Types of Insomnia

M L Perlis and P Gehrman, University of Pennsylvania, Philadelphia, PA, USA

Glossary

Difficulty initiating and maintaining sleep (DIMS): A broad term that includes insomnia with any of the subtypes described above.

Idiopathic insomnia: Lifelong insomnia with a presumed organic component.

Inadequate sleep hygiene insomnia: A form of insomnia that is conceptualized as being perpetuated, in large measure, by lifestyle issues.

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

Insomnia not otherwise specified (NOS): A form of insomnia that is conceptualized as being perpetuated by unknown factors.

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).

Paradoxical insomnia: A form of insomnia for which there is a profound discrepancy between the patient's experience of sleep continuity disturbance and the measure of insomnia severity by polysomnography.

Physiological insomnia: A form of insomnia that is conceptualized as being perpetuated, in large measure, by organic factors.

Psychophysiologic insomnia: A form of insomnia that is conceptualized as being perpetuated by both psychological (behavioral and cognitive) and physiological factors.

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

Insomnia is the first of the sleep disorders to be described as either a symptom or a disease. References to this form of sleeplessness may be found in the oldest documents known to man including Iliad, the Epic of Gilgamesh, the Torah, the New Testament, and the Koran. Interestingly, the term for insomnia, while generally ubiquitous, also has a variety of historical synonyms that, while not in current use, serve to illustrate the wisdom of the ages. For example, as cited in a Medical Dictionary published in 1892, insomnia is referred to as, in general, 'Agrypnia,' and the dictionary further specifies three subtypes.

Agrypnia – a term for wakefulness or sleeplessness; one of the premonitory signs of insanity.

Agrypnia excitata – sleeplessness due to mental excitement with listlessness as to surrounding objects.

Agrypnia pertoesa – sleeplessness from bodily disquiet, with attention alive to surrounding objects.

Agrypnia senilis – the sleeplessness of old age.

It is interesting to note that each of these classifications have direct modern analogs (not necessarily in name but in terms of theory). First, with respect to the general category, the claim that sleeplessness is a 'premonitory sign for insanity' is very consistent with modern research showing that insomnia is a significant risk factor for, and potentially a prodromal sign of, depression. Further, insomnia has been found to be a risk factor for other psychiatric disorders (though the evidence base is less well developed) such as anxiety and substance disorders. With respect to Agrypnia excitata, this classification appears to presage the relevance of cognitive arousal (including worry, rumination, intrusive thoughts, and attention bias) as a perpetuating factor for insomnia. With respect to Agrypnia pertoesa, this classification appears to presage the relevance of abnormal sensory and information processing as a perpetuating factor for insomnia. Finally, while controversial, Agrypnia senilis does seem to capture the common observation that sleep continuity declines with age.

There is also a historical precedent for typing insomnia in terms of the presenting complaint, that is in terms of the subtypes (or phenotypes) of insomnia which include initial, middle, and terminal insomnia. According to Kleitman, in his seminar work 'Sleep and Wakefulness' (1939/1963), the first reference to the insomnia subtypes was by Worster-Drought in 1927. While it is difficult to say how these terms became enshrined in the sleep and/or psychiatry lexicons, one likely possibility is the use of these classifications by Hamilton in the construction of his rating scale for depression (first published in 1960). The classical description of the insomnia subtypes, which are essentially unchanged from Kleitman's description, are provided below.

Initial or predormitional insomnia – where the onset of sleep is delayed.

Middle insomnia – (where sleep is) broken, choppy, intermittent, or lacunary.

Terminal or postdormitional (or late) insomnia – where the sleeper awakens up too early and is not able to fall asleep again.

Currently, insomnia is defined in each of the major nosologies that define human disease and mental illness including (as noted above) *Diagnostic and Statistical Manual for Psychiatric Disorders*, 4th edn, Text Revision (DSM-IV-TR) and the *International Classification of Sleep Disorders*, 2nd edn (ICSD-2) as well as the *International Classification of Disease*, 9th edn (ICD-9). These diagnostic classifications have been augmented with the delineation of formal research diagnostic criteria.

