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THEORETICAL REVIEW

The attention–intention–effort pathway in the development of psychophysiologic insomnia: A theoretical review

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Summary Psychophysiologic insomnia (PI) is the most common form of persistent primary insomnia. Its 'behavioral phenotype', comprising elements such as conditioned arousal, sleep-incompatible behavior and sleep preoccupation, has not changed markedly across several generations of diagnostic nosology. Moreover, a substantial outcome literature demonstrates that PI can be treated effectively using a range of psychological interventions. It seems evident that behavioral and cognitive factors play a part. What is less clear is exactly how PI develops and what are its crucial maintaining factors. This paper proposes an explanatory model, that we call the attention–intention–effort pathway. The argument is that sleep normalcy is a relatively automatic process. Consequently, it is vulnerable, and may be inhibited, by focused attention and by direct attempts to control its expression. Drawing upon parallels in the literature on adult psychopathology, and upon recent clinical and experimental studies on insomnia, the evidence for this pathway is considered and a research agenda is outlined. In particular, computerized tests of cognitive bias are seen as offering an objective means of appraising mental processes in insomnia. These may be applied concurrently with somatic measurements in future studies to better understand this common psycho-physiologic condition.

'Sleep (is like) a dove which has landed near one's hand and stays there as long as one does not pay any attention to it; if one attempts to grab it, it quickly flies away'

(Viktor E. Frankl (1965, p. 253): [Frankl VE. The Doctor and the soul. 2nd ed. New York: Knopf; 1965.] cited in Ansfield, Wegner and Bowser (1996) [Ansfield ME, Wegner DM, Bowser R. Ironic effects of sleep urgency. Behav Res Ther 1996;34:523–31.]

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Introduction

In this paper, we propose a possible pathway for the development and maintenance of persistent psychophysiologic insomnia. Our thinking is guided by predictions from recently described insomnia models, by the relevant literature, including work undertaken in other disorders, and by recent experimental data.

Psychophysiologic insomnia

Psychophysiologic insomnia (PI) is the most common insomnia sub-type, found in 1–2% of the general population, and in 12–15% of all patients seen at sleep centers. According to clinical nosologies[3–5] and research diagnostic criteria,[6] the central pillars of PI are heightened arousal and learned sleep-preventing associations, with patients exhibiting an excessive focus upon and anxiety about sleep. A number of models has been proposed, each placing somewhat differing emphases upon these features of insomnia, nevertheless, all appear to endorse the validity of the PI phenotype[7] for a comprehensive review). Many studies evaluating psychological interventions have also been generated, and there is now substantial evidence that cognitive behavioral methods, either singly or within multi-component therapy, yield sustained reductions in insomnia complaints.[8–10]

In spite of this progress, there is much that remains to be done. Not least, there is an outstanding need to investigate the mechanisms and processes underlying the development of insomnia; and to establish what are the critical components of behavioral insomnia therapies and how they achieve their effects.[11] A timely call has been made to adopt experimental psychopathology paradigms for the purposes of testing and developing theories in relation to insomnia.[12] This approach has been fruitful in other areas of mental health research, most notably in anxiety and depressive disorders.

In recent times, we have established such an experimental program at the University of Glasgow Sleep Research Laboratory. The invitation to write this review arose from a presentation made on some of this work by the first author at the 17th Congress of the European Sleep Research Society in Prague, October, 2004. In this paper, we propose one candidate process, in still an early stage of conceptual development, that seems to merit further experimental and clinical research evaluation in PI.

The attention–intention–effort pathway

This idea has its origins in the psychobiological inhibition model of insomnia (Espie, 2002),[13] which differs from most other conceptualisations in that it takes as its starting point a perspective upon normalcy rather than pathology. The model considers what it takes to upset the course of normal good sleep, and to prevent (inhibit) its recovery. Lundh and Broman (2000)[14] similarly reflected on the importance in insomnia research of having "a sufficiently adequate understanding of how the (sleep) process typically unfolds normally" (p. 303).

It is of course known that prolonged wakefulness reliably induces sleep, and that failure to obtain at least a core amount of sleep (sleep deprivation) leads to impaired function. Within the 'two process' system (process S, sleep homeostatic drive, process C, circadian variation), the sleep homeostat drives the sleep–wake schedule toward a balanced requirement in that prolonged wakefulness accrues 'sleep debt', and the circadian timer modulates sleep propensity on approximately a 24 h cycle.[15,16] We suggest, however, that there is an implicit ancillary process that is associated with the automatic regulation of sleep–wake patterns in good sleepers. The concept of automaticity[13] refers to the largely involuntary nature of the well-adjusted sleep schedule, and to the over-learned associations that may form part of a good sleep stimulus control paradigm.[17] In other words, we see the good sleeper as essentially passive because internal and external cues act as automated setting conditions for sleep, and these are further reinforced by rapid sleep-onset. Endogenous cues to sleep, such as physical and mental fatigue, are presumed to interact reciprocally with exogenous perhaps classically conditioned cues, in the bedroom environment; so that the good sleeper approaches sleep, just as s/he walks or talks—without thinking much about it and without a consciously explicit plan.[13]

Just as homeostatic and circadian mechanisms play a central role in understanding some sleep symptoms (e.g. excessive daytime sleepiness, phase disorders), this 'third process' of automaticity may be central to PI. We hypothesise that because the sleep–wake process is essentially self-regulatory, de-arousal and sleep engagement may be particularly vulnerable if for any reason the process is switched out of its natural automated

mode. We have used the term inhibition for this switching,[13] for two reasons. First, our focus is upon factors that might be preventing the expression of normal sleep and preventing its natural recovery. Second, we do not assume that PI is associated causally with any particular sleep pathology.[a] Rather we are inclined to the view that people with PI have the potential to sleep normally if inhibitory factors can be overcome. In this context, Cognitive behavior therapy (CBT) methods may be effective in PI because they serve to overcome inhibitory mechanisms and to re-establish setting conditions for normal involuntary sleep. To the extent that any CBT method enables an individual to abandon personal agency over sleep and to return to reliance upon involuntary sleep it may be likely to achieve a good therapeutic effect.[13]

More specifically, we now propose that sleep-wake automaticity can be inhibited by selectively attending to sleep, by explicitly intending to sleep, and by introducing effort into the sleep engagement process. We call this route into PI the attention-intention-effort (A-I-E) pathway.

Selective attention

Introduction

We can go back as far as William James[18] for a definition of selective attention:

> "Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalisation, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others."

Psychologists interested in information processing theory have researched human attention extensively. In short, we now know that stimuli that are salient to an individual are likely to attract attention. That is, there is an information processing bias toward salient stimuli. If you are engaged in the process of buying a new car of a certain model you will likely begin to notice other vehicles of that same type. It is not that more of these cars have appeared on the roads; but simply that you have developed an attention bias to something that has become a relevant stimulus.

Support has been found implicating attention bias in the perpetuation of a wide range of anxiety-related psychological disorders and concerns including panic disorder, hypochondriasis, eating disorders, obsessional disorders, generalized anxiety disorder, and PTSD[19,20] for reviews). In this field, attention bias toward potentially threatening stimuli has been of particular interest. Indeed, it has been argued that attention bias toward threat may have a causal role in anxiety disorders.[21] Evidence favouring attention bias in depression has been more equivocal,[22] with some studies demonstrating effects and some not. Some recent evidence, however, suggests that if depressed individuals do selectively attend to negative material it tends to be a more delayed, and possibly a more controlled, process than in anxiety disorders.[23]

The classic Beck cognitive model of emotional disorders assumes attention biases (as well as mnemonic and interpretive biases) are driven by negative beliefs stored in long-term memory. When activated these 'schema' guide information processing, including attention, toward stimuli congruent with them. Conviction in negative automatic thoughts is thereby increased, and hypervigilance is promoted. The anxious individual, therefore, remains preoccupied with danger and threat, and the depressed individual with failure and loss.[24] However, one of the limitations of schema theory, and the account of attention bias it offers, is that it views beliefs in a static rather than dynamic way by failing to acknowledge the top down influence of self-knowledge.[25] So-called 'metacognitive' models like the Self-Regulatory Executive Function model (S-REF)[26] account for cognitive self-regulation of attention, perception and memory, and, thus, may offer a more holistic, dynamic account. Psychological disorders are, according to S-REF, associated with a 'cognitive-attentional syndrome' that maintains attention focus on threat, promotes ruminative worry-based processing, and activates negative self-beliefs. Alternative processing routines are denied, adaptive self-knowledge is blocked and maladaptive self-knowledge is maintained. This S-REF approach is somewhat closer to our line of thinking about PI than 'classic' cognitive theory because S-REF helps to explain how intrusive, worrisome thought (and attention bias) may persist in insomnia.

It is very important to observe, however, that attention biases do not operate only in the context of threat. For example, expertise and personality have also been shown to mediate selective attention.[27,28] Likewise, in the illustration

[a] This is not to say that there is no neurobiological substrate to insomnia, but rather to suggest that such 'arousal' may be part of the expression of the A-I-E pathway itself.

introduced earlier, noticing more cars of a certain type on the roads would be unlikely to be motivated by threat. Rather, cognitive bias would probably be influenced by a more positive interest in making an eventual purchase! It is instructive, therefore, to look beyond the psychological disorders to consider how attention bias has been implicated in the perpetuation of habit/dependence disorders, including alcohol, heroin and nicotine. The stimuli that have been used in experiments on these disorders are clearly salient (related to the dependence) but if anything they tend to be reinforcers rather than threats. To take the example of alcohol, selective attention bias to behaviorally relevant word or picture stimuli has been found in alcoholics and problem drinkers, but not in social drinkers.[29,30] It seems that problem drinkers are more likely to notice alcohol-related stimuli in the environment, that this attention bias 'reminds' them of drinking, and that it may even mediate the maintenance of their addiction by producing 'craving'.[30] We will come back to ways in which both threat and incentive may be relevant to PI in conceptualization of selective attention in relation to PI.

Generally, an attention bias has been found in those who have a clinical diagnosis, contributing to the explanation of why anxiety disorders[31] and abuses/dependences[32] are frequently self-maintaining, and why relapse often occurs after an emergent treatment response. In explanations such as these, attention bias is conceived as an initial involuntary (unconscious, implicit) process that gives rise to voluntary (conscious, explicit) processes.[33] In other words, pre-attentive processing guides the early, automatic capture of relevant information even when conscious access to the information is not available. A good illustration of this was a study that successfully elicited both psychophysiological reactivity and subjective fear in phobic participants to pictures of their feared objects that were presented beyond their conscious awareness.[34]

The human attention system is clearly complex and intriguing. So what might happen to sleep if it became subject to such selective monitoring and scrutiny?

Conceptualization of selective attention in relation to PI

Attention biases reflect discrete changes in the direction of attention focus, in response to stimuli that are, in some sense, salient. Sleep is certainly salient to people with PI. Contemporary ICSD-2[4] diagnostic criteria for PI include:-

> "Excessive focus on and heightened anxiety about sleep" (Criterion C1)

The accompanying ICSD-2 text describes just how marked the preoccupation with sleep can become in PI:

> "Concerns about sleep grow progressively over months or years as sleep gradually deteriorates until the desire to obtain a good night's sleep becomes the person's major concern" (p. 1–6)

Interestingly, this statement conveys both a sense of incrementing distress associated with sleeplessness (cf. threat), and a preoccupying longing for sleep (cf. craving) that might serve as preconditions for attention bias. We have summarised in Table 1 what may be some of the key features of this incentive-threat comparison.

In 1943, Maslow published his influential paper on human motivation suggesting that a 'hierarchy of needs' act as motivators for human behavior.[35] According to Maslow's theory, basic needs are physiological; for example, hunger, thirst, sleep, etc. When these are satisfied they are replaced by safety needs reflecting the desire for protection against danger or deprivation. In this context, we can think of sleep as a primary reinforcer, reflecting basic physiological processes necessary for physical, intellectual and emotional well-being (Table 1). Thus, the A–I–E pathway in relation to sleep may parallel Maslow's commentary on hunger.

Table 1 Comparison of potential 'drivers' for sleep-related attention bias in Psychophysiologic Insomnia

Incentive	Threat
Sleep is a primary reinforcer	Inability to solve sleeplessness is threatening
Sleep is at top of the 'hierarchy of needs'	Safety needs come after primary physiological essentials
Sleep 'deprivation' produces craving	Sleep 'deprivation' produces worry
Hunger, thirst, oxygen as a model	Fear, anxiety as a model
Goal directed behavior is to obtain sleep	Goal directed behavior is to avoid being awake

> "For the man who is extremely and dangerously hungry, no other interest exists but food. He dreams food, he remembers food, he thinks about food, he emotes about food, he perceives only food and he wants only food (…) For our chronically and extremely hungry man, Utopia can be defined simply as a place where there is plenty of food. He tends to think that, if only he is guaranteed food for the rest of his life, he will be perfectly happy and will never want anything more. Life itself tends to be defined in terms of eating." (p. 374)

Similarly, we suggest that the person with PI experiences sleep disruption, sleep loss and perceived sleep inadequacy and so becomes atypically motivated by sleep, which is increasingly incentivised in proportion to the preoccupation associated with it. Just as food is more of a reinforcer when we are hungry, in PI we might expect that a much higher than normal value would be placed upon sleep. Of course, sleep would be a primary reinforcer for any individual, but presumably its reinforcement value might increase in relation to sleep requirement/deficit or perceived sleep requirement/deficit. The desire for sleep of good quality, therefore, may in this sense become a 'craving'.

However, consistent with the second level of Maslow's hierarchy, the perceived inability to sleep may also be conceptualised and experienced as a significant threat. Sleeplessness may be threatening. Bedroom arousal may develop in PI as a result of the conditioning of non-verbal (environmental) and verbal signals (e.g. thoughts about sleeplessness) as threat cues which impact on selective attention. But there is also another sense in which being unable to sleep might be experienced as a threat. Taking our principle of automaticity into account, people who sleep well do not usually know how they do so. Ask any normal sleeper what they do to sleep and they will probably appear rather bewildered. Sleep is not in this sense an enacted operant (cf.[17]), but rather it is passive and effortless. On the assumption that the person with PI started out as a normal sleeper, one can understand that to have apparently lost the capacity to sleep, not really knowing how you managed to sleep successfully before, might be rather threatening.

Harvey's model of insomnia[36] represents an adaptation of the cognitive perspective on psychological disorders, and finds common ground with the selective attention component of the A–I–E pathway. Harvey suggests that insomnia is maintained by a cascade of cognitive processes that includes selective attention and monitoring of the internal environment (e.g. alertness, bodily sensations) and external environment (e.g. clock-watching, environmental noise) that interact with negative beliefs, worry, misperception of sleep and the negative daytime sequelae of insomnia and the engagement of counterproductive 'safety behaviors'. Anxious people, for example, have been found to exhibit characteristic 'safety behaviors'.[37] These are overt and covert strategies that people develop in order to avoid feared outcomes. However, they generally prevent disconfirmation of catastrophic beliefs, and so in fact make feared outcomes more likely. For example, a social phobic fearful of spilling a drink in public, might grip the glass more tightly, thus (a) preventing unambiguous disconfirmation of the belief that spilling the drink is likely, and (b) increasing the likelihood of actually spilling. In insomnia, classic safety behaviors might include things like going to bed early or clock-watching. According to the Harvey model, increased monitoring for, or attention to sleep-related threat cues increases the chance of detecting such cues and thus establishes a mutually maintaining vicious cycle.[36]

So, it seems conceivable that selective attention to sleep-related cues might arise because of salience (unspecified), because of threat monitoring, because sleep is a reinforcer, or, of course, because of a combination of the latter reasons. Attention bias may be a signature of classically conditioned arousal in PI. In this respect, it would seem timely to re-consider the stimulus control model of insomnia in terms of classical/associative learning as well as its conceptualisation in terms of operant/instrumental learning.[17] It may also be worth considering which sleep parameters would be associated with different components of the A–I–E process. We suggest that increased sleep–onset latency (SOL) and wake time after sleep–onset (WASO), as the symptomatic representation of insomnia, could be indicative of conditioned arousal responses to intrinsic and extrinsic threat cues; whereas reduced sleep efficiency (SE) may reflect sleep craving through increased time in bed (TIB) (sleep opportunity) in the effort to increase total sleep time (TST). The contrast between good sleepers (GS) and those with insomnia is even clearer in the sub-group of GS who by choice mildly restrict their bedtime to 6–7 h/night on a fairly chronic basis. This applies to those busy people who accept the mild cost of some increased daytime tiredness for the choice of spending more time at work, with family, being entertained, etc. The attitude of these people towards sleep is not craving as the PI, nor even neutral as with the GS, but it may even be

somewhat negative and off-handed. For them, sleep may be seen as deserving relatively little attention.

