Insomnia

Placeholder – chapter table of contents

An insomnia disorder is defined as a persistent difficulty with sleep initiation, duration, or consolidation that occurs despite adequate opportunity and circumstances for sleep and results in concern, dissatisfaction, or perceived daytime impairment, such as fatigue, decreased mood or irritability, general malaise, or cognitive impairment. Among adults with insomnia disorder, sleep complaints typically include difficulties initiating or maintaining sleep. Among infants and young children, the complaint is voiced by a caregiver and may also include bedtime resistance and an inability to sleep independently without caregiver intervention.

Concerns about associated wake time impairment attributed to the nocturnal symptoms are referred to as daytime impairment. However, in some circumstances, such as shift work, the wake hours and impact of poor sleep may occur at night. Among adults with insomnia disorder, daytime impairment may impact social, familial, vocational, or educational functioning and reduce quality of life. Among children, it may also lead to inattention and cognitive deficits as well as behavioral and mood disturbances that impair daily functioning at school, at home, and in social situations. Other daytime symptoms may include muscle tension, palpitations, headache, and fatigue. Although less common and often confused with fatigue, daytime sleepiness, when present, may increase the risk for motor vehicle and occupational accidents. In the absence of reported or observed daytime impairment or dissatisfaction with sleep, individuals who describe prolonged sleep latency, recurrent or prolonged awakenings, or restricted sleep duration are not regarded as having an insomnia disorder that warrants clinical attention, other than education and reassurance.

Insomnia symptoms often accompany comorbid medical illnesses, mental disorders, and other sleep disorders. Insomnia symptoms may also arise with the use, abuse, or exposure to certain substances. A separate insomnia disorder diagnosis is warranted when the insomnia symptoms are persistent and result in distress or impairment.

The insomnia disorder nosology presented in ICSD-3 and maintained in this text revision represents a marked departure from the prior International Classification of Sleep Disorders, 2nd Edition classification system regarding its conceptual framework. The previous insomnia nosology of the International Classification of Sleep Disorders promoted the concept that insomnia can exist as a primary sleep disorder or arise as a secondary form of sleep disturbance related to an underlying primary psychiatric, medical, or substance use disorder. However, differentiation between primary and secondary subtypes is difficult, if not impossible. More importantly, even when another condition initially causes the insomnia, it often develops into an independent disease entity that merits clinical attention. Evidence suggests that

insomnia disorder, if left untreated, may adversely affect the outcome, and increase the risk of recurrence of comorbid conditions. Moreover, it appears that, in some cases, treatment of the insomnia disorder may improve the outcome of both the sleep disturbance and the comorbid conditions. Given these observations, insomnia disorder seems best viewed as a comorbid disorder that warrants separate treatment attention.

In addition to the primary vs. secondary insomnia distinction, prior editions of the International Classification of Sleep Disorders delineated multiple putative "primary insomnia" diagnostic subtypes. Specifically, the original 1990 version of the International Classification of Sleep Disorders and the International Classification of Sleep Disorders, 2nd Edition, described primary insomnia subtypes such as psychophysiological insomnia, idiopathic insomnia, inadequate sleep hygiene, paradoxical insomnia, and behavioral insomnia of childhood as discrete diagnostic entities. However, experience suggests that, in practice, it is rare to encounter patients who meet the diagnostic criteria for one of these subtypes exclusively. Moreover, many of the diagnostic criteria delineated for these subtypes represent generic characteristics of insomnia (e.g., engaging in sleep-disruptive habits, underestimating sleep time, demonstrating evidence of conditioned arousal), per se, and do not facilitate discrimination among these subtypes or between these subtypes and those presumed to have "secondary" forms of insomnia. Moreover, both clinical experience and a growing body of empirical findings have shown that the diagnostic distinctions advocated by previous versions of the International Classification of Sleep Disorders are difficult to ascertain reliably and are of questionable validity. Given such considerations, the current manual abandons the previously employed complex and highly specific insomnia classification scheme described by the original International Classification of Sleep Disorders and the 2nd Edition in favor of a more global and defensible nosology.

The chapter includes three diagnostic categories for insomnia: chronic, short-term, and other insomnia disorders. Chronic insomnia disorder is characterized by chronic sleep-onset or sleep-maintenance complaints with associated daytime impairment. The diagnosis is reserved for individuals whose sleep difficulties exceed minimal frequency and duration thresholds associated with clinically significant morbidity outcomes. Short-term insomnia disorder is characterized by sleep/wake difficulties that fail to meet the minimal frequency and duration criteria of chronic insomnia disorder but are associated with clinically significant sleep dissatisfaction or perceived daytime impairment. A diagnosis of other insomnia disorders should be assigned in rare cases that fail to meet criteria for chronic or short-term insomnia disorder yet have sufficient symptoms of insomnia to warrant clinical attention. Finally, while symptoms of insomnia in childhood and adolescents share some commonality with characteristics of adult insomnia disorder, there are unique features that characterize the disorder in younger patients. Moreover, there is substantial variation in the defining characteristics of insomnia disorder within this young age group. The section on Developmental Issues highlights these features.

Chronic Insomnia Disorder

ICD-9-CM code: 307.42

ICD-10-CM code: F51.01

Alternate Names

Historically the literature has used the following terms: primary insomnia, secondary insomnia, comorbid insomnia, disorder of initiating and maintaining sleep, behavioral insomnia of childhood, sleep-onset association disorder, limit-setting sleep disorder.

Diagnostic Criteria

Criteria A-F must be met

A. The patient reports, or the patient's parent or caregiver observes, one or more of the following:[1,2]
    1. Difficulty initiating sleep.
    2. Difficulty maintaining sleep.
    3. Final awakening earlier than desired.
    4. Resistance to going to bed on an appropriate schedule.
    5. Difficulty sleeping without parent or caregiver presence or intervention.
B. The patient reports, or the patient's parent or caregiver observes, one or more of the following related to the nighttime sleep difficulty:
    1. Fatigue/malaise.
    2. Impaired attention, concentration, or memory.
    3. Impaired social, family, occupational, or academic performance.
    4. Mood disturbance/irritability.
    5. Subjective daytime sleepiness.[3]
    6. Behavioral problems (e.g., hyperactivity, impulsivity, aggression).
    7. Reduced motivation/energy/initiative.
    8. Proneness for errors/accidents.
    9. Concerns about or dissatisfaction with sleep.
C. The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e., time allotted for sleep) or inadequate circumstances (i.e., safety, darkness, quiet, and comfort) for sleep.
D. The sleep disturbance and associated daytime symptoms occur at least three times per week.
E. The sleep disturbance and associated daytime symptoms have been present for at least three months.[4,5]
F. The sleep disturbance and associated daytime symptoms are not solely due to another current sleep disorder, medical disorder, mental disorder, or medication/substance use.[6,7]

Notes:

1. Reports of difficulty initiating or maintaining sleep or waking up too early can be encountered in all age groups. Resistance to going to bed on an appropriate schedule, including resisting age-appropriate naps, and difficulty sleeping without a caregiver's presence or intervention are most commonly seen in children and in adults with cognitive impairment.
2. The assessment of chronic insomnia disorder in children and adolescents should include information from both the child and their caregiver because the caregiver may not have complete knowledge about the child's experience of sleep difficulties, and the child may not have full awareness of the daytime consequences of their sleep difficulties.
3. Patients tend to use the terms sleepiness and fatigue interchangeably. Therefore, the assessment of subjective daytime sleepiness requires distinguishing the two symptoms. Fatigue refers to low physical or mental energy, whereas sleepiness refers to the propensity to fall asleep in conducive circumstances. Although patients with chronic insomnia disorder may report feeling subjectively "sleepy," extensive research indicates that most individuals with chronic insomnia disorder have low objective daytime sleepiness, as measured by the multiple sleep latency test.
4. Applying the adult frequency and duration criteria for chronic insomnia disorder may not always be appropriate in the first year of life. It is important to consider developmental sleep norms in defining this disorder in young children. For example, full-term infants are not expected to sleep through the night regularly (without caregiver intervention such as soothing or nursing) until they are three to six months of age. Therefore, the three-month duration criterion suggests that a diagnosis of chronic insomnia disorder might first be considered at six to nine months of age. However, sleep/wake patterns that are viewed as problematic by caregivers may begin to emerge in the first 12 months without meeting the required three-month duration.
5. Some patients with chronic insomnia may experience recurrent episodes of sleep/wake difficulties lasting several weeks at a time over several years, yet not meet the three-month duration criterion for any single episode. Nonetheless, a diagnosis of chronic insomnia disorder is merited in these cases, given the persistent sleep difficulties.
6. Comorbidity does not preclude the independent diagnosis of chronic insomnia disorder. Evidence has clearly shown that even when a co-occurring disorder has instigated the insomnia, the sleep disturbance often transforms into an independent, self-sustaining disorder. By the time such a patient presents with an insomnia complaint to a health care provider, the insomnia is usually either independent of the comorbidity or shares a reciprocal relationship with it. If another condition does not solely cause the patient's sleep/wake complaints, those complaints merit a diagnosis of chronic insomnia disorder and separate treatment attention.
7. Regular users of hypnotic medications who sleep well when they take the medications should still be assigned a diagnosis of chronic insomnia disorder. They may present clinically with concerns about their inability to sleep without sleep medications.

