Supplementary Materials

Table S1 – Commonly used models of insomnia.

These models are not mutually exclusive but complementary.

Mechanism	Description	Clinical implications
3P model[1]	Insomnia generated acutely by precipitating stressful factors, in the setting of predisposing physiologic or cognitive-behavioral factors, and becomes chronic through perpetuating cognitive-behavioral factors.	Helpful simplified model for implementing a clinical approach in identifying and treating primarily precipitating and perpetuating factors of insomnia. Basis for most other comprehensive models.
4P model	Extension of the 3P model by introducing classical conditioning wherein perpetuating behaviors lead to conditioned physiologic responses of arousal.	Adds a theoretical basis of treatment by dissociating specific stimuli from conditioned responses.
Microanalytic model[2]	Insomnia presented at the junction of four factors (a) arousal mechanisms, (b) consequences of insomnia, (c) dysfunctional cognitions, and (d) maladaptive behaviors, and their bidirectional relationship, which in turn accentuate insomnia symptoms through positive feedback loops.	Highlights the need to intervene in order to break the positive feedback of perpetuating symptoms. It further allows multi-component treatment approaches to be considered on all four factors.
Neurocognitive model[3]	Extension of the 3P/4P models, adding cortical arousal as a core factor contributing to insomnia and supplemented by cognitive and physical/somatic factors. It further weakens the link between hyperarousal on one hand and sleep onset and maintenance insomnia on the other, the latter accentuated by increased somatic and cognitive processing.	Facilitates the identification of specific behaviors and thought processes leading to cortical arousal and allows for explanation of sleep state misperception secondary to increase sensory and cognitive processing during NREM sleep, as observed in paradoxical insomnia.

Two factor model[4]	Simplified model where symptoms emerge along two orthogonal axes: sleep requirement and baseline arousal level.	Explains idiopathic and psychophysiological insomnia (high arousal, short sleep requirement), in contrast to paradoxical insomnia (high arousal, long sleep requirement), and further explains states of hypersomnia for low arousal states.
Sleep interfering-interpreting model[5]	Comprehensive two factor model wherein disturbance of sleep continuity and hyperarousal is mediated by a sleep-interfering factor and insomnia appraisal by a sleep-interpreting factor.	Allows multi-component integration of physiologic, cognitive and personality traits into two main factors that lead to chronic insomnia, but also explains why patients report varied complaints despite similar insomnia symptoms.
Psychobiologic inhibition model[6]	Integrates homeostatic and circadian sleep-wake regulating processes with a person's ability to respond to real life stressors. Chronicity emerges when attention shifts from life stressors to insomnia symptoms, promoting an intention to act on these symptoms, which in turn promotes effort towards sleep (an otherwise automatic process that does not involve effort).	Provides a useful breakdown of cognitive-behavioral processes that promote transition from acute to chronic insomnia. These thoughts and behaviors can individually be targeted during treatment. It also presents insomnia as a deficit of sleep-promoting processes, rather than direct enhancement of arousal, which in turn allows consideration of targeted pharmacotherapies in conjunction to cognitive behavioral therapy.
Cognitive model[7]	Variation of previous models focusing on anxiety-based theory, following on feedback loops wherein sleep-related anxiety cognitive processes perpetuate insomnia via physiologic hyperarousal.	Places an emphasis on worry as part of chronic insomnia generation, allowing for identification of stress-producing factors and cognitive processes that can be addressed with cognitive therapy, but may distract

		from other contributing factors (e.g., physiologic or even behavioral).
Neurobiological model[8]	Model focusing on the imbalance of specific sleep-wake regulating brain networks involved in insomnia, as well as aberrant activity of cortical and subcortical areas.	The first comprehensive model to follow the neurologic method of localizing symptoms to the brain. It allows for a neurobiological explanation of state misperceptions, decreased sensory and cognitive inhibition, as well as the cognitive-behavioral daytime effects of such dysregulations.
Parallel process model[9]	Neurophysiological and cognitive-behavioral processes working in parallel in the development of chronic insomnia, wherein factors reported in other models are accounted for.	Comprehensive model incorporating most factors included in other models, being the youngest offspring of the 3P model.

References

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