



Review

Insomnia: a practical clinical and therapeutic approach

Insomnio: enfoque clínico y terapéutico

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ABSTRACT

Insomnia is one of the most prevalent conditions in clinical practice, with significant mental, physical, and social consequences. This review aims to provide an updated and clinically oriented synthesis of current evidence on its diagnosis, pathophysiology, and therapeutic management, integrating cognitive perspectives and the role of shared decision-making. Its conceptualization has shifted from an unspecific symptom to a distinct disorder with well-established neurobiological foundations. Diagnosis is primarily clinical and requires assessment of functional impact, perpetuating factors, and patient expectations. International guidelines recommend cognitive-behavioral therapy for insomnia (CBT-I) as the first-line treatment, with pharmacotherapy reserved for selected cases. Sleep hygiene measures, while necessary, are insufficient when used in isolation. Dysfunctional beliefs and shared decision-making are central to optimizing adherence and preventing chronicity. Contemporary management demands an integral, personalized, and person-centered approach that combines educational strategies, psychotherapy, and rational pharmacological interventions.

RESUMEN

El insomnio es uno de los trastornos más frecuentes en la práctica médica, con importantes repercusiones en la salud mental, física y social. El objetivo de esta revisión es sintetizar de forma actualizada y práctica la evidencia sobre su diagnóstico, fisiopatología y enfoque terapéutico, incorporando la perspectiva cognitiva y la toma de decisiones compartida. Su conceptualización ha pasado de ser un síntoma inespecífico a reconocerse como un trastorno autónomo con bases neurobiológicas propias. El diagnóstico es esencialmente clínico y requiere valorar la repercusión funcional, los factores perpetuantes y las expectativas del paciente. Las guías internacionales recomiendan la terapia cognitivo-conductual para el insomnio (TCC-I) como tratamiento de primera línea, reservando la farmacoterapia para casos seleccionados. Las medidas de higiene del sueño, aunque necesarias, resultan insuficientes cuando se aplican de forma aislada. Las creencias disfuncionales y la toma de decisiones compartida son determinantes para la adherencia y la prevención de la cronificación. El enfoque actual exige un enfoque integral, personalizado y centrado en la persona.

Palabras clave:

Insomnio
Terapia cognitivo-conductual
Higiene del sueño
Expectativas del paciente
Farmacoterapia racional
Toma de decisiones compartida

Introduction

According to the International Classification of Sleep Disorders, Third Edition (ICSD-3) and the DSM-5-TR (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision), insomnia is defined as a persistent difficulty in initiating, maintaining or consolidating sleep, occurring despite adequate opportunity and conditions for sleep, and associated with clinically significant impairment in daytime functioning.^{1,2}

Insomnia is one of the most common reasons for consultation in both primary care and psychiatry, and one of the most prevalent complaints in the general population. Approximately one quarter of adults report dissatisfaction with their sleep quality; between 10% and 15% report insomnia symptoms accompanied by daytime consequences, and between 6% and 10% meet diagnostic criteria for an insomnia disorder, with higher incidence in women and older individuals.³ Far from being a trivial phenomenon, persistent insomnia is associated with substantial impairment in daytime functioning, an increased risk of depression, anxiety and suicide, as well as various somatic diseases, including cardiovascular and metabolic conditions.⁴

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Over recent decades, a profound conceptual shift has taken place: insomnia is no longer regarded merely as a secondary symptom, but rather as an independent disorder, with its own neurobiological mechanisms and a specific therapeutic approach.⁵ This review provides a practical update on the diagnosis and treatment of insomnia, with particular emphasis on the most effective interventions and on the role of patient expectations as a modulator of clinical course and therapeutic response.

The present article also aims to offer clinicians an integrative, evidence-based perspective to facilitate decision-making in everyday practice. This work corresponds to a narrative review oriented towards clinical practice. For its preparation, a non-systematic search was conducted in PubMed, Embase and Web of Science covering the period from 2000 to 2025, using terms related to “insomnia”, “CBT-I”, “hypnotics”, “orexin antagonists”, “sleep hygiene” and “shared decision-making”. International clinical guidelines, meta-analyses and studies with high methodological quality were prioritised. The selection of studies was based on their relevance and clinical applicability, without applying formal inclusion or exclusion criteria, in keeping with the narrative nature of the review.

Epidemiology and clinical burden

Insomnia is one of the most common sleep disorders in the general population and constitutes a major public health problem due to its high prevalence and functional impact.³ Approximately one third of adults experience insomnia symptoms, and between 9% and 15% meet clinical criteria when daytime impairment is required; the prevalence of chronic insomnia is around 6% according to formal diagnostic criteria.⁶

Rates are consistently higher in women (female-to-male ratio ~1.5:1) and increase with age, particularly after 45 years.⁶ In Spain, the population-based study by Ohayon and Sagales showed that 20.8% of adults reported insomnia symptoms and 6.4% met diagnostic criteria, with higher prevalence in women and older individuals.⁷ Despite its impact, substantial levels of under-recognition and undertreatment persist.

Insomnia is associated with marked impairment of attention and memory, an increased risk of domestic, occupational and traffic accidents,⁸ and reduced productivity. In addition, it represents an independent risk factor for depression, suicidal ideation and cardiovascular disease.^{9,10} These consequences confirm that insomnia is not a minor symptom, but rather a disorder with significant clinical and functional implications, whose early detection should be considered a priority.

