



## Improving sleep with mindfulness and acceptance: A metacognitive model of insomnia

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### ABSTRACT

While there is an accumulating evidence to suggest that therapies using mindfulness and acceptance-based approaches have benefits for improving the symptoms of insomnia, it is unclear how these treatments work. The goal of this paper is to present a conceptual framework for the cognitive mechanisms of insomnia based upon mindfulness and acceptance approaches. The existing cognitive and behavioral models of insomnia are first reviewed and a two-level model of cognitive (primary) and metacognitive (secondary) arousal is presented in the context of insomnia. We then focus on the role of metacognition in mindfulness and acceptance-based therapies, followed by a review of these therapies in the treatment of insomnia. A conceptual framework is presented detailing the mechanisms of metacognition in the context of insomnia treatments. This model proposes that increasing *awareness* of the mental and physical states that are present when experiencing insomnia symptoms and then learning how to *shift* mental processes can promote an adaptive *stance* to one's response to these symptoms. These metacognitive processes are characterized by balanced appraisals, cognitive flexibility, equanimity, and commitment to values and are posited to reduce sleep-related arousal, leading to remission from insomnia. We hope that this model will further the understanding and impact of mindfulness and acceptance-based approaches to insomnia.

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Novel treatments using mindfulness and acceptance-based approaches are a wave of rapidly emerging therapies. Many treatment studies and meta-analyses have demonstrated the benefits of these therapies on mental and physical health. A subset of these studies has examined the impact of mindfulness and acceptance-based therapies on sleep disturbance, collectively referred to here as metacognitive therapies for insomnia (MCTI). While there is an accumulating evidence to suggest that MCTIs have benefits for improving sleep, a conceptual model to explain their potential common mechanisms is lacking.

This paper proposes a conceptual framework for the cognitive mechanisms of insomnia based upon mindfulness and acceptance approaches. First, we review the existing cognitive and behavioral models of insomnia and present a re-conceptualization of sleep-related arousal to include metacognitive processes in addition to the cognitive processes that have been previously described. Second, we examine the literature on mindfulness and acceptance

which has identified metacognitive processes as a potential target for emotion regulation and reducing second order distress. Third, we present a treatment model to explain how MCTIs target secondary distress and lead to remission from an insomnia disorder. Studies that support psychopathological processes relevant to the components of the model are presented along with clinical implications for assessment and intervention. Finally, we discuss potential implications of this model and offer an agenda for future research. We hope that these concepts provide a framework that can stimulate empirical testing of the treatment mechanisms in MCTIs.

### Cognitive and behavioral models of insomnia: a historical context

Conceptualizations of insomnia have consistently focused on the notion that sleep disruptions are caused by elevated arousal. It has even been argued that insomnia is a disorder of hyperarousal that causes sleep disruption (Bonnet & Arand, 1997). Consequently, the early wave of behavioral treatments, delivered as a single component, had behavioral targets that directly or indirectly

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reduced arousal. Examples include progressive muscle relaxation (Jacobson, 1938) and biofeedback (Hauri, 1981) to reduce physiological arousal, paradoxical intention for reducing arousal that stems from performance anxiety (Ascher & Efran, 1978; Ascher & Turner, 1979; Turner & Ascher, 1979), stimulus control for reducing a conditioned arousal response to the bed (Bootzin, 1972), and sleep restriction therapy (Spielman, Saska, & Thorpy, 1987), which restricts time in bed to mobilize the homeostatic drive to sleep, thus offsetting hyperarousal. Integrating these concepts into a diathesis–stress–response model, Spielman, Caruso, and Glovinsky (1987) conceptualized chronic insomnia as involving predisposing, precipitating, and perpetuating factors. Perpetuating factors include behaviors that individuals enact in an effort to cope with sleep disturbances (e.g., staying in bed to gain more sleep), but which inadvertently create arousal and perpetuate the problem.

Emerging from a focus on cognitive arousal, the second wave of treatments targeted sleep-related cognitions. Investigation into sleep interfering cognitions revealed that people with insomnia hold maladaptive beliefs and attitudes about sleep (Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). Based on these findings, Morin presented an integrated conceptualization of insomnia, whereby emotional, cognitive, and physiologic hyperarousal were identified as the central mediating features of insomnia (Morin, 1993). He posited that both maladaptive behaviors and arousal-producing cognitions are dysfunctional responses to poor sleep and daytime fatigue and that these responses maintain the disorder by creating a vicious cycle of insomnia (Fig. 1). Morin proposed to add cognitive therapy strategies to the existing behavioral interventions in order to help patients modify their dysfunctional beliefs and attitudes about sleep. He emphasized targeting two common cognitive errors among individuals with insomnia – catastrophizing and probability over estimation (Morin, 1993). This combination of cognitive and behavioral components is now known as

cognitive–behavior therapy for insomnia (CBTI) and includes stimulus control, sleep restriction, education about sleep hygiene, and cognitive therapy to address cognitive errors.