Further relating attention bias to the concept of automaticity, we suggest that the good sleeper is like the experienced car driver who easily executes a complex series of operations with minimal attention load to the process. By comparison the person with PI is like the anxious learner driver—vigilant, deliberate and errorful.[13] The concept of automaticity in human learning has long been discussed as part of information-processing theory.[33] Some authors have suggested that the development of explicit, conscious processing is a relatively recent development in evolutionary terms.[38] Essentially, prior to humans gaining conscious thought, all learning would have flowed through an implicit acquisition process without the need for conscious, verbal reflection. Explicit, verbal learning may provide a means to 'short-cut' the development of some skills, such as driving; with the process of automatisation occurring as the skill is consolidated.

Paradoxically, it has been demonstrated, that proficient motor skills, may degrade if individuals are asked to verbalise their actions and turn their attention inwards towards the mechanics of their actions.[39,40] Perhaps, the most eloquent demonstration of this is paying attention to, and attempting to take conscious control of, the actions of your feet as you run downstairs. This is not to be recommended at the top of a flight of stairs. Thus, the development of sleep processes may be seen in much the same light. Setting conditions for sleep, responding to sleep cues, developing a sleep pattern are all part of infant training and should lead the development of sleep process that does not enter the realm of consciousness. However, if one encounters difficulties with sleep and attempts to 'take control' of the situation by directed attention, the disruption to the automaticity of the sleep process may parallel the difficulties encountered in consciously controlling one's legs whilst descending stairs. Respiration is another example of a similar phenomenon, although in this case there is no learned component. Respiration like sleep engagement, is normally an unconscious, passive, simple process. Yet, during a panic attack, thinking that not enough oxygen is being obtained induces hyperventilation that exacerbates the sensory dysphoria, light headedness, feelings of insufficient oxygen, which induces greater panic and a vicious cycle, at times leading to final unconsciousness.

Evidence of attention bias in PI

We have recently suggested that individuals with PI are characterised by high levels of metacognitive beliefs and plans for processing, which predispose them to appraise thoughts, experiences, and bodily states negatively.[41] This 'cognitive architecture', we hypothesise, promotes worry, rumination and attention bias in the pre-sleep period. We would predict, therefore, that metacognitive beliefs that promote negative appraisal of nocturnal intrusions (e.g. 'thinking at night keeps me awake') characterise the person with insomnia, as do associated metacognitive plans for processing, including those, which promote attention bias (e.g. to sleep, I must focus on how sleepy I feel).

Evidence of information processing bias in insomnia can be drawn from several sources, using differing methodologies. The most direct evidence comes from experimental studies specifically measuring or manipulating aspects of selective attention. We will review these studies after consideration of the less robust, but nevertheless interesting, descriptive literature comprising qualitative data, questionnaires and rating scales.

Phenomenological/descriptive studies

Many measures used in insomnia research contain items that reflect the person with insomnia's tendency to pre-occupying worry about sleep. These include the pre-sleep arousal scale (e.g. item 1 'worry about falling asleep'),[42] the dysfunctional beliefs and attitudes about sleep scale (DBAS) (e.g. item 4 'I am worried that if I go for 1 or 2 nights without sleep I may have a nervous breakdown'),[43] the sleep disturbance questionnaire (e.g. item 12 'I worry that I won't cope tomorrow if I don't sleep well')[44] the self-statement test: 60+ (e.g. item X 'if I don't get to sleep soon, I will feel very tired tomorrow')[45] and the anxiety and preoccupation about sleep questionnaire (e.g. item 1 'I worry about the amount of sleep I am going to get each night').[46] Interestingly, Watts et al. conducted a study comparing 'worried' and 'non-worried' insomniacs on pre-sleep mental activity.[47] The former group appeared preoccupied by work-related issues and general mental activity. In contrast, 'non-worried insomniacs' tended to focus on problems they were having with the sleep process itself. This direct focus on sleep is, of course, of particular relevance in relation to attention bias.

In a prospective study of pre-sleep mentation, Wicklow and Espie obtained voice-activated audiotape recordings of spontaneous thoughts, and sleep

actigraphic data from 21 poor sleepers over three consecutive nights.[48] Content analysis of over 1000 thought segments yielded eight categories of pre-sleep intrusion, and a regression model indicated that focusing on sleep and the anticipated consequences of poor sleep, along with general problem-solving were the strongest predictors of objective SOL. Thought content was subsumed under one of the three factors; 'active problem-solving' (e.g. rehearsing/planning events), 'present state monitoring' (e.g. thinking about-sleep/not sleeping, autonomic experiences, your own thinking) and 'environmental reactivity' (e.g. attending to external noises). Thirty-eight percent of thought segments represented present state monitoring.

The qualitative component of this study was partially replicated in a further investigation which also had a psychometric phase, leading to the development of the Glasgow content of thoughts inventory (GCTI).[49] The GCTI was found to have good internal consistency ($\alpha = 0.87$) and test–retest reliability (ICC = 0.88) and a score of 42 discriminated PI from GS groups with sensitivity of 100% and specificity of 83%. A principal components analysis of the GCTI found that present state monitoring emerged as an important factor accounting for 38% of explained variance.

Harvey conducted some parallel research.[50] Using a semi-structured interview, five areas of attention focus were investigated. It was found that people with insomnia relative to GS were more likely to attend to sensations of falling asleep and to worries/concerns, trying to solve problems and listening to noises. Good sleepers on the other hand were more likely to attend to 'nothing in particular' during their (relatively shorter) wake times. Neitzert-Semler and Harvey then reported two related studies. In the first of these, students meeting criteria for primary insomnia were compared with a GS control group using a semi-structured interview of sleep-related threat, negative thoughts, and safety behaviors.[51] People with insomnia reported more frequent monitoring, night and day, and they engaged in more safety behaviors. A path analysis suggested that monitoring may act as a driver for negative thinking in insomnia. In the second study reported in this paper some evidence emerged for the generalizability of these findings to a clinical insomnia sample.

This work was extended through the development of the sleep associated monitoring index (SAMI).[52] This 30-item scale of sleep-related threat monitoring shows good reliability ($\alpha = 0.87$) and positive correlation with the Pittsburgh sleep quality index (PSQI[132]). Importantly, moderate correlation ($r = 0.36$) with the Penn state worry questionnaire suggests that the SAMI score is not simply an index of generic aspects of worry. An eight component solution was obtained following principal components analysis on a large sample ($n = 400$) of university students and staff. These components included monitoring for body sensations (daytime, pre-sleep, and on waking, each loaded as separate components), clock time and the environment. This study also explored the relationship between the SAMI (monitoring) and two other constructs used in attentional theory. Both 'amplification' (the tendency to experience somatic sensation intensely) and 'self-focus' (awareness of internally generated information) correlated with the majority of the SAMI subscales, excepting calculation of time and pre-sleep clock monitoring.

Summarizing this work, it seems that questionnaire data are broadly supportive of the notion of attention bias in PI. However, two notes of caution seem appropriate.

First, mental arousal associated with sleep may not be as crucial to the conceptualisation of PI as some of the psychological theories suggest. In a recent comparison of PI, delayed sleep phase syndrome (DSPS) and GS, we found that several self-report measures of the construct of cognitive arousal were elevated in both PI and DSPS, relative to GS.[53] This raises the possibility that such arousal may be epiphenomenal to wakefulness in PI (rather than causal). DSPS is presumed to reflect an endogenous phase delay so there is no need to infer any psychological process. On the other hand, the mechanisms that trigger DSPS are often precipitated by life or social events and the effects of DSPS may lead to increased pre-sleep arousal when individuals with DSPS try to reset their clocks by attempting to sleep 'out of phase'. This could theoretically precipitate symptoms of, or the onset of PI. Conversely, when individuals with PI are unable to fall asleep, they may inadvertently entrain their sleep to a later time, resulting in an element of DSPS.[54] Of course, worry and rumination are transdiagnostic phenomena that present across a range of psychiatric disorders.[55] Also in many medical disorders, worry is an associated feature. What the research literature needs to determine is which features of cognitive arousal are directly involved in the genesis and maintenance of persistent insomnia.

Second, the studies reviewed so far were not designed specifically to test attention bias within the context of a controlled experiment. However, a number of such studies has emerged recently,

largely from Dr Allison Harvey's research group in Oxford[b] and from our own laboratory in Glasgow.

Real world experiments

Harvey's innovative work stems from her cognitive model of insomnia in which the importance of monitoring of sleep-related threat was posited (outlined above). This research team has conducted a series of controlled experiments, involving the manipulation of attention in order to test its causal role in increasing or decreasing insomnia symptoms.

Neitzert-Semler and Harvey[56] attempted to test the hypothesis that monitoring for sleep-related threat during the day would trigger a cycle of subsequent negative thinking, perceived impairment, and subjective sleepiness. Young people with insomnia were randomly assigned either to a condition involving monitoring of body sensation (to be closely aware of the feelings and sensations and to focus attention on internal reactions), to a condition involving distraction from such monitoring (focusing upon external environment and activities) or to a no instruction control group. Results largely confirmed the prediction that the monitoring group would report higher ratings for negative thoughts, safety behaviors and daytime sleepiness than the control condition. Daytime functioning, however, was not different between groups and, unfortunately, the no monitoring manipulation was less well designed and so was relatively unsuccessful. Nevertheless, this does not detract from the findings for the monitoring group.

Neitzert-Semler and Harvey[57] assigned 51 participants meeting DSM-IV criteria for insomnia to a self-focus group (viewing themselves on a TV monitor), to a monitoring group (similar to above but also focusing on thoughts and mood) or to a no instruction group. Participants were then exposed to a 60-min neuropsychological test battery. The purpose of the study was to index the effect of attentional focus on real versus perceived performance. As hypothesized, no differences were observed in the former comparison. However, the self-focus group perceived their performance as significantly worse on the majority of tasks than the no instruction group, providing confirmation of the potential role of self-focusing as a contributory factor to the perceived daytime impairments of people with primary insomnia. By contrast, the monitoring condition did not differ from the no instruction group on any subjective performance rating. The authors suggest that the self-monitoring condition, unlike the video-TV condition, in this experiment may have resulted in insufficient self-focused attention.

Tang et al.[58] considered the importance of clock monitoring in insomnia. In a first experiment, good and poor sleepers were instructed to monitor (or not) a clock as they were trying to get to sleep. Clock monitors, whether poor or good sleepers, reported a higher worry rating and had longer SOL as indexed by sleep diary and actigraphic data. A second experiment was conducted with a clinical sample, where monitoring per se was controlled by using a digit display monitoring task to isolate the specific effects of clock watching. The degree of worry and SOL overestimation demonstrated by the clock monitoring group was greater than the control condition, thus, lending further support to the idea that attentional bias, in the form of clock watching, is not conducive to sleep.

To summarise these real world experiments, it seems that insomnia is associated, at least in terms of self-report, with sleep-related self-monitoring tendencies.

One of the challenges for research in the area of attention is to separate out sleep-specific effects from heightened generic responding. For example, research on the 'orienting response', indexed physiologically by skin conductance levels, suggests that people with insomnia exhibit a general tendency to increased attentiveness. That is they are more responsive, particularly to emotional stimuli and stress, and take longer to habituate than do good sleepers.[59–61] Although, so far, psychophysiological measures have not been taken in parallel, studies employing reaction time as a dependent variable have now gone some way towards establishing responses to sleep stimuli over and above heightened orienting per se.

Computerized experimental studies

Several studies have explored selective attention bias in insomnia using measures of information processing speed (Table 2). These studies used computerized experimental protocols where both salient and neutral stimuli were presented to investigate any systematic processing differences between good sleepers and people with sleep disorder. The paradigm in such attentional tasks is that stimulus salience interferes with response time because of the 'grabbiness' (uncontrolled prolonged attention capture or focusing) of sleep-related word or picture stimuli relative to neutral stimuli. An advantage of this experimental approach is that it does not rely on self-report, but rather posits objective reaction time differentials as a proxy for cognitive arousal. Before

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Table 2 Experimental studies investigating attention bias in insomnia using information processing paradigms

Authors	Participant characteristics	Paradigm	Findings
Lundh et al.[68]	40 adults between ages of 20 and 65 and gender matched across two experimental groups (primary insomnia and good sleepers)	Stroop task	Repeated measures ANOVA examined the effects for each of the 3 stimulus types [i.e. sleep, physical threat and physical control words; and colour names versus a control sequence of letters (XXX…)]. Both the insomniacs and the controls responded more slowly to the sleep words, physical threat words and colour names, than to their matched control stimuli. There was no main effect of group with regard to any stimulus type and no significant interaction between group and sleep words [$F(1, 37)=0.21$)].
Taylor et al.[69]	33 Adults (23 F/10 M; mean age 47 years) with sleep-onset insomnia subsequent to cancer diagnosis. Mean time since diagnosis for the acute insomnia group was 2.0 months and was 14.3 months for the persistent insomnia group	Stroop task	Independent samples *t*-tests indicated no significant difference between the two groups for the cancer interference index ($t=0.91$, $p=0.37$) but there was a significant difference on the sleep interference index ($t=-2.44$, $p=0.02$). Both groups demonstrated interference for cancer words relative to neutral words, but only the persistent insomnia group exhibited interference for sleep words. Groups did not differ significantly on pre-sleep cognitive or somatic arousal (PSAS) and they used similar thought control strategies (TCQ: distraction, re-appraisal, social control)
MacMahon et al.[70]	63 adults (35 F/ 28 M; mean age 25 years). Across three experimental groups (primary insomnia (PI), delayed sleep phase syndrome (DSPS), good sleeper (GS)).	Dot probe	Orthogonal contrasts of PI versus DSPS and GS indicated a significant difference ($t=-1.88$, $p=0.03$), suggesting that participants with PI showed a greater attention bias to sleep related words than those with DSPS or GSs. A further contrast between DSPS and GS did not indicate a significant difference between these groups ($t=-1.27$, $p=0.10$), thereby supporting the hypothesis that attention bias plays a fundamental role in the disorder of PI. The possibility of an underlying trend in DSPS responses needs to be further investigated
Jones et al.[72]	192 Adults (50% F; mean age 32.1 years) age and gender matched adults across three experimental groups (poor sleepers, moderate sleepers good sleepers)	Flicker ICB	Independent samples *t*-tests revealed that Poor sleepers and Moderate sleepers detected the sleep-related change significantly quicker than Good Sleepers ($t=$ 3.33 and 2.90, both $p<0.01$). There was no difference in this change detection latency between Poor and Moderate sleepers. Poor sleepers detected the sleep-related change quicker than the neutral change [$F(1180)=7.11$, $p<0.01$] displaying a sleep-related attention bias. For Moderate sleepers this difference was not significant, and Good sleepers detected the change within the neutral objects significantly quicker than within the sleep-related objects [$F(1180)=6.21$, $p<.05$] showing a bias towards neutral rather than sleep-related objects

(*continued on next page*)

Table 2 *(continued)*

Authors	Participant characteristics	Paradigm	Findings
Macphee et al.[73]	90 Adults (50% F; mean age 22.8 years) across three experimental groups [(primary insomnia (PI), delayed sleep phase syndrome (DSPS), good sleeper (GS)]	Flicker ICB	Independent samples *t*-tests revealed that, at the level of PI, sleep-related change was detected significantly quicker than a sleep neutral change, ($t=13.10$, $p<0.0001$). No such differences were observed at the level of GS ($p=0.95$), or DSPS ($p=0.14$). At the level of sleep-related change, responses of PI were significantly quicker than GS and DSPS ($t=7.50$ and 4.80, both $p<0.0001$) and responses of DSPS were quicker than those of GS, ($t=2.66$, $p<0.01$). At the level of neutral change, responses of GS and DSPS were significantly quicker than PI ($t=5.70$ and 6.80, both $p<0.0001$). No difference was observed between GS and DSPS

reviewing the literature on the application of these experiments in insomnia research it may be helpful to describe the tasks themselves.

