MedicineToday 2011; 12(11): 29-40

PEER REVIEWED FEATURE
POINTS: 2 CPD/2 PDP

Breaking the cycle of insomnia

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Key points

- Insomnia is a disorder in itself but is frequently dismissed as being a symptom of a mood disorder or another medical condition. A bidirectional relation exists where untreated insomnia can lead to depression or anxiety and untreated depression or anxiety can result in insomnia.
- Important factors that contribute to insomnia development can be assessed using the three 'Ps': predisposing factors, precipitating factors and perpetuating factors.
- Excessive sleepiness (as opposed to fatigue) is uncommon in primary insomnia and, if present, other causes of sleep disruption (such as obstructive sleep apnoea and restless legs syndrome), medical and psychiatric conditions and substance abuse should be considered.
- Cognitive behavioural therapy (CBT) is the most efficacious treatment for patients with insomnia and can be success - fully used in general practice. Important elements of CBT include stimulus control, bed restriction, relaxation, cognitive therapy and healthy sleep practices. Pharmacotherapy is also efficacious but may be associated with significant side effects and should be used with caution.

Insomnia can be exacerbated by anxiety and anxiety can cause insomnia. Cognitive behavioural therapy and pharmacological therapy are important tools in breaking the cycle of insomnia.

Sleep is an essential element of mental and physical health, and difficulties with sleep result in suboptimal health and considerable personal distress. The patient presenting with insomnia in general practice is often difficult to treat due to the chronicity of the disorder and commonly comorbid negative mood ranging in severity from mild anxiety to major depression.[1,2] Insomnia is a disorder in itself but is frequently dismissed as being a symptom of a mood disorder or some other medical condition. In practice, a bidirectional relation exists where untreated insomnia can lead to depression or anxiety and untreated depression or anxiety can result in insomnia.[3,4] Prevalence studies show that psychiatric comorbidities are present in 28% of individuals with insomnia,[5] and 80% of individuals with a major depressive disorder experience insomnia.[6]

Interestingly, sleep anxiety (anxiety about the night and sleep) is extremely prevalent and a common complaint is 'if I could only sleep I would feel so much better' implying that poor sleep is the perceived cause of distress. Depression can further exacerbate the sense of helplessness and negatively impacts the patient's response to treatment.[7] Overall, insomnia is an independent entity but one that is commonly comorbid with mood disorders. Recognition of this interactive process is essential and initial treatment needs to target the most distressing disorder first.

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TABLE 1. DIAGNOSIS AND DIFFERENTIATION OF THE INSOMNIAS*

Insomnia categories	Essential features Complaint of insomnia plus the following:
Psychophysiological insomnia	Learned sleep-preventing associations, conditioned arousal, 'racing mind' phenomenon Often associated with a specific trigger
Paradoxical insomnia	Complaint of poor sleep disproportionate to sleep pattern and sleep duration
Idiopathic insomnia	Insomnia typically begins in childhood or from birth Unknown cause generally
Insomnia due to a mental disorder	Course of sleep disturbance concurrent with mental disorder
Inadequate sleep hygiene	Daily living activities inconsistent with maintaining good-quality sleep
Insomnia due to a medical medical disorder	Course of sleep disturbance concurrent with disorder
Insomnia due to drug or substance use	Sleep disruption caused by use of prescription medication, recreational drug, caffeine, alcohol or a particular food stuff
Adjustment insomnia	Presence of identifiable stressor Insomnia resolves or is expected to resolve when stressor removed

* Definitions are based on *International Classification of Sleep Disorders* (*ICSD*) Revised.[12]

DEFINITION OF INSOMNIA

Individuals with insomnia present with a distressing difficulty in sleep initiation, difficulty in sleep maintenance or early awaking, or a combination of these factors resulting in wakefulness in bed of more than 30 minutes and a perception of unmet sleep needs.[8] The quality of sleep in insomnia is described as being chronically nonrestorative. Insomnia is associated with increased absenteeism from work and reduced productivity[9] but when objectively measured, performance is not necessarily impaired.[10]

