Idiopathic Insomnia

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Glossary

Idiopathic insomnia (also referred to as childhood-onset insomnia): A form of insomnia that appears to have its onset early in life ('beginning in childhood if not at birth') and has a clinical course that is chronic and relatively invariant.

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

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 and/or wake after sleep-onset measures. The other use specifically refers to the class of variables (in contrast to sleep architecture) that measure sleep 'performance' including sleep latency (SL) and/or wake after sleep onset (WASO), number of awakenings (NWAK), total sleep time (TST), and sleep efficiency (a ratio of total sleep time to total time in bed; SE%).

History and Nomenclature

The term 'idiopathic insomnia' dates back to at least the 1800s. In that era, the diagnostic category did not appear to denote 'lifelong' insomnia with an insidious onset; instead, it denoted a form of initial insomnia with profound daytime effects, which were attributed to nonrestorative sleep. As summarized in 1885 in a book entitled *Brain Rest* by J. Leonard Corning:

> [The symptoms of Idiopathic insomnia] consistent [of] great lassitude during the day, coupled with a high degree of irritability, which is particularly well marked in the morning. [...] Uncontrollable yawning is present in almost every case. [...] On retiring the subject is unable to fall asleep. He tosses from side to side, removes the bed-clothes, changes his position continuously in the vain endeavor to become unconscious. When, as frequently happens, sleep at last supervenes, it is no longer physiologic in character but, on the contrary, perverted by dreams and unconscious cerebration to such a degree that it affords little or no refreshment.

Within the context of modern sleep research, there are two seminal studies by Peter Hauri conducted in the 1980s. The first study (1980) was a polysomnographic (PSG) investigation of sleep continuity and sleep architecture differences between patients with adult-onset ($n = 39$) and those with childhood-onset ($n = 20$) insomnia. As summarized in the abstract,

> the childhood-onset insomniacs took longer to fall asleep, slept less, and showed excessive amounts of REM sleep without eye movements. Adult-onset insomniacs showed more restless sleep. No differences between childhood- and adult-onset groups were found on personality inventories, but those with childhood-onset insomnia reported more evidence of possible 'soft' neurological impairment.

The second study (1983) was a cluster analysis of 89 patients with insomnia who had been extensively profiled via clinical interviews (e.g., sleep history questions), psychological testing (e.g., the Minnesota multiphasic personality inventory, depression and anxiety inventories; affect checklists; and medical symptom scales) and with polysomnography. This study identified five subgroups, two of which were related to idiopathic insomnia. The two subtypes were identified in terms of current illness severity (moderate vs. severe). The sleep continuity and architecture profiles are depicted in Table 1.

The groups also differed with respect to their tendency toward reverse first-night effects, history of attention deficit hyperactivity disorder, and personality measures.

The current use of the classification, consistent with its use throughout modern medicine, refers to the nature of the onset (arising from unknown causes) and carries with it the denotation that the onset is early in life and is related to biological causes which may have genetic underpinnings. It is noteworthy that the *International Classification of Sleep Disorders*, 2nd edn (ICSD-2) definition of idiopathic insomnia does not specify the subtype of insomnia (i.e., initial insomnia) even though such a presentation would be consistent with historical definitions, the age distribution of the insomnia subtypes, and recent clinical trials evaluating melatonin as a potential treatment modality for the disorder.

Demographics and Prevalence

As noted above, idiopathic insomnia is thought to be rare, occurring in <1% of the population. No information is available with respect to lifetime prevalence, annual incidence, or data regarding remission, recovery, and/or relapse rates. With respect to the last of the issues, this disorder is defined by its chronicity and thus it is implied that spontaneous remission does not occur, that treatment responses are small in magnitude, and that relapse is a highly probable event.

Onset, Ontogeny, and Clinical Course

To date, no work has been undertaken to assess the natural history (longitudinal prospective studies) of idiopathic insomnia. It is presumed/defined to have (1) an insidious onset, (2) a childhood onset, and (3) a chronic and unremitting course where illness severity is thought to be constant over time.