More recently, Espie (2002) has proposed a psychobiological inhibition model of insomnia that centers on the failure to de-arouse. In his model, a central process in insomnia is the loss of automaticity (i.e., sleep comes effortlessly) and plasticity (i.e., sleep is flexible) which are characteristics of good sleepers. Good sleepers are able to de-arouse at night, which facilitates the process of falling asleep. In contrast, people with insomnia have selective attention for sleep-related cues, make explicit intention to sleep, and have increased effort to sleep. This approach to sleep is maladaptive. It leads to a failure to de-arouse at night and thus inhibits the homeostatic and circadian processes that regulate sleep. Espie's model also considers how daytime factors could contribute to insomnia. For example, inaccurate attributions of impaired daytime functioning to poor sleep may increase sleep effort and lead to performance anxiety and worse sleep. Similar to Espie, Harvey (2002) applied cognitive theories of anxiety to a model of insomnia that recognizes cognitive processes at night and during the day that could contribute to increased arousal and interfere with sleep (see Fig. 2). These include worry, selective attention monitoring, misperception of sleep, unhelpful beliefs, and safety behaviors. Moreover, Harvey posits that these processes can occur with or without real deficits in sleep or daytime functioning but can lead to actual deficits in sleep and daytime function through increased arousal. Harvey has proposed and tested the use of “behavioral experiments” as a way to help people with insomnia test these sleep-related arousal inducing cognitions (Harvey, Sharpley, Ree, Stinson, & Clark, 2007). Maintaining a focus on the cognitive processes of insomnia, Lundh and Broman (2000) have proposed a theoretical model based on two cognitive processes: sleep-interfering processes and sleep-interpreting processes. They

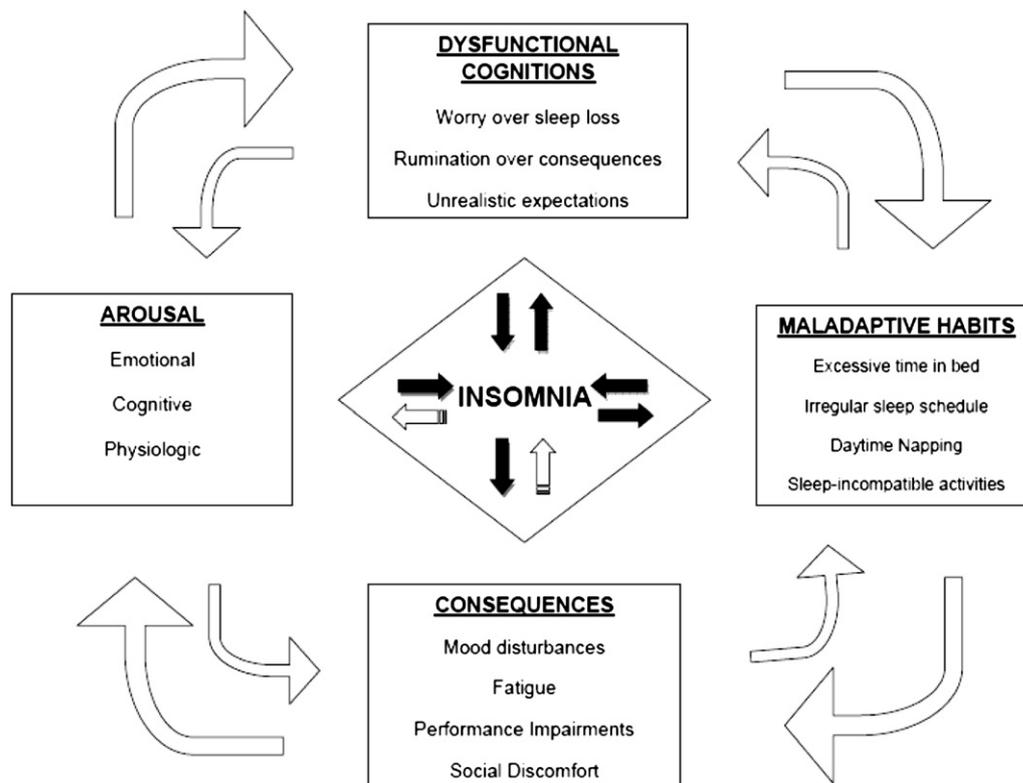


Fig. 1. Morin's cognitive–behavioral model of insomnia, depicting the role of arousal, cognitions, behaviors, and perceived consequences in creating a cycle of insomnia.

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