

Psychophysiological Insomnia

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Glossary

Difficulty initiating and maintaining sleep (DIMS):

A broad term that includes insomnia with any of the subtypes described below.

Initial insomnia: Difficulty falling asleep in the absence of middle or late insomnia (also referred to as sleep-onset insomnia).

Late insomnia: Difficulty with early morning awakenings in the absence of initial or middle insomnia (also referred to as terminal insomnia or sleep-offset insomnia).

Middle insomnia: Difficulty maintaining sleep in the absence of initial or late insomnia (also referred to as sleep-maintenance insomnia).

Psychophysiological insomnia: A form of insomnia that is conceptualized as being perpetuated by

both psychological (behavioral and cognitive) and physiological factors. While psychophysiological insomnia is not classified as having subtypes, it may be descriptively useful to take into account the presenting complaint.

Sleep continuity: This term is often used in two interrelated ways. One use is to refer to the extent to which sleep is efficient as regards sleep latency (SL) and/or wake after sleep-onset (WASO) measures. The other use specifically refers to the class of variables (in contrast to sleep architecture) that measure sleep 'performance' including SL and/or WASO, number of awakenings (NWAK) measures, total sleep time (TST), and sleep efficiency as a percentage measure of the ratio of TST to total time in bed (SE %).

History and Nomenclature

The general term 'psychophysiology' came into common use in the 1960s and 1970s and represented the effort to explore the psychological influences on physiological functioning via the use of electrophysiological measures (electroencephalogram, electromyogram, etc.). It is unclear how this term came to represent a type of insomnia (i.e., who adopted the expression and what their rationale was). This said, the term seems particularly apt in that it underscores the concept that this form of insomnia is determined by both psychological and physiological factors. Psychological factors include both cognitive and behavioral elements. Cognitive factors consist of perceived stress, worry, rumination, intrusive thoughts, and attention bias. Behavioral factors consist of altered sleep scheduling (i.e., expanded sleep opportunity and napping), and the tendency to stay in bed when awake. Physiological factors include conditioned arousal and/or wakefulness and increased neurohormonal activation. While it has yet to be demonstrated which of these factors is primary, the term itself suggests that physiological factors mediate, while psychological factors moderate, the disorder.

Demographics and Prevalence

While there is a fair amount of data regarding the overall prevalence of insomnia (generally defined) and how this varies by sex and age, there are little to no data on psychophysiological insomnia specifically for the variables of interest (point prevalence, lifetime prevalence, annual incidence, or data regarding remission, recovery, and/or relapse rates). In general, insomnia symptoms or acute insomnia is thought to occur in up to 30% of the population. Ten percent of the population is estimated to have chronic and severe insomnia, with

women and elderly patients reporting higher rates. Finally, with respect to insomnia generally defined, the annual incidence rate is ~5%, and untreated patients with chronic insomnia tend to remain ill at a rate of about 50% over follow-up periods of 1–20 years.

The only estimates that exist specifically for psychophysiological insomnia are from the second edition of the *International Classification of Sleep Disorders (ICSD-2)*, which specifies that point prevalence and in-clinic prevalence rates for this type of insomnia are 1–2% and 12–15%, respectively.

Onset, Ontogeny, and Clinical Course

As indicated earlier, no work has been undertaken to assess in specific the natural history of psychophysiological insomnia. It is presumed to have (1) an insidious or acute onset (known precipitant), (2) an adult onset (partly owing to how pediatric insomnia is defined), or (3) a chronic and unremitting course where illness severity is thought to intensify with time. Interestingly, while it is thought to be 'chronic and unremitting,' insomnia is not thought to necessarily occur each night. Most research studies tacitly acknowledge this night-to-night variability by using eligibility criteria that stipulate that the insomnia (to be considered severe) must occur with a frequency of three or more nights per week.

Etiology and Pathophysiology

As with issues related to its clinical course, there are little to no data relating specifically to the etiology and pathophysiology of psychophysiological insomnia. This said, there are a variety of models describing chronic and severe insomnia that appear to be characterizing this specific type of insomnia.

Predisposing, Precipitating and Perpetuating Factors

Chronic and severe insomnia has been posited to occur in relation to predisposing, precipitating, and perpetuating factors. In brief, the model, articulated by Spielman and colleagues in the 1980s, suggests that a variety of biopsychosocial factors predispose the individual to, and precipitate the development of, insomnia and that the chronic form of the disorder develops in association with one particular perpetuating factor: the behavioral tendency to extend sleep opportunity in the face of sleep loss. The net effect of this behavioral tendency is to produce a mismatch between 'sleep ability' and 'sleep opportunity' such that sleep continuity disturbance will persist unabated and largely in the absence of the factors that predisposed and/or precipitated the onset of the disorder. The model has high face validity, strong conceptual internal validity, and the treatments that derive from the model have been found to be highly efficacious. This said, the specific tenets of the model (especially in terms of the natural history of insomnia) have not been specifically assessed for their potential as causal factors.