Perhaps the most significant historical development to date with respect to the definition of insomnia and insomnia subtypes has been the effort to challenge the validity and utility of the diagnostic classification of 'secondary' insomnia by Lichstein and colleagues. At this juncture, many appear ready to doff the concept of 'secondary' insomnia in favor of the concept of 'comorbid' insomnia. This perspective appears to have been embraced by the framers of the DSM-V who have all but abolished the concept and category of secondary insomnia with the elimination of the requirement that primary insomnia be diagnosed *only in the absence* of concurrent medical and mental illness. The newer conceptualization is that when insomnia occurs with axis I and axis II disorders, the insomnia is to be considered a comorbid disorder. Finally, even the diagnostic term has been altered to reflect this change in emphasis. In the upcoming DSM-V, what was once called primary insomnia is now slated to be referred to as insomnia disorder.

Demographics and Prevalence

To our knowledge there are little to no demographic or epidemiologic data (lifetime prevalence, annual incidence, or data regarding remission, recovery, and/or relapse rates) for any of the types of insomnia. Data that do exist pertain to point prevalence and these data appear to be based largely on expert consensus. With this caveat in mind, the ICSD-2 specifies the point prevalence and in-clinic prevalence rates for each insomnia type.

Several studies have been conducted regarding the prevalence of the insomnia subtypes (both in the population at large and within clinical subgroups). In general, each of the three phenotypes appear to be equally represented (regardless of the sample), with some limited data to support the notion that sleep initiation problems occur predominately in young individuals and sleep maintenance problems (particularly early morning awakenings) occur predominately in the elderly.

Given the relative absence of demographic or epidemiologic data for the types of insomnia, the general data regarding insomnia (variably defined) may be illustrative. Acute insomnia (variably defined from days to months) is thought to occur in up to 30% of the population and inasmuch as 50% of patients assessed in primary-care practice. Chronic insomnia (variably defined as more than 2 weeks to more than 6 months) is thought to occur in 10% of the population.

Little is known about the annual incidence of insomnia (new onset cases per year) lifetime prevalence of the disorder, and/or how frequently patients with acute or chronic insomnia (1) spontaneously remit and/or relapse from such remissions or (2) relapse following self-help or evidence-based treatment. The best data to date suggest that

- the annual incidence rate for insomnia is ∼5%;

- patients with chronic insomnia tend to remain ill at a rate of about 50% over follow-up periods of 1–20 years.

Onset, Ontogeny, and Clinical Course

In the absence of data, it is presumed that each of the insomnia types (if not subtypes) have different natural histories. At one extreme end of the continuum it is likely that the onset of the disorder is presumed to be early in life (first two decades) and the clinical course is chronic and relatively invariant (idiopathic insomnia). At the other extreme end of the continuum it is likely that the onset, ontogeny, and clinical course are entirely situational or environmental (sleep hygiene insomnia disorder). For specifics regarding onset, ontogeny, and clinical course of the various types and subtypes of insomnia, the reader is referred to the articles that specifically define these disorders.

With respect to subtype differences vis-à-vis onset, ontogeny, and clinical course considerations, there is also a relative dearth of data, with one notable exception. There are at least two cross-sectional epidemiologic studies which suggest that initial and late insomnia are differentially prevalent in the young and the old (more initial insomnia in the young and more late insomnia in the old). Whether these represent cohort or aging effects remains to be determined.

Etiology and Pathophysiology

As with clinical course issues, there are little to no data regarding type and subtype differences with respect to etiology, pathophysiology, and pathogenesis. This said, it is possible (if not likely) that each of the insomnia types, and potentially the subtypes (Table 1), occur as a result of different biopsychosocial pathological processes. At one extreme end of the continuum the etiology and pathophysiology are likely to be largely organic in nature (i.e. idiopathic insomnia and/or physiological insomnia) and the expression of the disorder is presumably the result of genetic and/or biological abnormalities that give rise to fundamental dysregulations of the circadian system,

Table 1   Prevalence of insomnia subtypes

Type	Population prevalence (%)	In-clinic prevalence (%)
Idiopathic insomnia	0–1	<10
Psychophysiologic insomnia	1–2	12–15
Paradoxical insomnia	–	5
Inadequate sleep hygiene insomnia	1–2	5–10
Physiological insomnia	–	–
Insomnia NOS	–	–
Insomnia due to mental illness	3	**
Insomnia due to medical illness	0–1	3–4
Insomnia due to substance use/ abuse or withdrawal	0–1	3–4

All the entries in this table are from the ICSD-2 (stated as ranges).