Clinical diagnosis of insomnia

A positive diagnosis of insomnia is primarily clinical and is based on three pillars: a subjective complaint of difficulty initiating or maintaining sleep or early morning awakening, daytime impairment, and temporal persistence of symptoms at least three nights per week for more than three months.²

The main diagnostic systems converge on these essential elements, although they differ in certain conceptual nuances, particularly regarding the definition of daytime distress and the exclusion of secondary causes. [Table 1](#) summarises, in a comparative manner, the most relevant diagnostic criteria of the three classifications currently in use: DSM-5-TR, ICSD-3-TR and ICD-11 (International Classification of Diseases, 11th edition).^{1,2,11}

Clinical assessment

A structured clinical interview is the essential diagnostic tool. It should explore the sleep pattern (bedtime, sleep latency, nocturnal awakenings and perceived quality), precipitating and perpetuating factors, lifestyle habits, medication use and functional impact. A practical

approach is Spielman’s 3-P model (predisposing, precipitating and perpetuating factors), which is useful for guiding intervention.¹²

Among the available assessment instruments, the Insomnia Severity Index (ISI)¹³ and the Pittsburgh Sleep Quality Index (PSQI)¹⁴ are brief, self-administered paper-and-pencil questionnaires that are easy to interpret and sensitive to change, making them particularly useful both in research and in clinical practice to objectify insomnia severity and to monitor therapeutic response.

The ISI consists of seven items assessing difficulty initiating or maintaining sleep, satisfaction with sleep, daytime interference, concern about the problem and perceived distress. Each item is scored from 0 to 4, yielding a total score ranging from 0 to 28, which allows classification of severity: no insomnia (0–7), subthreshold insomnia (8–14), moderate insomnia (15–21) and severe insomnia (22–28). Validated Spanish versions with excellent psychometric properties have been published, both in the general population and in patients with psychiatric and medical disorders,¹⁵ making it a particularly useful tool for detection and follow-up in primary care, in contrast to the PSQI, which assesses overall sleep quality.

The PSQI is a broader tool comprising 19 items grouped into seven components (subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of hypnotic medication and daytime dysfunction). It generates a global score ranging from 0 to 21, with values >5 indicating poor sleep quality. It also has Spanish adaptations and validations with good internal consistency and construct validity.¹⁶

It is essential to exclude secondary causes or comorbidities that may explain the complaint of insomnia, such as chronic pain, medical illnesses, psychiatric disorders, alcohol or caffeine consumption, or the use of stimulant medications, as well as other sleep disorders (obstructive sleep apnoea, restless legs syndrome). Polysomnography is reserved for cases with suspected comorbid sleep disorders or lack of response to conventional treatment.¹⁷

Finally, assessment should consider the patient’s perception of their sleep, their expectations regarding rest and their degree of concern, as these cognitive factors substantially influence the persistence of the problem and adherence to therapeutic interventions.¹⁸

Pathophysiology and explanatory models

Spielman’s 3-P model remains the most useful framework for explaining the development and maintenance of insomnia, integrating predisposing, precipitating and perpetuating factors¹² (see [Table 2](#) for a conceptual summary). This framework helps to understand how an acute episode may evolve into a persistent problem in the presence of dysfunctional behaviours and cognitions.

From a neurobiological perspective, insomnia is related to a state of physiological and cognitive hyperarousal characterised by increased sympathetic tone, activation of the hypothalamic–pituitary–adrenal axis, and increased cortical activity even during sleep.¹⁹ Alterations have also been described in circadian and homeostatic sleep regulation systems, with dysfunction in pathways that modulate wakefulness (orexin, dopamine) and sleep (GABA, adenosine).^{20–24}

Contemporary cognitive models emphasise the role of attentional focus on internal sleep-related cues, concerns about the consequences of insufficient sleep, and dysfunctional beliefs, all of which contribute to the maintenance of hyperarousal and chronicity.¹⁸

Taken together, these mechanisms explain why effective treatment of insomnia must simultaneously address the behavioural, cognitive and physiological components of the disorder, forming the conceptual basis of current therapeutic interventions.

Treatment of insomnia

Management of insomnia should be individualised based on symptom severity, duration, and perpetuating factors, with a preference

Table 1
Diagnostic criteria for insomnia according to DSM-5-TR, ICSD-3-TR and ICD-11 (comparative summary).

Diagnostic system	Essential criteria	Duration and functional impact
DSM-5-TR (2022) ¹	Difficulty initiating or maintaining sleep, or early awakening with an inability to return to sleep, despite having adequate opportunity to sleep	Symptoms ≥ 3 nights/week for ≥ 3 months. Cause clinically significant distress or daytime functional impairment (fatigue, irritability, concentration difficulties). Not better explained by another mental or medical disorder or by a substance
ICSD-3-TR (2023) ²	Complaint of insomnia despite adequate opportunity and conditions for sleep; difficulty initiating, maintaining or consolidating sleep	Symptoms ≥ 3 nights/week for ≥ 3 months, with daytime consequences (fatigue, sleepiness, low mood, work errors, excessive concern about sleep)
ICD-11 (2019) ¹¹	Dissatisfaction with sleep quantity or quality associated with difficulty initiating, maintaining or consolidating sleep, or early morning awakening	Duration ≥ 3 months with clinically significant daytime impact (fatigue, cognitive or emotional disturbances, social or occupational impairment)

Sources: References 1,2,11.