Diagnosis

Psychophysiological insomnia, as defined in the ICSD-2, is a state of "heightened arousal and learned sleep preventing associations that result in a complaint of insomnia and associated decreased functioning during wakefulness." The specific criteria are as follows (page 7):

- Complaint of difficulty in initiating sleep or in maintaining sleep, waking up too early, or sleep that is chronically nonrestorative or of poor quality.
- The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.
- The insomnia is present for at least 1 month.
- The patient has evidence of conditioned sleep difficulty and/or heightened arousal in bed as indicated by excessive focus on, and heightened anxiety about, sleep; difficulty falling asleep in bed at the desired bedtime or during planned naps, but no difficulty during other monotonous activities when not intending to sleep; ability to sleep better away from home than at home; mental arousal in bed characterized either by intrusive thoughts or perceived inability to volitionally cease sleep-preventing mental activity; heightened somatic tension in bed reflected by a perceived inability to relax the body sufficiently to allow the onset of sleep.
- The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

Treatment

There is no evidence that any type or subtype of insomnia is differentially responsive to one or more treatment types. A general overview of treatment approaches is provided below.

Pharmacologic Approaches

In general there are four approaches to the medical treatment of insomnia. The first approach is via the use of sedative hypnotics (barbiturates (e.g., amobarbital), benzodiazepines (e.g., temazepam), and benzodiazepines receptor agonists

(e.g., zolpidem)). Of these classes, barbiturates are no longer considered to have a primary indication for the treatment of insomnia, owing to their low therapeutic index. Currently, there are no data to suggest that benzodiazepine receptor agonists have superior efficacy or safety profiles as compared to benzodiazepines, although it is generally believed that benzodiazepine receptor agonists have a higher therapeutic index. The second approach is via the use of melatonin agonists. Currently, there is only one compound with an Food and Drug Administration indication for the treatment of insomnia (ramelteon). While there are no data regarding this medication's relative efficacy, it has been shown to have larger effects on polysomnography measures as compared to prospective self-report measures (sleep diaries). The third approach is via the use of low-dose doxepin (Silenor). This compound, originally developed and marketed as an antidepressant, is thought to provide good efficacy while having a reduced risk for side effects and tolerance, especially in elderly patients. The fourth approach includes a variety of off-label approaches using antidepressants (e.g., trazodone) and/or antipsychotic (quetiapine) medications. At present, the limited data that exist do not suggest that any approach has superior efficacy and/or better safety profiles than the benzodiazepines or benzodiazepine receptor agonists.

Cognitive Behavioral Approaches

The primary cognitive behavioral treatment of insomnia is referred to as CBT-I. This is a multicomponent behavioral therapy that usually comprises three core treatments including (in order of priority) stimulus control, sleep restriction, and sleep hygiene therapies. Interestingly, and despite the 'C' in CBT-I, it is often the case that formal cognitive therapy is not part of the CBT-I intervention.

- Stimulus control therapy – Stimulus control instructions (at their core) (1) restrict the behaviors that occur in the bedroom to sleep and sex, (2) limit the amount of time patients spend awake in bed or in the bedroom, and (3) promote counterconditioning by ensuring that the bed and bedroom environment are tightly coupled with sleepiness and sleep.
- Sleep restriction therapy (SRT) – This requires patients to limit the time they spend in bed to an amount equal to their average total sleep time. When sleep proves to be efficient, total sleep time is incrementally increased.
- Sleep hygiene therapy – This intervention requires that the clinician and patient review a set of instructions geared toward helping the patient maintain good sleep habits. Sleep hygiene instructions, it should be noted, are not helpful when provided as a monotherapy.

See also: **Descriptions of Insomnia:** Insomnia Due to Mental Illness; **Treatment of Insomnia:** Cognitive Therapy for Insomnia; Pharmacologic Treatment of Insomnia.

Further Reading

- Morin CM, et al. (2009) The natural history of insomnia: A population-based 3-year longitudinal study. *Archives of Internal Medicine* 169(5): 447–453.
- Perlis M, Smith M, Jungquist C, and Posner D (eds.) (2005) *The Cognitive-Behavioral Treatment of Insomnia: A Session by Session Guide*. New York: Springer.
- Sateia M and Buysse D (eds.) (2009) *Insomnia: Diagnosis and Treatment*. New York: Informa Healthcare.