Note: the total of the population prevalence is consistent with the 10% overall prevalence rate of chronic insomnia.

**Two studies estimate that 44% and 46% of patients with chronic insomnia assessed in clinic have concurrent mental illness.

the sleep homeostasis system, and/or the underlying neural circuitry of sleep and/or wake control (note: the drosophila model of insomnia, as developed by Shaw and colleagues, serves as an excellent illustration of such phenomena). At the other extreme end of the continuum it is likely that the etiology and pathophysiology are entirely related to so-called lifestyle factors (i.e., sleep hygiene insomnia disorder) or behaviors that adversely affect the individual's sleep and/or wake control system (i.e., behaviors that exceed the natural 'automaticity' and 'plasticity' of the sleep system). When considering the insomnia types dimensionally, it is important to note that psychophysiologic insomnia represents the middle of the continuum. In this case, the disorder is clearly conceptualized as occurring as a result of reciprocally interactive biological and psychological factors (including cognitive, behavioral, and conditioning effects). For specifics regarding the etiology, pathophysiology, and pathogenesis of the various insomnia types, the reader is referred to the articles in this text that specifically define these disorders (and to the recommended readings listed at the end of this article).

Given the relative dearth of information regarding the etiology and pathophysiology regarding insomnia types, it should come as no surprise that there are also little to no data that show how the insomnia subtypes differ with respect to genetic and/or biological factors, regulation of the circadian and sleep homeostasis systems, and/or the functioning of the neural circuitry of sleep and/or wake control. This said, extreme forms of initial and terminal insomnia have been exhaustively researched as independent disorders of the circadian system. Specifically, delayed sleep phase disorder (corresponding to initial insomnia) and advanced sleep phase disorder (corresponding to terminal insomnia). In these cases, the pathogenesis of the disorders are thought to be related to differences in the circadian control of sleep and wakefulness and there is behavioral and molecular genetic research to suggest that these disorders are heritable and are related to specific gene alterations (e.g., alterations in the expression of the *PER-2* and *PER-3* genes). For specifics regarding delayed sleep phase disorder and advanced sleep phase disorder, the reader is referred to the articles in this text that specifically define these disorders.

Associative, Predisposing, and Precipitating Factors

As a general entity, chronic insomnia has been extensively described in almost precisely these terms. That is, chronic insomnia (presumably as classified as primary insomnia in the DSM-IV-TR or as defined as psychophysiological insomnia in the ICSD-2) has been posited to occur in relation to predisposing (as opposed to associative), 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 natural history of insomnia) have not been specifically assessed for their potential as causal factors.

The only model, to our knowledge, that appears to address how the insomnia types differ with respect to predisposing, precipitating, and perpetuating factors is the neurocognitive model. In this instance, it is suggested that paradoxical insomnia (or at least the phenomenon of sleep state misperception) occurs in association with a 'second-order' perpetuating factor: conditioned cortical arousal. It is hypothesized that with time the neurophysiologic arousal that occurs with insomnia becomes a conditioned phenomenon (i.e., cortical/subcortical activation occurs as a conditioned response to sleep-related stimuli). The result of abnormal levels of activation is to permit high levels of sensory and information processing at or around sleep onset and during non-rapid eye movement (NREM) sleep and the formation of some degree of long-term memory for these periods of time. Taken together (brain activation and the consequences of this activation), and given that such events do not alter sleep staging, it follows that there would be a discordance between the patient's perception and the objective measure of sleep. Empirical studies of the tenets of this model have provided some supportive data for the perspective.

