

Insomnia: prevalence, consequences and effective treatment

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Insomnia is a very common disorder that has significant long-term health consequences. Australian population surveys have shown that 13%–33% of the adult population have regular difficulty either getting to sleep or staying asleep.^{1,2} Insomnia can occur as a primary disorder or, more commonly, it can be comorbid with other physical or mental disorders. Around 50% of patients with depression have comorbid insomnia, and depression and sleep disturbance are, respectively, the first and third most common psychological reasons for patient encounters in general practice.³ Insomnia doubles the risk of future development of depression, and insomnia symptoms together with shortened sleep are associated with hypertension.^{4,5}

Insomnia is defined in the fifth edition of the *Diagnostic and statistical manual of mental disorders* (DSM-5) as difficulty getting to sleep, staying asleep or having non-restorative sleep despite having adequate opportunity for sleep, together with associated impairment of daytime functioning, with symptoms being present for at least 4 weeks.⁶ Having a sleep experience that does not meet our expectation, such as with some transient awakenings but with good daytime functioning, does not constitute insomnia.

Acute versus chronic insomnia

Acute insomnia is defined as sleep disturbance meeting the DSM-5 definition of insomnia, but with symptoms occurring for less than 4 weeks.⁶ Generally, acute insomnia is triggered by precipitating events such as ill health, change of medication or circumstances, or stress. Once the precipitating event passes, sleep settles back to its usual pattern. Hence, treatment for acute insomnia is focused on avoiding or withdrawing the precipitant, if possible, and supporting the acute distress of not sleeping with short-term use of hypnotics if symptoms are significant. This is the usual approach in primary care, with 95% of general practitioner consultations for insomnia resulting in the prescription of a hypnotic, usually a benzodiazepine.⁷

However, if patients have repeated episodes of acute insomnia or ongoing comorbidities, insomnia symptoms can persist and evolve into chronic insomnia, which requires a different treatment approach. Once people have had difficulty sleeping for over 4 weeks, they have usually begun to behave and think about sleep differently, in ways that are maladaptive and perpetuate their sleep difficulties.⁸ The long-term course is then generally one of relapse and remission rather than resolution,⁹ which continues well after the acute precipitating circumstances have passed. Therefore, the treatment approach needs to match this, with a chronic disease management model educating and upskilling patients on how best to manage their insomnia symptoms over time. Health care

Summary

- Insomnia is common and can have serious consequences, such as increased risk of depression and hypertension.
- Acute and chronic insomnia require different management approaches.
- Chronic insomnia is unlikely to spontaneously remit, and over time will be characterised by cycles of relapse and remission or persistent symptoms.
- Chronic insomnia is best managed using non-drug strategies such as cognitive behaviour therapy.
- For patients with ongoing symptoms, there may be a role for adjunctive use of medications such as hypnotics.

providers need to see insomnia as a chronic illness and emphasise the role of strategies to prevent relapses, rather than focusing on treatment of acute episodes or crises.

Assessment and diagnosis of insomnia

The assessment and diagnosis of insomnia is formulated mainly from a systematic sleep history. To assist in establishing premorbid baseline sleeping patterns and formulating treatment goals, clinicians must ask patients about their typical sleeping pattern before they developed insomnia.

Insomnia assessment involves understanding the patient's typical sleep pattern at night and over a time frame of weeks to months. Therefore, part of the sleep assessment is asking for the patient's narrative of typical bedtime, time taken to fall asleep after lights out (sleep latency), frequency and rough duration of awakenings in the middle of the night, and what time the patient gets out of bed. Are there times when sleep returns to normal? Was there an initial trigger or did the symptom arise spontaneously? Was it related to a period of stress, anxiety or depression? Did it start during childhood and continue thereafter? Are there lifestyle factors contributing to insomnia, such as too much caffeine or exercise late in the day, television or pets in the bedroom, or use of alcohol or nicotine? Knowing the patient's cognitions, beliefs and worries about sleep, which are often apparent in the language and emotion used when they describe their sleep, can assist in the formulation of specific behavioural and calming approaches to assist with sleep.

It is important to assess the effects of poor sleep on the patient. Common daytime consequences include mood lowering, irritability, poor memory, fatigue, lack of energy and general malaise. These can manifest as work absenteeism, with insomnia being one of its leading medical causes.¹⁰ It is also imperative to ask for risky consequences of insomnia, including accidents and sleepiness while driving.

