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Original article

Understanding insomnia through cognitive modelling

Colin A. Espie*

University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research, Glasgow, United Kingdom

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Abstract

Cognitive models of insomnia have received growing support in recent years and are embraced by the current diagnostic framework. Many people with insomnia report that mental events, such as intrusive thoughts or a racing mind, prevent them from achieving or maintaining sleep. Dysfunctional cognition may play an important role in perpetuating insomnia, with many individuals with psychophysiological insomnia reporting a distorted perception of sleep. Neurocognitive studies have indicated that high-frequency EEG activity associated with cognitive processes is enhanced in patients with insomnia at or around sleep onset, which may distort the individual's judgement about sleep initiation and duration. A subtype of psychophysiological insomnia has been proposed – attention–intention–effort (AIE) syndrome – that takes into consideration the interaction between behavioral and cognitive factors in the development and maintenance of insomnia. A series of studies from the University of Glasgow Sleep Centre using cognitive probe tasks has provided insight into this pathway, particularly with regard to the role of attention bias towards sleep stimuli in mediating insomnia. Further research is required to explore the cortical correlates of attention bias, investigate AIE as a potential causal mechanism of insomnia and examine AIE in other insomnia groups.
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Keywords: Psychophysiological insomnia; Cognitive factors; Attention–intention–effort pathway

Insomnia is common[1] as both a disorder in its own right (primary insomnia) and as a symptom of other disorders (comorbid insomnia). It has been associated with impaired concentration and memory, reduced ability to perform daily tasks and decreased enjoyment of interpersonal relationships.[2] In addition, insomnia may play a causal role in the development of other psychopathological conditions, notably depression and anxiety.[3]

The underlying basis for insomnia has been attributed to physiological,[4] cognitive[5-7] and behavioral[8,9] elements. While it is likely that all three of these elements may play a role in the initiation or maintenance of insomnia, cognitive models have received growing support in recent years.

This review will evaluate the evidence from cognitive models of insomnia and will consider further avenues for researching aspects of this condition.

Why is a cognitive model of insomnia relevant?

Many people with insomnia report that mental events prevent them from sleeping.[6,10,11] Such cognitive activity can include uncontrollable worry, intrusive thoughts and an overly active or 'racing' mind. Furthermore, because 'normal' sleep is a relatively automatic, subconscious process that cannot be forced by will, it is vulnerable to disruption by patients consciously thinking about sleep and by direct attempts to control the process.

A cognitive model of insomnia is embraced by the current diagnostic framework. Indeed, the essential feature of psychophysiological insomnia (also referred to as conditioned or learned insomnia) is heightened arousal and learned sleep-preventing associations, according to the 2nd edition of the International Classification of Sleep Disorders.[12] Cognitive aspects of psychophysiological insomnia include dysfunctional thinking, heightened anxiety about sleep, cortical arousal, sleep misperception, automaticity, and attentional processing.

The natural history of persistent insomnia has been presented by Spielman as the end-stage of a series of predisposing, precipitating and perpetuating factors that

* Tel.: +44 141 232 7700; fax: +44 141 232 7697
E-mail address: c.espie@clinmed.gla.ac.uk

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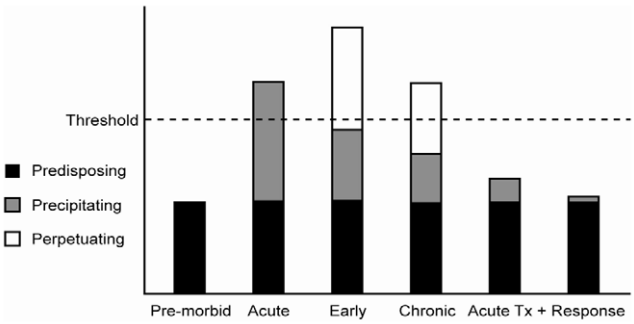

Fig. 1. The natural history of insomnia. Adapted from Spielman 1991.[13] Springer and Kluwer Academic/Case Studies in Insomnia, 1991. p1-15, The Varied Nature of Insomnia, Spielman AJ, Glovinsky PB, Figure 2, Copyright 2007, with kind permission from Springer Science and Business Media. Tx = treatment.

may push an individual over the threshold required for effective sleep (Figure 1).[13] For example, a predisposing factor could be a susceptibility to anxiety, a precipitating factor could be a promotion at work to a position with added responsibility and a perpetuating factor could be anxiety over daytime deficits or expectation of a poor sleep. This model shows that insomnia develops through acute, early and chronic stages, each with differing proportions of predisposing, precipitating and perpetuating components.