To date, there are no studies or theories that seek to account for the different phenotypic expressions of insomnia. In fact, the descriptive and diagnostic utility of subtyping is in and of itself rather controversial. The primary objection to the relevance of the subtypes comes from the observation (one study and routine clinical observation) that the subtypes are unstable over time (change from one to another) and thus don't represent a trait or phenotype. A useful way of thinking about such variability is to ask (1) what was the initial expression of the insomnia and (2) what factors precipitated the change. The former may represent a trait disposition and the latter may implicate factors related to conditioning and/or factors that impinge on the circadian and homeostatic regulation of sleep.

Diagnosis

Each of the types of insomnia has formal definitions. To date there is no agreed-upon definitions for the insomnia subtypes. For a brief description of the insomnia types, please refer to the glossary entries. For detailed information about the various diagnostic entities, the reader is referred to the articles in this text that are devoted to each classification.

Treatment

As a continuing theme of this review, there is no evidence that any type or subtype of insomnia is differentially responsive to one or more treatment types. This said, a general overview of treatment approaches is provided below.

Table 2   Medications commonly used for sleep

Generic	Brand	T$_{1/2}$ (h)	Dose (mg)	Drug class
Flurazepam	Dalmane	48–120	15–30	BZD
Temazepam	Restoril	8–20	15–30	BZD
Triazolam	Halcion	2–6	0.125–0.25	BZD
Estazolam	Prosom	8–24	1–2	BZD
Quazepam	Doral	48–120	7.5–15	BZD
Zolpidem	Ambien	1.5–2.4	5–10	Non-BZD
Zaleplon	Sonata	1	5–20	Non-BZD
Eszopiclone	Lunesta	5–7	1–3	Non-BZD
Zolpidem ext. rel.	Ambien CR	1.5–2.4	6.25–12.5	Non-BZD

Adapted from a slide by Daniel Buysse and colleagues.

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 its 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 (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 polysomnographic (PSG) measures as compared to prospective self-report measures (sleep diaries). The *third* approach is via the use of low-dose doxepin (Silenoir). This compound, originally developed and marketed as an antidepressant, is thought to provide good efficacy while providing a reduced risk for side effects and tolerance, especially in elderly patients. The fourth approach includes a variety of off-label approaches using antidepressant (e.g., trazodone) and/or antipsychotic (quetiapine) medications. At present, the limited data that exist do not suggest that either approach has superior efficacy and/or better safety profiles than the benzodiazepines or benzodiazepine receptor agonists. Table 2 presents detailed information on some of the most commonly prescribed hypnotics.

Cognitive behavioral approaches

The primary cognitive behavioral treatment of insomnia (CBT-I) is a multicomponent behavioral therapy that usually comprises three core treatments: 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 (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 counterconditioning by insuring that the bed and bedroom environment are tightly coupled with sleepiness and sleep.

Sleep restriction

Sleep restriction therapy (SRT) requires patients to limit the amount of time they spend in bed to an amount equal to their average total sleep time (TST). When sleep proves to be efficient, TST is incrementally increased.

Sleep hygiene

This intervention requires that the clinician and patient review a set of instructions which are 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: **Psychiatric Disorders:** Mood Disorders and Sleep.

Further Reading

Edinger JD, et al. (2004) Derivation of research diagnostic criteria for insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep* 27(8): 1567–1596.

Kleitman N (1963) *Sleep and Wakefulness*, revised edition. Chicago, IL: University of Chicago Press.

Lichstein KL (2006) Secondary insomnia: A myth dismissed. *Sleep Medicine Reviews* 10(1): 3–5.

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, Shaw P, Cano G, and Espie C (2011) Models of insomnia. In: Kryger, Roth & Dement (eds.) (Section Editor: Buysse D) *Principles and Practice of Sleep Medicine*, 5th edn., section 10, ch. 850–865. Philadelphia, PA: Elsevier & Saunders Co.

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.) (2010) *Insomnia: Diagnosis and Treatment*. New York: Informa Health Care.