Attention bias tasks. Three different methods have been applied to the study of insomnia.

First, the emotional Stroop task, which has been described as the hallmark measure of attention,[62] has been used to assess selective attention bias in a wide range of conditions. The Stroop task involves target (salient) and control (neutral) words being presented at random in different ink colours. Subjects are asked to respond quickly to the presented colour by pressing the corresponding coloured button on a response box. They are instructed to ignore the actual meaning of the words. Response latencies for colour identification are automatically recorded for each stimulus. Longer response latency is thought to suggest increased attention bias because automatic processing of word meaning for the salient words is likely to interfere with (slow down) colour naming relative to response time for the neutral words: the so-called interference effect.

There has been debate in the literature over whether the Stroop task actually measures increased vigilance or simply reflects the impact of heightened arousal interfering with information processing when salient stimuli are presented.[63] Thus, Stroop data alone may be insufficient to conclude that attention bias is present in PI. A second test of cognitive bias toward semantic material, the dot-probe task, has been posited as one solution to this problem.[63] In this task, words are simultaneously presented (typically for 500 ms) to two areas on a computer screen. The ensuing distribution of visual attention is measured by recording detection latency for a visual probe that could appear in the spatial location of either word, immediately after the display of that word has terminated. Thus, the task bypasses limitations of the Stroop, by using a neutral response (a keypress) to a neutral stimulus (a 'dot'). The trials providing the data of interest are those in which one of the words is salient. By examining the impact of sets of such words on the relative probe detection latencies in the two spatial areas, it is possible to determine whether visual attention has shifted toward or away from such stimuli.

There are, however, some limitations to research on attention bias that uses word stimuli. As Yiend and Mathews[64] and others have pointed out, although words can be unequivocally negative in valence it is far from clear that they constitute a severe or highly salient threat. For this reason attention bias studies investigating state and trait

anxiety sometimes use picture stimuli (e.g. weapons, corpses, dangerous animals) that are known to evoke both subjective and physiological reactions.[65] Pictures of this kind are fairly generic threatening stimuli, but ones that nonetheless elicit greater attentional responses in anxious individuals. In the context of insomnia, it is somewhat difficult to represent sleeplessness through objects in this graphic way. Besides, we are not committed at this point to an explanation for attention bias in insomnia that is motivated solely through threat monitoring.

Nevertheless, it is possible to explore attention bias with digitised objects using a flicker paradigm featuring a perceptual phenomenon called induced change blindness (ICB).[66,67] Research using this third method reveals that when a change is made to a visual scene (and the process of change is hidden from view), it is more difficult to detect than might be expected. Normally in this paradigm, a single feature of a visual scene is changed between successively repeated brief presentations until the change is detected—essentially the ICB is a spot the difference task. Change-detection latency, measured by the number of flickers it takes for the change to be identified, is explained by a change's 'grabbiness' and this depends not just on the object's physical feature that carry the change but also on the viewer's history in relation to that object. So, for example, in the alcohol field, problem drinkers take fewer flickers to detect an alcohol-related change within the visual array than a neutral change and are faster to detect such changes than control subjects.

Experiments using computerized tasks

The first study to be published was that of Lundh, Froding, Gyllenhammar et al.[68] This was a pioneering piece of work because it translated the emotional Stroop task into the insomnia field. Lundh et al. found that people with insomnia had prolonged response latency for sleep-related words (Table 2). However, this effect was also evident in a control population of good sleepers, and there was no group difference on the Stroop interference index; a result inconsistent with the attention bias hypothesis. Lundh et al. suggested that sleep-related words might have emotional valence for people that may or may not be directly related to sleep problems. However, the extensive literature on the Stroop task would not predict experimental effects in normal control groups. Of course, recruited controls who are good sleepers may have a particular interest in sleep, and this might yield a bias. Also, in this study no measure of affective state (which is known to influence Stroop findings) was taken, and diagnostic criteria were not reported for the insomnia group.

Lundh et al.'s pioneering work in Sweden, therefore, yielded somewhat equivocal findings. However, since then, our research group in Glasgow has completed four experiments using each of the three attention bias paradigms described above (Stroop, dot probe, Induced Change Blindness) that generally indicate the presence of selective attention bias in insomnia.

In our first study, we also used the Stroop paradigm, selecting a cancer population because our primary purpose was to investigate the development of insomnia associated with a stressor in people, who had previously been good sleepers.[69] None of the participants had insomnia prior to their cancer diagnosis; that is they were a 'true' secondary insomnia population rather than people whose (pre-existing) insomnia had been exacerbated. Insomnia is common in cancer populations, so this seemed to be a valid population group to study. Two groups of people with cancer and insomnia, 0–3 months and 12–18 months after cancer diagnosis, completed the computerized emotional Stroop task comprising cancer-related, sleep-related and neutral word cues. Both groups demonstrated attention bias for cancer-related words but only the persistent insomnia group demonstrated attention bias for sleep-related words (Table 2). The fact that interference effects for sleep words were absent at 0–3 months but were evident at 12–18 months, suggests that selective attention bias towards sleep may play a role in the transition from adjustment insomnia to psychophysiological insomnia.

According to ICSD-2[4] the essential feature of Adjustment Insomnia is

> "…the presence of insomnia in association with an identifiable stressor. The sleep disturbance of Adjustment Insomnia has a relatively short duration, typically a few days to a few weeks" (p. 1–3)

In Fig. 1 we have illustrated how persistent psychophysiologic insomnia may evolve from Adjustment Insomnia following the experience of a series of stressors (such as illness). It is assumed that experiencing stress is associated with both psychological (mental, behavioral, emotional, etc.) and physiological (autonomic, cortical, metabolic, etc.) responses. These are likely to inhibit normal sleep-related de-arousal, and so produce transient sleep disturbance. If cognitive and physiological arousal becomes sustained, insomnia may persist as a symptom. If not, then the insomnia symptoms would dissipate and normal sleep would return. Our

assumption, consistent with the Taylor et al. data,[69] is that attention bias (whether implicit or explicit) during Adjustment Insomnia is selective towards the perceived source of the stress. Indeed, because of that selectivity, the insomnia symptoms per se are unlikely to grab attention whilst the stressor is active.

However, continuing the description of Adjustment Insomnia from ICSD-2:

> "…the sleep disturbance resolves, or is expected to resolve, when the specific stressor resolves, or when the individual adapts to the stressor" (p. 1–3)

Consistent with this statement, we suggest that, close to the point of normal resolution of the adjustment insomnia, selective attention upon the stressor might reduce markedly. However, in circumstances where insomnia symptoms still persist, there could be an increased risk that an attention bias towards sleep-related cues might develop. That is, attention might shift from the resolving stressor to any persisting sleep disturbance at this point. Furthermore, the transition to sleep-related implicit attention bias could be prepared by the frequent prior conditioning of sleep cues with sleeplessness during preceding weeks. Thus we may have the start of the A–I–E pathway as a self-perpetuating persistent PI, even when the original stressors have resolved or diminished.

The Taylor et al. study, however, was of a cross-sectional rather than a longitudinal design and we did not have a control group of good sleepers without medical problems. Moreover, the paradigm employed presented word stimuli for the standard supraliminal 500 ms duration. Thus it is not possible to determine to what extent the bias was pre-attentive/automatic, i.e. occurred involuntarily without intention or conscious control. The results, therefore, need to be interpreted with some caution.

We have recently completed another attention bias experiment study using the dot-probe task.[70] Sixty-three young adults across three experimental groups (PI, DSPS, GS) participated (Table 2). PI and DSPS participants met ICSD criteria for their respective disorders following extensive assessment comprising clinical interviews, the use of self-report scales, and sleep diary and actigraphy monitoring. The DSPS group was employed as a further, clinical, control sample of people who, like our PI participants, had sleep-onset problems, but whom we would not expect to exhibit cognitive arousal as an explanatory mechanism for their continued wakefulness. Rather, circadian factors are presumed to explain the emergence and maintenance of DSPS. Consequently, those with

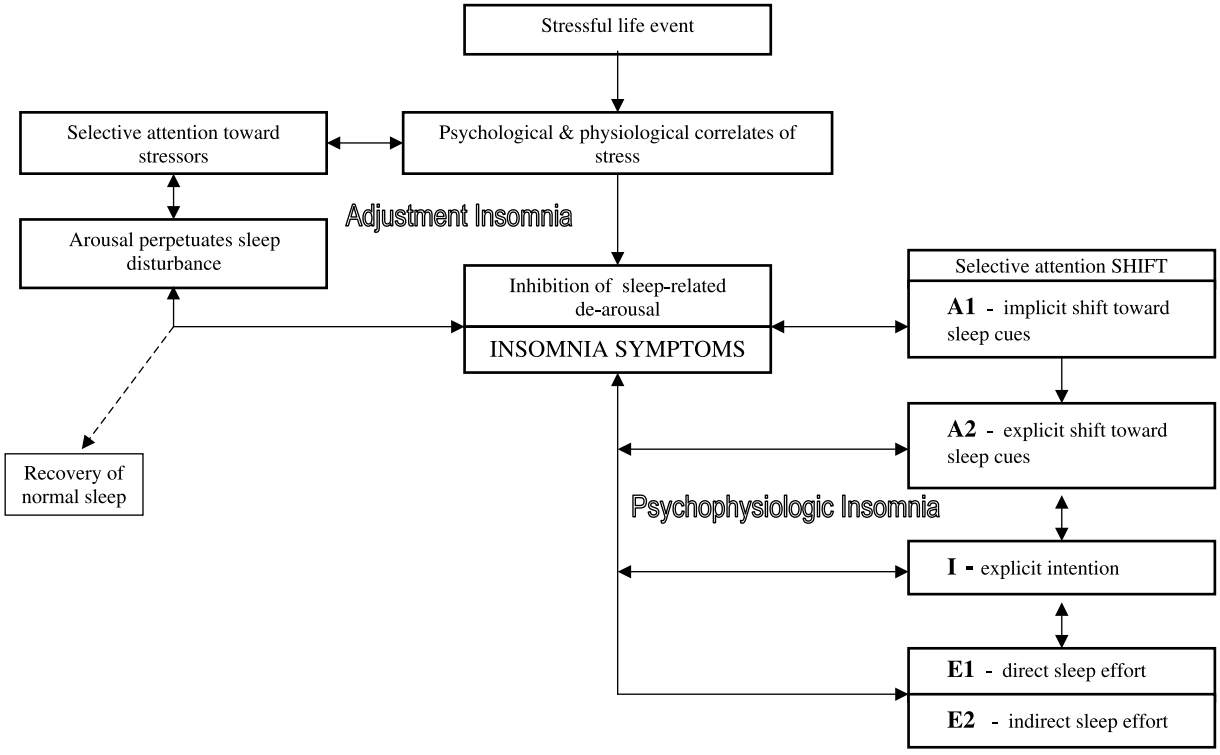

Figure 1 Proposed evolution of psychophysiologic insomnia from adjustment insomnia following the A–I–E pathway.

DSPS would not be predicted to show a cognitive processing bias to sleep-related stimuli. Results supported our predictions, with those in the PI group showing a significantly greater processing bias toward sleep-related words (in comparison to neutral words) when compared to the GS and DSPS groups (Table 2). Notably, the GS and DSPS groups did not differ from each other, suggesting that the underpinning mechanism maintaining DSPS is not an attention bias to sleep-related stimuli.

Finally, we have conducted two experiments using the ICB task. Interest in the control that sleep-related objects might have over sleep behavior is long-established. For example, within a conditioning framework, bedroom environment objects might become discriminative stimuli for sleep,[17] but when the bedroom-sleep contingencies are broken, they might become discriminative stimulus for wakefulness. In that regard it is interesting that less than one-quarter of the sleep-related words in our Stroop and dot probe studies were objects. Consequently, we felt that the ICB paradigm may be better suited to investigating the possible influence of the bedroom environment on sleep. We were also interested in the fact that, using this technique, a differential attention bias between two 'levels' of social use of alcohol and cannabis has been found.[71] In the insomnia context, we wanted to extend this approach to explore differential attention bias along the sleep problems continuum. If attention is implicated in the development of persistent insomnia we might expect to find a systematically changing attention bias, not just at the clinical pole.

In our first ICB study, 192 participants (mean age 32 years) were selected for this totally between subjects experiment[72] Table 2). Participants completed the 15-min ICB task, after which they were assessed for sleep quality and other characteristics. Importantly, therefore, retrospective group assignment was blind to the dependent variable of the analyses, change detection latency. A different flicker pair of stimuli was used for each of the two levels of the factor, nature of change (sleep-related and neutral). Each pair contained the same original stimulus comprising seven sleep-related objects and an equal number of neutral objects arranged in two collections on either side of the scene midline. The second stimulus of each pair was identical to the original stimulus but for one small change: a sleep-related change (removing one of the pair of slippers) or a neutral change (removing one of the pair of gloves). The two changed stimuli are shown in Fig. 2 along with their common originating stimulus. The two stimuli of a pair were then presented in continuous succession (each replacing the other) until the change was detected. A brief 'mask' was inserted in between the flicker pairs to suppress visual transients. We selected the sleep stimuli using a comprehensive process designed to identify objects associated with 'going to bed to sleep'. Clearly none of the objects is intrinsically threatening, and slippers emerged with the highest mean sleep-relatedness score.