Understanding the etiology of insomnia has a considerable impact on the selection of a therapy and its subsequent effectiveness. There is now substantial evidence that psychophysiological insomnia can be successfully treated with a range of psychological interventions including cognitive–behavioral therapy (CBT). CBT is based on the concept that cognition, emotion and behavior all interact and that maladaptive thoughts can cause negative feelings which can result in changes in behavior. The cognitive component of CBT addresses such negative thought processes, and in turn promotes more sleep-promoting behavior. Likewise, behavior influences thought processes. The behavioral component of CBT addresses unhelpful behaviors to make them more adaptive, and so provides evidence that helps to alter dysfunctional thinking. Using the Spielman model of insomnia,[13] it is possible to understand the role of CBT in the treatment plan of an individual with insomnia. CBT produces a response primarily by removing the perpetuating factors, and secondarily by addressing potential precipitating components.

Limitations of assessment of psychophysiological insomnia and cognition

While it is evident that insomnia has a psychophysiological basis in many individuals, it is unclear exactly how it develops and what factors are crucial to its maintenance. One particular problem is that the insomnia phenotype lacks objective markers. This is especially true for the important domain of cognitive arousal. Ideally, just as sleep itself can be measured in the laboratory and by subjective experience, cognitive factors associated with insomnia would be measurable using both self-report and objective, performance-based tests. Self-report instruments which yield scores for the mental and somatic components of the pre-sleep state do exist (e.g. Pre-Sleep Arousal Scale).[14] It is proposed at a later point in this paper that computerized testing of information-processing bias may offer an objective means of appraising mental processes in insomnia.

A cognitive perspective on insomnia

Dysfunctional beliefs and attitudes about sleep are presumed to play an important mediating role in perpetuating insomnia. Morin[15] devised an integrative model in which insomnia is viewed as a result of dynamic interaction between dysfunctional cognition, maladaptive behavior (or habits), concerns about the consequences of lack of sleep (mood, fatigue, performance), and arousal (emotional, cognitive, physiological). The role of dysfunctional cognition in the perpetuation of insomnia has been supported by a number of studies. For example, in an investigation of the impact of CBT and pharmacological treatments for chronic primary insomnia on sleep-related beliefs and attitudes, CBT, alone or in combination with pharmacotherapy, resulted in greater improvements on the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale at post-treatment than pharmacotherapy alone or placebo.[16] Interestingly, improvements in sleep efficiency (measured by sleep diaries and polysomnography) were significantly correlated with reductions in DBAS scores, and the presence of more adaptive beliefs and attitudes about sleep following treatment was associated with better maintenance of sleep improvements at the 12- and 24-month follow-ups. Furthermore, experimental manipulation of pre-sleep stress by Haynes and colleagues have shown that insomnia can be induced in normal sleepers by exposure to cognitive stressors.[17] In this study, 11 normal sleepers were asked to perform arithmetic problems once they had gone to bed and lights had been turned off. Compared with when they were allowed to sleep undisturbed, these individuals experienced significant increases in subjective and objective sleep latency. Interestingly, when 10 individuals with sleep-onset insomnia were exposed to the same cognitive problems, sleep latency decreased. This may be because the experimental manipulation allowed disruption or distraction from normal pre-sleep cognitive stressors (such as ruminative cognition or concerns about sleep) in those with insomnia, thus allowing faster onset of sleep.

Another cognitive model of insomnia has been proposed by Lundh and Broman.[18] In their view, sleep-interfering psychological processes (such as arousability, slow recuperation after stress, worry and emotional conflicts) and sleep-interpreting psychological processes (such as sleep-related beliefs, attitudes and perfectionist standards) are involved in an integrative fashion in the development and