DSM-5-TR: *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision*; ICSD-3-TR: *International Classification of Sleep Disorders, Third Edition, Text Revision*; ICD-11: *International Classification of Diseases*, 11th edition.

Table 2
Spielman's 3 Ps model: predisposing, precipitating and perpetuating factors of insomnia (conceptual summary).

Factor	Description	Clinical examples
Predisposing	Biological, psychological, or social vulnerabilities that increase susceptibility to insomnia	Hyperarousal tendency, anxious or perfectionistic traits, family history of insomnia, older age, female sex
Precipitating factors	Events or circumstances that trigger an acute episode of insomnia	Occupational or academic stress, interpersonal problems, bereavement, medical illness, hospitalisation, schedule changes or night shifts
Perpetuating factors	Dysfunctional behaviours and cognitions that maintain insomnia once the precipitating factor has resolved	Lying awake in bed, compensating with daytime naps, excessive caffeine intake, catastrophic thoughts about the consequences of not sleeping

Source: Spielman AJ, et al.¹²

for non-pharmacological interventions and pharmacotherapy reserved for selected cases. Current clinical guidelines consistently recommend cognitive behavioural therapy for insomnia (CBT-I) as the first-line treatment for chronic insomnia.^{4,5}

General measures and sleep hygiene

Sleep hygiene measures constitute the foundation of any therapeutic intervention, although on their own they are usually insufficient.²⁴ They include maintaining regular sleep schedules, avoiding caffeine, alcohol or nicotine in the hours preceding bedtime, limiting daytime naps, reducing screen exposure before going to bed, and ensuring an appropriate bedroom environment.²⁵

Education on healthy habits should be accompanied by a review of dysfunctional compensatory behaviours, such as spending excessive time in bed or attempting to sleep "by force", which perpetuate the problem. These recommendations, although simple, help prepare the ground for more specific interventions.

Table 3 summarises the main evidence-based sleep hygiene measures recommended by international guidelines.

Cognitive behavioural therapy for insomnia (CBT-I)

CBT-I is a structured intervention that integrates behavioural and cognitive techniques to reduce hyperarousal, modify maladaptive sleep behaviours, and restructure dysfunctional beliefs. Its efficacy is well established in meta-analyses and international guidelines, with remission rates of 60–70% in chronic insomnia and sustained long-term improvement.^{26,27}

The main components of CBT-I are:

- **Stimulus control:** strengthening the association between bed and sleep by restricting its use to sleep or sexual activity.
- **Sleep restriction:** limiting time in bed to the average effective sleep time and gradually extending it as sleep efficiency improves.
- **Cognitive restructuring:** identifying and replacing dysfunctional thoughts ("if I don't sleep, I won't be able to function tomorrow") with more realistic interpretations.
- **Relaxation and mindfulness techniques:** reducing physiological and cognitive arousal.
- **Sleep hygiene education:** consolidating the healthy habits described above.

Digital or self-guided versions of CBT-I have demonstrated efficacy comparable to face-to-face therapy in controlled trials²⁸ and represent a cost-effective alternative, particularly useful in primary care.

Despite its high efficacy in clinical trials, the implementation of CBT-I in real-world practice faces relevant limitations: the availability of trained professionals is limited, waiting times are often long, and many patients encounter logistical difficulties in completing a structured programme. This mismatch between evidence and implementation has driven the development of digital or self-guided versions that improve accessibility, although they do not fully replace in-person clinical support. These barriers should be considered when planning individual therapeutic management.

Pharmacological treatment

Pharmacological treatment of insomnia should be considered a complement to non-pharmacological interventions and reserved for cases in which CBT-I is not available, is insufficient, or a short-term intervention is required to relieve symptoms. The aim is not only to induce sleep, but to restore a healthy sleep pattern and enable the effective implemen-

Table 3
Sleep hygiene measures and basic behavioural recommendations.

Aspect	Recommendation	Rationale
Schedule and regularity	Maintain fixed bedtimes and wake-up times, even on weekends	Reinforces circadian rhythm and improves sleep efficiency
Bedroom environment	Ensure a dark, quiet environment with a comfortable temperature (18–20 °C) and a comfortable bed	Minimises physiological arousal and interruptions
Use of the bed	Reserve the bed exclusively for sleep or sexual activity. Avoid reading, watching television or using a mobile phone in bed	Strengthens the bed–sleep association and reduces negative conditioning
Pre-sleep routine	Establish a relaxing routine (warm bath, breathing exercises, light reading). Avoid screens for at least one hour before bedtime	Promotes physiological and cognitive deactivation
Physical activity	Engage in regular exercise, preferably in the morning or early afternoon. Avoid intense evening exercise	Improves sleep quality and regulates circadian rhythm
Substance consumption	Avoid caffeine, nicotine, or alcohol in the hours before bedtime	These substances disrupt sleep latency and sleep architecture
Diet	Avoid heavy meals before bedtime. If hungry, opt for a light snack	Facilitates digestion and reduces reflux or nocturnal discomfort
Naps	Limit naps to less than 30 min and avoid them after 16:00	Prevents reduction in homeostatic sleep pressure
Night-time awakenings	If sleep does not occur within 15–20 min, get out of bed and engage in a quiet activity until feeling sleepy	Reduces the association between bed and frustration at not being able to sleep
Relationship with sleep	Avoid clock-watching during the night and accept that one poor night's sleep does not imply serious consequences	Reduces anticipatory anxiety and cognitive hyperarousal

Sources: Adapted from references 4,25.

tation of behavioural strategies.^{3,5} The basic principles for the rational and safe use of hypnotics are summarised in Table 4.