Insomnia may be difficult to define, because it is both a symptom and a disorder with varying types, durations and aetiologies (Table 1). The duration may be acute, chronic or intermittent, although substantial disagreement exists as to when it becomes a chronic disorder. The *Diagnostic and Statistical Manual* (*DSM*) IV-TR defines insomnia as chronic when symptoms are present for at least one month occurring more than three times per week.[11] However, the *International Classification of Sleep Disorders* (*ICSD*) Revised states that symptoms need to be present for longer than six months.[12] Central to the definitions in both classification systems is the presence of daytime symptoms and impaired social and/or occupational functioning. Individuals feel sleep deprived but generally have sufficient opportunity to sleep compared with true sleep deprivation where sleep opportunity is restricted either voluntarily or involuntarily to only a few hours each night.

Secondary insomnia is defined as insomnia occurring as a consequence of either a primary medical (e.g. pain-related conditions, neurological disorders such as Parkinson's disease, or cardiovascular or respiratory diseases) or psychiatric disorders (e.g. major depressive disorders, dysthymic disorder, bipolar disorder and a range of anxiety disorders).[12] However, this categorisation may not be relevant in terms of treatment interventions because cognitive behavioural therapy (CBT) can be equally efficacious in patients with either primary or secondary insomnia.[13] Individuals with both types of insomnia are high users of healthcare services, which is thought to be related to their level of psychological distress.[14,15]

DEFINING ANXIETY AND DEPRESSION IN RELATION TO INSOMNIA

Anxiety is a future-oriented negative emotional state associated with unpredictable and/or uncontrollable outcomes.[16] Anxiety occurring on more days than not over a six-month period is defined as generalised anxiety disorder and is commonly associated with nonrestorative sleep with a lifetime prevalence ranging between 10 and 25%.[17] Increased somatic tension and excessive worry are defining features of generalised anxiety disorder and are also common behaviours in individuals with insomnia. Anxiety and stress symptoms may precede insomnia or occur as a consequence of insomnia.[15] It can be argued that as long as both disorders are recognised and patients are treated, the order of treatment is irrelevant. Unfortunately, both insomnia and anxiety are frequently untreated in the early stages, which is exactly when treatment is more efficacious.

Depression is a common condition with a lifetime prevalence of major depression estimated at 17.1% with more women affected than men.[18] Loss of interest or pleasure in daily activities is a frequent component of depressive symptoms, as is insomnia. Feelings of worthlessness and hopelessness, psychomotor agitation, diminished ability to concentrate and fatigue are also common in patients with depression and there is overlap with some

symptoms of insomnia.[11] It is important to treat patients with both insomnia and depression because even with successful treatment of a major depressive episode, the most common residual symptom is sleep disturbance.[19]

DEVELOPMENT OF INSOMNIA

A useful way of assessing the development of insomnia is to explore the three 'Ps':[20]

- predisposing factors
- precipitating factors
- perpetuating factors.

The Spielman Model is a three-factor 'stress-diathesis model' applicable to both patients with acute and chronic insomnia (Figure).[21] Most individuals have at least one potential predisposing factor for insomnia, such as:

- demographic factors (female gender and older age)
- a personal or family history of insomnia
- psychological factors (including previous anxiety and depression and personality traits)
- physiological and lifestyle factors including somatic tension.

The precipitating factors push the individual over the insomnia threshold and predominantly include stressful life and health events (such as pain, cardiovascular disease or mental health problems).