Essential Features

The essential feature of chronic insomnia disorder is a frequent and persistent difficulty initiating or maintaining sleep that results in general sleep dissatisfaction or perceived impairment, reported by the

patient or a caregiver. Distress about poor sleep or impairment in family, social, vocational, academic, or other important functions accompanies the sleep complaint. Furthermore, the sleep disturbance and associated waking symptoms occur despite having adequate time and circumstances to obtain necessary sleep.

The sleep complaints that comprise chronic insomnia disorder include difficulties initiating or maintaining sleep. The latter may consist of waking up during the night with difficulty returning to sleep or having a final awakening occurring too early, well before the desired rising time. Individuals' sleep complaints may also change over time. Those with solely sleep-onset complaints may subsequently develop sleep-maintenance complaints and vice versa. In addition, those who present initially with mixed sleep-onset and sleep-maintenance difficulties may later evidence only one or the other of these difficulties. Complaints about poor-quality, unrefreshing, or nonrestorative sleep often accompany sleep-onset and maintenance complaints. However, complaints of this nature do not merit a diagnosis of chronic insomnia disorder in the absence of sleep-onset or maintenance difficulties (see Unresolved Issues and Further Directions).

The degree of sleep disturbance required to assign a chronic insomnia disorder diagnosis is somewhat arbitrary in that it relies primarily on an individual's or a caregiver's sleep complaints. Moreover, the degree of sleep disturbance that is clinically significant varies across age groups. Empirical data suggest that among adults, onset latencies and periods of wakefulness during sleep > 30 minutes typically connote clinical significance. Early morning awakening typically entails the termination of sleep at least 30 minutes before the desired rising time and a concomitant reduction in total sleep time compared with the usual premorbid sleep pattern. However, the exact time at which early morning awakenings occur may vary considerably as a function of usual bedtimes. For example, a complaint of final awakening at 4:00 a.m. and a desire to sleep more is likely to connote clinical significance when the usual bedtime is 11:00 p.m. but not when it occurs at 9:00 p.m. There are fewer empirical data concerning thresholds for sleep-onset latency and frequency of night awakenings in infants and children, as these are age-dependent. Among very young children, clinical significance also depends on the level of caregiver involvement needed for the child to sleep.

Qualitative research indicates that adults experience chronic insomnia disorder as a 24-hour problem and report multiple waking symptoms. Common waking symptoms include fatigue, reduced motivation, concentration, attention, and memory functioning, and irritability or low mood. Complaints of subjective daytime sleepiness need to be distinguished from fatigue. Whereas fatigue manifests mainly as a lack of mental or physical energy and desire to reduce or limit activity levels, subjective sleepiness is characterized by a reduced level of alertness. Despite a desire to nap, many individuals with insomnia are unable to do so. However, unintentional dozing, primarily in the evening, may be present. Frequent, involuntary daytime sleep episodes are more characteristic of other types of sleep disorders such as sleep-related breathing disorders, narcolepsy, idiopathic hypersomnia, or insufficient sleep syndrome. Individuals with insomnia also do not exhibit objective sleepiness, as indicated by their longer latency to sleep onset on the multiple sleep latency test compared to age-matched healthy sleepers with similar sleep duration. Reports of reduced performance at work or school or impaired social functioning are also

common. Some affected individuals attribute errors or accidents at work to their sleep difficulties. Somatic symptoms such as headaches or gastrointestinal dysfunction are occasionally attributed to ongoing sleep difficulties. Some evidence indicates that the experience of difficulties with both initiating and maintaining sleep is associated with more significant daytime impairment than the experience of either one of these nocturnal symptoms alone.

Although transient and episodic forms of insomnia occur, the clinically significant daytime consequences of a chronic insomnia disorder and the longer-term morbidity outcomes typically develop when the sleep difficulties occur at least three times per week and persist for at least three months. For this reason, these frequency and duration criteria must be met to assign a chronic insomnia disorder diagnosis (see Criteria Notes 4 and 5 regarding exceptions to the duration criterion). More acute and episodic forms of insomnia may cause significant distress and functional impairment and require clinical attention. Cases that meet all criteria except the frequency or duration criteria for chronic insomnia disorder should be assigned a diagnosis of short-term insomnia disorder.

Chronic insomnia disorder can occur in isolation, although it is commonly comorbid with a mental or medical disorder, another sleep disorder, or substance use. Insomnia symptoms can be solely due to a comorbidity (e.g., patients with obstructive sleep apnea and complaints of repeated awakenings that resolve with positive airway pressure). However, as noted above, the symptoms often evolve into a chronic insomnia disorder that is at least partially independent from the comorbidity and requires independent assessment and treatment.

Associated Features

Individuals with chronic insomnia disorder typically note feelings of reduced well-being, general malaise during the day, physical tension at night, and physiological, cognitive, or emotional hyperarousal at night. Complaints such as: “I can’t shut my mind off” or feeling “wound up” or anxious often characterize the cognitive and emotional hyperarousal, respectively. In addition, excessive focus on and worry about ongoing sleep difficulties and their associated daytime consequences are common. Thoughts about ongoing sleep difficulties may occur throughout the day and may be amplified as bedtime approaches. Performance anxiety about sleep is common. Anxious thoughts and worries experienced by individuals with chronic insomnia disorder often focus on the sleep difficulty, including concerns about potential consequences of poor sleep. A comorbid anxiety disorder may be present when anxiety and worry are more pervasive and not solely focused on sleep problems. However, even in the absence of anxiety disorders, rumination about events of the day or anxious thoughts about a future event may make it difficult to sleep. Whereas for good sleepers, the bed and bedroom are cues for sleep, the sleep environment becomes a cue for wakefulness for many with chronic insomnia disorder. Such individuals may fall asleep easily in settings outside of their bedrooms when not trying to sleep but show a pattern of cognitive and physiological arousal when lying down in their beds intending to fall asleep. Some patients with this pattern may report sleeping better when away from home. Unlike good sleepers, for whom sleep is an automatic process, individuals with chronic insomnia disorder often express conscious intentions

and excessive effort to sleep, only to find sleep difficult to initiate under such circumstances. They tend to have the same experience when trying to nap and are typically unable to do so.

In infants and toddlers, sleep difficulties can also manifest as difficulty settling into or sustaining naps. In children, daytime behavioral problems, mood dysregulation, and limit-setting difficulties during the day may accompany the sleep disturbance. In addition, the nighttime sleep disturbance often results in poor caregiver's sleep and associated daytime impairment. Marital conflicts about responding to or intervening with the child's ongoing sleep problem may arise. Caregivers may also develop negative feelings toward the child who disrupts their sleep and demands attention during the night.

Clinical and Pathophysiological Subtypes

Previous nosology's have described various clinical and pathophysiological subtypes of chronic insomnia disorders. The American Psychiatric Association's Diagnostic and Statistical Manual, 4th Edition, Text Revision (DSM-IV-TR) and the International Classification of Sleep Disorders, 2nd Edition described independently occurring or *primary* forms of insomnia. Within DSM-IV-TR, the global term, p*rimary insomnia*, is applied to all forms of insomnia that are not better explained by a coincident psychiatric, medical, or substance use/abuse disorder. The International Classification of Sleep Disorders, 2nd Edition delineated multiple primary insomnia subtypes. For reasons described in the introduction and below, ICSD-3 abandoned this detailed classification and listed all presentations within a single diagnosis of chronic insomnia disorder. Thus, the ICSD-3-TR does not support classification into subtypes but retains descriptions below to illustrate settings in which chronic insomnia disorder may arise and for educational and research purposes.

Psychophysiological insomnia is characterized primarily by heightened arousal and learned sleep-preventing associations that result in a complaint of insomnia. Patients presumed to have this type of insomnia often have sleep difficulty when trying to sleep in their usual sleep setting at home but may fall asleep easily in a novel sleep setting or when not trying to sleep. They also demonstrate an excessive focus on and worry about sleep and suffer from elevated levels of cognitive and somatic arousal, particularly at bedtime.