Although widely used, some pharmacological options have important limitations that must be considered in real-world practice. Prolonged-release melatonin shows modest efficacy, with clinical benefit mainly confined to individuals over 55 years of age or cases of mild insomnia, and therefore does not constitute a first-line alternative for most patients.^{29,30} Z-drugs provide rapid relief, but their efficacy decreases over time, and their safety profile—complex parasomnias, cognitive impairment and risk of misuse—limits prolonged use.^{31,32} Dual orexin receptor antagonists (DORAs) represent a novel option with better tolerability and less cognitive interference, although their cost, availability and limited long-term experience currently constrain their use in daily practice.^{33–35} These considerations highlight the need to individualise hypnotic choice and to periodically reassess clinical usefulness.

European and North American guidelines recommend using hypnotic drugs at the minimum effective dose and for the shortest possible time, periodically reviewing the need to continue treatment and promoting gradual withdrawal when feasible.^{3,9}

Benzodiazepine hypnotics

These include lorazepam, lormetazepam, temazepam, flurazepam, and triazolam, among others. They act as positive allosteric modulators of the GABA-A receptor, reducing sleep latency and increasing sleep continuity. They are effective in the short term, but prolonged use is associated with tolerance, dependence, cognitive impairment, falls and residual daytime sleepiness.^{36,37}

Short- or intermediate-acting compounds (such as lormetazepam or temazepam) are preferred, chronic use should be avoided, and discontinuation should be gradual, reducing the dose by 10–25% every 1–2 weeks.^{38,39} They should be avoided in patients with COPD, sleep apnoea, or concomitant alcohol consumption.

Z-drugs (zolpidem, zopiclone, eszopiclone)

These are selective GABA-A $\alpha 1$ receptor agonists, with a more predictable pharmacokinetic profile and less disruption of sleep architecture than benzodiazepines. Although they show better tolerability, they may cause tolerance, complex parasomnias (sleepwalking or sleep-related eating) and cognitive impairment with prolonged use.³¹

Treatment duration should be limited to ≤ 4 weeks. Eszopiclone has demonstrated efficacy for up to six months in controlled studies, always with close clinical monitoring.³²

Sedating antidepressants

These can be used when insomnia coexists with depressive or anxiety symptoms.

- Trazodone (25–100 mg/night): improves sleep latency and maintenance, with a favourable safety profile and no risk of dependence.⁴⁰
- Mirtazapine (7.5–15 mg): effective in patients with weight loss or anxiety, although it may cause daytime somnolence and weight gain.⁴¹
- Doxepin (3–6 mg): a tricyclic antidepressant approved for sleep maintenance insomnia, with minimal anticholinergic activity.⁴²
- Amitriptyline: frequently used off-label but not recommended due to lack of evidence and risk of adverse effects.⁴³

Melatonin and analogues

Prolonged-release melatonin (2 mg) has shown modest efficacy, particularly in individuals over 55 years of age or in cases of mild sleep-onset insomnia⁴¹. It carries no risk of dependence or residual effects, making it suitable for polymedicated patients or those with chronic illnesses.

Other melatonin receptor agonists, such as ramelteon (not available in Spain) and tasimelteon, have demonstrated efficacy in circadian rhythm disorders, but their use in primary insomnia is limited.^{29,30}

Table 4
General principles for the use of hypnotic drugs in the treatment of insomnia.

Principle	Practical recommendation
Indication	Use only when CBT-I is unavailable, insufficient, or a short-term intervention is required
Dose and duration	Use the minimum effective dose for a period not exceeding 2–4 weeks, unless clinically justified
Clinical follow-up	Periodically review efficacy, adverse effects, tolerance, and signs of dependence or abuse
Polypharmacy	Avoid concomitant use of other central nervous system depressants (alcohol, opioids, antihistamines, antipsychotics)
Older adults	Reassess with particular caution in older people, due to the increased risk of falls, confusion and cognitive impairment ³¹

Sources: Adapted from references ^{4,17}.

CBT-I: cognitive behavioural therapy for insomnia; CNS: central nervous system.

≥: Greater than or equal to.

≤: Less than or equal to.

Table 5
Practical outline for the pharmacological management of insomnia in adults.

Clinical scenario	First choice	Alternatives	Comments
Chronic insomnia without psychiatric comorbidity	CBT-I (face-to-face or digital)	—	Assess sleep hygiene and perpetuating factors
Limited access to or partial response to CBT-I	Z-drugs (zolpidem, zopiclone) ≤4 weeks	Intermediate half-life benzodiazepine or PR melatonin 2 mg	Avoid prolonged use and combine with CBT-I
Insomnia with depression or anxiety	CBT-I + sedating antidepressant (trazodone, mirtazapine)	Doxepin 3–6 mg	Reassess after 4–6 weeks; avoid polypharmacy
Sleep maintenance insomnia in older adult	PR melatonin 2 mg or doxepin 3–6 mg	Daridorexant (if available)	Monitor for falls, confusion, and residual effects
Treatment-resistant insomnia or tolerance to hypnotics	DORA (daridorexant, suvorexant, lemborexant)	—	Adjust dose according to hepatic function and maintain fixed dosing time

Sources: Adapted from references ^{4,5,17,48,49}.