Insomnia is then perpetuated by maladaptive sleep behaviours, such as excessive time in bed, daytime napping, medication use and clock-watching. Concerns about lack of sleep, both initiation and maintenance, create a cycle of increased worry, anxiety and physiological arousal, which then feeds the insomnia on a nightly basis. Effective and early treatment can break this cycle and revert patients to their premorbid, non-insomnia state. Once perpetuating behaviours become entrenched, however, insomnia can persist even when the 'trigger(s)' have been resolved.[22] The clinical presentation is typically a frustrated patient who now has a chronic health problem, often with accompanying negative personality traits associated with inadequate treatment interventions[17] and worsening physical health.[23]

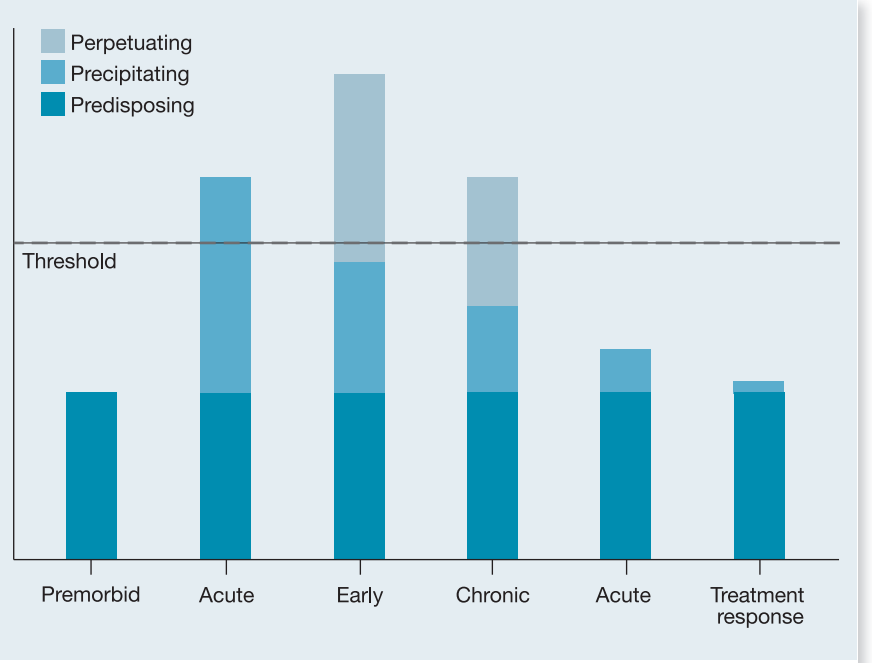
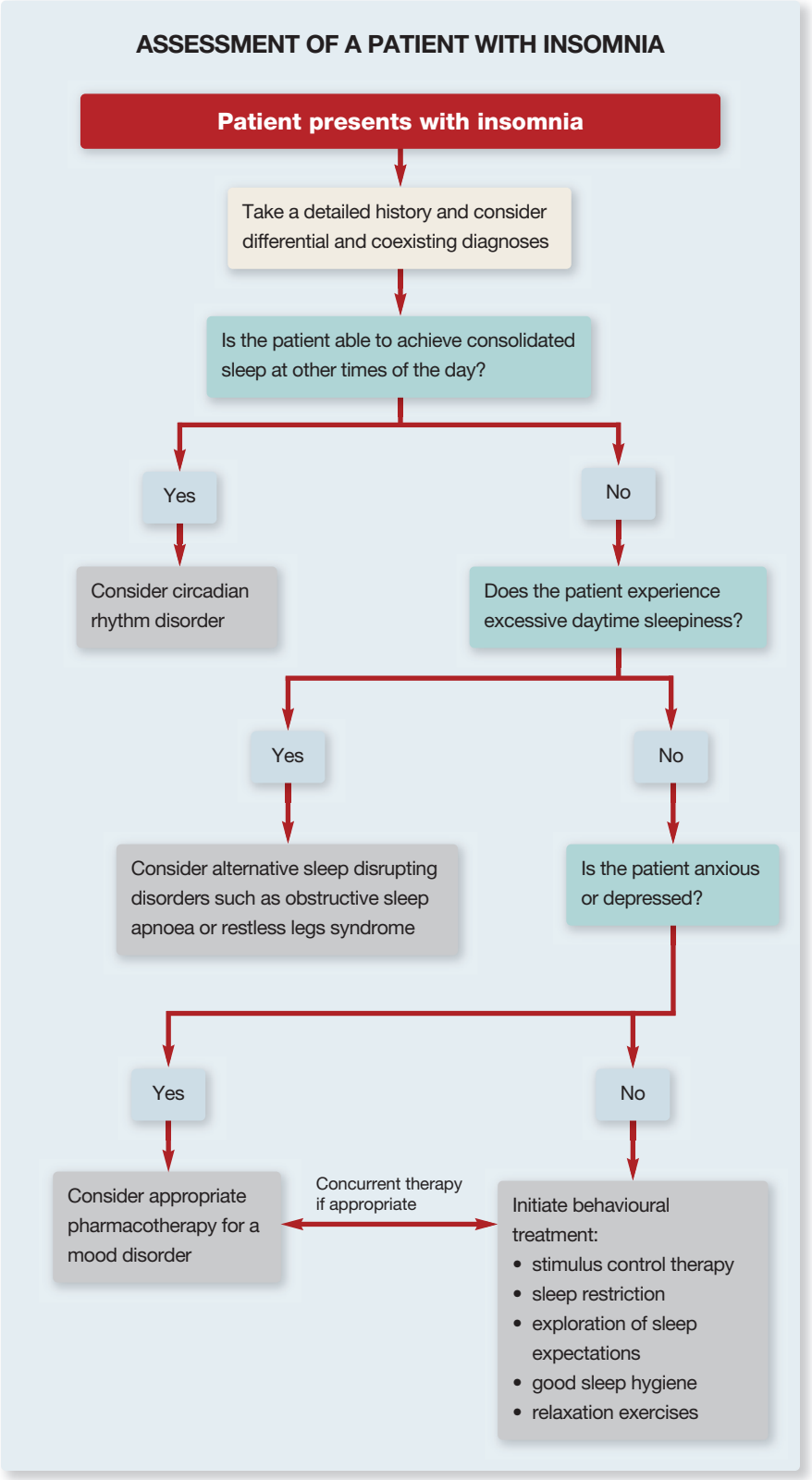