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Identifying the body clock type of the patient is crucial in excluding circadian rhythm disorders. A commonly undiagnosed condition, delayed sleep phase disorder is a body clock variation where the patient is biologically inclined to go to sleep much later than usual (typically after midnight), yet generally sleeps well after sleep onset, with a natural wake time that is much later than for most people and is often incompatible with normal school or work start times.

It is also important to look for comorbid conditions that can present with insomnia, such as depression and anxiety, chronic medical conditions, and other sleep disorders. Comorbid conditions have a bidirectional relationship with insomnia, with each influencing or exacerbating the other and requiring concurrent assessment and management. The Auckland Sleep Questionnaire, a validated sleep screening questionnaire in primary care, is one tool that can assist in identifying these disorders.¹¹ Other validated questionnaires such as the Insomnia Severity Index can help to document the severity of patients' symptoms and assess their response to treatment.¹²

Since many people with insomnia overestimate their sleep disruption and underestimate actual sleep time, a 2-week sleep diary is a very helpful assessment tool as it assists the sleep clinician to get a more accurate snapshot of sleep compared with a pure verbal account.¹³ For some, a sleep diary is revealing in that they realise that they do get some sleep, albeit fragmented or superficial. This can provide the basis for discussion. There are several downloadable sleep diaries online — for example, <http://yoursleep.aasmnet.org/pdf/sleepdiary.pdf>. If patients have difficulty completing a sleep diary, or there is significant misperception of sleep suspected, actigraphy (using a device worn on the wrist to monitor sleep-wake cycles) can be used to objectively measure sleep.

Although an overnight sleep study or polysomnography is not routinely indicated in diagnosing insomnia, it can be helpful in diagnosing several conditions, including obstructive sleep apnoea, sleep-related movement disorders, parasomnias, or insomnias that are treatment-resistant.¹³ A routine physical and mental status examination can give clues regarding comorbid medical and or mental health conditions. Other tests including laboratory and radiographic procedures are not routinely indicated in chronic insomnia.¹³

Non-pharmacological treatment of insomnia

Cognitive behaviour therapy aimed at treating insomnia (CBT-i) targets maladaptive behaviour and thoughts that may have developed during insomnia or have contributed to its development. CBT-i is considered to be the gold standard in treating insomnia, with effect sizes similar to or greater than those seen with hypnotic drugs and, unlike with hypnotics, maintenance of effect after cessation of therapy.^{14,15} These effects are seen in both primary and comorbid insomnia.¹⁶

The implementation of individual face-to-face CBT-i is typically delivered by a trained health professional, which

makes it expensive, labour intensive and therefore beyond the reach of many. Patients with insomnia are eligible for Medicare rebates for psychological treatment if they are referred under the Chronic Disease Management or Better Access to Mental Health Care initiatives. Telephone and online delivery of CBT-i have been shown in clinical trials to be as effective as face-to-face CBT-i.^{17,18} While these different treatment delivery models have the potential to markedly improve access to CBT-i, they need to be investigated further with respect to their long-term reliability and effectiveness. They might be best used as part of a stepped-care approach.¹⁹ Some patients may need little guidance, while others may need more personal treatment and guidance.

CBT-i consists of five major components: stimulus control, sleep restriction (also known as sleep consolidation or bed restriction), relaxation techniques, cognitive therapy and sleep hygiene education (Box). Typically, CBT-i is delivered in four to 10 sessions, either individually or in a group setting, ideally involving four to eight participants.

Stimulus control is a reconditioning treatment forcing discrimination between daytime and sleeping environments.²⁰ For the poor sleeper, the bedroom triggers associations with being awake and aroused. Treatment involves removing all stimuli that are potentially sleep-incompatible (reading, watching television and use of computers) and excluding sleep from living areas. The individual is instructed to get up if he or she is not asleep within 15–20 minutes, or when wakeful during the night or experiencing increasing distress, and not return to bed until feeling sleepy.