Idiopathic insomnia is characterized by a longstanding complaint of sleep difficulties with insidious onset occurring during infancy or early childhood. It is presumed to arise in infancy or early childhood without discernible cause and persist over time without sustained periods of remission. Given its early onset, stability over time, and lifelong course, this disorder is thought to result from either genetically determined or congenital aberrations in the sleep-inducing or arousal systems in the brain. However, no consistent genetic markers or neural pathology have been identified among those presumed to suffer from this condition.

Paradoxical insomnia, which has previously been called sleep state misperception, is characterized by a complaint of severe sleep disturbance despite objective evidence of relatively normal sleep initiation,

maintenance, and duration. Those presumed to have this form of sleep difficulty have a marked propensity to underestimate the amount of sleep they are obtaining. Essentially, they perceive much of the time they are asleep as wakefulness. Some studies using neuroimaging or sleep electroencephalograph (EEG) spectral analysis techniques have suggested that an altered sleep/wake arousal system may explain the apparent mismatch between their conventional objective sleep measures and subjective sleep reports.

Inadequate sleep hygiene is presumed to result from or be sustained by daily living activities that are inconsistent with the maintenance of good-quality sleep and normal daytime alertness. These practices include daytime napping; maintaining a highly variable sleep/wake schedule; routinely using sleep-disruptive products (caffeine, tobacco, alcohol) too close to bedtime; engaging in mentally or physically activating or emotionally upsetting activities too close to bedtime; routinely using the bed and bedroom for activities other than sleep; and failing to maintain an environment that is conducive to sleep.

Behavioral insomnia of childhood is presumed to result from improper sleep training or limit-setting by parents or caretakers. Several subtypes have been described within this broader diagnosis. *Sleep-onset association type* is characterized by the child's dependency on specific stimulation, objects, or settings for initiating or returning to sleep following an awakening; sleep onset is significantly delayed in the absence of these conditions. *Limit-setting type* is characterized by bedtime stalling or bedtime refusal resulting from inadequate limit-setting by a caregiver. A *mixed type* characterized by features of sleep-onset association difficulties and bedtime resistance represents the third subtype within this diagnostic category.

The DSM-IV-TR and International Classification of Sleep Disorders, 2nd Edition also described several so-called *secondary insomnias* arising from co-occurring primary or causative conditions. Among these are the following:

Insomnia due to (another) mental disorder is caused by or secondary to a co-occurring psychiatric condition. Insomnia is a common complaint among those with mental disorders, particularly among patients with mood disorders and anxiety disorders. Likewise, psychotic spectrum disorders and various Axis II (personality) disorders are frequently associated with sleep disturbance. Among those individuals presumed to have insomnia due to a mental disorder, insomnia has historically been regarded as a secondary symptom caused by the mental disorder itself.

Insomnia due to (a) medical condition is caused by or secondary to a co-occurring medical condition. Like mental disorders, many medical conditions may have an ongoing insomnia complaint associated with them. In particular, those conditions that cause some form of persistent pain or discomfort, mobility limitation, or breathing disturbance commonly have insomnia complaints accompanying them. Among those individuals presumed to have insomnia due to a medical disorder, insomnia has historically been regarded as a secondary symptom caused by the medical disorder itself.

Insomnia due to drug or substance is caused by or secondary to use of or withdrawal from a drug or substance. Many forms of prescription and nonprescription medications, street drugs, and other commonly used substances may produce sleep disturbances during periods of use or upon withdrawal. Given these effects, insomnia complaints may arise during periods when these substances are used or following their discontinuance. Among those individuals presumed to have insomnia due to drug or substance, insomnia has historically been regarded as a secondary symptom caused by the drug or substance itself.

Research has provided minimal support for the reliability and validity of most of these specific subtypes. It is also difficult to discriminate among them in clinical practice. Many of the defining features of these previously defined subtypes, such as conditioned arousal, poor sleep hygiene practices, and underestimation of sleep duration, are ubiquitous among individuals with chronic insomnia disorder. They are therefore not specific to one subgroup. There is also limited support for an insomnia subtype due to another medical or psychiatric disorder. Although these conditions may precipitate sleep difficulties, the sleep difficulties are maintained over time by compensatory behaviors and thoughts and result in daytime impairment. When this occurs, the symptoms are independent of the comorbid disorder and instead become a self-sustaining insomnia disorder that impacts and is impacted by the co-occurring disorder. As a result, it is practically impossible to draw firm conclusions about the direction of causality between insomnia and such co-occurring conditions.

The International Classification of Sleep Disorders, 2nd Edition additionally delineated the separate pediatric diagnosis, *behavioral insomnia of childhood*, characterized by sleep difficulties resulting from inappropriate sleep associations or inadequate limit setting by parents or caregivers. As a descriptive entity, this designation remains clinically useful when treating young children because it defines etiologic factors that can guide the formulation of targeted behavioral intervention strategies. However, there is limited empirical support for this diagnostic subtype. Moreover, inappropriate sleep-onset associations and inadequate limit-setting may be observed across the age spectrum. For example, adults with chronic insomnia disorder may report intolerance for quiet and dark environments and may be unable to fall asleep unless the television is on. Similarly, limit-setting issues may be manifested among cognitively impaired adults when caregivers fail to set limits on daytime napping that predisposes to disruptive awakenings and night wandering. Nevertheless, as discussed earlier, there are unique features of chronic insomnia disorder experienced by children and adolescents that are discussed in the Developmental Issues section below.

In addition to these various subtypes, a distinct subtype of *insomnia with objectively short sleep duration* has been described. This patient group is characterized by insomnia complaints, along with an objectively documented average sleep time of fewer than six hours per night and elevated morbidity risk. Although evidence supporting this chronic insomnia disorder subtype is growing, it is premature to delineate a separate insomnia category for this presentation.

Given these considerations, the ICSD-3 has chosen a more global and defensible approach for diagnosing those with chronic insomnia complaints. Specifically, the single diagnosis of chronic insomnia disorder

applies to all patients who have persistent and frequent insomnia complaints, whether they occur in the presence or absence of a potentially sleep-disruptive comorbid psychiatric illness, medical disorder, or pattern of substance use.

Demographics

The complete clinical syndrome of chronic insomnia disorder occurs in about 10% of the population, but the prevalence of transient insomnia symptoms is much higher (30% to 35% of the population). Chronic insomnia disorder is more common in women; those with medical, psychiatric, or substance use disorders; and in people in lower socioeconomic strata. A substantial majority of people with insomnia also experience an intrusive co-occurring medical or psychiatric condition. About 30% of people with chronic insomnia disorder also have obstructive sleep apnea, and nearly 40% of people with obstructive sleep apnea also have insomnia symptoms. Chronic insomnia disorder may occur at any age but is more commonly diagnosed in older adults, in part due to age-related cognitive decline/dementia, deterioration in sleep continuity, and an increase in medical comorbidities and medication use that increase insomnia risk.

Chronic insomnia disorder is estimated to occur in 10% to 30% of children, depending on the exact definition used and age. Specific subgroups of children, including those with chronic medical disorders, psychiatric comorbidities, and neurodevelopmental disorders such as attention-deficit/hyperactivity disorder and autism spectrum disorder (ASD) have a higher prevalence of insomnia symptoms. For example, current data suggest that up to 80% of pediatric patients diagnosed with ASD have significant sleep complaints. There is also a high prevalence of chronic insomnia disorder among children and adolescents with genetic (e.g., Smith-Magenis and Angelman syndromes) or acquired (e.g., fetal alcohol spectrum) disorders. Studies on adolescents indicate prevalence rates of 3% to 39%, depending on the diagnostic criteria utilized and the age range studied. Recent estimates employing DSM-5 criteria for the 16 to 18-year-old age group are 12% in boys and 18% in girls.

Predisposing and Precipitating Factors

Predisposition refers to longstanding vulnerability to developing an insomnia disorder. Individuals who have difficulty sleeping during stressful times or report being habitual light sleepers appear to have an elevated propensity to develop chronic insomnia disorder. Prior transient episodes of sleep difficulties elevate the risk for subsequent development of a chronic insomnia disorder. A personal tendency to be excessively anxious or overly concerned about health, general well-being, or daytime functioning may serve as predisposing characteristics. Indeed, individuals with chronic insomnia disorder often display an excessive preoccupation with daytime consequences of poor sleep and devote particular effort to what they assume to be sleep-promoting practices. A history of trauma can also predispose individuals to develop chronic insomnia disorder due to increased hypervigilance.