Table 1 Sleep continuity and architecture profiles

	Moderate	*Severe*
SL	48.5	161.4
WASO	60.5	36.5
TST		
SE%	78.6	61.0
STG1%	13.0	14.1
SWS%	3.8	0.6
REM%	18.8	18.3
RL	92.5	76.6

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 idiopathic insomnia. It is presumed that the condition arises from a fundamental dysregulation of the circadian system, the sleep homeostasis system, and/or the underlying neural circuitry of sleep and/or wake control. Further, this form of insomnia may have a strong genetic determination to the extent that (1) it is more often than not expressed at a very young age, (2) insomnia (in general) and idiopathic insomnia (in particular) is heritable (runs in families), and (3) sleep continuity disturbance (and in particular prolonged sleep latency) may be amplified over successive generations using laboratory selection in animal models. Interestingly, of these potential etiologic factors, treatment research has focused primarily on circadian considerations.

Associative, Predisposing, and Precipitating Factors

As noted earlier, idiopathic insomnia is conceptualized as an organic, and potentially genetic, disorder. At present it is unknown if the disorder is preponderantly expressed at birth or during the first decade of life. In the former case, it is not necessary to postulate what the precipitating factors are. In the latter case, it may be fruitful to identify the environmental factors that interact with the predisposition to cause the expression of the disorder. In either case, it is likely that state-wise exacerbation is possible and that traditional perpetuating factors may moderate illness severity.

Diagnosis

Idiopathic insomnia is defined in the ICSD-2 as follows.

A. The patient's symptoms meet the following criteria for insomnia:
   - There are complaints of difficulty initiating sleep and maintaining sleep, or 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.
   - There is evidence of daytime impairment.

B. The course of the disorder is chronic as indicated by each of the following:
   - Onset during infancy or childhood.
   - No identifiable precipitant or cause.
   - Persistent course with no periods of sustained remission.

C. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

Treatment

As a continuing theme of this article, there is no evidence that any type or subtype of insomnia is differentially responsive to one or more treatment types. This said, idiopathic insomnia might be expected to be less responsive to traditional treatments (therapy with benzodiazepine receptor agonists (BZRAs) and/or cognitive behavioral treatment of insomnia (CBT-I)). Given this concern and given

1. that the disorder may be related to dysregulation of the circadian system (amongst other things), and
2. the clinical profile similarities between idiopathic insomnia and delayed sleep phase disorder (DSPS (including early life onset, the tendency toward severe initial insomnia, and a sleep architecture profile that is reminiscent of phase delay)),

it follows that a chronobiotic approach, alone and/or in combination with standard methods, may be indicated for the management of idiopathic insomnia.

To date, only two studies have been conducted evaluating a chronobiologic approach. Both studies were undertaken in children (6–12 years of age) with 'chronic idiopathic childhood sleep-onset insomnia.' In both studies, spearheaded by Marcel Smits (2003, 2010), it was found that treatment with melatonin significantly advanced sleep onset (~1 h) and reduced diary-assessed sleep latency by about 50%. Interestingly, in both investigations, the post-sleep latencies remained at ~30 min. To our knowledge, no studies of melatonin or melatonin agonists have been undertaken in adults with idiopathic insomnia.

Standard Pharmacologic Approaches

In general, there are three 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 BZRAs (e.g., zolpidem)). Of these classes, barbiturates are no longer considered to have a primary indication for the treatment of insomnia, owing to its low therapeutic index. Currently, there are no data to suggest that BZRAs have superior efficacy or safety profiles as compared to benzodiazepines, although it is generally believed that BZRAs 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 (FDA) 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 PSG measures as compared to prospective self-report measures (sleep diaries). The third 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 of these approaches have superior efficacy and/or better safety profiles than the benzodiazepines or BZRAs.

Standard 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 the bedroom, and (3) promote counter-conditioning 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 towards helping the patient maintain good sleep habits. Sleep hygiene instructions, it should be noted, are not helpful when provided as a monotherapy.

For additional information on the assessment and treatment of insomnia in children, the reader should refer to the chapters in this Encyclopedia that specifically address the pediatric insomnias.

See also: **Descriptions of Insomnia:** Behavioral Insomnia of Childhood; **Special Conditions, Disorders, and Clinical Issues for Hypersomnias:** Hypersomnias in Children; **Special Conditions, Disorders, and Clinical Issues for Insomnia:** Insomnia in Children; **Treatment of Insomnia:** Cognitive Therapy for Insomnia.

Further Reading

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