Figure. The Spielman Model of the three 'Ps.[21]

EPIDEMIOLOGY OF INSOMNIA

Reported prevalence rates of insomnia range from 4 to 48% of the population depending on the criteria used. In the UK, insomnia has been reported to have a prevalence rate ranging from 8 to 37% in general practice depending on diagnostic criteria used.[14] A review of the epidemiology of insomnia found that 33% of people reported experiencing at least one insomnia symptom, such as sleep-onset difficulties at the time of the study.[6] A survey conducted in NSW in 2004 found a similar prevalence of 32%.[24] Prevalence of insomnia symptoms is reduced when daytime dysfunction is included in the definition, dropping to between 9 and 15%, whereas the prevalence increased to 8 to 18% with the addition of sleep dissatisfaction as a symptom.[6]

Age and gender influence the prevalence of insomnia. Women are more likely to report insomnia than men and prevalence increases in the perimenopausal and postmenopausal phases. Insomnia is more prevalent in older adults compared with younger adults.[25] The relation between increasing age and sleep difficulties is also less clear when daytime consequences and sleep dissatisfaction are included. However, it does appear that older adults are more vulnerable to persistent symptoms, especially when pain-related conditions are present.[26] Interestingly, older active healthy adults have a prevalence of insomnia similar to the general population.[27] Although sleep disturbances are commonly reported in perimenopausal women, menopause status itself is not significantly associated with sleep quality when objectively assessed. Hot flushes, symptoms of depression, menstrual bleeding patterns and reproductive hormone levels are stronger predictors of poor sleep in this group of patients and therefore these symptoms should be targeted when considering treatment.[28,29]



Treatment approaches

The flowchart on this page may be a useful adjunct in determining the best approach to insomnia treatments.