CBT-I: cognitive behavioural therapy for insomnia; DORA: dual orexin receptor antagonist; PR: prolonged-release; Z-drugs: non-benzodiazepine hypnotics from the Z group (zolpidem, zopiclone).

Dual orexin receptor antagonists (DORAs)

Orexin receptor antagonists (suvorexant, lemborexant and daridorexant) exert their effects by inhibiting the orexin A and B neuropeptides, leading to reduced activation of the arousal system.³³

Phase III clinical trials have demonstrated efficacy in both sleep-onset latency and sleep maintenance, with few residual effects and minimal cognitive interference.³⁴

Daridorexant, recently authorised in Europe, shows a significant improvement in quality of life and daytime functioning, with a low risk of dependence or withdrawal.³⁵

They are particularly useful in older patients or those with medical comorbidities in whom traditional hypnotics are contraindicated.

Other drugs

- **Antihistamines** (hydroxyzine, diphenhydramine): sedating, but associated with rapid tolerance, anticholinergic effects and daytime somnolence; not recommended for long-term use.⁴⁴
- **Sedative antipsychotics** (quetiapine, olanzapine): should not be used unless there is significant psychiatric comorbidity.⁴⁵
- **Cannabinoids, valerian and phytotherapy**: insufficient evidence to recommend routine use.^{46,47}

Pharmacological treatment of insomnia should be understood as a temporary and complementary tool alongside cognitive-behavioural interventions. Its effectiveness increases when accompanied by appropriate education, review of expectations and shared decision-making

between clinician and patient, with the aim of achieving functional and sustainable sleep.

Treatment selection for insomnia should take into account patient age, comorbidities and insomnia subtype, with cognitive behavioural interventions consistently prioritised. When pharmacotherapy is indicated, the choice of agent, dosage and treatment duration should be tailored to the individual patient and potential risks.

Table 5 presents a practical algorithm for the pharmacological management of insomnia in adults, adapted from the main international clinical guidelines and the available evidence, which may guide therapeutic decision-making in clinical practice.

Expectations, beliefs and shared decision-making

Patients' expectations regarding sleep and its treatment play a decisive role in both the chronicity of insomnia and therapeutic response.^{3,20} Dysfunctional beliefs—such as considering eight hours of sleep essential or assuming that insomnia always requires medication—contribute to anticipatory anxiety, increased cognitive hyperarousal and, ultimately, dependence on hypnotics.⁵⁰

Therefore, reviewing and aligning expectations is an essential component of the cognitive intervention within CBT-I. Clinicians should actively explore what patients expect from treatment, clarify realistic goals—for example, prioritising improvement in daytime functioning over achieving “perfect” sleep—and address erroneous beliefs about rest and medication.⁵⁰

Recently, the so-called Sleep Expectation–Reality Gap has been described, referring to the discrepancy between desired and perceived sleep duration or quality, which may generate distress, worry and cognitive

distortion regarding one's own sleep. This phenomenon has been identified in primary care patients, where unrealistic sleep expectations are associated with greater dissatisfaction and poorer adaptation to treatment.⁵¹

Shared decision-making is likewise a central element in modern insomnia management. Active patient participation in choosing interventions—behavioural or pharmacological—improves adherence, therapeutic satisfaction and clinical outcomes.^{52,53} Studies in mental health settings have shown that patient involvement in decision-making is associated with greater trust in treatment, better adherence and reduced treatment discontinuation, particularly when empathic, bidirectional communication exists between clinician and patient.⁵⁴

Ultimately, addressing insomnia involves not only treating a symptom, but rebuilding the patient's relationship with sleep, helping them understand its mechanisms, moderate expectations and actively participate in therapeutic decisions. This collaborative, person-centred approach integrates the different treatment modalities—from CBT-I to pharmacotherapy—in a rational manner and strengthens autonomy and the therapeutic alliance.

Conclusions and future perspectives

Insomnia is a highly prevalent disorder with significant functional impact that requires an integrated approach based on understanding its multiple biological, psychological and behavioural determinants. Spielman's 3-P model and the concept of hyperarousal provide a coherent framework for understanding its pathophysiology and guiding treatment.

CBT-I represents the first-line intervention, with proven efficacy and sustained effects, while pharmacotherapy should be reserved for selected cases and limited periods. The incorporation of new strategies, such as digital CBT-I platforms and orexin system modulators, broadens therapeutic options and favours a more individualised approach. However, treatment success largely depends on reviewing patient expectations and beliefs and on shared decision-making that strengthens adherence and promotes self-care. Future research should further explore the neurobiological and cognitive mechanisms underlying insomnia chronicity and evaluate integrated interventions combining psychological, pharmacological and educational therapies in real-world clinical settings.