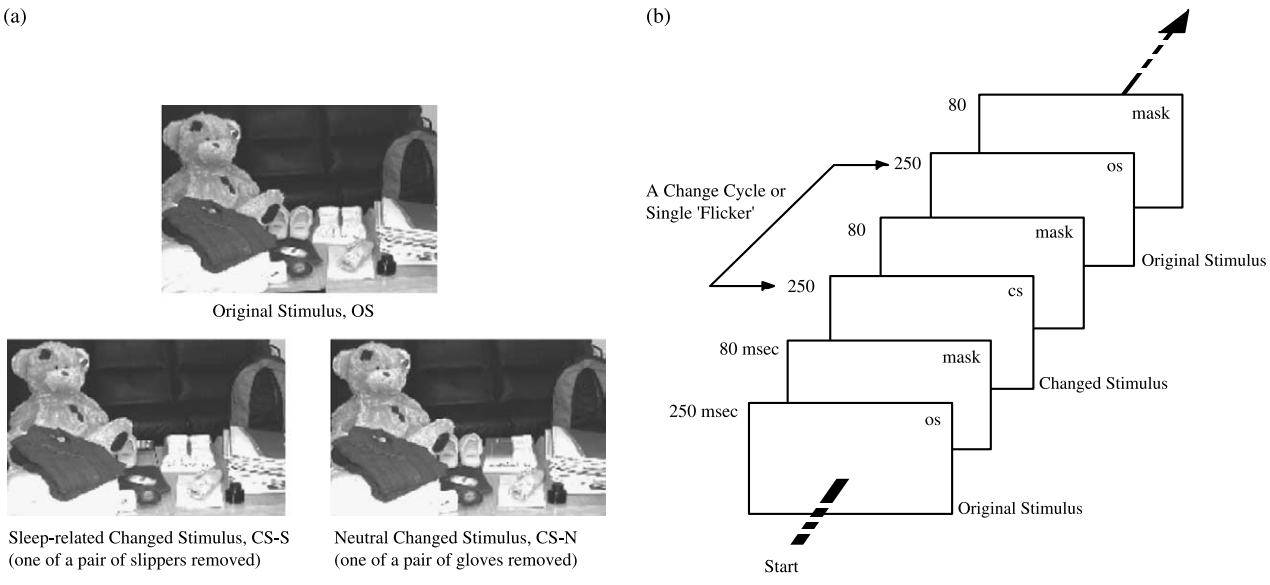

Figure 2 (a) Greyscale versions of the full color stimuli used in ICB experiment [72] Original stimulus (OS) and the two changed stimuli for each of the two levels of the factor, nature of change-sleep-related change (CS-S) and neutral change (CS-N) and (b) a Flicker paradigm for inducing change blindness illustrating a change cycle or single 'Flicker'.

Results revealed significant differences in change detection latency between poor, moderate and GS for the sleep-related change. Only the poor sleepers, who detected sleep-related change quicker than neutral change, demonstrated selective attention bias for sleep salient stimuli. Moderate sleepers showed a trend in the same direction. By contrast, GS detected the change with the neutral objects significantly quicker. Hierarchical regression was then applied to test the relationship between change detection latency and a continuous representation of the global PSQI score. This evidenced a systematically changing effect of sleep quality upon attention bias, independent of age, gender and depressive symptom level.

In simple terms, when competing for attentional resources with matched neutral stimuli, poor sleepers appeared to prioritise sleep-related stimuli. The findings for GS (of relative prioritisation of neutral stimuli) may be explained by differences in the physical saliencies of all the stimuli in the scene. That is, the neutral half of the scene may have been more salient in general, or may have included one highly salient single item, as well as relative positional and configurational aspects. Because all sleep quality groups were presented with the same complex scene, we suggest that an attentional force that is greater than existing physical saliencies is likely to have driven the responses of poor sleepers.

We have recently completed a second ICB experiment to replicate and extend the above work.[73] In this study, we improved diagnostic methods by including a clinical interview and actigraphy in the protocol, and strengthened the primary analysis by incorporating DSPS as a clinical control group. In this experiment we used different change stimuli; respectively, a teddy bear and a mug, to rule out the possibility of idiosyncratic effects to previously used stimuli. A 2 (experimental condition) by 3 (group) between-participants design was employed. Participants ($n=90$) were within one of three groups (PI, DSPS, GS) and each sleep quality group was consequently split in half at random to receive the sleep-related change, or a sleep-neutral. Group allocation was not fully known to the experimenter until the ICB experiment was completed. As predicted, the stimulus change/sleep quality interaction was significant and PI detected the sleep-related change significantly quicker than the sleep-neutral change. No such difference was observed between the sleep-related and sleep-neutral changes for either DSPS or GS participants (Table 2). Post hoc testing also revealed that, for the sleep-related change, responses of PI were significantly quicker than GS and DSPS, and that responses of DSPS were significantly quicker than those of GS. By comparison, for the neutral change, responses of GS and DSPS were significantly quicker than PI and no difference was observed between GS and DSPS.

The results of this experiment provide further evidence of attention biases to sleep-related stimuli in insomniacs. However, unlike our dot probe study[70], we also found that DSPS participants detected a sleep-related change significantly quicker than GS. We propose several possible explanations for this effect. First, DSPS, particularly in younger people, may comprise two distinct sub-groups, a socially driven DSPS and an inherent/genetic DSPS, whose responses to attentional measures may differ. Second, results may depend on whether DSPS participants were sleeping in phase or out of phase at the time of the experiment. In the latter case one might expect more insomnia symptoms. Third, and as we previously discussed[54], suggest that PI and DSPS may often share overlap symptoms. Sleep-onset PI may facilitate phase delay, and phase delay may contribute to anxiety and preoccupation about sleep initiation.

To date then, our work has shown that attention bias presents in individuals with persistent PI. The extent to which such biases are maintaining of PI, or merely epiphenomenal, remains unclear, as does whether attention bias can predispose to PI. Although some of our data suggest that attention may shift from stressor to sleep as adjustment insomnia becomes chronic, longitudinal work will be needed to confirm this. Sleep-related attention bias may be a common factor in all sleep disorders or it may help to differentiate insomnia subtypes, e.g. psychophysiologic insomnia from insomnia due to mental disorder. Again further work is needed on this.

Evoked potential studies

Two recent insomnia studies employing event-related potential (ERP) measurement of cortical arousal also merit consideration. Devoto et al.[130,131] have shown, in participants with PI, that cortical hyperarousal is not constant in poor sleepers. Rather it fluctuates depending on the quality of the previous night's sleep. Variation in P300 amplitude is thought to reflect neuroelectric activity related to cognitive processes such as attention allocation and immediate memory; larger P300 amplitudes being associated with poorer nights of sleep. These data, therefore, are important because they further evidence an association between disturbed sleep and attention processes. They suggest that P300

measurement may be of potential benefit in the further study of attention bias. Would, for example, biased attention to sleep cues in a Stroop paradigm map onto enhanced ERP activity? There is already psychophysiological evidence to this effect, on attention to emotion cues.[29]

In summary to this point, we suggest that there is a developing body of evidence that supports the proposition that sleep-related attention bias may be implicated in the development and persistence of PI. This evidence comes from both qualitative and quantitative studies. Recent experimental data from studies manipulating attentional variables, and from studies investigating information processing speed and evoked potentials, offer the most direct evidence and such data are consistent with clinical impressions of sleep preoccupation, and possibly of sleep-related conditioning, in PI.

Explicit intention

Introduction

We propose that the next stage of the inhibitory A–I–E pathway is that of developing an explicit intention to sleep. That is, an attention-for-action mechanism associates attentional focus upon sleep and sleeplessness, with intentive actions designed to deliver sleep and to eliminate wakefulness (Fig. 1). We will argue that these intentions are antithetical to the behavior of good sleepers, as they are with those seeking skilled performance in other domains,[39,40] that they further inhibit the automaticity of normal sleep (beyond the effects of attention/monitoring alone), and that, because they are usually ineffective, they lead to the engagement of sleep effort.

So far we have considered the process of attention. Now it seems helpful to consider its purpose. Why is selective attention so important? There seem to be two, related answers to this question. One is that attention has to be selective because the brain is a limited capacity processor. The other is that selectivity actually confers distinct advantages, irrespective of capacity.

At any given time, only a small proportion of information available in the environment can be selected and identified for conscious processing. Optimally, this selection should be based on the information necessary for the execution of current and planned behavior. Thus, it is adaptive to focus upon a threat, so that you can take avoidant action. Likewise, it is adaptive to focus upon an unmet need (to crave), so that you can develop a plan to meet that need. It is less urgent and less important to focus elsewhere.

Allport[74] pointed out that the majority of research in this area considered the limited information-processing capacity of the brain as the fundamental constraint underlying all operations of attention. Thus the selection function of attention arises necessarily from the notion of limited capacity. However, diverging from the idea that attention operates primarily as a mechanism for coping with central limited capacities of cognitive processing,[75,76] Allport also emphasized constraints in preparation and control of action. The idea behind this attention-for-action perspective is that integrated actions require the selection of particular aspects or attributes from the environment that are relevant to the action at hand. At the same time, any information irrelevant to the action should be ignored. Thus, attentional processes may be seen as the selection of action-relevant events or stimuli relying on particular action plans. This echoes the concluding phrase of James'[18] definition, presented earlier, that

> "(selective attention) … implies withdrawal from some things in order to deal effectively with others" (present authors' emphasis).

Likewise, Posner[77] reflects upon an early metaphor that:

"Thinking, like swinging a bat, has a "point of no return"—once committed in a particular direction, thought is ballistic in that it cannot be altered." (p. 3)

It seems then that specific aspects of the environment are overtly and covertly selected and become integrated in goal-directed action planning. This is what we mean by 'intention'. We attend so that we can intend. For the majority of human behavior this works well as an active process. Focusing and directed, purposive behavior is helpful, and it generally improves performance. However, there are certain circumstances where explicit intention is counter-productive. These arise, not least, with bodily functions that are designed to operate automatically.

A good example of this is the human sexual response. Erectile responsivity is not essentially purposive, and it can be inhibited by attention and intention, particularly when fuelled by anxiety or worry about performance failure (threat) or by desire to respond normally (incentive). There is a considerable literature demonstrating that the human sexual response is undermined by self-monitoring and self-observation, and that it is promoted by focusing away from, rather than directly upon, physiological 'performance'.[78,79]

Techniques such as 'sensate focus' have been applied successfully for 30 years or more to reduce this maladaptive self-referential monitoring of sexual response. Using this approach involves focusing upon (selectively attending to) sensory experiences (of touch) and away from overtly sexual experiences (sexual arousal). The explicit intention of responding sexually is, thereby, removed, permitting arousal to re-emerge spontaneously.

There is a long tradition of psychological theory and practice, dating back to the work of Victor Frankl[1] involving techniques such as paradox and dereflection.[80,81] These have been applied to situations where there is excessive concern about the frequency of a response occurring too often (e.g. blushing, excessive sweating) or not often enough (e.g. sexual response). This literature illustrates that attending to, and intending to produce, a desired behavioral goal can be inhibiting; and that, in turn, that by instructing patients to intend the opposite, the original desired goal may be achieved more easily.

Conceptualisation of explicit intention in relation to insomnia

To re-iterate, we propose that a dysfunctional explicit intention to sleep develops in the context of an attention-for-action mechanism that associates sleep/sleeplessness with behavior specifically designed to deliver sleep/eliminate wakefulness (Fig. 1). Thus, the automaticity of normal sleep initiation is further challenged by the emergence of a specific purpose-to sleep. Again it is instructive to think of sleep normalcy.

We suggest that normal sleepers do not exhibit a well-developed explicit intention to sleep. Rather their intentions may be more implicit than explicit. That is, it is implicit in going to bed, putting the light out, adjusting body position and so on that the purpose is to sleep. We have previously referred to these as automated setting conditions. Such preparatory behaviors certainly reflect an implicit intention—they are not indifferent to or neglectful of sleep—but they are not done with the express purpose of sleep. Indeed, exception to the general principle that good sleepers lack intentive purpose associated with bedtime behavior, may arise when their explicit intention is, in fact, to remain awake. The notion of 'abandonment of wakefulness' seems more apposite to normal good sleep.

Take the example of reading in bed. Good sleepers read in bed, so why should we discourage people with insomnia from reading in bed, as per stimulus control instructions?[17] Is reading in bed a sleep-incompatible behavior, or is it not? This may, of course, depend more upon the nature of conditioned behavioral associations, than upon the behaviors per se. However, another explanation of the essential difference between good sleepers and people with insomnia in relation to reading, is that good sleepers are more likely to have the explicit intention of remaining awake, in order to read; whereas people with insomnia are more likely to read, with the explicit intention of falling asleep. Therefore, the situation may arise that the good sleeper gets to the point of quitting reading in order to sleep, simply because they are already lapsing into micro-sleeps from time to time. Indeed, they may fight sleep off for a while before giving in (passively) to sleep. By comparison the person with insomnia is more likely to be filling wakeful time, hoping that this (or another) strategy might work to help them sleep. The contrast, therefore, is that for the good sleeper the precursor to sleep is abandoning wakefulness; whereas, for the person with PI it is trying to initiate sleep.

It also appears from the diagnostic schedule that explicitly intending to sleep is problematic for the person with PI. Although ICSD-2[4] criteria for PI are based in part on expert clinician evidence and await scientific validation, they do suggest PI patients fall asleep when they do not intend to. One of the PI criteria is:

> "Difficulty falling asleep in bed at the desired bedtime or during planned naps, but no difficulty falling asleep during other monotonous activities, when not intending to sleep." (criterion C2; p. 1–7)

The implication is that desire, planning and intention are counterproductive in PI. There is also the implication that people with PI may more readily fall asleep when they do not have the explicit intention to do so. This is consistent with the literature on paradox.

By definition, people with PI have difficulty falling asleep and remaining asleep, and by convention in insomnia research and in clinical practice we ask people to record these difficulties in a sleep diary. This raises an interesting point in relation to the measurement of sleep intention. Sleep-onset latency (SOL) is usually taken as the length of time it takes to fall asleep, after settling down with the intention of sleeping (after lights out). This fits with the insomnia model, because this is what they do. However, it would be interesting to know, from normal sleepers, on what proportion of nights they fell asleep before they ever intended to. We lack data on this, but our prediction would be that the true SOL values for some good sleeper

nights are in fact negative values, if we were to use the above definition of SOL literally.

The fact that we routinely ask patients to monitor sleep pattern on diaries raises another important point. According to our A-I-E model, to pay directed attention to the involuntary response that is sleep may impair its very automaticity. As therapists then, are we not likely to exacerbate patients sleep problems merely by asking them to complete diaries? Successful completion does require directed attention to sleep. This could be examined, experimentally, by comparing one group of PI patients on standard diary completion with a second PI group, whose sleep is monitored using a method not requiring direct participant involvement with their sleep data. We might expect however that the strongest inhibition of sleep would occur with direct monitoring of sleep during the sleep-onset period.

We would also note at this point that we believe the A-I-E pathway may be equally applicable to sleep maintenance problems. WASO is the primary measure of sleep (dis)continuity, representing the cumulative time taken to re-initiate sleep after night-time arousals. Our model would suggest that the selective attention process may be active before sleep, that it may remain active after sleep-onset and that it may re-activate during sleep, particularly during light sleep and its associated arousals. Because selective attention may be represented as a conditioned involuntary response to arousal, it does not require the person to be conscious or fully conscious. Indeed, the literature on evoked potentials suggests that the brain's capacity to discriminate the intrinsic significance or semantic content of a stimulus may persist in stage 2 and REM sleep.[82] More specifically then, in relation to WASO, it is possible that the vigilant scanning associated with selective attention becomes switched on during light sleep producing vulnerability to arousal and accentuating 'pre-existing' normal and transient nocturnal arousals. Consequently, arousals may be more likely to extend into consciousness and to frank awakenings. Whereas, it has been known for some time that even in normal sleepers there is significantly shorter awakening latency to meaningful stimuli presented during sleep,[133] it remains unclear from the research literature available at this time whether or not PI is associated with increased frequency of awakening from sleep.[134] Further research in this area is required.