EXCLUDING OTHER SLEEP DISORDERS

The insomnia patient is usually 'wired and tired' but not sleepy. A score of more than eight on the Epworth Sleepiness Scale (likelihood of going to sleep in passive situations with a top score of 24)[30] is suggestive of another sleep disorder such as obstructive sleep apnoea, restless legs syndrome, periodic limb movement disorder or circadian rhythm sleep disorders (delayed sleep phase disorder and advanced sleep phase disorder). Pain, depression and substance abuse are all associated with increased sleepiness (Table 1). Between April 2006 and March 2008, the Australian Bettering the Evaluation and Care of Health (BEACH) program documented that GPs managed sleep disorders 2987 times (a rate of 1.6 contacts per 100 encounters), which extrapolates to about 1.7 million times annually across Australia. Insomnia was by far the most common sleep disorder found in the BEACH report, representing eight out of 10 sleep disorders managed by GPs.

NONPHARMACOLOGICAL TREATMENTS FOR INSOMNIA

CBT is the most effective nonpharmacological treatment option for patients with insomnia.[31-33] Despite this, medications are the most commonly offered intervention when a patient seeks treatment for insomnia. The BEACH program found that a GP consultation for insomnia resulted in pharmacotherapy 95% of the time and these patients are less likely to receive advice or counselling than patients in a standard GP encounter.[15]

CBT is often perceived as being too time-consuming in the GP setting. However, a simpler version taking less time can still be very effective. A very useful starting point is to challenge and increase

TABLE 2. DESCRIPTION OF COGNITIVE BEHAVIOURAL THERAPY COMPONENTS

Intervention	General description	Specific techniques
Stimulus control	Bed = sleep Set of instructions aimed at conditioning the patient to expect that bed is for sleeping and not stimulating activities with the exception of sexual activity. Aim is to promote a positive association between the 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 to 20 minutes (without watching the clock), leave the bed and bedroom and go to another room and do a nonstimulating activity. Return to bed only when comfortable enough to sleep again. Do not read, watch TV, talk on the phone, use the computer, worry or plan activities while in bed.
Sleep/bed restriction therapy	A technique that increases sleep 'drive' and reduces time in bed lying awake. It 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 to 30 minutes when the time spent asleep is at least 85% of the allowed time in bed. Keep a fixed wake-up time, regardless of actual sleep duration.
Relaxation techniques	Various breathing techniques, use of visual imagery and use of meditation. Aim to reduce somatic and cognitive arousal.	Progressive muscle relaxation (practice at least daily) and encourage shorter relaxation periods (two minutes) several times per day. Use of breathing and self-hypnosis techniques.
Cognitive therapy	Identification and targeting of beliefs that may be interfering with adherence to stimulus control and sleep restriction. Use of 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	Sleep hygiene emphasises environmental factors, physiological factors, behaviours and habits that promote sound sleep.[20]	Avoid long naps in the daytime, short naps (less than 30 minutes) are okay. Do regular exercise. Maintain regular sleep-wake schedule seven days per week. Avoid stimulants (such as caffeine and nicotine). Limit alcohol intake, especially before bed. Avoid visual access to clock when in bed. Keep bedroom dark, quiet, clean and comfortable.

the individual's awareness that what he or she is currently doing is not working.

Effective behavioural treatments for insomnia

The two most effective behavioural treatments for insomnia are stimulus control therapy and sleep (bed) restriction (Table 2).[34,35] The following treatment interventions are designed to re-associate bed with sleep rather than wakefulness, worry and sleep anxiety.

Stimulus control therapy or the quarter-hour rule

Stimulus control therapy is a form of reconditioning that is useful when the bedroom has become a place of wakefulness and often out-of-control-arousal.[1,36] All stimulating wake-promoting factors (such as television, radio, mp3 players, mobile phones, computers and a thinking/worrying brain) need to be removed from the bedroom environment. The individual is encouraged to get up out of bed if unable to instigate sleep within about a quarter of an hour. The concept that bed is for sleep and sex only may also require some exploration.

Sleep or bed restriction

Individuals compensate for poor-quality sleep by increasing their time in bed. This results in less consolidated sleep, more time awake and an associated increased anxiety about not sleeping. Matching time in bed with reported sleep time requires a reduction of time in bed to as

few as five hours (patients stay up later and/or get up earlier) in order to increase the homeostatic drive for sleep.[37] This can be followed by a gradual increase in time in bed as sleep time and confidence about sleep increase, and bed can be seen as a safe sleeping place. Good sleepers have a sleep efficiency of more than 85%, where sleep time and time in bed are relatively matched. (Sleep efficiency is calculated by time asleep/time in bed x 100.)