Looking ahead, priority should be given to expanding and validating digital CBT-I platforms to overcome access limitations to face-to-face therapy, as well as to developing strategies to rationalise hypnotic use, with particular attention to structured deprescribing programmes. The integration of hybrid models—digital psychoeducation, brief interventions and focused clinical follow-up—could improve the real-world applicability of evidence-based recommendations and contribute to safer and more efficient management of insomnia in clinical practice.

CRedit authorship contribution statement

Carlos De las Cuevas Castresana conceived the idea, conducted the literature search, drafted the manuscript, and approved the final version.

Ethical considerations

The present work is a narrative review of the literature and does not involve experiments with human beings or the use of individual-level data. Approval by a research ethics committee was not required.

Declaration of Generative AI and AI-assisted technologies in the writing process

The author declares that generative artificial intelligence has not been used in the writing of the scientific text or in the interpretation of the data.

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ReferenciasReferences

References

1. American Academy of Sleep Medicine. *International Classification of Sleep Disorders*. 3rd ed. Darien, IL: AASM; 2023. Text Revision (ICSD-3-TR).
2. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: APA Publishing; 2022. Text Revision (DSM-5-TR).
3. Morin CM, Benca R. Chronic insomnia. *Lancet*. 2012;379(9821):1129–1141, [http://dx.doi.org/10.1016/s0140-6736\(11\)60750-2](http://dx.doi.org/10.1016/s0140-6736(11)60750-2).
4. Riemann D, Espie CA, Altena E, et al. The European Insomnia Guideline: an update on the diagnosis and treatment of insomnia 2023. *J Sleep Res*. 2023;32(6):e14035, <http://dx.doi.org/10.1111/jsr.14035>.
5. Clinical Guidelines Committee of the American College of Physicians Qaseem A, Kansagara D, Forcica MA, Cooke M, Denberg TD. Management of Chronic Insomnia Disorder in Adults: A Clinical Practice Guideline From the American College of Physicians. Management of Chronic Insomnia Disorder in Adults: A Clinical Practice Guideline From the American College of Physicians. *Ann Intern Med*. 2016;165(2):125–133, <http://dx.doi.org/10.7326/m15-2175>.
6. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev*. 2002;6(2):97–111, <http://dx.doi.org/10.1053/smr.2002.0186>.
7. Ohayon MM, Sagales T. Prevalence of insomnia and sleep characteristics in the general population of Spain. *Sleep Med*. 2010;11(10):1010–1018, <http://dx.doi.org/10.1016/j.sleep.2010.02.018>.
8. Léger D, Bayon V, Ohayon MM, et al. Insomnia and accidents: cross-sectional study (EQUINOX) on sleep-related home, work and car accidents in 5293 subjects with insomnia from 10 countries. *J Sleep Res*. 2014;23(2):143–152, <http://dx.doi.org/10.1111/jsr.12104>.
9. Baglioni C, Battagliese G, Feige B, et al. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. *J Affect Disord*. 2011;135(1-3):10–19, <http://dx.doi.org/10.1016/j.jad.2011.01.011>.
10. Sofi F, Cesari F, Casini A, Macchi C, Abbate R, Gensini GF. Insomnia and risk of cardiovascular disease: a meta-analysis. *Eur J Prev Cardiol*. 2014;21(1):57–64, <http://dx.doi.org/10.1177/2047487312460020>.
11. World Health Organization. *ICD-11 for Mortality and Morbidity Statistics*. Geneva: WHO; 2019.
12. Spielman AJ, Caruso LS, Glovinsky PB. A behavioral perspective on insomnia treatment. *Psychiatr Clin North Am*. 1987;10(4):541–553. PMID: 3332317.
13. Bastien CH, Vallières A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med*. 2001;2(4):297–307, [http://dx.doi.org/10.1016/s1389-9457\(00\)00065-4](http://dx.doi.org/10.1016/s1389-9457(00)00065-4).
14. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;28(2):193–213, [http://dx.doi.org/10.1016/0165-1781\(89\)90047-4](http://dx.doi.org/10.1016/0165-1781(89)90047-4).
15. Fernandez-Mendoza J, Rodriguez-Muñoz A, Vela-Bueno A, et al. The Spanish version of the insomnia severity index: a confirmatory factor analysis. *Sleep Med*. 2012;13(2):207–210, <http://dx.doi.org/10.1016/j.sleep.2011.06.019>.
16. de la Vega R, Tomé-Pires C, Solé E, et al. The Pittsburgh sleep quality index: validity and factor structure in young people. *Psychol Assess*. 2015;27(4):e22–27, <http://dx.doi.org/10.1037/pas0000128>.
17. Sateia MJ, Buysse DJ, Krystal AD, Neubauer DN, Heald JL. Clinical practice guideline for the pharmacologic treatment of chronic insomnia in adults: an American academy of sleep medicine clinical practice guideline. *J Clin Sleep Med*. 2017;13(2):307–349, <http://dx.doi.org/10.5664/jcsm.6470>.
18. Harvey AG. A cognitive model of insomnia. *Behav Res Ther*. 2002;40(8):869–893, [http://dx.doi.org/10.1016/s0005-7967\(01\)00061-4](http://dx.doi.org/10.1016/s0005-7967(01)00061-4).
19. Riemann D, Spiegelhalder K, Feige B, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep Med Rev*. 2010;14(1):19–31, <http://dx.doi.org/10.1016/j.smrv.2009.04.002>.
20. Schwartz MD, Kilduff TS. The neurobiology of sleep and wakefulness. *Psychiatr Clin North Am*. 2015;38(4):615–644, <http://dx.doi.org/10.1016/j.psc.2015.07.002>.