However, once awake, somewhat less controversially, we propose that the person with sleep maintenance PI is then in exactly the same position as the person with sleep-onset PI. We are not the first to suggest that a central component of sleep-maintenance insomnia relates to a problem of re-initiating sleep rather than solely a disorder of intermittent wakening per se.[83] The explicit intention to get back to sleep would then apply during the night inhibiting de-arousal in the same way as at (the first) sleep-onset.

In summary, then we suggest that explicitly intending to sleep is (a) not what normal good sleepers do, (b) it is dysfunctional because it inhibits normal de-arousal, and by extension of these points (c) that normal sleep may be restored when intention to sleep is neutralized.

Evidence of explicit intention in PI

There are few studies that directly address sleep intention in PI. However, evidence from a number of sources is suggestive that explicit intention to sleep plays a part in PI.

The multiple sleep latency test (MSLT) is not routinely recommended for insomnia, partly because people with PI generally have daytime sleep latencies in the normal range or even longer.[84,85] This has been taken as support for a variety of different positions—that PI has no true daytime consequences, that people with PI are hyperaroused around the 24 h clock, and that people with insomnia find it difficult to sleep in labs or when observed or when trying to sleep. We would develop this last point by commenting that it may be important that, in the MSLT, people are explicitly trying to sleep. The typical task instruction is:

> "Please lie quietly, keep your eyes closed and try to fall asleep"
>
> (p. 1418)[86]

This could be why people with PI are unable to sleep under MSLT conditions. You might then wonder why explicitly trying to sleep would not inhibit people with other sleep disorders from sleeping during MSLT naps? Our suggestion here would be in terms of our concept of inhibitory sufficiency.[13] An explicit intention to sleep simply may not be sufficient to inhibit sleep in someone who has strong homeostatic pressure to sleep (e.g. narcolepsy) or who is otherwise sleep deprived (e.g. sleep apnea). However in PI, homeostatic pressure to sleep may be relatively weak (until sleep restriction is applied) or at best it may be highly variable; and people with PI are not objectively sleep deprived. Further experimental work varying the instructional set for the MSLT would therefore be informative. Similarly, a comparison of GS and PI

on the maintenance of wakefulness test, or simply using a non-instructional condition (e.g. 'We just want to calibrate the equipment for a few minutes with your eyes closed while you are laying there awake') could prove informative about potential underlying mechanisms.

The inhibitory properties of explicit intention in PI are also supported indirectly by a body of research on paradox and ironic control in insomnia. It has been suggested that anxiety responses may be conditioned not only to external, situational cues but also to the individual's behavior.[87-89] Fear of a performance failure (insomnia) and of anticipated negative consequences of that failure is described as performance anxiety. In the treatment known as paradoxical intention, counter-productive attempts to fall asleep are replaced by the intention of remaining passively awake or by giving up any direct intention to fall asleep.[90,91] This rationale is supported by the fact that good sleepers do not use any strategies to fall asleep. Typical instructions for paradoxical intention therapy have been summarised as follows by Morin and Espie (pp 95–97)[92]

1. When you are in bed lie in a comfortable position and put the light out.
2. In the darkened room, keep your eyes open, and try to keep them open 'just for just a little while longer'. That's your catch phrase.
3. As time goes by congratulate yourself on staying awake but relaxed.
4. Remind yourself not to try to sleep but to let sleep overtake you, as you gently try to resist it.
5. Keep this mind set going as long as you can, and if you get worried at staying awake remind yourself that that is the general idea, so you are succeeding.
6. Don't actively prevent sleep by trying to rouse yourself. Be like the good sleeper, let sleep come to you.

The emphasis, therefore, in paradoxical intention is upon natural sleep initiation and the patient is encouraged to take a passive, accepting role. Setting conditions for sleep are established (bed, comfortable, dark) but the explicit intention is to remain awake, thus obviating attempts to sleep. It is, of course, consistent with the view of wakefulness as accruing sleep debt that one of the most reliable ways to guarantee sleep is to remain awake. Paradoxical intention therapy takes advantage of this principle by prescribing wakefulness as a precursor to successful sleep. Paradoxical intention has demonstrated efficacy as a single therapy in controlled trials,[93,94] and is regarded as an intervention that reflects a 'moderate degree of clinical certainty' according to AASM practice criteria.[95,96]

Of course treatment outcome studies are not designed to test mechanisms of action, so data from such sources must be regarded as preliminary. Rather, both experimental comparison studies (GS, PI) and experimental manipulation studies[12] (in GS) are required to look at specific causal mechanisms. Four experimentally-based studies have been published that yield some evidence about the role of intention.

First, Gross and Borkovec[97] allocated good sleepers to one of three experimental conditions. All participants were instructed to 'go to sleep as quickly as possible' (p. 113) during a daytime nap opportunity. However, in one group they were told that they would have to make a speech at the end of the experiment on an unspecified subject, and in another that the speech had to be on a specific topic. The third group was a control condition with no manipulation of pre-sleep mental content. They found that the speech plus topic group had the longest mean SOL, suggesting that trying to sleep in circumstances where cognitive arousal/anxiety was increased led to greater sleep difficulty. In the Gross and Borkovec experiment it was not possible to separate out the effects of intention and cognitive task demands.

Second, Ansfield et al.[2] explored the effects of different sleep-onset instructions in good sleepers under high or low 'mental load'. This was an elegant study where two factors were systematically investigated. Good sleepers were instructed to fall asleep either "…as quickly as possible … in record time" or "… whenever you would like" (p. 526) under conditions of either "… stirring … marching band music" or "sleep-conducive … music containing restful, outdoor sounds …". Paradoxical wakefulness was found amongst those actively attempting to sleep while listening to the sleep-inhibiting music. This result was interpreted in terms of Wegner's[98] theory that the thwarted attempt to control a particular mental state can yield the opposite of what is desired. Ansfield et al. hypothesized that failure to fall asleep on a few occasions could occur when sleep is attempted under transitory mental loads, such as at times of stress. Eventually a person's thoughts about being unable to sleep could constitute a debilitating mental load which, when combined with the continuing frustrated desire to fall asleep, could lead to chronic insomnia. Interestingly, Ansfield et al. also found that explicit intention to sleep plus sleep-conducive music did not delay SOL, suggesting the importance of the interaction of

intention and mental load in insomnia. Furthermore, this group actually fell asleep more rapidly. This finding could reflect an experimental treatment effect where a relaxation response to calming music counteracted the explicit intention to sleep and delivered a sleep-promoting benefit. That is, although Ansfield et al. intended the calm music to be a neutral condition relative to the impact of the marching music, it seems plausible that it had a converse therapeutic effect.

Third, Harvey[99] has explored the effects of suppressing pre-sleep cognitive activity on SOL. People with insomnia and good sleepers were allocated either to a suppression condition ('suppress the thought most likely to dominate your thinking as you get into bed') or a non-suppression condition ('think about anything as you get into bed, including the thought you would most likely think about as you go to sleep'). Interestingly, 'suppress' participants reported longer sleep latencies and poorer sleep quality, regardless of whether they had insomnia or were good sleepers. Harvey concluded that thought suppression, whilst attempting to turn off pre-sleep intrusive thoughts, appeared to have the opposite effect in that it prevented sleep-onset, in a manner consistent with Wegner's theory of ironic mental control.[98] Wegner uses the terms 'intentional operating process' to refer to the search for mental content that will yield a desired state (e.g. drowsiness), and 'ironic monitoring process' to refer to the search for evidence of failure to achieve a desired state (e.g alertness). There are clear parallels here with Harvey's views on the negative impact of sleep-associated monitoring[36,52] and also with our concept of present state monitoring[13,48] that we now suggest forms part of the A-I-E pathway.

Before considering the final experimental study relevant to explicit intention, it is interesting that several preliminary findings show that thought control strategies may characterise people with insomnia. Harvey has examined beliefs about pre-sleep worry using the utility of pre-sleep worry questionnaire.[100] Relative to good sleepers, people with insomnia endorsed more positive belief statements about worry (e.g. 'worry in bed helps me get things sorted out in my mind'). The number of negative belief statements endorsed was not different between the groups. Harvey using the Thought Control Questionnaire for Insomnia also reported that people with insomnia used more reappraisal, worry and thought suppression strategies to control pre-sleep intrusions, relative to good sleepers.[101] Similarly, Ellis and Cropley found worry and punishment strategies to be the strategies most commonly used by people with insomnia.[102] Such attempts amongst people with insomnia to control their thoughts (so that they can clear the way for sleep), therefore, may fuel further intrusions, and so maintain sleep disturbance. We suggest that the motivation for thought control may be because, unlike normal good sleepers, people with insomnia are actively engaged with intentional sleep.

Fourth, Lundh and Hindmarsh[103] described a clinical experiment that has relevance to this thought control literature, and to what we have previously described as metacognition. Forty participants were instructed simply to monitor, but not to respond to, their thoughts and emotional state during the pre-sleep phase. This 'meta-cognitive observation' task was found to significantly reduce sleep latency. Although lacking a comparison group, the study provides a demonstration of the potential benefit to sleep of this 'mindfulness' approach, which in turn may mimic normal pre-sleep cognition.

Taking the body of available evidence as a whole then, we suggest that there is preliminary evidence to support both the presence of explicit sleep intentive processes in insomnia, and the utility of neutralizing/normalizing strategies to counteract their effects.

Sleep effort

Introduction

The third component of the proposed A-I-E pathway is what we have called sleep effort. Because, this is a developmental pathway, we conceive of A-I-E as comprising not so much discrete components as overlapping stages. Consequently, there is not a point at which intention ceases and effort commences (Fig. 1). Rather, we regard effort as a further development of intention, to the extent that the 'end state' of persistent PI (leaving aside the potential for depression to develop) could be described as a sleep effort syndrome.

Indeed, for the past century the Yerkes-Dodson Law has suggested that high physiological arousal can be disruptive to intended behaviors presumably by an excess of neural noise interfering with the sequences of choice points necessary in initiating behaviors. The decreases in signal to noise ratios at all these choice or decision points result in disruptions to skilled behaviors or voluntarily directed behaviors. Anxiety clearly increases arousal level, but so does any 'effort'. Whereas a certain amount of arousal can be a constructive behavioral

motivator, too much (or indeed too little) is likely to work against the organism. Because increased arousal in response to effort is an extremely well-learned response, we would argue why should it be different with increased effort to fall asleep?

In evidence of explicit intention in PI we illustrated explicit intention and how it interfered with sexual responsivity. An analogy for effortful preoccupation and its inhibitory consequences would be the phenomenon of stammering. It is rare for people with speech impediments of this type to have specific physiological or motor/mechanical disorder, although the 'causes' of stammering are best regarded as multi-factorial.[104] Deliberate and effortful speech production is typical, and automatic, fluent speech appears to be inhibited or compromised. Particular difficulty is evident in responsive speech to concrete questions where awareness of the correct answer and attempts to produce it lead to blocking and stammering on letters or syllables. Also, like sexual dysfunction and insomnia, there is marked frustration at being unable to accomplish what seems like a relatively simple thing—in this case to articulate words fluently. The automatic, over-learned nature of competent, fluent speech is so unremarkable that comment would seldom be made on successful everyday conversation. Consequently, difficulties with speech are of magnified concern to the stammerer. However, the 'good speaker', like the good sleeper is entirely unaware of the process, and exhibits no qualities or special competencies that are immediately obvious.

Attention to speech, intention to speak and effort to control spoken output seem to characterize the problem. This contributes to what may be one hallmark feature of effortful preoccupation, performance anxiety. Performance anxiety is found in a wide range of symptoms and disorders, particularly where psychological and physiological factors interact. Other examples include blushing, where social context triggers vasodilation but attentional and controlling responses exacerbate the symptoms, and psychogenic urinary retention where self-observation and active efforts to urinate restrict sphincter relaxation.[80] The criterion of performance is clear in each case (e.g. erection, speech, social encounter, urinating) thus making it easy for the person with the problem to identify inadequate performance or performance failure.

The impact of effortful processes has also been shown in experimental studies. For example, Wegner, Broome and Blumberg demonstrated using electrodermal measurement that trying to remember a 9-digit number during progressive muscle relaxation increased skin conductance level, whereas skin conductance decreased with relaxation alone.[105] This combination of intention to do one thing (relax) in the face of oppositional factors (mental load) parallels our concept of effort arising out of intention. 'Striving' would be a good synonym for our concept of effort.

It is also instructive that successful treatment for disorders where effort and conscious control are part of the problem does not involve reinforcement of effort as a strategy. On the contrary, disrupting the inhibition of what in our terms is the A-I-E pathway can yield good outcome. Many of these strategies are in fact paradoxical in nature. For example, in stammering, delayed auditory feedback disrupts self-observation and forced speech, and distractor or rhythmic procedures are used to enhance natural fluency rather than emphasising speech production per se.[106] At a more generic level, there is emerging evidence of the health value of the principle of acceptance (e.g.[107]). Of course, this is not a new idea because many philosophies and world religions embrace the importance of accepting certain situations instead of struggling against them. The principle has been particularly well articulated in what has become known as the mindfulness approach (e.g.[108]). where cognitive/emotional processes are observed without any ambition to change them. Although mindfulness is a much wider system of thinking and therapy than is actually required for the present purposes of understanding PI, it does lend further construct validity to intention and effort as maladaptive strategies.

Conceptualisation of sleep effort in relation to insomnia

Our suggestion is that the development of PI follows a pathway from an implicit information processing bias (pre-attentive perceptual) to an explicit processing bias (conscious mental), then to an explicit intention (responsive mental) and then to an effortful preoccupation (responsive/proactive behavioral). If selective attention is scanning mode and explicit intention is planning mode, then sleep effort is performing mode. Sleep effort is seen as comprising two, related processes—one that is direct (e.g. actively trying to sleep) and one that is indirect (e.g. increasing sleep opportunity). This conceptualisation therefore characterises the development of the behavioral response to the developing PI problem and is illustrated in Figs. 1 and 3.

A mechanical analogy may be helpful. We propose that good sleep is fully automated, in the

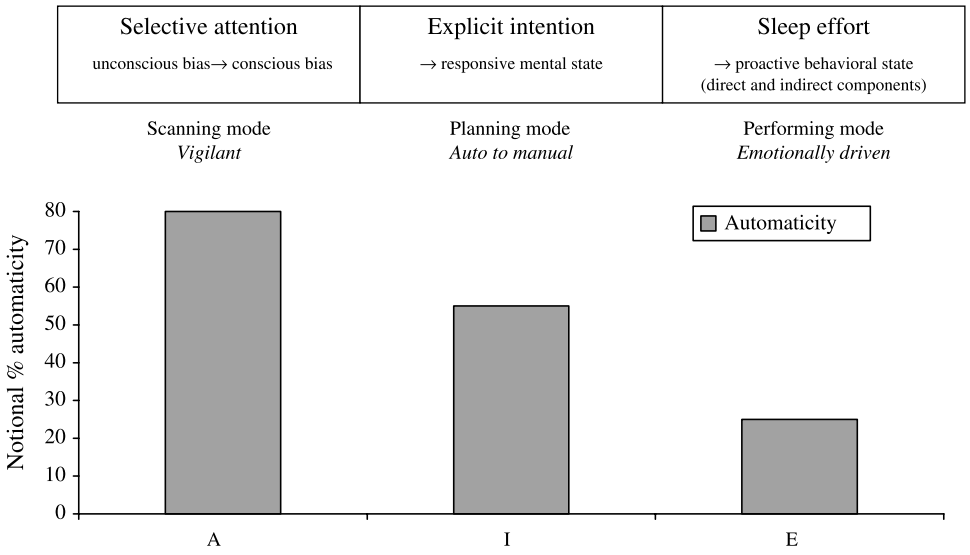

Figure 3 The development of psychophysiological insomnia following the attention–intention–effort pathway and its impact on automaticity.

context of appropriate setting conditions, but that selective attention partially impairs this automaticity. We suggest that it is then further impaired by a crucial switch towards 'manual' operation during the intentive stage. However, the lasting damage, resulting in persistent PI, may be done when a compelling need to take control and fix the problem develops. We suggest that this stage is more emotionally driven and so it will be particularly associated with evaluative considerations. This is what we mean by using the term performing mode (Fig. 3). Sleep has now become an enactment that is observed, analysed and performance reviewed. Dissatisfaction in these circumstances is likely to undermine not only sleep but also self-efficacy. Fig. 3 also illustrates notionally the cumulative effect of the A–I–E pathway upon the impairment of automaticity of the healthy sleep process.