Heritability is a strong predisposing factor for insomnia. Concordance rates among monozygotic twins are higher than in dizygotic twins. The rate of insomnia in first-degree relatives of people with insomnia symptoms is higher than in first-degree relatives of people without insomnia. There is an estimated 40% genetic influence in symptomatic insomnia. The genetic weight specifically in chronic insomnia disorder has yet to be determined.

Concerning the pediatric age group, caregivers who have unrealistic sleep expectations for their children may predispose them to insomnia by putting them in bed too early or assigning them too much time in bed each night. Similarly, parents and caregivers of young children who fail to set limits on the child's sleep behaviors also create an unstructured sleep environment that predisposes young children to develop chronic insomnia disorder.

Precipitating factors are immediate instigators of sleep disturbance. Job-related or financial stress and factors such as the death of a loved one, divorce, a marked change in work schedule, job loss, and other significant life changes are often precipitating circumstances for chronic insomnia disorder. Other contributing factors include unstable home situations, safety concerns, and physical, mental, or sexual abuse or domestic violence exposure. Mild-to-moderate stressors can also precipitate insomnia. Psychiatric conditions, particularly mood and anxiety disorders, may precipitate chronic insomnia disorder. Likewise, comorbid restless legs syndrome or medical disorders such as gastroesophageal reflux disease or conditions that result in chronic pain, breathing difficulties, or immobility can also lead to chronic insomnia disorder. Consumption of a large quantity of alcohol at night may contribute to insomnia, particularly sleep-maintenance problems. A pattern of alcohol dependence/abuse, excessive use of caffeine (such as energy drinks) or other stimulants, and excessive evening use of electronic media may increase the risk for chronic insomnia disorder. This latter group of risk factors is prevalent among adolescents.

In children, life stresses that impact sleep include the development of a medical or psychiatric illness; physical, emotional, or sexual abuse; death of a parent; exposure to negative parental interactions, separation, or divorce; and placement in an alternate care setting, such as foster care.

In many cases, predisposing or precipitating factors cannot be identified. This is particularly true in longstanding insomnia.

Familial Patterns

The familial pattern of insomnia is not well documented. However, family studies indicate that the prevalence of insomnia is higher among first-degree relatives of people with chronic insomnia disorder (~35-55%) than among good sleepers. Mothers are the most frequently afflicted first-degree relative. Evidence from several twin studies indicates that heritability estimates for insomnia fall in the moderate range for adults and younger children but are somewhat lower for adolescents. Genetic influences are more important in the etiology of insomnia symptoms for females than males and are largely stable over

time. Candidate gene approaches have identified genetic variants across numerous systems, but few have been studied in detail, and findings have often not been replicated. Genome-wide association studies of chronic insomnia disorder and individual nocturnal insomnia symptom phenotypes using multiple biobank databases (UK Biobank, Partners Biobank, HUNT study) have identified several insomnia-related genes, including *MEIS1, CACNA1C,* and *RBFOX3*. These findings have been replicated across different populations. Evidence for shared genetic factors between insomnia and a range of health conditions, including psychiatric and cardiometabolic disorders, has been consistently identified. However, further work is needed to determine the extent to which familial aggregation represents shared genetic predisposition, shared environment, learned behavior (e.g., by observations of parental behavior), or a by-product of psychopathology.

Onset, Course, and Complications

Onset may be insidious or acute. In the former case, individuals may report insomnia symptoms in early life or young adulthood. Among adults, the onset of chronic insomnia disorder often has a clear precipitating major life event (see Predisposing and Precipitating Factors). However, in some cases, the origin of insomnia cannot be identified.

The *course* of insomnia can be episodic or persistent. The specific type of sleep complaint may also change over time. Individuals who complain of difficulty falling asleep at one time may later complain of difficulty maintaining sleep and vice versa (see Essential Features). Although short-term insomnia caused by a clear precipitating event or situation may remit when the precipitating event subsides or the individual adapts to it, sleep difficulties may persist over time even after the initial triggering factor has disappeared. When left untreated, persistent insomnia disorder may worsen over time. Concerns about sleep and daytime impairments, apprehension about poor sleep, and engagement in counter-productive behaviors may worsen sleep disturbance. Chronic insomnia disorder can also follow an intermittent course, with recurrent episodes of sleep difficulties that worsen with life stresses. In general, individuals with chronic insomnia disorder experience night-to-night variability of symptoms, with an occasional good night's sleep interspersed with several nights of poor sleep. Approximately 70% of individuals with insomnia at a given time report insomnia a year later, and 50% still have insomnia three years later.

Complications of persistent insomnia include increased risks for new onset or recurrence of depressive and other psychiatric disorders, as well as suicidality. Chronic insomnia also confers risk for work disability and prolonged use of prescription or over-the-counter sleep aids. In addition, there is some evidence for increased risk of hypertension among individuals with chronic insomnia disorder who have short objectively measured sleep duration, defined as under 6 hours.

Among children, chronic insomnia disorder may have its onset at any time from late infancy through the childhood years. The course of chronic insomnia disorder in young children varies and depends on the reasons for the sleeplessness. When limit-setting factors and negative sleep associations resolve, sleep often improves. In adolescence, caregivers may cease to set limits, leading some teens, particularly those

with eveningness chronotype, to maintain a delayed or irregular sleep schedule. In turn, this may precipitate or exacerbate sleep difficulties, sleep loss, and associated consequences. While studies suggest that young children with early onset insomnia are more likely to have persistent or recurrent insomnia throughout childhood, the trajectory linking childhood insomnia to adult insomnia is less clear and requires further study.

Developmental Issues

Several unique developmental issues are highly relevant to the conceptualization of chronic insomnia disorder in infants, children, and adolescents.

The *first* involves the importance of viewing insomnia symptoms within the context of age-related sleep norms and the developmental trajectory of changes in sleep and circadian biology across this age spectrum. During childhood, there is a gradual decline in the normative duration of nocturnal and daytime sleep. Early childhood is associated with a decrease in the frequency of normal nighttime awakenings (paralleling the gradual lengthening of the ultradian cycle). Later in childhood, bedtime is progressively delayed, and, by adolescence, circadian and behavioral processes contribute to increased prevalence of eveningness chronotype. This preference for a later bedtime, in turn, contributes to shorter sleep duration on school nights and extended duration on weekends (see below).

Second, developmental progression across domains, including motor milestones, language acquisition, social/emotional, and cognitive development, all affect the types and manifestations of insomnia across childhood. For example, developmentally normative separation anxiety and fear of being in the dark may contribute to children requesting the presence of a caregiver in the bedroom, refusing to sleep in their own bed or delaying going to bed. In cases where appropriate limits are not set, the child may become dependent on the presence of a caregiver for initiating sleep at the beginning or middle of the night. In the absence of these conditions, both sleep onset at bedtime and return to sleep after normal night awakenings are often significantly prolonged. If the conditions associated with falling asleep are reestablished, the child usually resumes sleep relatively quickly. Because maladaptive sleep-onset associations are highly prevalent among young children, a diagnosis of chronic insomnia disorder is merited only when both of the following conditions are present: (1) the associations are prolonged, demanding and typically require caregiver intervention/presence (e.g., extended rocking or car rides), and (2) in the absence of these specific conditions, sleep onset is significantly prolonged, or night awakenings are extended and disruptive.

Third, caregivers' behaviors, bedtime interactions, and expectations regarding appropriate child behavior and definitions of "good parenting" can impact chronic insomnia disorder. These beliefs and behaviors, in turn, often reflect the culture and environment of the family. Caregivers' issues are relevant to chronic insomnia disorder across childhood and adolescence as well as in adults who need care for daily function. A significant caregiver issue relates to limit setting. Inadequate limit setting occurs when caregivers institute inadequate developmentally-appropriate limits or when the limits are instituted inconsistently or unpredictably. Consequences of poor limit setting manifest as stalling or refusing to go to bed or return

to sleep during the night. While most commonly presenting as a sleep-onset complaint, limit-setting sleep problems may also result in prolonged nocturnal awakenings, depending on caregiver response during the night. In early childhood, limit-setting issues tend to intensify when the child starts climbing out of the crib or is moved into a bed. In children, as well as in adults with dementia, inadequate limit setting can also occur when the caregiver permits excessive sleeping during the day or establishes a sleep opportunity window that is too long. Extended time in bed may lead to difficulty with sleep onset or maintenance, including fragmented sleep or waking at an undesirably early time in the morning.