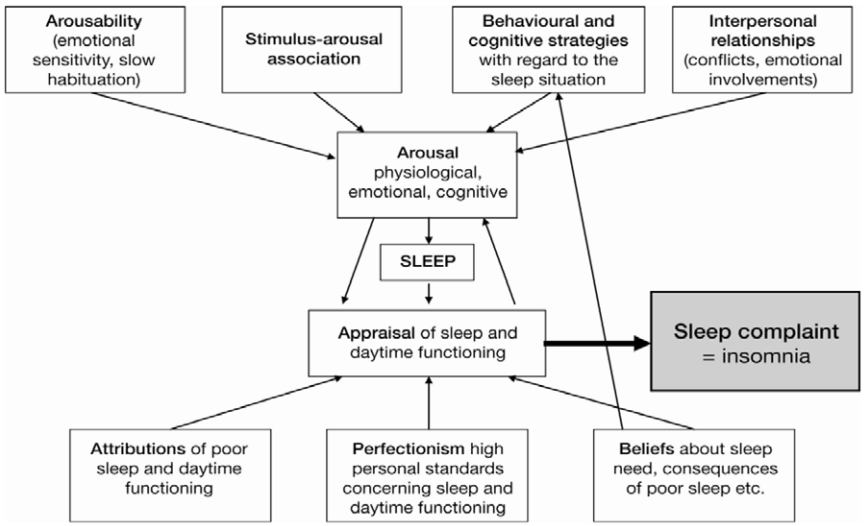

Fig. 2. Lundh and Broman's model of sleep-interfering and sleep-interpreting processes.[18] Reprinted from Journal of Psychosomatic Research, Vol 49, Lundh LG, Broman JE, Insomnia as an interaction between sleep-interfering and sleep-interpreting processes, p299-310, Copyright 2007, with permission from Elsevier.

maintenance of insomnia. Using data from empirical studies to develop a theoretical model, Lundh and Broman suggest that psychological vulnerability may predispose an individual to respond to stress with sleep-interfering processes and to engage in dysfunctional sleep-interpreting processes (Figure 2). The extent to which any of these processes interfere with sleep may explain variations in the presentation of insomnia and the requirement for different treatment strategies.

While anxiety may not be a requisite part of insomnia,[19] a model proposed by Harvey[7] suggests that people with insomnia tend to be worried about sleep and the consequences of lack of sleep. This triggers autonomic arousal and emotional distress or anxiety that results in a distortion of the perception of sleep deficit as well as counter-productive behavior. Escalating anxiety can eventually culminate in a real deficit in sleep and daytime functioning, and a distorted perception of sleep can also have an impact on sleeping patterns. For example, in a study designed to correct distorted perceptions of sleep in patients with primary insomnia, some patients were shown the discrepancy between their self-reported and objective sleep data. Patients shown the discrepancy then estimated their sleep more accurately and reported less anxiety and preoccupation about sleep than those not shown the discrepancy.[20]

A neurocognitive perspective on insomnia

However, insomnia is not merely a psychological disorder of misperception. Perlis and colleagues have demonstrated neurophysiological differences between insomnia and normal sleep that may help to explain the experiential report of the person with psychophysiological insomnia. In their neurocognitive model it is proposed that, in patients with insomnia, high-frequency EEG activity

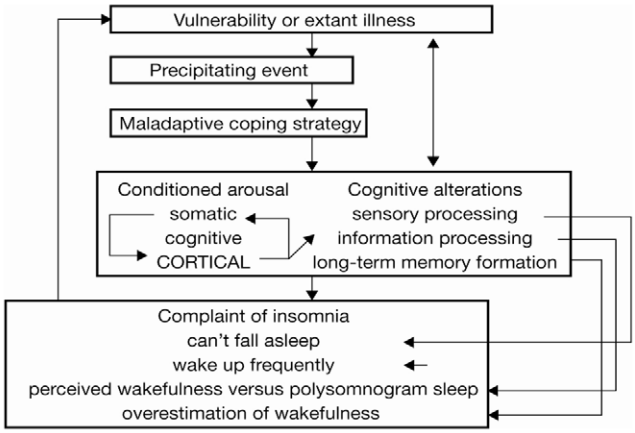

Fig. 3. Neurocognitive perspective on insomnia. Adapted from Perlis et al 1997.[21] Reprinted from Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt JK. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J Sleep Res 1997;6:179-88, with permission from Blackwell Publishing Ltd.