Returning to ICSD-2[4] descriptions of PI, the following segments seem relevant to the concept of sleep effort.

> "Learned associations are marked by overconcern with the inability to sleep. A cycle develops in which the more one strives to sleep, the more agitated one becomes, and the less able one is to fall asleep" (p. 1–6)

> "... individuals with insomnia characteristically demonstrate effortful preoccupation with both the consequences of and the potential solutions for their sleep problems" (p. 1–6)

> "A sense of repeated failure to resolve sleep problems often leads to intermittent periods of resigned helplessness and help-seeking behaviors" (p. 1–7)

We suggest that the sleep effort response presents in two ways; first, as increased direct effort to get to sleep, or to get back to sleep when awake in bed; and second, as increased indirect effort to sleep by manipulating the setting conditions for normal sleep. Furthermore, 'efforts' may be both cognitive and behavioral. We will briefly describe these ideas.

Direct behavioral effort may include things like trying to force sleep to come, tossing and turning to find a sleep position, lying particularly still as if asleep, and being unwilling to 'give in' and get up when not sleeping. We have previously mentioned reading in bed with the express purpose of sleeping in this context, and people tiring themselves out by exercising vigorously is another example. Examples of direct cognitive effort include thought management, counting sheep, suppressing thoughts and distraction techniques. As will be apparent, direct efforts to sleep may also include well-intentioned strategies that have some basis in behavioral sleep medicine, e.g. the use of relaxation exercises as a sleep-inducer. Our point is that, to the extent these activities involve the direct effort to sleep, they may actually contribute towards the maintenance of PI, rather than towards its resolution. It is anecdotal, but nevertheless typical, of the person with PI to report that they have 'tried everything' to help them sleep!

Indirect sleep efforts may be particularly important in PI. This would be where maladaptive steps are taken to influence the probability of

sleep. In terms of our model, this not only upsets the default setting conditions for good sleep, but also reduces the statistical probability of sleep. At the cognitive level, people with insomnia often try to manage or contain levels of mental and emotional stimulation so that they are likely to sleep better. Sleep becomes such a priority that it is anticipated throughout the waking day. The most common behavioral responses are to increase sleep opportunity by going to bed earlier, staying in bed later, and trying to 'catch up' on lost sleep. We emphasized earlier how craving for sleep might result in increased time spent in bed. So, ironically, in an indirect behavioral effort to obtain more sleep, the net effect is to reduce sleep efficiency.

To illustrate this, consider the data presented in Table 3. This is a fictional case of a person averaging 63 min of wakefulness and with sleep efficiency of 85% based on 7 h in bed (week A). This sleep pattern is at the margin between normal sleep and insomnia, but let us suppose that the person is concerned about their sleep and its consequences, and so increases time in bed by 1 h per night for the next 7 nights. The data in week B assume that this actually results in some benefit to sleep duration (illustrated by a stable increase in sleep of 15 min per night). However, the impact on sleep efficiency reduces the average to 78% (range 66–86%). Although, this is only a modest 7% reduction from the week A value, for the homeostat to return efficiency to the 85% value, based on the new behavior of spending 8 h in bed, would require an average sleep of 408 min (480×0.85). Inspection of the raw data over the 14 nights in Table 3 show that this length of sleep was only ever obtained on 1 occasion, and that, in spite of sleeping a little more, average sleep in week B is some 36 min below that target.

Direct efforts to initiate sleep we suggest are more likely to be affect laden—e.g. lying awake trying to get to sleep. Indirect efforts such as increasing sleep opportunity may be less affect laden. Indeed, they may initially reduce anxiety because they offer the reassuring possibility of more sleep, and may actually deliver some. However, as well as undermining automaticity, they have the capacity to exacerbate insomnia symptomatology and place sleep efficiency outwith the range within which it can spontaneously recover.

Most commentators regard stimulus control/-sleep restriction as the core components of an effective CBT programs (e.g.[92]). One of the foremost reasons for this is that these interventions quickly and effectively tackle the problem of extended sleep opportunity. We cannot say at this stage by what means these procedures achieve their effects,[13] however, it should be noted that these, essentially behavioral, approaches are not inconsistent with the A-I-E pathway. Both stimulus control and sleep restriction therapies involve (a) establishing setting conditions for sleep that are largely determined by sleep needs, rather than sleep desires; (b) strengthening homeostasis and circadian timing, rather than personal agency over sleep; and (c) precluding the need for, or quickly abandoning, direct attempts to sleep, rather than trying to initiate sleep. Consistent with sleep normalcy, stimulus control and sleep restriction help to re-engage the 'two process' functions of sleep drive and timing. Consistent with the A-I-E pathway they also reinforce the implicit third process of automaticity.

Evidence of sleep effort in PI

Evidence of sleep effort in insomnia can be drawn from several sources.

In the context of paradoxical intention therapy, the outcome literature that we reviewed in the section on Explicit intention, includes three studies that provide some evidence of effortful processes and mechanisms.

Table 3 Comparison of 2 weeks of fictional sleep diary data illustrating the impact of extending sleep opportunity by 1 h

		Night 1	Night 2	Night 3	Night 4	Night 5	Night 6	Night 7	Mean
Week A	TST	380	360	390	320	350	400	300	357
	TIB	420	420	420	420	420	420	420	420
	SE	90	86	93	76	83	98	71	85
Week B	TST	395	375	405	335	365	415	315	372
	TIB	480	480	480	480	480	480	480	480
	SE	82	78	84	70	76	86	66	78

TST, total sleep time (min); TIB, time in bed (min); SE, sleep efficiency % (TST/TIB×100).

Fogle and Dyall[91] compared different approaches to delivering paradoxical instructions. They found that the instruction to 'give up trying' to sleep was just as effective in treating insomnia as the more explicitly paradoxical 'try to remain awake' instruction. In other words, ceasing to try may be the essential element in the paradoxical approach, which would be consistent with reversal of the effort component of the A-I-E pathway.

Our own early work using paradox yielded some unexpected findings. We found that it could be a very effective treatment for insomnia,[94] yet for some individuals the paradoxical directive seemed to re-focus performance anxiety and lead them to quite literally remain awake.[88] We interpreted this as an interaction between the performance focus of the individual (initially upon sleep) and the demand characteristics of the therapeutic environment, where they were being asked to stay awake (instead of to sleep), such that they tried too hard (to implement therapy). In other words, effort gets in the way. There may be parallels here with Lundh et al.'s suggestion that perfectionistic traits may be predispositional in some people with insomnia.[109]

Recently, we have completed another study on paradox.[110] This was an experimental trial that examined the effect of paradoxical intention on effort to sleep, on sleep performance anxiety, and on both objectively-estimated and subjectively-estimated SOL. Sleep effort was measured by a rating of 'When I went to bed last night, I tried really hard to get to sleep' ranging from 0 'not at all' to 6 'very much', and performance anxiety about sleep was rated using a preliminary version of the Glasgow sleep effort scale (GSES).[c] Following a seven-night baseline, 34 participants (mean age 25 years) with persistent sleep-onset insomnia (mean duration 6 years) were randomly allocated to fourteen nights of paradox, or to a control (sleep as usual) condition. The intervention period was deliberately short because we were primarily interested in the impact of paradoxical instruction upon sleep effort. Consistent with this model, participants allocated to paradox, relative to controls, showed a significant reduction in sleep effort and sleep performance anxiety. A developing trend for significantly lowered subjective (sleep diary) SOL in PI participants was also demonstrated within the brief treatment period. The important finding here was the observation that paradoxical intention appeared to operate by reducing sleep effort/anxiety. Effort change significantly correlated with SOL change when sleep anxiety was partialled out ($rp = 0.42$, $p = 0.016$). In contrast, when effort change was partialled out, sleep anxiety was not associated with SOL change ($rp = 0.08$, $p > 0.1$). This further supports the model of explicit sleep intention as inhibitory.

Some rating scales that are commonly used in insomnia research and in clinical practice contain items that suggest that an effortful approach to sleep might be implicated in PI. The dysfunctional beliefs and attitudes about sleep scale[43] is designed to measure cognitive distortions and thinking errors in insomnia. DBAS Item 7 ('When I have trouble falling asleep or getting back to sleep, I should stay in bed and try harder') forms part of a constellation of mental symptoms that Morin[111] originally related to 'faulty beliefs about sleep-promoting practices'. This item was retained (as Item 4) in a recent psychometric analysis of the DBAS that produced a shortened 10-item version comprising beliefs that demonstrably changed in response to CBT intervention.[112] A Principal Components Analysis (PCA) loaded this item on Factor III 'beliefs about the need for control over insomnia'.

Three of the 12 items in the Sleep Disturbance Questionnaire (SDQ) are also relevant to the concept of effort. In the original study using the SDQ the items 'I try too hard to get to sleep', 'I get too "worked up" at not sleeping' and 'I worry that I won't cope tomorrow if I don't sleep well' were used to select patients for paradoxical intention therapy because they were felt to reflect sleep effort and sleep performance anxiety.[44] Furthermore, Principal Components Analyses have demonstrated that these items do load together on the same construct.[44,112] However, the lack of a specific validated measure of sleep effort led us to develop a new self-report measure—the glasgow sleep effort scale (GSES).[113]

Work on the GSES began in the context of a study investigating the sensitivity and specificity of commonly-used insomnia research tools in discriminating PI, insomnia associated with mental disorder (I-MD) and GS.[114] Fifty-four adults (mean age 40 years; $n = 18$ per group) participated by completing a set of six psychometrically robust insomnia self-report instruments, along with the Beck Anxiety Scale and the Beck Depression Scale. Although the experimental groups differed on the majority of these measures, logistic regression analysis indicated that 'effortful preoccupation with sleep' (as measured by the GSES) discriminated PI from GS (with 100% sensitivity, and 94% specificity) and the GSES also discriminated I-MD from GS (100%, 100%). Furthermore, only depressive symptomatology (on the Beck Depression

[c] The final version of the GSES is described in greater detail below.

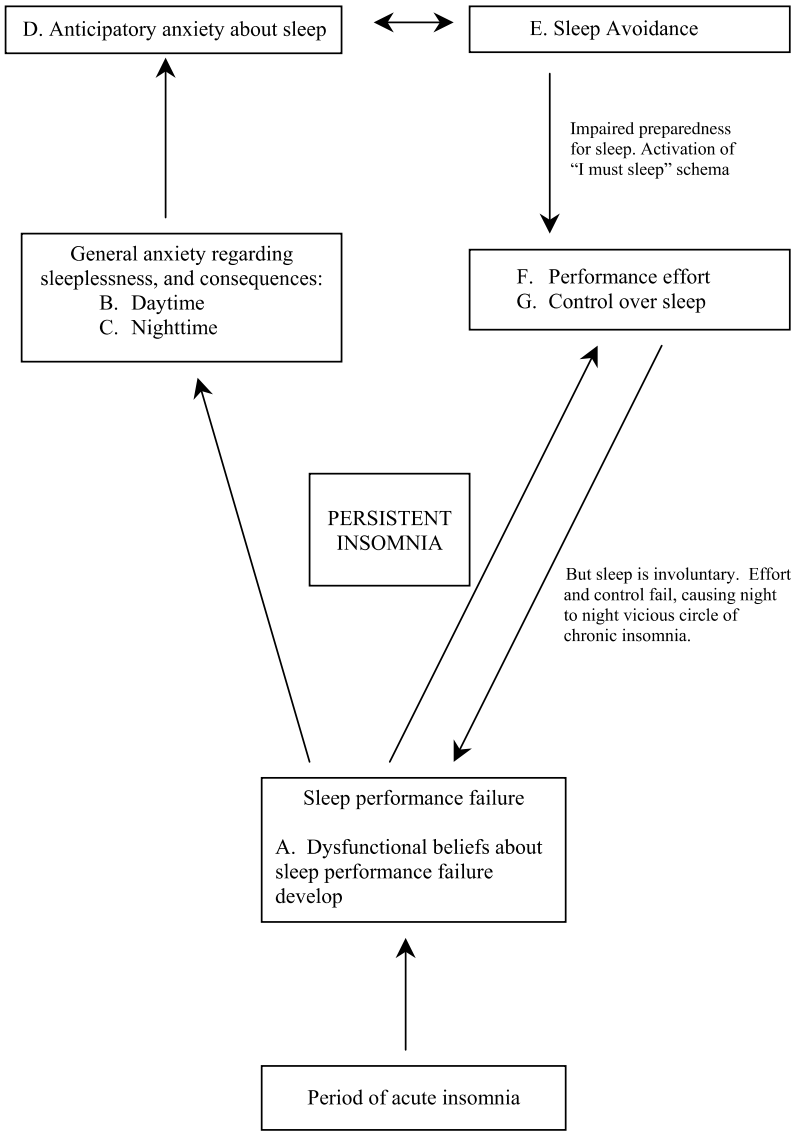

Figure 4 A preliminary working model of direct sleep effort in persistent Psychophysiologic Insomnia (reproduced from[113]).

Inventory (BDI)) discriminated I-MD from PI. No other variables entered any of the regression models.

These results not only suggest that sleep effort is an important concept in PI, but also that other mental and behavioral measurements may be less specific to PI as a disorder. Furthermore, the fact that these findings held also for insomnia associated with depression raises the interesting possibility that a continuum may exist across 'primary' and 'secondary' insomnia (at least where it is associated with depression/anxiety). This possibility is supported by other recent work on symptom reports in severe chronic insomnia which have found that 'depression-related insomnia' and PI were separable only by characteristic symptoms of depression.[115] On the other hand, Broman and Hetta[116] reported that cognitive and somatic pre-sleep arousal was not correlated with insomnia associated with affective disorder. Clearly, further work in this area is required.

We were encouraged by these results to conduct a formal validation study of the GSES.[113] Therefore, a working model was developed, integrating what we felt were the seven core subjective components of sleep effort[d] and each component was assigned a single item (Fig. 4 and Table 4). Fig. 4, thus, represents the final stage of the A-I-E pathway and illustrates what we mean by performing mode (cf. Figs. 1 and 3 and associated text). This proactive

[d] That is direct sleep effort. Indirect sleep effort (e.g. by increasing time in bed) is best measured behaviorally, on a sleep diary, rather than on a Likert Scale.