Lastly, there are specific developmental factors relevant to chronic insomnia disorder during adolescence. Adolescents typically seek greater independence from caregivers, resulting in the adolescent's resistance to following adult-prescribed recommendations regarding sleep or in caregivers relinquishing oversight of sleep practices. Both occurrences may contribute to chronic insomnia disorder. In addition, executive functioning in adolescents is still evolving. As a result, adolescents may engage in numerous sleep-interfering practices such as spending excessive time and using electronics in bed. Emergent chronobiological and social factors contribute to a propensity for delaying bedtimes ("eveningness" preference) compared to pre-pubertal children. Early rise times, dictated by school start times, may require bedtimes earlier than circadian sleep propensity. Thus, if the adolescent tries to fall asleep at an earlier bedtime to obtain sufficient sleep, repeated unsuccessful attempts at sleep initiation may precipitate a chronic insomnia disorder (which may be comorbid with a delayed sleep-wake phase disorder).

The specific symptoms of chronic insomnia disorder, as well as precipitating and perpetuating factors, may vary across the adult age range. These changes partly reflect developmental changes across adulthood, such as a phase delay of endogenous circadian rhythms in adolescence and young adulthood, a phase advance and increased number of awakenings among healthy older adults, and a breakdown of circadian rhythms among adults with neurodegenerative disorders. Epidemiological studies confirm that sleep-onset difficulties and nonrestorative sleep are most common among young adults with chronic insomnia disorder. In contrast, sleep-maintenance insomnia and early morning awakening are more common in middle-aged and older adults. Objective daytime sleepiness is uncommon in younger adults with chronic insomnia disorder but more common in older adults, possibly related to increased prevalence of other comorbid medical and sleep disorders. Precipitating factors for chronic insomnia disorder may also change across the adult age range, with medical and medication factors assuming a more significant role in older adults. Indeed, hypnotic medications are disproportionately prescribed to older adults, often with limited benefit and the potential for rebound and withdrawal insomnia. Likewise, perpetuating factors may vary with the life circumstances of adults of different ages. For example, older adults may spend large amounts of time in bed after retirement, adversely affecting sleep continuity. In addition, medical disorders, and symptoms (e.g., pain, dyspnea, and impaired mobility), as well as cognitive impairment, play a more prominent role in the perpetuation of insomnia among older adults. Likewise, medications for comorbid conditions that have known negative impacts on sleep may contribute to developing and maintaining a chronic insomnia disorder.

Pathology and Pathophysiology

Studies of the pathophysiology of chronic insomnia disorder have focused on various dimensions of hyperarousal during sleep and wakefulness. These studies contrast groups of people with insomnia, typically those without significant comorbidities, with healthy controls, using a variety of physiological measures. Many of these studies have small sample sizes and have not been replicated. Collectively these studies suggest increased physiological arousal among individuals with insomnia, characterized by measures such as increased heart rate; altered cardiovascular autonomic activity; increased whole-body metabolic rate; elevated cortisol, adrenocorticotropic hormone, and corticotropin-releasing factor (CRF) levels (particularly near sleep onset); increased body temperature; and increased high-frequency electroencephalographic (EEG) activity during non-rapid eye movement (NREM) sleep. Markers of physiological arousal have been associated with insomnia in children and adolescents, although data are much more limited than for adults. A few studies have documented alterations in cortisol and inflammatory markers in pediatric insomnia in both young children and adolescents. These studies imply heightened sympathetic nervous system and hypothalamic-pituitary-adrenal axis activity across sleep and wakefulness that perpetuates sleep/wake dysfunction. Some evidence also suggests that physiological dysregulation may be more evident in specific subgroups of individuals with insomnia (e.g., those with extreme subjective-objective sleep discrepancies or those with insomnia and short objective sleep duration). There is also evidence that these findings may not generalize to insomnia comorbid with mental disorders, which may have different pathophysiological findings. Emerging findings from neuroimaging and circadian-related studies further suggest that a more complex model than sleep-wake hyperarousal is needed to fully explain the pathophysiology of insomnia.

No discrete structural brain pathology can be identified in most individuals with insomnia. Patients with insomnia comorbid with neurological disorders such as stroke, brain trauma, and multiple sclerosis may have identifiable brain lesions, but insomnia is rarely their sole neurological symptom. Studies have provided conflicting evidence regarding the finding of reduced hippocampal or anterior cingulate volume, with most studies reporting negative results.

Objective Findings

Polysomnography (PSG) and the Multiple Sleep Latency Test (MSLT) are not indicated in the routine evaluation of insomnia; however, they are indicated to rule out other sleep disorders when the clinical presentation warrants. For example, sleep testing should be considered when symptoms or bed partner observations of a sleep-related breathing disorder or periodic limb movements during sleep are present. In addition, patients who present with insomnia symptoms accompanied by excessive daytime sleepiness may warrant PSG and MSLT testing, particularly if comorbid narcolepsy is suspected. Finally, sleep testing should also be considered to rule out a comorbid sleep disorder in patients who show acceptable adherence to trials of well-established insomnia therapies but fail to show adequate treatment response.
In the absence of other sleep disorders, results of PSG monitoring of patients with chronic insomnia disorder may show increased sleep latency or increased wake time after sleep onset, coupled with

reduced sleep efficiency in comparison to good sleeper controls. Sleep-onset latency or wake time after sleep onset often exceeds 30 minutes, although one-hour to two-hour periods of wakefulness in bed are not uncommon. Sleep duration may also be reduced compared with good sleeper controls. A subset of insomnia patients sleeps less than six hours per night. Some patients show altered sleep architecture with an increase in stage N1 sleep and a decrease in slow-wave sleep and REM sleep. Patients with a pronounced conditioned sleep difficulty in the home environment may show a reverse first-night effect in the sleep laboratory (i.e., better sleep on the first vs. subsequent recording nights).

Patients with chronic insomnia disorder typically underestimate sleep duration and overestimate sleep latency and awakenings. In contrast, good sleepers tend to overestimate sleep duration and underestimate sleep latency and awakenings relative to PSG. However, some patients with chronic insomnia disorder demonstrate a marked subjective-objective discrepancy. Despite significant subjective complaints, PSG reveals age-appropriate sleep duration and an absence of sleep-onset or maintenance difficulties. This presentation of chronic insomnia disorder has previously been categorized as subjective insomnia, sleep state misperception, or paradoxical insomnia. Some insomnia patients have alterations in quantitative measures of the sleep EEG compared with individuals without sleep complaints. Specifically, these patients often show greater power in the high-frequency bandwidths (especially beta) during wakefulness, NREM, and REM sleep. Some studies have shown that elevated high-frequency power is especially characteristic of individuals with chronic insomnia disorder with marked subjective-objective discrepancies in sleep measures, compared to those with obvious objective sleep disturbances or without sleep complaints.

Young children with chronic insomnia disorder typically show essentially normal sleep during PSG monitoring when the caregiver is present and appropriate limits are set in the laboratory. Objective studies of sleep among adolescents have demonstrated mixed findings. Some studies report longer sleep latency and wake after sleep onset among those with insomnia than those without, while others report no difference between the two groups.

Serial monitoring with PSG or actigraphy in individuals with insomnia typically shows marked night-to-night variability in all sleep measures and recorded bed and rising times, compared to good sleepers. Studies using actigraphy have not found reliable parameters to differentiate people with insomnia from good sleepers.

Results of the MSLT usually show normal daytime alertness. In several studies, patients with chronic insomnia disorder have longer mean MSLT values (i.e., less sleepiness) than control subjects, suggesting hyper-alertness or hyperarousal. A minority of insomnia patients, particularly older adults with chronic insomnia disorder, have reduced mean MSLT values indicating increased sleepiness. Such a finding should prompt consideration of other concurrent sleep disorders such as obstructive sleep apnea.

Patients with chronic insomnia disorder can also be differentiated from good sleepers on several other physiological measures, including cortisol levels and rhythmicity, heart rate/sympathetic activity, blood pressure, and metabolic rate, among others (see above). Some of these studies find more significant

physiological abnormalities among insomnia patients with objective short sleep duration and improvement in some of these parameters with treatment.

Patients with insomnia frequently complain of daytime cognitive impairments. Studies generally support small to moderate deficits in the focused cognitive domains of complex attention, problem-solving, episodic memory, and manipulation/retention in working memory.