Education about healthy sleep habits or sleep hygiene

There are a number of misperceptions about sleep in general. A summary of the different stages of sleep is important for individuals to gain a better understanding of how we cycle through the stages of sleep every 90 minutes, often waking from dream sleep or light sleep. Waking is a normal part of sleep and these brief awakenings will not impact on the individual the following day.

Reassurance can be given that deep sleep is usually maintained in patients with insomnia but they often perceive light sleep as being wakeful and 45 to 55% of the night is in fact spent in light sleep. Addressing behaviours that are known to interfere with sleep, such as drinking caffeine or alcohol, nicotine smoking, daytime napping, timing of exercise and making the bedroom comfortable, quiet and dark are useful adjuncts but are not as effective as bed restriction and stimulus control. Few individuals allow for wind-down time before bed or are able to self-soothe. Sometimes seeking outside help to address these issues can be vital.

Boundary setting for exercise, light and getting up times

Exercise not only reduces muscle tension and physiological arousal but potentially improves mood with the release of endorphins. The introduction of exercise generally improves sleep and allows the individual to 'do something', especially in the mornings rather than lying awake in the morning waiting for more sleep. Exercising in the evening artificially raises core body temperature and if completed three to four hours before the expected bed time (to allow the body to cool down) can be helpful for sleep onset.

Getting up at the same time each day regardless of the length and quality of the previous night's sleep is a crucial component in setting sleep boundaries. Getting up at the same time is more important than a regular bedtime, which does not guarantee sleep onset. If this intervention is combined with exposure to early morning light (40 minutes in the first two hours after getting up), the brain sleep clock can be reset with the suppression of the nighttime sleep hormone melatonin.

Relaxation and learning to deal with uncomfortable feelings

Relaxation alone is not an effective stand alone treatment compared with a combination of the other treatments. Relaxation techniques are useful to reduce muscle tension and stress during the daytime and also for helping individuals to reduce their overarousal responses.

Relaxation techniques include progressive muscle relaxation, focused breathing strategies, imagery training, meditation and hypnosis. However, using relaxation methods to go to sleep may paradoxically increase sleep effort.[1] Having 'time out' and doing something positive that is not sleep related is a useful step in preparing for sleep. Reading, listening to music and avoiding work, emails and stressful telephone conversations are useful strategies.

Cognitive therapy – challenging negative sleep thoughts

Cognitive therapy works on the premise that how we feel is a result of how we think.[38] Challenging individuals to recognise how their sleep is being sabotaged by their thinking can easily be done in general practice. In psychology, there is a tendency to encourage individuals to recognise negative thoughts and learn to replace them with positive thoughts; however, this approach is often unrealistic when dealing with a chronic disorder. A more useful approach is to help the patient be aware of what is a helpful thought or approach compared with an unhelpful one. Common unhelpful beliefs about sleep such as: 'I must have eight hours of sleep to be able to function the next day' can be modified to 'maybe I don't always need eight hours of sleep and I have managed to function with less than that in the past'. This reframing of unhelpful thoughts is a key factor in improving self-efficacy (belief about seeing oneself undertake a therapy successfully in the future) in regard to sleep.[39]

PHARMACOLOGICAL TREATMENTS FOR INSOMNIA

Pharmacotherapy may be indicated for the short-term management of patients with insomnia but is often not the best first-line treatment option.[40] However, it can be effective in inducing, maintaining and consolidating sleep (as measured by polysomnography or self reports) and can be used in the treatment of patients with primary or secondary insomnia.[41]

Many different pharmacological agents have been used to initiate and maintain sleep. The benzodiazepines (such as temazepam and nitrazepam) and the nonbenzodiazepine gamma-aminobutyric acid $(GABA)_A$ receptor agonists (such as zolpidem and zopiclone) are the most common drugs used in Australia, with the nonbenzodiazepine $GABA_A$ receptor agonists demonstrating a more favourable safety profile.

