or ability to stay awake?	Not at all	A little	Somewhat	Much	Very Much
7....troubled you in general?	Not at all	A little	Somewhat	Much	Very Much
Finally ...					
8....how long have you had a problem with your sleep?	I don't have a problem/< 1 mo	1-2 mo	3-6 mo	7-12 mo	> 1 yr

Appendix H: Sleep Disorders Screening Questionnaire

Sleep disorders screening questionnaire

1. Narcolepsy

a. Do you sometimes fall asleep in the daytime completely without warning?

- ☐ **Yes**
- ☐ **No**

If answer is No, go straight to question 2.

b. Is it literally impossible to resist 'sleep attacks' during the day?

- ☐ **Yes**
- ☐ **No**

c. Do you have collapses or extreme muscle weakness triggered by extreme emotion?

- ☐ **Yes**
- ☐ **No**

d. Do you have visual hallucinations, either just as you fall asleep or when you wake in the morning?

- ☐ **Yes**
- ☐ **No**

e. Are you paralysed and unable to move when you wake up from your sleep?

- ☐ **Yes**
- ☐ **No**

2. Sleep breathing disorder

a. Are you a very heavy snorer?

- ☐ **Yes**
- ☐ **No**

If answer is No, go straight to question 3.

b. Does your partner say that you sometimes stop breathing?

- ☐ **Yes**
- ☐ **No**

c. Do you often wake up gasping for a breath?

- ☐ **Yes**
- ☐ **No**

d. Are you often excessively sleepy during the day or fall asleep without wanting to?

- ☐ **Yes**
- ☐ **No**

3. PLMS/ RLS
a. Do your legs often twitch or jerk or can't keep still in bed?

☐ **Yes**
☐ **No**

If answer is No, go straight to question 4

b. Is it very difficult to get to sleep because of repeated muscle jerks?

☐ **Yes**
☐ **No**

c. Do you frequently wake from sleep with sudden jerky movements or with a compulsion to move your legs?

☐ **Yes**
☐ **No**

d. Do you simply have to get out of bed and pace around to get rid of these feelings?

4. Circadian Rhythm Sleep Disorder
a. Do you tend to sleep well but just at the ''wrong times''?

☐ **Yes**
☐ **No**

If answer is No, go straight to question 5.

b. Can you sleep well enough, but only if you stay up very late?

☐ **Yes**
☐ **No**

c. Are you in a very sound sleep at normal waking time and could sleep on for hours more?

☐ **Yes**
☐ **No**

d. Can you sleep well enough, but only if you go to bed very early?

☐ **Yes**
☐ **No**

e. Do you wake very early, bright and alert and no longer sleepy?

☐ **Yes**
☐ **No**

5. **Parasomnia**
a. Do you have unusual behaviours associated with your sleep that trouble you or that are dangerous?

☐ **Yes**
☐ **No**

If answer is No, do not complete any of the remaining questions.

b. Do you sleepwalk frequently and run the risk of injuring yourself or others?

☐ **Yes**
☐ **No**

c. Do you have frequent night terrors when you are extremely distressed but not properly awake?

☐ **Yes**
☐ **No**

d. Do you act out your dreams and risk injuring yourself or others?

☐ **Yes**
☐ **No**

e. Do you have terrible recurring nightmares?

☐ **Yes**
☐ **No**

Appendix I: Dysfunctional Beliefs and Attitudes about Sleep (DBAS-16)

DYSFUNCTIONAL BELIEFS AND ATTITUDES ABOUT SLEEP

Statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement circle the number according to your PERSONAL rating falls.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

DISAGREE AGREE

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

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

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

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

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

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

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

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

15. Medication is probably the only solution to sleeplessness.

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

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

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

Appendix J: Depression, Anxiety and Stress Scale (DASS-21)

DASS

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of the time
3 Applied to me very much, or most of the time

I found myself getting upset over quite trivial things	0	1	2	3
I was aware of dryness of my mouth	0	1	2	3
I couldn't seem to experience any positive feeling at all	0	1	2	3
I experienced breathing difficulty (e.g. excessively rapid breathing, breathlessness in the absence of physical exertion	0	1	2	3
I just couldn't seem to get going	0	1	2	3
I tended to over-react to situations	0	1	2	3
I had a feeling of shakiness (e.g. legs going to give way)	0	1	2	3
I found it difficult to relax	0	1	2	3
I found myself in situations that made me so anxious I was most relieved when they ended	0	1	2	3
I felt that I had nothing to look forward to	0	1	2	3
I found myself getting upset rather easily	0	1	2	3
I felt that I was using a lot of nervous energy	0	1	2	3
I felt sad and depressed	0	1	2	3
I found myself getting impatient when I was delayed in any way (e.g. lifts, traffic lights, being kept waiting)	0	1	2	3
I had a feeling of faintness	0	1	2	3
I felt that I had lost interest in just about everything	0	1	2	3
I felt I wasn't worth much as a person	0	1	2	3
I felt that I was rather touchy	0	1	2	3
I perspired noticeably (e.g. hands sweaty) in the absence of high temperatures or physical exertion	0	1	2	3
I felt scared without any good reason	0	1	2	3
I felt that life wasn't worthwhile	0	1	2	3
I find it hard to wind down	0	1	2	3
I had difficulty in swallowing	0	1	2	3
I couldn't seem to get any enjoyment out of the things I did	0	1	2	3
I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat)	0	1	2	3
I felt down-hearted and blue	0	1	2	3
I found that I was very irritable	0	1	2	3
I felt I was close to panic	0	1	2	3
I found it hard to calm down after something upset me	0	1	2	3
I feared that I would be "thrown" by some trivial but unfamiliar task.	0	1	2	3
I was unable to become enthusiastic about anything	0	1	2	3
I found it difficult to tolerate interruptions to what I	0	1	2	3