21. Rosenwasser AM. Functional neuroanatomy of sleep and circadian rhythms. *Brain Res Rev.* 2009;61(2):281–306, <http://dx.doi.org/10.1016/j.brainres-rev.2009.08.001>.
22. Riemann D, Nissen C, Palagini L, Otte A, Perlis ML, Spiegelhalter K. The neurobiology, investigation, and treatment of chronic insomnia. *Lancet Neurol.* 2015;14(5):547–558, [http://dx.doi.org/10.1016/s1474-4422\(15\)00021-6](http://dx.doi.org/10.1016/s1474-4422(15)00021-6).
23. Perlis M, Pigeon W, Gehrman P, Findley J, Drummond S. Neurobiological mechanisms in chronic insomnia. *Sleep Med Clin.* 2009;4(4):549–558, <http://dx.doi.org/10.1016/j.jsmc.2009.07.002>.
24. Yanagisawa M. Toward the mysteries of sleep. *Keio J Med.* 2019;68(1):27, <http://dx.doi.org/10.2302/kjm.68-001-abst>.
25. Irish LA, Kline CE, Gunn HE, Buysse DJ, Hall MH. The role of sleep hygiene in promoting public health: a review of empirical evidence. *Sleep Med Rev.* 2015;22:23–36, <http://dx.doi.org/10.1016/j.smrv.2014.10.001>.
26. Wu JQ, Appleman ER, Salazar RD, Ong JC. Cognitive behavioral therapy for insomnia comorbid with psychiatric and medical conditions: a meta-analysis. *JAMA Intern Med.* 2015;175(9):1461–1472, <http://dx.doi.org/10.1001/jamainternmed.2015.3006>.
27. Trauer JM, Qian MY, Doyle JS, Rajaratnam SM, Cunningham D. Cognitive behavioral therapy for chronic insomnia: a systematic review and meta-analysis. *Ann Intern Med.* 2015;163(3):191–204, <http://dx.doi.org/10.7326/m14-2841>.
28. Espie CA, Emsley R, Kyle SD, et al. Effect of digital cognitive behavioral therapy for insomnia on health, psychological well-being, and sleep-related quality of life: a randomized clinical trial. *JAMA Psychiatry.* 2019;76(1):21–30, <http://dx.doi.org/10.1001/jamapsychiatry.2018.2745>.
29. Lemoine P, Wade AG, Katz A, Nir T, Zisapel N. Efficacy and safety of prolonged-release melatonin for insomnia in middle-aged and elderly patients with hypertension: a combined analysis of controlled clinical trials. *Integr Blood Press Control.* 2012;5:9–17, <http://dx.doi.org/10.2147/ibpc.s27240>.
30. Żelabowski K, Pichowicz W, Skowron I, et al. The efficacy of melatonergic receptor agonists used in clinical practice in insomnia treatment: melatonin, tasimelteon, ramelteon, agomelatine, and selected herbs. *Molecules.* 2025;30(18):3814, <http://dx.doi.org/10.3390/molecules30183814>.
31. Gunja N. The clinical and forensic toxicology of Z-drugs. *J Med Toxicol.* 2013;9(2):155–162, <http://dx.doi.org/10.1007/s13181-013-0292-0>.
32. Krystal AD, Walsh JK, Laska E, et al. Sustained efficacy of eszopiclone over 6 months of nightly treatment: results of a randomized, double-blind, placebo-controlled study in adults with chronic insomnia. *Sleep.* 2003;26(7):793–799, <http://dx.doi.org/10.1093/sleep/26.7.793>.
33. Żelabowski K, Petrov W, Wojtyśiak K, et al. Targeting the orexin system in the pharmacological management of insomnia and other diseases: suvorexant, lemborexant, daridorexant, and novel experimental agents. *Int J Mol Sci.* 2025;26(17):8700, <http://dx.doi.org/10.3390/ijms26178700>.
34. Fietze I, Bassetti CLA, Mayleben DW, Pain S, Seboek Kinter D, McCall WV. Efficacy and safety of daridorexant in older and younger adults with insomnia disorder: a secondary analysis of a randomised placebo-controlled trial. *Drugs Aging.* 2022;39(10):795–810, <http://dx.doi.org/10.1007/s40266-022-00977-4>.
35. Kunz D, Dauvilliers Y, Benes H, et al. Long-term safety and tolerability of daridorexant in patients with insomnia disorder. *CNS Drugs.* 2023;37(1):93–106, <http://dx.doi.org/10.1007/s40263-022-00980-8>.
36. Glass J, Lanctôt KL, Herrmann N, Sproule BA, Busto UE. Sedative hypnotics in older people with insomnia: meta-analysis of risks and benefits. *BMJ.* 2005;331(7526):1169, <http://dx.doi.org/10.1136/bmj.38623.768588.47>.
37. Barker MJ, Greenwood KM, Jackson M, Crowe SF. Cognitive effects of long-term benzodiazepine use: a meta-analysis. *CNS Drugs.* 2004;18(1):37–48, <http://dx.doi.org/10.2165/00023210-200418010-00004>.
38. Kripke DF. Greater incidence of depression with hypnotic use than with placebo. *BMC Psychiatry.* 2007;7:42, <http://dx.doi.org/10.1186/1471-244x-7-42>.