Finally, an increasing number of structural and functional controlled imaging studies have been conducted during sleep and wakefulness in insomnia. Findings across these studies provide some support, though inconsistent, for the hyperarousal model of insomnia. In addition, structural magnetic resonance imaging (MRI) studies suggest other factors such as gray matter atrophy in the orbitofrontal and temporal areas of the cortex, precuneus, and hippocampus, and volume increase in the anterior cingulate cortex (ACC) in patients with chronic insomnia disorder compared to good sleepers. Controlled functional imaging studies suggest regionally specific alterations of relative glucose metabolism in chronic insomnia disorder. Specifically, these studies show smaller wake to NREM decreases of relative glucose metabolism in sleep/wake-regulating regions, including the thalamus, upper brainstem, anterior cingulate, and limbic cortex. Self-reported and objective sleep disruptions correlate with increased relative metabolism in these regions. These findings are similar to regional activation patterns during sleep in an animal model of stress-induced insomnia. Other studies using nuclear magnetic resonance spectroscopy have identified reduced gamma-aminobutyric acid (GABA) signaling in sleep-regulating regions in insomnia that correlates with objective measures of sleep continuity. Studies examining task-related changes in blood flow using blood oxygenation level-dependent functional magnetic resonance imaging (BOLD fMRI) paradigms have shown reduced activation of specific brain regions in individuals with chronic insomnia disorder compared with good sleeper controls. Task-related activation changes in the direction of "normalization" following cognitive behavioral treatment. Studies using newer imaging techniques and analytic approaches suggest alterations in the structure and connectivity of brain networks in patients with insomnia compared to good sleepers. Still, there remains a lack of convergent findings between structural and functional disturbances among insomnia patients, resulting from heterogeneity in sample populations and methodological approaches.

Differential Diagnosis

Circadian rhythm sleep disorders Sleep initiation problems are present in individuals with *delayed sleep-wake phase disorder (DSWPD).* Among those with *DSWPD*, sleep initiation is consistently later than desired or required because the individual's endogenous circadian rhythm is delayed relative to the desired bedtime. Sleep-onset difficulties develop when they attempt to initiate sleep earlier than allowed for sleep onset by the delayed endogenous circadian rhythm. Unlike patients with a chronic insomnia disorder, patients with *DSWPD* are typically able to fall asleep with less difficulty and sleep a normal amount of time when sleeping in phase with their delayed endogenous rhythm by selecting late bed and rising times. By comparison, those with a chronic insomnia disorder are unable to sleep regardless of the timing of their bed and rise times. Given the prevalence of delayed sleep-wake phase disorder in teenagers

and young adults, it is essential to consider the possibility of *DSWPD* as an alternate or comorbid diagnosis when evaluating individuals from these age groups presenting with sleep-onset difficulties. It is estimated that a little over half of adolescents with delayed sleep-wake phase disorder have comorbid insomnia symptoms.

A chronic insomnia disorder that presents as difficulty maintaining sleep with premature morning rise times should be differentiated from a*dvanced sleep-wake phase disorder*. Among those with an advanced sleep-wake phase disorder, sleep initiation is consistently earlier than desired because the individual's endogenous circadian rhythm is advanced relative to the desired sleep schedule. However, total sleep time is adequate when the individual chooses early bed and rise times that coincide with the advanced endogenous circadian rhythm. In contrast, those with chronic insomnia disorder may display sleep-maintenance difficulties with premature morning rise time regardless of the sleep schedule they select. An early sleep-wake pattern tends to be more common in older adults.

There can be an overlap between chronic insomnia disorder and both d*elayed and advanced sleep-wake phase disorders*. For example, patients with a delayed sleep pattern may become chronically frustrated or anxious about their inability to initiate sleep at their desired times. This frustration or anxiety may disrupt sleep and delay sleep onset well beyond the time supported by the endogenous rhythm. Similarly, individuals with an advanced sleep pattern may experience early morning awakenings that are even earlier than dictated by their endogenous rhythm. In such circumstances, both a circadian rhythm sleep-wake disorder diagnosis and a chronic insomnia disorder diagnosis may apply and should be assigned.

Sleep-disruptive environmental circumstances A variety of environmental factors, including excessive noise or light and extreme temperatures, will disrupt the sleep of most individuals. Also, sleeping in an area where there is an imminent threat or danger to one's safety can be disruptive to sleep. Bed partners who snore loudly, move excessively during sleep, or have parasomnias may also disrupt one's sleep. When an individual reports environmental circumstances that would be regarded as disruptive to the sleep of most individuals, yet sleep well in another less disruptive environments, a chronic insomnia disorder should not be assigned. Chronic insomnia disorder only applies when the individual reports sleep difficulty in the context of a sleep-conducive environment or when the insomnia symptoms show some independence from the environmental factors. When environmental circumstances are a primary cause of sleep disturbance, a diagnosis of *other sleep disorder* may apply.

Chronic insufficient sleep syndrome Chronic insomnia disorder should also be differentiated from inadequate sleep duration due to work and other responsibilities or social and recreational schedules. Daytime sleepiness and fatigue typically occur as a result of insufficient sleep. However, when these individuals allow sufficient time to sleep, they can initiate and maintain sleep easily and for a normal duration. In contrast, those with chronic insomnia disorder have difficulties initiating and maintaining sleep and reduced sleep duration despite routinely allotting sufficient time to sleep. Moreover, unless other comorbid sleep disorders are present, chronic insomnia disorder is not typically associated with the excessive daytime sleepiness and unintentional daytime sleep episodes often observed in those with voluntary sleep restriction.

Sleep-related breathing disorders *Obstructive sleep apnea* may provoke sleep-onset or maintenance difficulties. Daytime sleepiness that routinely accompanies obstructive sleep apnea may be misattributed to these insomnia symptoms and mask sleep apnea. Chronic insomnia disorder may co-occur with obstructive sleep apnea, but the critical distinguishing features of objective daytime sleepiness and snoring are hallmarks of obstructive sleep apnea and are not common in chronic insomnia disorder. Individuals complaining of insomnia symptoms will often report being very sleepy during the day, but careful assessment will reveal that this is a complaint of fatigue rather than sleepiness. The Epworth Sleepiness Scale may help discriminate between sleepiness and fatigue. The presence of marked daytime sleepiness suggests that another sleep disorder, most often obstructive sleep apnea, may be present and justifies further assessment, including sleep testing. If a diagnosis of sleep apnea is confirmed, this does not necessarily rule out the presence of an independent chronic insomnia disorder. Ongoing assessment following treatment of the obstructive apnea may be required to determine if a persistent insomnia disorder is present. Patients with *central sleep apnea* may also complain of insomnia symptoms. These patients typically manifest related comorbid conditions such as heart failure, neurologic disorders, or opioid use, which give rise to central sleep apnea.

Narcolepsy Many patients with narcolepsy experience sleep-maintenance difficulties. Some may mistakenly attribute their daytime sleepiness to nighttime sleep disturbance. The presence of pronounced daytime sleepiness merits further investigation and sleep testing.

Unresolved Issues and Further Directions

Further research that elucidates the pathophysiology of chronic insomnia disorder will also shed light on the persistent issue of subtyping the disorder; specifically, whether the current unitary diagnostic approach should be maintained or replaced by well-defined subtypes. Although the consensus to date has favored the unitary approach, there remains serious consideration that chronic insomnia disorder may, in fact, represent multiple separate disorders with similar outward presentations. If the latter is the case, then the designation of subtypes can guide the development of novel interventions. For example, it will be important to determine if paradoxical insomnia represents the extreme end of the distribution of subjective-objective discrepancy in sleep outcome measures among individuals with chronic insomnia disorder or a distinct subtype of insomnia that may need specialized treatment. Similarly, recent attention has been given to insomnia with short sleep duration as a potential novel subtype. It will be critical to clarify if this presentation represents merely the extreme end of hyperarousal in chronic insomnia disorder or a distinct subtype. In the latter case, it will be essential to know how to reliably define “chronic insomnia disorder with short sleep duration,” both in terms of sleep duration cutoff and the need for polysomnography to ascertain the diagnosis.

Additional research is also needed to clarify how best to define chronic insomnia disorder in the pediatric population. There are apparent developmental factors unique to the causes and expression of chronic insomnia disorder from early childhood to adolescence. As research into the etiology of chronic insomnia

disorder in these age groups grows, it will help to clarify if a diagnosis of behavioral insomnia of childhood, which was present in the previous editions of the International Classification of Sleep Disorders, should be re-instated.

Previous editions of the International Classification of Sleep Disorders allowed for the assignment of an insomnia diagnosis to patients who present exclusively with complaints of nonrestorative sleep; that is, patients who complain only of sleep that is poor in quality or unrefreshing, in the absence of sleep -onset or maintenance complaints. The third edition of this nosology excludes such patients from the insomnia diagnostic categories. This decision was based on several considerations. Most insomnia patients present with complaints of sleep-initiation or maintenance difficulties. Although a small subset of patients presents solely with complaints of nonrestorative sleep, this complaint more commonly arises in association with other symptoms of insomnia or in conjunction with -sleep-related breathing disorders, other sleep disorders, or certain chronic medical conditions (e.g., fibromyalgia or chronic fatigue syndrome). Several epidemiological studies have identified subgroups of individuals that present solely with a complaint that their sleep is nonrestorative or unrefreshing. These studies typically did not include polysomnography to rule out other occult sleep disorders that might explain this presentation. Finally, a complaint of nonrestorative sleep is based mainly on daytime symptoms such as fatigue or anergia. However, it remains clinically challenging to determine whether these symptoms are attributable to disturbed sleep or another non-sleep-related cause, particularly in the absence of sleep testing. Given these considerations, the isolated complaint of nonrestorative sleep was not retained as part of the diagnostic definition of chronic insomnia disorders delineated in this chapter.

