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Sleep Medicine 8 Suppl. 4 (2007) S3–S8

SLEEP MEDICINE

www.elsevier.com/locate/sleep

Original article

Understanding insomnia through cognitive modelling

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Available online January 2008

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.

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

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doi: 10.1016/j.sleep.2007.04.011

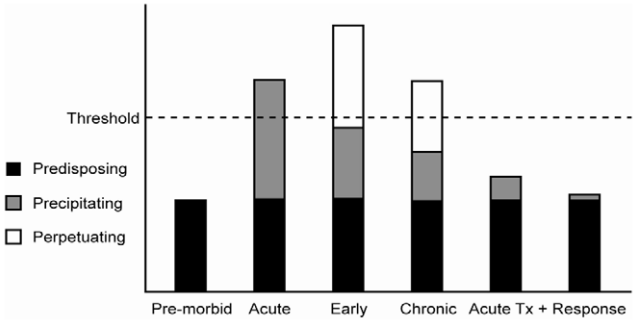

Fig. 1. The natural history of insomnia. Adapted from Spielman 1991.[13] Springer and Kluwer Academic/Case Studies in Insomnia, 1991. p1-15, The Varied Nature of Insomnia, Spielman AJ, Glovinsky PB, Figure 2, Copyright 2007, with kind permission from Springer Science and Business Media. Tx = treatment.

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