Sleep restriction relates to better matching the time spent in bed to the average nightly sleep duration.²¹ Patients keep a sleep diary to determine average sleep duration. They are then allowed a period of time in bed equal to this plus 30 minutes, and set a regular arising time. As some patients can underperceive the amount of sleep, the time in bed should never be set at less than 5 hours. As sleep becomes more consolidated, the length of time in bed can be gradually increased in 15–30 minute increments. This effective intervention induces natural sleepiness (reduced time in bed) and gives the individual a sense of assurance that bed is now a safe place to sleep. Bed restriction has recently been shown to be an effective intervention in primary care.²²

Relaxation techniques include progressive relaxation, imagery training, biofeedback, meditation, hypnosis and autogenic training, with little evidence to indicate superiority for any one approach. Patients are encouraged to practice relaxation techniques throughout the day and early evening. Even a few minutes two to four times a day is useful. A last-minute relaxation attempt minutes before sleep will not work miracles. Muscular tension and cognitive arousal (eg, a “chattering” mind) are incompatible with sleep. At the cognitive level, these techniques may act by distraction. Relaxation reduces physical and mental arousal but is less effective as a stand-alone treatment and is better used in combination with other treatment interventions.

Cognitive behaviour therapy for insomnia		
Intervention	General description	Specific instructions
Stimulus control	BED = SLEEP. Set of instructions aimed at conditioning the patient to expect that bed is for sleeping and not other stimulating activities. Only exception is sexual activity. Aim is to promote a positive association between bedroom environment and sleepiness	Go to bed only when sleepy/comfortable and intending to fall asleep. If unable to sleep within what feels like 15–20 minutes (without watching the clock), leave the bed and bedroom and go to another room and do non-stimulating activity. Return to bed only when comfortable enough to sleep again. Do not read, watch television, talk on phone, pay bills, use electronic social media, worry or plan activities in bed
Sleep-restriction therapy	Increases sleep drive and reduces time in bed lying awake. Limits the time in bed to match the patient's average reported actual sleep time. Slowly allows more time in bed as sleep improves	Set strict bedtime and rising schedule, limited to average expected hours of sleep reported in the average night. Increase time in bed by 15–30 minutes when the time spent asleep is at least 85% of the allowed time in bed. Keep a fixed wake time, regardless of actual sleep duration
Relaxation techniques	Various breathing techniques, visual imagery, meditation	Practise progressive muscle relaxation (at least daily). Take shorter relaxation periods (2 minutes) a number of times per day. Use breathing and self-hypnosis techniques
Cognitive therapy	Identifies and targets beliefs that may be interfering with adherence to stimulus control and sleep restriction. Uses mindfulness to alter approach to sleep	Unhelpful beliefs can include overestimation of hours of sleep required each night to maintain health; overestimation of the power of sleeping tablets; underestimation of actual sleep obtained; fear of stimulus control or sleep restriction for fear of missing the time when sleep will come
Sleep hygiene education	Emphasises environmental factors, physiological factors, behaviour, habits that promote sound sleep	Avoid long naps in daytime — short naps (less than half an hour) are acceptable. Exercise regularly. Maintain regular sleep–wake schedule 7 days per week (particularly wake times). Avoid stimulants (caffeine and nicotine). Limit alcohol intake, especially before bed. Avoid visual access to clock when in bed. Keep bedroom dark, quiet, clean and comfortable

Cognitive therapy involves enabling the patient to recognise how unhelpful and negative thinking about sleep increases physiological and psychological arousal levels. Setting aside 15–20 minutes in the early part of the evening to write down any worries, make plans for the following day and address any concerns that might arise during the night allows the day to be put to rest. It is helpful to challenge thoughts that arise at night with “I have already addressed this and now I can let go of it!”. “Time out” — some form of soothing activity before bed — can be useful in reducing arousal levels. Thought-stopping attempts or blocking techniques, such as repeating the word “the” every 3 seconds, occupy the short-term memory store (used in processing information), potentially allowing sleep to happen. Cognitive restructuring challenges unhelpful beliefs, such as “if I don’t get enough sleep tonight, tomorrow is going to be a disaster”, which maintain both wakefulness and helplessness. Another cognitive and behavioural technique is paradoxical intention. Clients are encouraged to put the effort into remaining wakeful rather than trying to fall asleep (decatastrophising), thereby strengthening the sleep drive and reducing performance effort.¹⁴

There is limited evidence to suggest that, on its own, sleep hygiene is efficacious.¹⁴ However, it is an essential component of CBT-i and involves “cleaning up” or improving an individual’s sleep environment and behaviour to promote better sleep quality and duration.²³

Mindfulness and insomnia

In recent years, the technique of mindfulness has become increasingly popular and is likely to be efficacious in helping to promote sleep by reducing cognitive and physiological arousal. Mindfulness treatment interventions have demonstrated statistically and clinically significant improvements in several night-time symptoms of insomnia, as well as reductions in presleep arousal, sleep

effort and dysfunctional sleep-related cognitions.²⁴ In many cases, mindfulness is combined with CBT-i.^{24,25} As an adjunct to CBT-i, it can be used for psychoeducation to help the client develop a more functional schematic model of sleep and for dealing with sleeplessness, including the detrimental role of hyperarousal. Typically, the chattering mind is focused on past or future events, whereas mindfulness emphasises being non-judgemental in the present, which potentially can reduce mind activation.