39. Reeve E, Ong M, Wu A, Jansen J, Petrovic M, Gnjidic D. A systematic review of interventions to deprescribe benzodiazepines and other hypnotics among older people. *Eur J Clin Pharmacol.* 2017;73(8):927–935, <http://dx.doi.org/10.1007/s00228-017-2257-8>.
40. Mendelson WB. A review of the evidence for the efficacy and safety of trazodone in insomnia. *J Clin Psychiatry.* 2005;66(4):469–476, <http://dx.doi.org/10.4088/jcp.v66n0409>.
41. Nguyen PV, Dang-Vu TT, Forest G, et al. Mirtazapine for chronic insomnia in older adults: a randomised double-blind placebo-controlled trial—the MIRAGE study. *Age Ageing.* 2025;54(3):afaf050, <http://dx.doi.org/10.1093/ageing/afaf050>.
42. Krystal AD, Lankford A, Durrence HH, et al. Efficacy and safety of doxepin 3 and 6 mg in a 35-day sleep laboratory trial in adults with chronic primary insomnia. *Sleep.* 2011;34(10):1433–1442, <http://dx.doi.org/10.5665/sleep.1294>.
43. Rauwerda N, van Straten A, Zondervan A, et al. Low dose amitriptyline versus cognitive behavioral therapy for insomnia in patients with medical comorbidity: results of a randomized controlled multicenter non inferiority trial. *Sleep.* 2025;zsaf176, <http://dx.doi.org/10.1093/sleep/zsaf176>.
44. Burgazli CR, Rana KB, Brown JN, Tillman F 3rd. Efficacy and safety of hydroxyzine for sleep in adults: systematic review. *Hum Psychopharmacol.* 2023;38(2):e2864, <http://dx.doi.org/10.1002/hup.2864>.
45. Thompson W, Quay TAW, Rojas-Fernandez C, Farrell B, Bjerre LM. Atypical antipsychotics for insomnia: a systematic review. *Sleep Med.* 2016;22:13–17, <http://dx.doi.org/10.1016/j.sleep.2016.04.003>.
46. Cong J, Zhang H, Xing W. Effectiveness and safety of chinese herbal medicine in treatment of perimenopausal insomnia: a systematic review and meta-analysis of randomized controlled trials. *Holist Nurs Pract.* 2026;40(1):3–15, <http://dx.doi.org/10.1097/hnp.0000000000000743>.
47. Ranum RM, Whipple MO, Croghan I, Bauer B, Toussaint LL, Vincent A. Use of cannabidiol in the management of insomnia: a systematic review. *Cannabis Cannabinoid Res.* 2023;8(2):213–229, <http://dx.doi.org/10.1089/can.2022.0122>.
48. McCall WV, Mercado K, Dzuryny TN, et al. Insomnia and the effect of zolpidem-extended-release on the sleep items of the Hamilton Rating Scale for Depression in outpatients with Depression, insomnia, and suicidal ideation: Relationship to patient age. *J Psychopharmacol.* 2024;38(9):827–831, <http://dx.doi.org/10.1177/02698811241268900>.
49. Mignot E, Mayleben D, Fietze I, et al. Safety and efficacy of daridorexant in patients with insomnia disorder: results from two multicentre, randomised, double-blind, placebo-controlled, phase 3 trials. *Lancet Neurol.* 2022;21(2):125–139, [http://dx.doi.org/10.1016/s1474-4422\(21\)00436-1](http://dx.doi.org/10.1016/s1474-4422(21)00436-1).
50. Harvey AG, Dong L, Bélanger L, Morin CM. Mediators and treatment matching in behavior therapy, cognitive therapy and cognitive behavior therapy for chronic insomnia. *J Consult Clin Psychol.* 2017;85(10):975–987, <http://dx.doi.org/10.1037/ccp0000244>.
51. De las Cuevas C, Segovia M. The sleep expectation-reality gap: exploring discrepancies between perceived and ideal sleep duration in primary care patients. *Healthcare (Basel).* 2025;13(6):650, <http://dx.doi.org/10.3390/healthcare13060650>.
52. De las Cuevas C, Peñate W, de Rivera L. To what extent is treatment adherence of psychiatric patients influenced by their participation in shared decision making? *Patient Prefer Adherence.* 2014;8:1547–1553, <http://dx.doi.org/10.2147/ppa.s73029>.
53. Charles C, Gafni A, Whelan T. Decision-making in the physician-patient encounter: revisiting the shared treatment decision-making model. *Soc Sci Med.* 1999;49(5):651–661, [http://dx.doi.org/10.1016/s0277-9536\(99\)00145-8](http://dx.doi.org/10.1016/s0277-9536(99)00145-8).
54. De las Cuevas C, Peñate W. To what extent psychiatric patients feel involved in decision making about their mental health care? Relationships with socio-demographic, clinical, and psychological variables. *Acta Neuropsychiatr.* 2014;26(6):372–381, <http://dx.doi.org/10.1017/neu.2014.21>.