was doing				
I was in a state of nervous tension	0	1	2	3
I felt I was pretty worthless	0	1	2	3
I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
I felt terrified	0	1	2	3
I could see nothing in the future to be hopeful about	0	1	2	3
I felt that life is meaningless	0	1	2	3
I found myself getting agitated	0	1	2	3
I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
I experienced trembling (e.g. in the hands)	0	1	2	3
I found it difficult to work up the initiative to do things	0	1	2	3

Appendix K: Consensus Sleep Diary-Morning Administration

Sleep Diary Instructions (CSD-M)

General Instructions

What is a Sleep Diary? A sleep diary is designed to gather information about your daily sleep pattern.

How often and when do I fill out the sleep diary? It is necessary for you to complete your sleep diary every day. If possible, the sleep diary should be completed within one hour of getting out of bed in the morning.

What should I do if I miss a day? If you forget to fill in the diary or are unable to finish it, leave the diary blank for that day.

What if something unusual affects my sleep or how I feel in the daytime? If your sleep or daytime functioning is affected by some unusual event (such as an illness, or an emergency) you may make brief notes on your diary.

What do the words "bed" and "day" mean on the diary? This diary can be used for people who are awake or asleep at unusual times. In the sleep diary, the word "day" is the time when you choose or are required to be awake. The term "bed" means the place where you usually sleep.

Will answering these questions about my sleep keep me awake? This is not usually a problem. You should not worry about giving exact times, and you should not watch the clock. Just give your best estimate.

Sleep Diary Item Instructions

Use the guide below to clarify what is being asked for each item of the Sleep Diary.

Date.: Write the date of the morning you are filling out the diary.

1. What time did you get into bed? Write the time that you got into bed. This may not be the time you began "trying" to fall asleep.

2. What time did you try to go to sleep? Record the time that you began "trying" to fall asleep.

3. How long did it take you to fall asleep? Beginning at the time you wrote in question 2, how long did it take you to fall asleep.

4. How many times did you wake up, not counting your final awakening? How many times did you wake up between the time you first fell asleep and your final awakening?

5. In total, how long did these awakenings last? What was the total time you were awake between the time you first fell asleep and your final awakening. For example, if you woke 3 times for 20 minutes, 35 minutes, and 15 minutes, add them all up (20+35+15= 70 min or 1 hr and 10 min).

6a. What time was your final awakening? Record the last time you woke up in the morning.

6b. After your final awakening, how long did you spend in bed trying to sleep? After the last time you woke-up (Item #6a), how many minutes did you spend in bed trying to sleep? For example, if you woke up at 8 am but continued to try and sleep until 9 am, record 1 hour.

6c. Did you wake up earlier than you planned? If you woke up or were awakened earlier than you planned, check yes. If you woke up at your planned time, check no.

6d. If yes, how much earlier? If you answered "yes" to question 6c, write the number of

minutes you woke up earlier than you had planned on waking up. For example, if you woke up 15 minutes before the alarm went off, record 15 minutes here.

7. What time did you get out of bed for the day? What time did you get out of bed with no further attempt at sleeping? This may be different from your final awakening time (e.g. you may have woken up at 6:35 a.m. but did not get out of bed to start your day until 7:20 a.m.)

8. In total, how long did you sleep? This should just be your best estimate, based on when you went to bed and woke up, how long it took you to fall asleep, and how long you were awake. You do not need to calculate this by adding and subtracting; just give your best estimate.

9. How would you rate the quality of your sleep? "Sleep Quality" is your sense of whether your sleep was good or poor.

10. How restful or refreshed did you feel when you woke up for the day? This refers to how you felt after you were done sleeping for the night, during the first few minutes that you were awake.

11a. How many times did you nap or doze? A nap is a time you decided to sleep during the day, whether in bed or not in bed. "Dozing" is a time you may have nodded off for a few minutes, without meaning to, such as while watching TV. Count all the times you napped or dozed at any time from when you first got out of bed in the morning until you got into bed again at night.