Benzodiazepines

Benzodiazepines are $GABA_A$ receptor agonists and stimulate the pentameric $GABA_A$ receptor complex to hyperpolarise neurons of the CNS. In short-term, randomised, double-blind, placebo-controlled trials, benzodiazepines have been shown to reduce sleep onset times, thereby increasing total sleep duration and improving sleep continuity.[42]

Benzodiazepines induce a change to

TABLE 3. CHARACTERISTICS OF COMMON ORAL BENZODIAZEPINES AND NONBENZODIAZEPINES[44]

Generic name	Receptor binding specificity	Dose range (mg)*	Time to peak action (hours)	Elimination half-life (hours)
Flunitrazepam	Nonspecific	0.5 to 2	0.5 to 3	36 to 200†
Midazolam	Nonspecific	2.5 to 5	0.5 to 1	2 to 6
Nitrazepam	Nonspecific	5 to 10	0.5 to 7	15 to 38
Oxazepam	Nonspecific	10 to 30	1.5 to 3	4 to 15
Temazepam	Nonspecific	10 to 30	0.5 to 3	8 to 22
Triazolam	Nonspecific	0.125 to 0.25	0.5 to 2	2 to 6
Eszopiclone (currently not available in Australia but is likely to become available soon)	$GABA_A$ α1-3	1 to 3	0.5 to 1	6
Zopiclone	$GABA_A$ α1-3, possibly α5	3.75 to 15	1 to 2	4 to 6
Zaleplon (currently not available in Australia)	$GABA_A$ α1	5 to 20	0.5 to 1	1
Zolpidem	$GABA_A$ α1	5 to 10	1.5 to 2	1.5 to 2.5

* Note substantial inter-individual variations on doses.
† Refers to the elimination half-life of the active metabolite.

sleep architecture with reduced proportions of slow wave sleep and rapid eye movement (REM) sleep and a corresponding increase in lighter stage two sleep.[41,43] At doses within the therapeutic range, a relatively flat dose-response curve is observed; that is, little additional benefit is gained by increasing the dose.

Benzodiazepines differ mainly in their pharmacokinetic properties (Table 3).[44] Temazepam and oxazepam are commonly used because of their relatively short half-lives, which minimise the residual daytime drowsiness and psychomotor impairment that can be a problem with the longer-acting agents. However, agents with short half-lives may be less effective in patients with predominant sleep-maintenance insomnia (who may wake after the effect of the drug has worn off) compared with those with sleep-initiation insomnia.

Common adverse effects of benzodiazepines include over-sedation, lightheadedness, memory loss and slurred speech – all of which may be more common in the elderly. Enhanced sedation and respiratory depression is possible with concurrent use of other common CNS depressants such as alcohol and antidepressants. The CNS-depressive effects of benzodiazepines may also increase the severity of sleep apnoea in patients with coexisting sleep-disordered breathing.

Benzodiazepines should be used for the shortest time possible with the duration of use defined and contracted with the patient from the outset. Tolerance, dependence and rebound insomnia may occur in patients taking benzodiazepines. Tolerance to the hypnotic effects of benzodiazepines develops rapidly on repeated administration. Dependence is rare in patients taking normal therapeutic doses for short periods, but patients on long-term treatment often have difficulty reducing and stopping their benzodiazepine.[45] Rebound insomnia is characterised by a worsening of sleep relative to baseline and is more marked with regular consumption of benzodiazepines but can occur after as little as one week of low-dose administration. Withdrawal from long-term use needs to be slow, taking place over several weeks with small dosage reductions.

Nonbenzodiazepine $GABA_A$ receptor agonists

The so-called 'z-drugs' – zopiclone and zolpidem – are sedative hypnotics with a similar mode of action to benzodiazepines. These drugs have marked specificity to a particular $GABA_A$ receptor subtype and therefore have reduced anxiolytic, muscle relaxant and anticonvulsant effects compared with traditional benzodiazepines but preserved hypnotic effects.