In addition to pathophysiologic considerations, it will also be essential to gain better insight into cross-cultural differences in the presentation of chronic insomnia disorder and their potential relevance to health outcomes and treatment.

Bibliography

Baglioni C, Regen W, Teghen A, et al. Sleep changes in the disorder of insomnia: a meta-analysis of polysomnographic studies. *Sleep medicine reviews*. 2014;18(3):195-213.

Barclay NL, Kocevska D, Bramer WM, Van Someren EJ, Gehrman P. The heritability of insomnia: A meta-analysis of twin studies. *Genes, Brain and Behavior*. 2020;20(4): e12717.

Bastien CH, Morin CM. Familial incidence of insomnia. *Journal of sleep research*. 2000;9(1):49-54.

Beaulieu-Bonneau S, LeBlanc M, Mérette C, Dauvilliers Y, Morin CM. Family history of insomnia in a population-based sample. *Sleep*. 2007;30(12):1739-45.

Bei B, Wiley JF, Trinder J, Manber R. Beyond the mean: a systematic review on the correlates of daily intraindividual variability of sleep/wake patterns. *Sleep medicine reviews*. 2016;28:108-24.

Bonnet MH, Burton GG, Arand DL. Physiological and medical findings in insomnia: implications for diagnosis and care. *Sleep medicine reviews*. 2014;18(2):111-22.

Chaput J-P, Yau J, Rao DP, Morin CM. Prevalence of insomnia for Canadians aged 6 to 79. *Health reports*. 2018;29(12):16-21.

Edinger JD, Wyatt JK, Stepanski EJ, et al. Testing the reliability and validity of DSM-IV-TR and ICSD-2 insomnia diagnoses: results of a multitrait-multimethod analysis. *Archives of general psychiatry*. 2011;68(10):992-1002.

Ellis JG, Perlis ML, Espie CA, et al. The natural history of insomnia: predisposing, precipitating, coping, and perpetuating factors over the early developmental course of insomnia. *Sleep*. 2021;44(9):zsab095.

Espie CA, Kyle SD, Hames P, Cyhlarova E, Benzeval M. The daytime Impact of DSM-5 insomnia disorder: comparative analysis of insomnia subtypes from the great British sleep survey. *The Journal of clinical psychiatry*. 2012;73(12):0-0.

Fernandez-Mendoza J, Bourchtein E, Calhoun S, et al. Natural history of insomnia symptoms in the transition from childhood to adolescence: population rates, health disparities, and risk factors. *Sleep*. 2021;44(3):zsaa187.

Flynn-Evans EE, Shekleton JA, Miller B, et al. Circadian phase and phase angle disorders in primary insomnia. *Sleep*. 2017;40(12):zsx163.

Gehrman P, Sengupta A, Harders E, Ubeydullah E, Pack AI, Weljie A. Altered diurnal states in insomnia reflect peripheral hyperarousal and metabolic desynchrony: a preliminary study. *Sleep*. 2018;41(5):zsy043.

Jarrin DC, Alvaro PK, Bouchard M-A, Jarrin SD, Drake CL, Morin CM. Insomnia and hypertension: a systematic review. *Sleep medicine reviews*. 2018; 41: 3-38.

Juan J. Madrid-Valero, María Rubio-Aparicio, Alice M. Gregory, Julio Sanchez-Meca, Juan R. Ordonana (2021). The heritability of insomnia: Systematic review and meta-analysis of twin studies. Sleep Medicine Reviews, 2021,58:1-9.

Levenson JC, Kay DB, Buysse DJ. The pathophysiology of insomnia. *Chest*. 2015;147(4):1179-92.

Logan RW, Hasler BP, Forbes EE, et al. Impact of sleep and circadian rhythms on addiction vulnerability in adolescents. *Biological psychiatry*. 2018;83(12):987-96.

Mindell JA, Sadeh A, Wiegand B, How TH, Goh DY. Cross-cultural differences in infant and toddler sleep. *Sleep medicine*. 2010;11(3):274-80.

Morin CM, Bélanger L, LeBlanc M, et al. The natural history of insomnia: a population-based 3-year longitudinal study. *Archives of internal medicine*. 2009;169(5):447-53.

Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep medicine reviews*. 2002;6(2):97-111.

Riemann et al. (2015). The neurobiology, investigation, and treatment of chronic insomnia. Lancet, 14, 547-558.

Roberts RE, Ramsay Roberts C, Chan W. Persistence and change in symptoms of insomnia among adolescents. *Sleep*. 2008;31(2):177-84.

Sánchez-Ortuño MM, Edinger JD. Internight sleep variability: its clinical significance and responsiveness to treatment in primary and comorbid insomnia. *Journal of Sleep Research*. 2012;21(5):527-34.

Smith MT, McCrae CS, Cheung J, et al. Use of actigraphy for the evaluation of sleep disorders and circadian rhythm sleep-wake disorders: an American Academy of Sleep Medicine clinical practice guideline. Journal of Clinical Sleep Medicine. 2018;14(7):1231-37.

Spiegelhalder K, Regen W, Baglioni C, Nissen C, Riemann D, Kyle SD. Neuroimaging insights into insomnia. *Current neurology and neuroscience reports*. 2015;15(3):9.

Touitou Y, Touitou D, Reinberg A. Disruption of adolescents' circadian clock: The vicious circle of media use, exposure to light at night, sleep loss and risk behaviors. *Journal of Physiology-Paris*. 2016;110(4):467-79.

Zhao W, Van Someren EJ, Li C, et al. EEG spectral analysis in insomnia disorder: A systematic review and meta-analysis. *Sleep Medicine Reviews*. 2021:101457.

Short-Term Insomnia Disorder

ICD-9-CM code: 307.41

ICD-10-CM code: F51.02

Alternate Names

Acute insomnia, adjustment insomnia.

Diagnostic Criteria

Criteria A-E must be met

A. The patient reports, or the patient's parent or caregiver observes, one or more of the following:[1,2]
    1. Difficulty initiating sleep.
    2. Difficulty maintaining sleep.
    3. Waking up earlier than desired.
    4. Resistance to going to bed on an appropriate schedule.
    5. Difficulty sleeping without parent or caregiver intervention.
B. The patient reports, or the patient's parent or caregiver observes, one or more of the following related to the nighttime sleep difficulty:
    1. Fatigue/malaise.
    2. Impaired attention, concentration, or memory.
    3. Impaired social, family, vocational, or academic performance.
    4. Mood disturbance/irritability.

5. Subjective daytime sleepiness.[3]
6. Behavioral problems (e.g., hyperactivity, impulsivity, aggression).
7. Reduced motivation/energy/initiative.
8. Proneness for errors/accidents.
9. Concerns about or dissatisfaction with sleep.

C. The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e., time allotted for sleep) or inadequate circumstances (i.e., safety, darkness, quiet, and comfort) for sleep.
D. The sleep disturbance and associated daytime symptoms have been present for less than three months.
E. The sleep disturbance and associated daytime symptoms are not solely due to another current sleep disorder, medical disorder, mental disorder, or medication/substance use.[4,5]

Notes

1. Reports of difficulties initiating sleep, difficulties maintaining sleep, or waking up too early can be seen in all age groups. Resistance to going to bed on an appropriate schedule and difficulty sleeping without parent or caregiver intervention is seen most commonly in children and older adults who require the supervision of a caretaker due to a significant level of functional impairment (e.g., those with dementia).
2. The assessment of short-term insomnia disorder in children and adolescents should include information from both the child and their caregiver because the caregiver may not have complete knowledge about the child's experience of sleep difficulties, and the child may not have full awareness of the daytime consequences of their sleep difficulties.
3. Patients tend to use the terms sleepiness and fatigue interchangeably. Therefore, the assessment of subjective daytime sleepiness requires distinguishing the two symptoms. Fatigue refers to low physical or mental energy, while sleepiness refers to the propensity to fall asleep in conducive circumstances.
4. Many conditions such as grief, acute pain, or other acute stressors are often associated with insomnia symptoms. When such conditions are the sole cause of the sleep difficulty, a separate insomnia diagnosis may not apply. The primary factor in determining the application of a short-term or adjustment insomnia diagnosis is the extent to which the sleep disturbance becomes a significant source of distress and hence warrants independent clinical attention.
5. Regular users of hypnotic medications who sleep well when they take the medications should still be assigned a diagnosis of short-term insomnia disorder. They may present clinically with concerns about their inability to sleep without sleep medications.