Table 4 *The Glasgow sleep effort scale*[113]

The Glasgow sleep effort scale The following seven statements relate to your night-time sleep pattern *in the past week*. Please indicate by circling one response how true each statement is for you				
1.	I put too much effort into sleeping when it should come naturally	Very much	To some extent	Not at all
2.	I feel I should be able to control my sleep	Very much	To some extent	Not at all
3.	I put off going to bed at night for fear of not being able to sleep	Very much	To some extent	Not at all
4.	I worry about not sleeping if I cannot sleep	Very much	To some extent	Not at all
	I am no good at sleeping	Very much	To some extent	Not at all
6.	I get anxious about sleeping before I go to bed	Very much	To some extent	Not at all
7.	I worry about the consequences of not sleeping	Very much	To some extent	Not at all

Relationship of Items 1–7 to components A–G in Fig. 4: 1–F; 2–G; 3–E; 4–C; 5–A; 6–D; 7–B.

state is emotionally driven in an effort to solve the problem of sleeplessness. Each core component was allocated one item in the GSES and we field-tested the scale on 89 insomnia patients and 102 good sleepers. The GSES was found to have good internal consistency ($\alpha = 77$) and discriminant validity. Mean total GSES score for PI was 7.06 (SD = 3.58) and for GS was 1.22 (SD = 1.35) [$t = 15.27$, $p < 0.0001$]. Importantly, sensitivity/specificity analysis found that a cut off score of only two correctly identified 93.3% of insomnia patients, and 87.3% of good sleepers. This result is supportive of the concept of automaticity in normal good sleep. GS simply do not endorse the items because the idea of trying to sleep is quite alien to them. There was also evidence of the construct validity of 'sleep effort' because PCA yielded a single principal component (Eigenvalue = 4.38) accounting for 62.6% of total variance, and each of the seven items loaded similarly and significantly on this factor (range 0.64–0.85).

Further work is clearly required on the GSES. Nevertheless, it has the potential to be a quick screening method to identify people with PI in the community, and used alongside other measures, to contribute to further research investigating diagnostic components of PI.

Discussion

In this paper, we have argued for an expansion of experimental cognitive research on insomnia, and have focused upon evidence relevant to the appraisal of what we believe may represent (for Psychophysiologic Insomnia at least) one critically important sleep inhibitory process: the attention–intention–effort pathway. Consistent with our starting point of understanding how PI differs from sleep normalcy,[13] we have suggested that the involuntary and automatic nature of the 'two process' sleep system is first compromised by selective attention to sleep, then imperilled by explicit intention to sleep, and finally dysregulated by a destructive combination of direct and indirect sleep effort. Therefore, PI in its end state as a 'sleep effort syndrome', may be characterised by attention bias, sleep preoccupation, and a panoply of mental and behavioral strategies designed to deliver sleep and to avoid sleeplessness, none of which would be typical in good sleepers. Of course, we must bear in mind that the true end state of insomnia may lie, not in intractable insomnia alone, but in the dysregulation of affect, because insomnia is an independent risk factor for depressive disorder.[117,118]

Evidence to support an A–I–E model is only beginning to emerge and there is much work still to do. Indeed, for now, whilst there is reasonable support for the attention component of the model, there is less evidence for the second and third components. The information processing literature on anxiety disorders emphasises attention bias toward emotionally threatening stimuli. This concept of 'sleep cue as threat' may apply to PI, although salience may be conferred on sleep cues for other reasons, not the least of which might be a 'craving' for sleep. Because, we are interested in the specificity of attention bias in insomnia, it will be important to partition and compare responses (within subjects) to threat words (e.g. tired,

wakeful, restless) and non-threat words (e.g. rested, relaxed, sleeping) using a verbal attention bias paradigm. We have also argued that attention is first 'allocated' to sleep cues as part of an unconscious process, akin to conditioning. This implies that people may be already incubating an insomnia response before they are aware that sleep and sleeplessness are grabbing their attention, as in the transition from acute/adjustment insomnia to persistent insomnia. Exploration of attention bias at the implicit or pre-attentive level is therefore required, and can be achieved using subliminal variants of attention bias probe tasks.[119]

The selective element of attention is also important to consider, because inevitably as attention is focused more in one direction, it is focused less in another direction. Posner has suggested that the attention system is not unitary, but comprises measurable cognitive components (shift, engage, disengage),[77] which are sub-served by specific, neural sub-systems[120,121] and, which are open to modulation by negative emotional stimuli.[29] In recent years, anxiety researchers have begun to apply Posner's attention model to determine whether salient threat stimuli attract attention i.e. modulate the engagement component of covert attention, and/or hold attention, i.e. modulate the disengage component.[122] Most findings for anxiety emphasise slowed disengagement from threat, i.e. a holding function.[123,124] In insomnia research, it would be useful, therefore, to determine which components of attention comprise the bias for sleep words that we have observed. Use of a modified cue-target paradigm would be appropriate for this purpose.

Similarly, we have reprised the notion of 'attention for action'.[74] That is selective attention has a purpose. In general terms, it confers evolutionary advantage by prioritising and directing activity through intention and goal-directed behavior. However, sleep may not be a response that is facilitated by such direct action; rather it may be inhibited. It remains to be demonstrated through experimental study that threat of sleeplessness and/or desire for sleep would drive explicit intention to sleep, although the idea is consistent with clinical experience and with contemporary diagnostic criteria for PI. The glasgow sleep effort scale, however, includes items that may span the A-I-E construct and reflect this 'dynamic' force of attention for action. The GSES has some encouraging sensitivity/specificity data[113,114] and may prove to be a useful measure for identifying individuals with PI. Ideally, however, sleep intention and sleep effort should not be measured solely by a self-report instrument. Some additional measure of mental/behavioral response tendency is desirable. We have illustrated how extending sleep opportunity by increasing time in bed could be a behavioral correlate of indirect sleep effort, leading to homeostatic dysregulation. In parallel, direct sleep effort (trying to force sleep to come) is likely to increase cognitive and emotional arousal.

The interrelationship between attention, intention and effort in the emergence and persistence of PI also needs to be studied. For example, researchers might consider exploring whether sleep intention and sleep effort can be reliably induced, experimentally, in good sleepers through manipulation of sleep monitoring instructions. It may also be possible to study, longitudinally, following an acute stressor event, the pattern of sleep change and associated development of attention, intention and effort, in individuals predisposed to PI. Extending the cross-sectional paradigm employed by Taylor et al.[69] in cancer patients would be useful here. Importantly, the work of Harvey and colleagues is leading the way by emphasizing the application of controlled experimental methods to tease out causal pathways in insomnia.[56–58] Computerised experimental tasks seem complementary to this work because they offer an objective index of sleep-related cognitive arousal.[69,70,72,73]

Related to these matters is the question of whether psychological treatment impacts upon the A-I-E pathway. For example, attention biases reduce following CBT therapy for anxiety disorders.[125,126] Demonstrating that established psychological treatments such as stimulus control or multi-component CBT impact attention bias in PI would add strength to the argument that such biases play a critical role. We are currently gathering data considering the impact of CBT on attention bias in individuals suffering sleep disturbance secondary to cancer, and will also have some outcome data on the GSES from other clinical trials.

We have suggested that, to the extent that any CBT method enables an individual to abandon personal agency over sleep and to return to total reliance upon involuntary sleep it may be likely to achieve a good therapeutic effect. This might result from a cognitive manipulation, aimed at changing the person's perspective upon sleep. Mindfulness approaches, which have recently been applied to insomnia treatment,[103] and other 'acceptance' based approaches, are of relevance here, as are attention-retraining methods (already used in the treatment of anxiety disorder[127]), and the more established cognitive and paradoxical

techniques that form part of standard CBT for insomnia.[92] However, we would point out that simple behavioral manipulations might be expected to achieve the end-point of involuntary sleep more quickly. Both sleep restriction and stimulus control (particularly the quarter of an hour rule) involve resisting or giving up efforts to initiate sleep and replacing them with an ultimate abandonment of wakefulness due to sleep pressure. This seems typical of normal good sleepers.

Finally, it seems important to examine the physiological substrate of the A-I-E pathway. In general, few studies have collected psychophysiological data associated with attention bias mechanisms.[122] Most employ only reaction time, despite the direct measurement potential of autonomic indices such as heart rate, skin conductance, as well as gaze direction. Additionally, examining attention allocation in PI using bedroom based (ambulatory) autonomic measurement would better explore conditioned arousal patterns in association with attention bias. Several studies have shown that differential attention responses can be activated by differing sensory and affective properties of the cue, and can be indexed using autonomic measures such as heart rate.[65] For example, deployment of attention towards sleep cues in PI may give rise to an orienting response associated with cardiac deceleration, whereas rapid deployment away might be associated with a defensive response accompanied by cardiac acceleration.[128] Importantly, this cognitive-psychophysiological framework should extend to exploration of the relationship between cognitive and cortical arousal, with data on the latter indexed from the EEG power spectrum (cf.[129]). If the attention bias paradigm provides a direct measure of cognitive arousal, and skin conductance or heart rate variability offer a parallel measure of autonomic function, then comparisons of such data with quantitative EEG parameters could prove particularly informative in exploring the underpinnings of the PI phenotype.

Practice points

1. Computerized information-processing tasks offer a novel objective method for appraising mental arousal associated with insomnia, particularly the sleep-related attention and monitoring component of arousal.
2. The A-I-E pathway offers a framework for evaluating the development of psychophysiologic insomnia as a persistent disorder following acute, stress-related sleep disturbance.
3. The Glasgow sleep effort scale is a brief measure that discriminates psychophysiologic insomnia, and operationalises this component of sleep preoccupation. It may be useful for diagnostic purposes.
4. The A-I-E pathway suggests that targeted CBT intervention to modify the attentional, the intentional or the effortful processes, which maintain insomnia may be sufficient to reduce sleep disturbance and to restore normal sleep.
5. The A-I-E model as a pathway suggests that novel interventions may be applied early to prevent the development of severe and persistent insomnia.

Research agenda

1. Prospective, longitudinal study of the A-I-E pathway is required to investigate its role in the etiology of insomnia.
2. Further studies are required to investigate the specificity of sleep-related attention bias to Psychophysiologic Insomnia, as opposed to other forms of sleep disorder.
3. Investigation of the somatic correlates of attention bias is required to understand insomnia as a psychophysiologic disorder with autonomic, cortical and cognitive components.
4. Future studies need to consider the components of attention (e.g. engagement, disengagement) that comprise selective attention bias to sleep cues in people with insomnia.
5. There is need to investigate the impact that psychological intervention has upon the A-I-E pathway. In particular, to consider whether measures of sleep-related information-processing bias and sleep effort reduce following CBT.

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References

1. Frankl VE. *The doctor and the soul*. 2nd ed. New York: Knopf; 1965.
* 2. Ansfield ME, Wegner DM, Bowser R. Ironic effects of sleep urgency. *Behav Res Ther* 1996;**34**:523–31.
3. American Sleep Disorders Association. *International classification of sleep disorders: diagnostic and coding manual (Revised)*. Rochester, MA: ASDA; 1997.
4. American Academy of Sleep Medicine. *International classification of sleep disorders: diagnostic and coding manual*. 2nd ed. Westchester, IL: AASM; 2005.
5. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. Washington, DC: APA; 1994.
6. Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, Espie CA, et al. Derivation of research diagnostic criteria for insomnia: report of an american academy of sleep medicine work group. *Sleep* 2004;**27**: 1567–96.
7. Perlis ML, Pigeon W, Smith MT. Etiology and pathophysiology of insomnia. In: Kryger MH, Roth T, Dement WC, editors. *The principles and practice of sleep medicine*. 4th ed. Philadelphia, PA: Saunders; 2005. p. 714–25.
8. Morin CM, Culbert JP, Schwartz MS. Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *Am J Psychiatry* 1994;**151**:1172–80.
9. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. *J Consult Clin Psychol* 1995;**63**:79–89.
10. Smith MT, Perlis ML, Park A, Giles DE, Pennington JA, Buysse DJ. Behavioral treatment vs pharmacotherapy for insomnia—a comparative meta-analyses. *Am J Psychiatry* 2002;**159**:5–11.
11. Edinger JD, Wohlgemuth WK. The significance and management of primary insomnia: the past, present and future of behavioral insomnia therapies. *Sleep Med Rev* 1999;**3**: 101–18.
12. Harvey AG, Tang NKJ. Cognitive behaviour therapy for insomnia: can we rest yet? *Sleep Med Rev* 2003;**7**:237–62.
* 13. Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Annu Rev Psychol* 2002;**53**:215–43.
* 14. Lundh LG, Broman JE. Insomnia as an interaction between sleep-interfering and sleep-interpreting processes. *J Psychosom Res* 2000;**49**:299–310.
15. Borbely AA, Ackermann. Sleep homeostasis and models of sleep regulation. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 3rd ed. Philadelphia, PA: Saunders; 2000. p. 377–90.
16. Dijk DJ, Czeisler CA. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *J Neurosci* 1995;**15**:3526–38.
17. Bootzin RR, Epstein D, Wood JM. Stimulus control instructions. In: Hauri P, editor. *Case studies in insomnia*. New York: Plenum Press; 1991. p. 19–28.
18. James W. *Principles of psychology*, vol. 1. New York: Holt; 1890.
19. Mogg K, Bradley BP. A cognitive—motivational analysis of anxiety. *Behav Res Ther* 1998;**36**:809–48.
20. Williams JMG, Watts FN, MacLeod C, Mathews A. *Cognitive psychology and emotional disorders*. 2nd ed. New York: Wiley; 1997.
* 21. Mathews A, MacLeod C. Cognitive approaches to emotion and emotional disorders. *Annu Rev Psychol* 1994;**45**:25–50.
22. Dalgleish T, Watts FN. Biases of attention and memory in disorders of anxiety and depression. *Clin Psychol Rev* 1990; **10**:589–604.
23. Harvey AG. Attention. In: Harvey AG, Watkins E, Mansell W, Safran R, editors. *Cognitive behavioural processes across psychological disorders: a transdiagnostic approach to assessment and treatment*. Oxford: Oxford University Press; 2004. p. 25–71.
24. Beck AT, Clark DA. An information processing model of anxiety: automatic and strategic processes. *Behav Res Ther* 1997;**35**:49–58.
25. Wells, Mathews G. *Attention and emotion: a clinical perspective*. Hillsdale, NJ: LEA; 1994.
26. Wells. *Emotional disorders and metacognition*. New York: Wiley; 2000.
27. Dalgleish T. Performance on the emotional stroop task in groups of anxious, expert and control participants. *Cognit Emotion* 1995;**9**:341–62.
28. Segerstrom S. Optimism and attentional bias for negative and positive stimuli. *Pers Soc Psychol Bull* 2001;**27**: 1334–43.
29. Stormark KM, Nordby H, Hugdahl K. Attentional shifts to emotionally charged cues: behavioural and ERP data. *Cognit Emotion* 1995;**9**:507–23.
30. Lusher J, Chandler C, Ball D. Alcohol dependence and the alcohol stroop paradigm: evidence and issues. *Drug Alcohol Dependence* 2004;**75**:225–31.
31. Mogg K, Mathews A, Bird C, MacGregor-Morris R. Effects of stress and anxiety on the processing of threat stimuli. *J Pers Soc Psychol* 1990;**59**:1230–7.
32. Drummond DC, Tiffany ST, Glautier S, Remington B. *Addictive behaviour: cue exposure theory and practice*. New York: Wiley; 1995.
33. Shiffrin RM, Schneider W. Controlled and automatic human information processing. II: perceptual learning, automatic attending, and a general theory. *Psychol Rev* 1977;**84**: 127–90.
34. Öhman A, Soares JJF. Unconscious anxiety: phobic responses to masked stimuli. *J Abnorm Psychol* 1994;**103**: 231–40.
35. Maslow. A theory of human motivation. *Psychol Rev* 1943; **50**:370–96.
* 36. Harvey AG. A cognitive model of insomnia. *Behav Res Therapy* 2002;**40**:869–93.
37. Salkovskis PM. The importance of behaviour in the maintenance of anxiety and panic: a cognitive account. *Behav Psychother* 1991;**19**:6–19.
38. Reber AS. An evolutionary context for the cognitive unconscious. *Philos Psychol* 1992;**5**:33–51.
39. Masters RSW, Polman RCJ, Hammond NV. Reinvestment: a dimension of personality implicated in skill breakdown under pressure. *Pers Indiv Differ* 1993;**14**:655–66.
40. Masters RSW, MacMahon KMA, Pall HS. Implicit motor learning in Parkinson's disease. *Rehabil Psychol* 2004;**49**: 79–82.
41. Broomfield NM, Gumley AI. and Espie CA. Candidate cognitive processes in insomnia. *J Cogn Psychother Int Q.* 2005; **19**:3–15.