Zopiclone and zolpidem have comparable efficacy with benzodiazepines in reducing sleep latency, decreasing nocturnal awakenings and increasing total sleep time. Oral zopiclone has a rapid onset

(15 to 30 minutes) and its elimination half-life is four to six hours, increasing with age.[46] Zolpidem has a similar onset of action (30 minutes), but its elimination half-life is shorter (1.5 to 2.5 hours). Zopiclone has been shown to maintain its hypnotic efficacy in studies of eight to 17 weeks of treatment, and tolerance does not appear to develop to the hypnotic effects of zolpidem, as demonstrated in clinical trials of up to one year in duration. Rebound is less frequent and milder than that seen after the discontinuation of benzodiazepines.

Common adverse effects of nonbenzodiazepine $GABA_A$ receptor agonists include a bitter taste with zopiclone use, and diarrhoea and dizziness with zolpidem use. These drugs cause less residual morning sedation and psychomotor impairment than many benzodiazepines with fewer reports of dependency and misuse. Reports of sleep-walking and other sleep activities in association with zolpidem have been widely reported by the media but are in fact rare.[47]

Antidepressants

Antidepressants with sedative effects are occasionally prescribed to patients with insomnia (off-label use) but are taken at a lower dose than used for treating patients with depression. Amitriptyline and doxepin are most commonly used, and their sedative action is primarily an anticholinergic effect.[48] Side effects of these drugs, particularly in the elderly, include anticholinergic effects (such as dry mouth, blurred vision, constipation, urinary retention and delirium) and α-adrenergic effects (such as orthostatic hypotension and dizziness). Additionally, many antidepressants can exacerbate periodic limb movements. Use of most serotonin selective reuptake inhibitors (SSRIs) will also exacerbate insomnia during the first few weeks of use.

Mirtazapine is a tetracycline antidepressant with anxiolytic and marked hypnotic effects. There are currently no randomised controlled trials to support the use of mirtazapine in the treatment of patients with primary insomnia; however, its use in patients with major depression and disturbed sleep is supported by small open-label studies.[49,50]

Antihistamines

Antihistamines are generally less effective than benzodiazepines in the treatment of patients with insomnia (off-label use) and induce daytime drowsiness and anticholinergic effects. There is limited evidence of their value over placebo and clear evidence of their side effects.[51]

Over-the-counter therapies

Valerian is commonly used as a sleep aid and is available over the counter. Evidence for its efficacy in the treatment of patients with insomnia is inconclusive.[52] Similarly, melatonin, a popular dietary supplement, cannot be recommended as a treatment for patients with primary insomnia on the basis of current evidence.[41] Melatonin does, however, have a role in the management of patients with insomnia caused by circadian disruption (e.g. jet lag or delayed sleep phase syndrome) through its circadian phase-shifting effects.[53] Emerging evidence suggests that prolonged-release melatonin taken in small doses at carefully set times may be effective in treating primary insomnia in older patients (aged 55 to 80 years).[54,55]

Novel drug therapies

There are several hypnotic agents currently under investigation, including:

- gaboxadol – a GABA agonist
- indiplon – a nonbenzodiazepine compound that binds to the benzodiazepine 1 site on GABA receptors
- ramelteon – a melatonin receptor agonist.

CONCLUSION

Insomnia and mood disorders commonly coexist and the relation between these disorders is frequently bidirectional. Treatment needs to be tailored specifically to patient characteristics and comorbidities. In addition, it is important to recognise that depression may also be present in individuals with excessive daytime sleepiness.

Cognitive behavioural treatments are the most efficacious treatment of patients with insomnia both in the short and ong term. Most GPs have considerable knowledge of CBT and other behavioural management strategies but are not always sure how to instigate these treatments. A common misperception on the part of the medical practitioner is that patients expect a prescription for hypnotics when they present with insomnia, but this is often not the case.[56] The need for clear communication from both sides of the consultation is essential in the management of patients with insomnia. MT

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A list of references is available on request to the editorial office.

COMPETING INTERESTS: None.

Breaking the cycle of insomnia

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