Essential Features

The essential feature of short-term insomnia disorder is recently emergent difficulty initiating or maintaining sleep that results in general sleep dissatisfaction or perceived impairment, reported by the patient or a caregiver. Daytime distress about poor sleep or impairment in family, social, occupational,

academic, or other important functions accompanies the sleep complaint. Furthermore, the sleep disturbance and associated daytime symptoms occur despite having adequate time and circumstances each night to obtain necessary sleep. Short-term insomnia disorder can occur in isolation or may be comorbid with a mental disorder, medical condition, or substance use. In many cases of short-term insomnia, there is an identifiable cause that serves as the precipitant. In other cases, insomnia occurs episodically, often coincident with daytime stressors that account for the sleep disturbance.

Short-term insomnia disorder may be characterized solely by sleep-onset or maintenance complaints, or both. Individuals' sleep complaints may vary, such that sleep-onset difficulties are apparent on some nights, whereas sleep-maintenance difficulties are present on other nights. Complaints about poor-quality, unrefreshing, or nonrestorative sleep may accompany sleep-onset and maintenance complaints but do not meet the definition of this condition when they occur in isolation.

The waking symptoms in short-term insomnia disorder are, likely, similar to those of chronic insomnia disorder, although limited data are available.

Associated Features

When insomnia arises in reaction to a stressful life event, such as the loss of a loved one, major illness, or divorce, the associated features may include anxiety, worry, ruminative thoughts, sadness, or depression related to the specific stressor. In addition, if the individual uses alcohol, illicit drugs, or medications for self-treatment, additional symptoms related to these substances may be present.

Clinical and Pathophysiological Subtypes

None.

Demographics

The exact prevalence of short-term insomnia disorder is unknown. The one-year prevalence of short-term insomnia disorder among adults appears to be in the range of 15% to 20%. Like chronic insomnia disorder, short-term insomnia disorder is more prevalent in women than in men and in older age groups.

Predisposing and Precipitating Factors

Affected individuals may note a lifelong tendency toward light sleep or difficulty sleeping during times of stress. A previous history of anxiety or depressive symptoms and disorders may also predispose an individual to develop a short-term insomnia disorder. Concerning children, caregivers who have unrealistic sleep expectations for their children may predispose them to insomnia by putting them in bed

too early or assigning them too much time in bed each night. Similarly, parents and caregivers of young children who fail to set limits on the child's sleep behaviors also create an unstructured sleep environment that predisposes young children to develop insomnia.

An acute, identifiable event or stressor usually precipitates this form of insomnia. Common precipitating events include changes or disputes in interpersonal relationships, occupational stress, personal losses, bereavement, diagnosis, or onset of a new medical condition, visiting or moving to a new location, or physical changes to the usual sleep environment or schedule. Changes or stresses with a positive emotional valence also may serve as precipitating events. In children or dependent adults, short-term insomnia can arise when the caretaker makes abrupt changes in the child's or adult's sleep routines or schedule.

Familial Patterns

Familial patterns are less well documented for cases of short-term insomnia disorder than they are for individuals meeting the criteria for chronic insomnia disorder. Nonetheless, the familial aggregation found for chronic insomnia disorders is likely to occur among individuals with more transient forms of insomnia. Data support a greater genetic diathesis to psychophysiological arousal among specific individuals in response to stressors. This diathesis may, in turn, suggest a constitutional predisposition to short-term insomnia in such individuals.

Onset, Course, and Complications

Many individuals who develop short-term insomnia disorder experience remission of their insomnia symptoms over time. This occurs as distress over the precipitating event diminishes, or the stressor resolves. However, a portion of those who initially experience short-term insomnia may develop a more chronic form of insomnia and subsequently meet the criteria for chronic insomnia disorder. These individuals will then have the course and complications described for chronic insomnia disorder.

Developmental Issues

As is the case for chronic insomnia disorder, short-term insomnia disorder should not be considered until after a child reaches at least six months of age.

The propensity to experience sleep disturbance in response to stress may dispose young and older individuals toward acute forms of insomnia. As is the case for chronic insomnia disorder, sleep-onset difficulties are most common among young adults with short-term insomnia. In contrast, sleep-maintenance complaints and early morning awakening are more common in middle-aged and older adults. Certain developmental life transitions may be associated with short-term insomnia disorder. For

example, balancing job and family stressors may contribute to intermittent short-term insomnia problems in younger and middle-aged adults. In contrast, older adults may have short-term insomnia disorder related to exacerbations in comorbid chronic medical conditions or due to loss of loved ones that commonly occurs with advancing age.

Pathology and Pathophysiology

Research on the pathology and pathophysiology of short-term variants of insomnia disorder is scant because most studies addressing these issues have focused on samples with chronic insomnia disorder.

Objective Findings

Systematic research into objective outcomes associated with short-term insomnia disorder has not been undertaken because studies have generally included only samples of individuals with chronic insomnia disorder.

Differential Diagnosis

Chronic insomnia disorder Short-term insomnia disorder shares many features with *chronic insomnia disorder*. The primary differences between the two conditions are the duration and frequency criteria. Unlike chronic insomnia disorder, the duration of short-term insomnia disorder is less than three months, and the diagnosis has no frequency criterion.

Circadian rhythm sleep-wake disorders Short-term insomnia disorder should also be distinguished from circadian rhythm sleep-wake disorders resulting from rotating *shift work* and *jet lag.* The sleep disturbance associated with shift work and jet lag arises from a sleep-wake schedule alteration that results in a mismatch between the endogenous circadian rhythm and the desired or required sleep-wake schedule - for example, having to sleep in the daytime rather than during the nighttime. In the case of short-term insomnia disorder, no such mismatch exists; yet the individual demonstrates sleep-onset or maintenance difficulties.

Bibliography

Johnson KA, Gordon CJ, Chapman JL, et al. The association of insomnia disorder characterised by objective short sleep duration with hypertension, diabetes and body mass index: A systematic review and meta-analysis. *Sleep Medicine Reviews*. 2021:101456.

Vgontzas AN, Fernandez-Mendoza J, Liao D, Bixler EO. Insomnia with objective short sleep duration: the most biologically severe phenotype of the disorder. *Sleep medicine reviews*. 2013;17(4):241-54.

Other Insomnia Disorder

ICD-9-CM code: 307.49

ICD-10-CM code: F51.09

This diagnosis is reserved for individuals who complain of difficulty initiating and maintaining sleep and do not meet the full criteria for chronic or short-term insomnia disorder. In some cases, this diagnosis may be assigned on a provisional basis when more information is needed to establish a diagnosis of chronic insomnia disorder or short-term insomnia disorder. However, this diagnosis should be used sparingly, given its nonspecific nature.

Isolated Symptoms and Normal Variants

Excessive Time in Bed

Some individuals may report prolonged sleep latency or long periods of wakefulness during the night, yet not complain of insomnia nor show daytime impairments. In children and cognitively impaired adults, this pattern may emerge when parents or caregivers have unrealistic expectations for the sleep needs and routinely allot too much time in bed for the child or adult they care for. In adults without cognitive impairment, this pattern is perhaps most common in noncomplaining groups who routinely allocate significantly more time in bed than needed for sleep. For example, some retired or unemployed individuals may routinely spend excessive time in bed each night and are not bothered by the expanded periods of wakefulness they routinely experience. Adverse consequences similar to those reported for chronic insomnia disorder have not been described in association with excessive time in bed.

Short Sleeper

Some individuals routinely obtain less than six hours of sleep per night yet have no sleep/wake complaints and show no apparent daytime dysfunction. Such individuals are considered normal short sleepers. Among these individuals, the observed relatively low average sleep duration does not result from chronic volitional sleep restriction, as in the case of insufficient sleep syndrome, but rather indicates a constitutional disposition for reduced sleep requirement. The clinical significance and possible subtypes of chronic short sleep duration remain undetermined. Various epidemiological studies have linked short sleep duration with metabolic, cardiovascular, and other forms of medical morbidity. However, these studies typically do not distinguish between individuals who have short sleep in the context of insomnia

or another sleep disorder, those who voluntarily restrict their sleep, and those who may have a naturally short sleep. Short sleep resulting from different causes may have different pathophysiological significance. At present, those who demonstrate less than six hours of sleep per night should not be assigned an insomnia diagnosis unless they also meet the criteria for one of the insomnia disorder diagnoses described above.