associated with cognitive processes is enhanced at or around sleep onset,[21] a phenomenon that has been observed in a number of other studies.[22-30] This cortical arousal may blur the distinction between sleep and wakefulness, and so distort the patient's judgment about sleep initiation and duration (Figure 3).[21] Furthermore, this activity suggests that the individual with insomnia may be sensory and information processing or forming long-term memories during the peri-sleep period – processes which are generally attenuated at this time in the normal sleeper.[21] Consistent with this hypothesis, a recent study comparing EEG event-release potentials (ERPs) relating to attention and sleepiness in patients with chronic primary insomnia and good sleepers showed that patients with insomnia had a lower ERP relating to sleepiness in the evening and a higher ERP

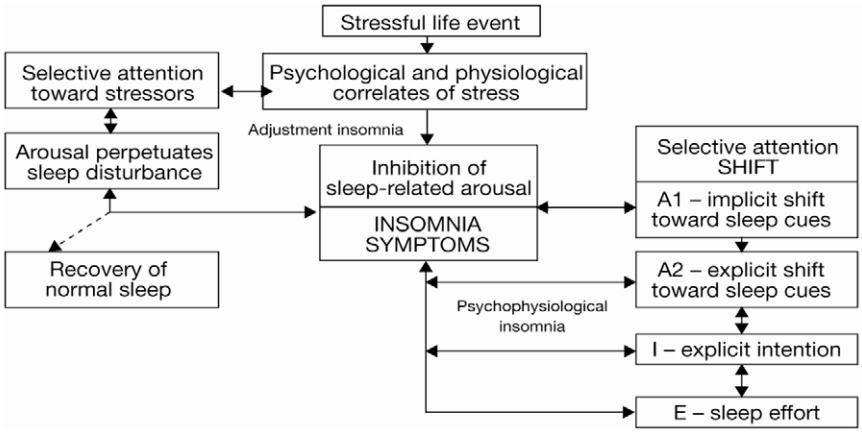

Fig. 4. Model of the attention–intention–effort pathway.[35] Reprinted from Sleep Medicine Reviews, Vol 10, Espie CA, Broomfield NM, MacMahon KM, Macphee LM, Taylor LM, The attention-intention-effort pathway in the development of psychophysiologic insomnia: a theoretical review, p215-45, Copyright 2007, with permission from Elsevier.

relating to attention in the morning.[31] This suggests that both an inability to initiate normal sleep processes and higher cortical activation might interfere with sleep in individuals with insomnia.

Brain imaging studies have also provided interesting insight into the neurocognitive basis for insomnia. Nofzinger and colleagues characterized the functional–anatomical correlates of β-EEG activity during nonREM sleep in normal sleepers using concurrent EEG sleep studies and positron emission tomography scans. The study showed that β-EEG activity is inversely correlated with sleep quality, but correlated with ventromedial prefrontal cortex metabolism, suggesting that the ventromedial prefrontal cortex plays a significant role in mediating one aspect of dysfunctional arousal.[32] Many other areas of the brain have also been shown to have overactive arousal systems (hyperarousal).[33] Thus, the inability to fall asleep may be related to a failure of the usual deactivation of arousal mechanisms during the transitional phase between wakefulness and sleep.

The attention–intention–effort (AIE) pathway to psychophysiological insomnia

The findings of EEG studies in insomnia are consistent with Espie's psychobiological inhibition model of insomnia, in which insomnia is viewed as a result of inhibition of de-arousal processes associated with normal sleep.[34] Sleep homeostatic and circadian factors are compromised by impairment of the automaticity and plasticity associated with good sleep and those cognitive–affective processes activate the clinical complaint of insomnia. Central to this model is a contrast between sleep normalcy and insomnia. The cognitive differences between normal sleep and psychophysiological insomnia are shown in Table 1. Essentially, it is suggested that good sleep is a function of an unimpeded de-arousal process that permits homeostatic and circadian imperatives to engage sleep automatically. Insomnia, on the other, hand may be a product of heightened arousal associated with attempts deliberately to engage sleep.

Espie's proposed AIE pathway (Figure 4) is a more detailed articulation of the psychobiological inhibition model.[35] Normal sleep may be a relatively involuntary psychophysiological process, but one that is vulnerable to, and has the potential to be compromised by, focused attention and direct attempts to control its expression. This pathway then suggests that persistent insomnia is a product of selective attention bias to sleep, leading to active intention to sleep, and ultimately to what amounts to a sleep effort syndrome, where there is pervasive sleep pre-occupation and a host of mental and behavioral strategies are used with the aim of inducing sleep.