Bright light exposure (natural or artificial)

Educating the patient about sleep and the importance of bright light is an important aspect of treating insomnia. Good objective information about sleep, sleep loss and the body clock are helpful starting points for self-management. Bright light is a potent synchroniser for human circadian rhythm. In particular, morning light, which can be combined with exercise such as walking, can be helpful in consolidating night-time sleep and reducing morning sleep inertia.²⁶

Pharmacological treatment of insomnia

Although psychological and behavioural interventions are indispensable and effective for most insomnia sufferers, some will still need the extra help from pharmacological agents. Current medications and natural products used for insomnia include benzodiazepine-receptor agonists, melatonin and variants, antidepressants, antipsychotics and antihistamines.

Hypnotic drugs that act on the γ -aminobutyric acid receptor include benzodiazepines, such as temazepam, as well as the benzodiazepine-receptor agonists, such as zopiclone and zolpidem. Medications of this group have been studied in randomised controlled trials, with efficacy over 6 months²⁷ and longer in open-label exten-

sions.²⁸ Many doctors avoid prescribing medications from this family, mainly because of concern regarding dependence and tolerance. However, long-term trials of eszopiclone (not available in Australia) and extended-release zolpidem have shown sustained response with no tolerance and dependence after 6 months of daily use.²⁷⁻²⁹ Despite these findings, the concern remains that there are vulnerable patients who may become dependent on hypnotic drugs. To limit the risk of tolerance and dependence, the prescriber can instruct the patient to use the medication on a scheduled basis; for example, only on alternating nights, or three times a week and at the lowest effective dose possible for a limited time (ie, a month).²⁷ Zolpidem has been associated with parasomnias, so clinicians need to warn patients about unusual sleep behaviours as a side effect. Sudden discontinuation of this class of medications can result in a rebound insomnia that can be mitigated by a gradual taper.

Despite the similarity in the mode of action and pharmacokinetics of these agents, patients react differently to each product. Lack of response to one agent does not mean that others of the same group will not work. Similarly, an adverse effect of one does not mean that others will cause the same reaction. The decision whether or not to prescribe hypnotics should rely on a careful risk-benefit analysis by both the doctor and the patient. In addition to the perceived risk of dependence and tolerance, clinicians should consider the risks of untreated insomnia.

Melatonin has been shown to be effective in treating insomnia, particularly among people aged over 55 years.³⁰ However, melatonin is more effective as a chronobiotic for treating body clock conditions like jetlag and delayed sleep phase disorder than as a treatment for chronic insomnia.³¹

Sedating antidepressants (eg, doxepin, amitriptyline, mirtazapine, trimipramine), sedating antipsychotics (eg, quetiapine, olanzapine) and antihistamines are used off-label as sleep medications, despite insufficient evidence.^{13,32,33} Many clinicians prefer prescribing these medications over hypnotics, because of perceived concerns regarding the risks of dependence and tolerance associated with hypnotics, and despite antidepressants, antipsychotics and antihistamines also having serious side effects including weight gain, anticholinergic side effects and diabetes. The decision to prescribe this group of medications for insomnia should be based on a careful risk-benefit analysis, not solely on concerns regarding the risks associated with hypnotics.

Among herbal and alternative medication choices for treating insomnia, valerian has the most evidence showing possible mild improvements in sleep latency, with inconsistent effects on the rest of the objective sleep parameters.¹³ Although valerian shows some promise in improving sleep latency without side effects, the clinical trials are poorly designed and generally of short duration.³⁴

Conclusion

Insomnia is complex and usually chronic by the time the individual consults a health practitioner, with cognitive, behavioural and social factors involved in its maintenance. Simple instructions, such as avoiding stress, or short-term use of hypnotics are usually not effective. CBT-i is an effective intervention with long-term efficacy that enables patients to better manage and live with their insomnia symptoms. The development of online delivery of CBT-i markedly improves access to treatment and can be readily used in primary care as first-line treatment for most patients, with specialised sleep services managing more complex cases, those with ongoing symptoms and those who require person-to-person care.

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