* The most important references are denoted by an asterisk.

42. Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behav Res Ther* 1985;**23**: 263–71.
43. Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol Ageing* 1993;**8**:463–7.
44. Espie CA, Brooks DN, Lindsay WR. An evaluation of tailored psychological treatment of insomnia. *J Behav Ther Exp Psychiatry* 1989;**20**:143–53.
45. Fichten, Libman E, Creti L, Amsel R, Tagalakis V, Brender W. Thoughts during awake times in older good and poor sleepers: the self statement test: 60+. *Cognit Ther Res* 1998;**22**:1–20.
46. Tang NKY, Harvey AG. Correcting distorted perception of sleep in insomnia: a novel behavioural experiment? *Behav Res Ther* 2004;**42**:27–39.
47. Watts FN, Coyle K, East MP. The contribution of worry to insomnia. *Br J Clin Psychol* 1994;**33**:211–20.
48. Wicklow, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav Res Ther* 2000;**38**:679–93.
49. Harvey KJ, Espie CA. Development and preliminary validation of the glasgow content of thoughts inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *Br J Clin Psychol* 2004;**43**:409–20.
50. Harvey AG. Pre-sleep cognitive activity: a comparison of sleep-onset insomniacs and good sleepers. *Br J Clin Psychol* 2000;**39**:275–86.
51. Neitzert Semler C, Harvey AG. An investigation of monitoring for sleep-related threat in primary insomnia. *Behav Res Ther* 2004;**42**:1403–20.
* 52. Neitzert Semler C, Harvey AG. Monitoring of sleep-related threat in primary insomnia: development and validation of the sleep associated monitoring inventory (SAMI). *Psychosomatic Med* 2004;**66**:242–50.
53. Minto JM, Espie CA. Is presleep cognitive over-arousal epiphenomenal in primary (psychophysiologic) sleep-onset insomnia? Under review.
54. Lack L, Bootzin RR. Circadian rhythm factors in insomnia and their treatment. In: Perlis ML, Lichstein KL, editors. *Treating sleep disorders: principles and practice of behavioral sleep medicine*. New Jersey: Wiley; 2003.
55. Harvey AG, Watkins E, Mansell W, Safran R. *Cognitive behavioural processes across psychological disorders: a transdiagnostic approach to assessment and treatment*. Oxford: Oxford University Press; 2004.
56. Neitzert Semler C, Harvey AG. An experimental investigation of daytime monitoring for sleep-related threat in primary insomnia. *Cognit Emotion*; in press.
57. Neitzert Semler C, Harvey AG. Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? *Behav Sleep Med*. 2006; **4**: 85–103.
58. Tang NKJ, Harvey AG, Schmidt DA. Sleeping with the enemy: clock monitoring in the maintenance of insomnia. *J Behav Ther Exp Psychiatry*; in press.
59. Broman JE, Hetta J. Electrodermal activity in patients with persistent insomnia. *J Sleep Res* 1994;**13**:165–70.
60. Lichstein KL, Fanning J. Cognitive anxiety in insomnia: an analogue test. *Stress Med* 1990;**6**:47–51.
61. Waters WF, Adams SG, Binsk P, Varnado P. Attention, stress and negative emotion in persistent sleep-onset and sleep-maintenance insomnia. *Sleep* 1993;**16**:128–36.
62. MacLeod CM. Half a century of research on the stroop effect: an integrative review. *Psychol Bull* 1991;**120**:3–24.
63. MacLeod CM, Mathews A, Tata P. Attentional bias in emotional disorders. *J Abnorm Psychol* 1986;**95**:15–20.
64. Yiend J, Mathews A. Anxiety and attention to threatening pictures. *Q J Exp Psychol Hum Exp Psychol* 2001;**54**:665–81.
* 65. Lang PJ, Bradley MM, Cuthbert BN. Motivated attention: affect, activation and action. In: Lang PJ, Balabam M, Simons RF, editors. *Attention and orienting: sensory and motivational processes*. Hillsdale, NJ: Earlbaum; 1997.
66. Rensink RA. Change detection. *Annu Rev Psychol* 2002;**53**: 245–77.
67. Simons DJ. Current approaches to change blindness. *Vis Cogn* 2000;**7**:1–15.
68. Lundh L-G, Froding A, Gyllenhammer L, Broman J-K, Hetta J. Cognitive bias and memory performance in patients with persistent insomnia. *Scand J Behav Ther* 1997;**26**:27–35.
* 69. Taylor LM, Espie CA, White CA. A comparative experimental investigation of attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behav Sleep Med* 2003;**1**:200–12.
70. MacMahon KMA, Broomfield NM, Macphee LM, Espie CA. Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome, *Sleep*; in press.
71. Jones BT, Jones BC, Smith H, Copely N. A flicker paradigm for inducing change blindness reveals alcohol and cannabis information processing biases in social users. *Addiction* 2003;**98**:235–44.
* 72. Jones BT, Macphee LM, Broomfield NM, Jones BC, Espie CA. Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. *J Abnorm Psychol* 2005;**114**: 249–58.
* 73. Macphee LM, Biello SM, Broomfield NM, MacMahon KMA, Espie CA. Who is pre-occupied with sleep?: a comparison of attention bias in people with psychophysiologic insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm *J Sleep Res* 2006;**15**: 1–10.
74. Allport DA. Patterns and actions: cognitive mechanisms are content specific. In: Claxton GL, editor. *Cognitive psychology: new directions*. London: Routledge & Kegan Paul; 1980. p. 26–64.
75. Broadbent. *Perception and communication*. London: Pergamon Press; 1958.
76. Deutsch J, Deutsch D. Attention: some theoretical considerations. *Psychol Rev* 1963;**70**:80–90.
77. Posner MI. Orienting of attention. *Q J Exp Psychol* 1980;**32**: 3–25.
78. Masters WH, Johnson VE. *Human sexual response*. New York: Lippincott/Williams & Wilkins; 1966.
79. Heiman JR, Meston CM. Empirically validated treatments for sexual dysfunction. In: Dobson KS, Craig KD, editors. *Empirically supported therapies: best practice in professional psychology*. New York: Sage Publications; 1998. p. 259–303.
80. Seltzer LF. *Paradoxical strategies in psychotherapy*. New York: Wiley; 1986.
81. Strong SR. Experimental studies in explicitly paradoxical interventions: results and implications. *J Behav Ther Exp Psychiatry* 1984;**15**:189–94.
82. Bastuji H, Garcia-Larrea L. Evoked potentials as a tool for the investigation of human sleep. *Sleep Med Rev* 1999;**3**: 23–45.
83. Thacher PV, Pigeon WR, Perlis ML. Do patients with sleep maintenance problems have a problem with sleep maintenance. *Behav Sleep Med*; in press.
84. Chambers MJ, Keller B. Alert insomniacs: are they really sleep deprived? *Clin Psychol Rev* 1993;**13**:649–66.

85. Stepanski EJ, Zorick F, Peters M, Roth T. Effects of sleep deprivation on alertness in chronic insomnia. *Sleep Res* 1990;**19**:297.
86. Mitler MM, Carskadon MA, Hirshkowitz M. Evaluating sleepiness. In: Kryger MH, Roth T, W.C. Dement, editors. *Principles and practice of sleep medicine*. 4th ed. Philadelphia, PA: Saunders; 2005.
87. Ascher LM, Turner RM. Paradoxical intention and insomnia: an experimental investigation. *Behav Res Ther* 1979;**17**: 408–11.
88. Espie CA, Lindsay WR. Paradoxical intention in the treatment of chronic insomnia: six case studies illustrating variability in the therapeutic response. *Behav Res Ther* 1985;**23**:703–9.
89. Espie CA. *The psychological treatment of insomnia*. Chichester: Wiley; 1991.
90. Ascher LM, Turner RM. A comparison of two methods for the administration of paradoxical intention. *Behav Res Ther* 1980;**18**:121–6.
91. Fogle DO, Dyall JA. Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. *Psychother Theory Res Pract* 1983;**20**:21–30.
92. Morin CM, Espie CA. *Insomnia: a clinical guide to assessment and treatment*. New York: Kluwer/Plenum Publishers; 2003.
93. Turner RM, Ascher LM. Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. *J Consult Clin Psychol* 1979;**47**: 500–8.
94. Espie CA, Lindsay WR, Brooks DN, Hood EM, Turvey T. A controlled comparative investigation of psychological treatments of chronic sleep-onset insomnia. *Behav Res Ther* 1989;**27**:79–88.
95. Morin CM, Hauri P, Espie CA, Spielman A, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia: an American academy of sleep medicine review. *Sleep* 1999;**22**:1134–56.
96. Chesson AL, Anderson WM, Littner M, Davila D, Hartse K, Johnson S, et al. Practice parameters for the nonpharmacologic treatment of chronic insomnia. *Sleep* 1999;**22**: 1128–33.
97. Gross RT, Borkovec TD. Effects of a cognitive intrusion manipulation on sleep-onset latency of good sleepers. *Behav Ther* 1982;**13**:117–24.
98. Wegner DM. Ironic processes of mental control. *Psychol Rev* 1994;**101**:34–52.
99. Harvey AG. Attempted suppression of pre-sleep cognitive activity in insomnia. *Cognit Ther Res* 2001;**27**:593–602.
100. Harvey AG. Beliefs about the utility of pre-sleep worry: an investigation of individuals with insomnia and good sleepers. *Cognit Ther Res* 2003;**27**:403–14.
101. Harvey AG. I cant sleep, my mind is racing! an investigation of strategies of thought control in insomnia. *Behav Cognit Psychother* 2001;**29**:3–11.
102. Ellis J, Cropley M. An examination of thought control strategies employed by acute and chronic insomniacs. *Sleep Med* 2002;**3**:293–300.
103. Lundh L-G, Hindmarsh H. Can metacognitive observation be used in the treatment of insomnia? A pilot study of a cognitive-emotional self-observation task. *Behav Cognit Psychother* 2002;**30**:233–6.
104. Wingate M. *Foundations of stuttering*. New York: Academic Press; 2002.
105. Wegner DM, Broome A, Blumberg SJ. Ironic effects of trying to relax under stress. *Behav Res Ther* 1997;**35**:11–21.
106. Andrews G, Guitar B, Howie P. Meta-analysis of the effects of stuttering treatment. *J Speech Hear Disord* 1980;**45**: 287–307.
107. Hayes SC, Strosahl KD, Wilson KG. *Acceptance and commitment therapy: an experiential approach to behavior change*. New York: Guilford Press; 1999.
108. Segal ZV, Williams MG, Teasdale JD. *Mindfulness-based cognitive therapy for depression: a new approach to preventing relapse*. New York: Guilford Press; 2001.
109. Lundh L-G, Broman JE, Hetta J, Saboonchi F. Perfectionism and insomnia. *Scand J Behav Ther* 1994;**23**:3–18.
110. Broomfield NM, Espie CA. Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behav Cognit Psychother* 2003; **31**:313–24.
111. Morin CM. *Insomnia: psychological assessment and management*. New York: Guilford Press; 1993.
112. Espie CA, Inglis SJ, Harvey L, Tessier S. Insomniacs' attributions: psychometric properties of the dysfunctional beliefs and attitudes about sleep scale and the sleep disturbance questionnaire. *J Psychosom Res* 2000;**48**: 141–8.
*113. Broomfield NM, Espie CA. Toward a valid, reliable measure of sleep effort. *J Sleep Res* 2005;**14**:401–7.
114. Kohn L, Espie CA. Sensitivity and specificity of measures of the insomnia experience: a comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. *Sleep* 2005;**28**:104–12.
115. Moul DE, Nofzinger EA, Pilkonis PA, Houck PR, Miewald JM, Buysse DJ. Symptom reports in severe chronic insomnia. *Sleep* 2002;**25**:553–63.
116. Broman JE, Hetta J. Perceived pre-sleep arousal in patients with persistent psychophysiologic insomnia and psychiatric insomnia. *Nord J Psychiatry* 1994;**48**:203–7.
117. Riemann, Voderholzer U. Primary insomnia: a risk factor to develop depression? *J Affect Disord* 2003; **76**:255–9.
118. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. *Am J Psychiatry* 2003;**160**:1147–56.
119. Bradley BP, Mogg K, Lee S. Attentional biases for negative information in induced and naturally occurring dysphoria. *Behav Res Ther* 1997;**35**:911–27.
120. Posner MI, Peterson SE. The attention system of the human brain. *Annu Rev Neurosci* 1990;**13**:25–42.
121. Posner MI, Petersen SE, Fox, Raichle ME. Localisation of cognitive operations in the human brain. *Science* 1988;**240**: 1627–31.
122. Broomfield NM, Turpin G. Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. *Biol Psychol* 2005;**68**:179–200.
123. Fox, Russo R, Bowles R, Dutton K. Do threatening stimuli draw or hold attention in sub clinical anxiety? *J Exp Psychol Gen* 2001;**130**:681–700.
124. Fox, Russo R, Dutton K. Attentional bias for threat: evidence for slowed disengagement from emotional faces. *Cognit Emotion* 2002;**16**:355–79.
125. Mathews, Mogg K, Kentish J, Eysenck M. Effects of psychological treatment on cognitive bias in generalised anxiety disorder. *Behav Res Ther* 1995;**33**:293–303.
126. Mogg K, Bradley BP, Millar N, White J. Cognitive bias in generalised anxiety disorder: a follow up study. *Behav Res Ther* 1995;**33**:927–35.
127. Wells, White J, Carter K. Attention retraining: effects on anxiety and beliefs in panic and social phobia. *Clin Psychol Psychother* 1997;**4**:226–32.

128. Cook EW, Turpin G. Differentiating orienting, startle and defense responses: the role of affect and its implications for psychopathology. In: Lang PJ, Balabam M, Simons RF, editors. *Attention and orienting: sensory and motivational processes*. Hillsdale, NJ: Erlbaum; 1997.
129. Perlis ML, Smith MT, Orff HJ, Andrews PJ, Giles DE. Beta/gamma activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep* 2001;**24**:110–7.
130. Devoto A, Vialano C, Lucidi F, Lombardo C. P300 amplitude in subjects with primary insomnia is modulated by their sleep quality. *J Psychosom Res* 2003;**54**:3–10.
131. Devoto A, Mangelli S, Lucidi F, Lombardo C, Russo PM, Violani C. Quality of sleep and P300 amplitude in primary insomnia: a preliminary study. *Sleep* 2005;**28**:863–9.
132. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The pittsburgh sleep quality index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;**28**:193–213.
133. Langford GW, Meddis R, Pearson AJD. Awakening latency from sleep for meaningful and non-meaningful stimuli. *Psychophysiology* 1974;**11**:1–6.
134. Akerstedt T, Billiard M, Bonnet M, Ficca G, Garma L, Mariotti M, et al. Awakening from sleep. *Sleep Med Rev* 2002;**6**:267–86.