While evidence for the AIE pathway is still relatively limited, the University of Glasgow Sleep Centre has conducted a series of studies that have investigated this model. Broomfield and Espie conducted a validation study of the Glasgow Sleep Effort Scale (GSES), which demonstrated that "effortful preoccupation with sleep" was able to distinguish those with psychophysiological insomnia from good sleepers with 100% sensitivity and 94% specificity.[36] Kohn and Espie also investigated the sensitivity and specificity of commonly used insomnia research tools in discriminating psychophysiological insomnia, insomnia associated with mental disorders, and good sleepers. The GSES emerged as the best tool to separate the groups, suggesting that sleep effort is indeed a useful construct. Furthermore, results indicated that psychophysiological insomnia and insomnia associated with mental disorders may exist on a continuum of insomnia severity, rather than being categorically distinct.[37]

The Glasgow group have also pioneered the use of computerized cognitive probe tasks to index the AIE pathway. In their first study, Taylor and colleagues evaluated the role of attention bias in the development of acute insomnia or persistent insomnia associated with cancer.[38]

Table 1

Cognitive differences between normal sleep and psychophysiological insomnia. Adapted from Espie 2002.[34]

Normal sleep	Psychophysiological insomnia
Minimal attention	Selective attention to sleep, sleeplessness and sleep consequences
Minimal intention	Activated intention to sleep, purposive
Minimal effort	Sleep effort, both direct and indirect
Minimal concern	Sleep preoccupation, persistent insomnia complaint

Using the computerized emotional Stroop task, individuals were shown 20 sleep-related words, 20 cancer-related words and 40 neutral words. While both groups demonstrated an attention bias towards the cancer-related words, only those with persistent insomnia had developed an attention bias towards sleep-related words. These findings indicate that secondary, sleep-related mental preoccupation may be associated with the transition from acute to persistent insomnia. Potentially, attention bias may also contribute to the perpetuation and possibly precipitation of primary insomnia. Using the induced-change blindness (ICB) paradigm, Jones and colleagues likewise demonstrated sleep-related attention bias in those with primary insomnia but not in good sleepers.[39] On this task, patients with insomnia were more likely to have an attention bias towards a representative bedroom object than a neutral object, while good sleepers did not demonstrate any notable difference between object categories. Marchetti and colleagues also compared attention bias in good sleepers and people with psychophysiological insomnia or delayed sleep phase syndrome using the ICB task[40] and so demonstrated some specificity of attention bias to insomnia in particular. Similar results were obtained in a further comparison of insomnia, delayed sleep phase syndrome and good sleepers on the Dot Probe task.[41] Interestingly, another study by Marchetti suggested that the attention bias to sleep-related stimuli detected in patients with psychophysiological insomnia may be driven by 'threat'.[42] Using a modified Posner paradigm that involved intrinsically threatening sleep words (e.g. fatigue), non-threatening sleep words (e.g. pillow) and neutral words, those with psychophysiological insomnia showed a differential response to threatening words than non-threatening or neutral words. In other words, psychophysiological insomnia patients were slower to disengage their attention from threatening sleep words than neutral words.

It is evident that there is still much of the AIE pathway that remains to be validated; nevertheless, these initial probe studies provide interesting insight into the potential role of selective attention bias in driving psychophysiological insomnia, and these paradigms offer an objective means of indexing cognitive processes in insomnia.

Conclusion

Psychophysiological insomnia may be a primary disorder caused by the inhibition of sleep automaticity and, at the very least, there may be a subtype of insomnia – AIE syndrome. Objective measures of attentional processing using cognitive probe tasks suggest there is selective attention bias in psychophysiological insomnia relative to both normal sleepers and those with delayed sleep phase syndrome. Further studies are required to explore the cortical correlates of attention bias, to investigate AIE as a potential causal mechanism, and to explore AIE in other insomnia groups. Future research should also include studies to further define the psychophysiological insomnia phenotype; assessment of methodologies to study cognitive arousal; prospective studies to investigate the natural history of psychophysiological insomnia; experimental manipulation studies to further elucidate mechanisms; consideration of the chronobiological perspective in insomnia; and integration of brain, systemic and psychological models. As our understanding of this debilitating condition grows, so too should our ability to treat it effectively.

Conflict of interest

The author has declared no conflict of interest.

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