11b. In total, how long did you nap or doze? Estimate the total amount of time you spent napping or dozing, in hours and minutes. For instance, if you napped twice, once for 30 minutes and once for 60 minutes, and dozed for 10 minutes, you would answer "1 hour 40 minutes." If you did not nap or doze, write "N/A" (not applicable).

12a. How many drinks containing alcohol did you have? Enter the number of alcoholic drinks you had where 1 drink is defined as one 12 oz beer (can), 5 oz wine, or 1.5 oz liquor (one shot).

12b. What time was your last drink? If you had an alcoholic drink yesterday, enter the time of day in hours and minutes of your last drink. If you did not have a drink, write "N/A" (not applicable).

13a. How many caffeinated drinks (coffee, tea, soda, energy drinks) did you have? Enter the number of caffeinated drinks (coffee, tea, soda, energy drinks) you had where for coffee and tea, one drink = 6-8 oz; while for caffeinated soda one drink = 12 oz.

13b. What time was your last caffeinated drink? If you had a caffeinated drink, enter the time of day in hours and minutes of your last drink. If you did not have a caffeinated drink, write "N/A" (not applicable).

14. Did you take any over-the-counter or prescription medication(s) to help you sleep? If so, list medication(s), dose, and time taken: List the medication name, how much and when you took EACH different medication you took tonight to help you sleep. Include medication available over the counter, prescription medications, and herbals (example: "Sleepwell 50 mg 11 pm"). If every night is the same, write "same" after the first day

15. *Comments*: If you have anything that you would like to say that is relevant to your sleep feel free to write it here.

Consensus Sleep Diary-M (Please Complete Upon Awakening) **ID/NAME:** _____

	Sample					
Today's Date	4/5/11					
1. What time did you get into bed?	10:15 p.m.					
2. What time did you try to go to sleep?	11:30 p.m.					
3. How long did it take you to fall asleep?	55 min.					
4. How many times did you wake up, not counting your final awakening?	6 times					
5. In total, how long did these awakenings last?	2 hours 5 min.					
6a. What time was your final awakening?	6:35 a.m.					
6b. After your final awakening, how long did you spend in bed trying to sleep?	45 min.					
6c. Did you wake up earlier than you planned?	☑ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No
6d. If yes, how much earlier?	1 hour					
7. What time did you get out of bed for the day?	7:20 a.m.					
8. In total, how long did you sleep?	4 hours 10 min.					
9. How would you rate the quality of your sleep?	□ Very poor ☑ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good
10. How rested or refreshed did you feel when you woke-up for the day?	□ Not at all rested ☑ Slightly rested □ Somewhat rested	□ Not at all rested □ Slightly rested □ Somewhat rested	□ Not at all rested □ Slightly rested □ Somewhat rested	□ Not at all rested □ Slightly rested □ Somewhat rested	□ Not at all rested □ Slightly rested □ Somewhat rested	□ Not at all rested □ Slightly rested □ Somewhat rested

	□ Well-rested □ Very well-rested	□ Well-rested □ Very well-rested	□ Well-rested □ Very well-rested	□ Well-rested □ Very well-rested	□ Well-rested □ Very well-rested	□ Well-rested □ Very well-rested
11. Comments (if applicable)	I have a cold					

Appendix L: Visual Analogue Rating Scales

DAY X—MORNING: **TIME:**

Questions about your sleep last night

Please place a mark along the line for each question.

1. Last night, as you were attempting to fall asleep or return to sleep, did thoughts keep running through your mind?

Not at all Very much so

2. Last night, as you were attempting to fall asleep or return to sleep, did you experience a jittery, nervous feeling in your body?

Not at all Very much so

3. How much effort did you put into sleeping last night?

No effort A lot of effort

4. How would you describe your mood right now?

Very bad mood Very good mood

Appendix M: Glasgow Content of Thoughts Inventory (GCTI)

Glasgow Content of Thoughts Inventory

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Here are some thoughts that people have when they can't sleep. Please indicate by placing a tick in the appropriate box *to what extent the following thoughts kept you awake last night*.

		Not at all	A little	A fair amount	A great deal
1	Things in the future				
2	How tired/sleepy you felt				
3	Things that had happened that day				
4	How nervous/anxious you felt				
5	How mentally awake you felt				
6	Checking the time				
7	Trivial things				
8	How you couldn't stop your mind from racing				
9	How long you'd been awake				
10	Your health				
11	Ways you could get to sleep				
12	Things that you had to do tomorrow				
13	How hot/cold you felt				
14	Your work/responsibilities				
15	How frustrated/annoyed you felt				
16	How light/dark the room was				
17	Noises you heard				
18	Being awake all night				
19	Pictures in your mind				
20	The effects of not sleeping well				
21	Your personal life				
22	How thinking too much was the problem				
23	Things in your past				
24	How bad you were at sleeping				
25	Things to do to help you sleep				