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INSOMNIA DISORDER IN PRIMARY CARE: DIAGNOSIS AND EVIDENCE-BASED MANAGEMENT

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ABSTRACT

Chronic insomnia disorder affects approximately one in ten adults in Europe. Despite this prevalence, it remains substantially underdiagnosed and undertreated in primary care, where pharmacotherapy is prescribed far more frequently than the evidence-based first-line intervention, cognitive behavioural therapy for insomnia (CBT-I) [8,12]. In Poland, subjective insomnia is reported by approximately 50.5% of the adult population (58.9% in women, 41.4% in men) [6]. Estimates of clinically defined insomnia disorder vary depending on the diagnostic criteria applied. This narrative review provides primary care physicians with a practical framework for the diagnosis and management of chronic insomnia disorder. Recommendations are drawn from four current guidelines: the European Insomnia Guideline 2023 [8], the VA/DoD Clinical Practice Guideline 2025 [3], the AASM Behavioural Treatment Guideline 2021 [2], and the Polish recommendations of the PTBS, PTMR, and PTP 2023 [1]. Diagnosis relies on clinical grounds alone [3,8]. Before initiating treatment, a structured five-domain differential evaluation should be completed- covering mental health, somatic conditions, medications, lifestyle, and primary sleep disorders. CBT-I, delivered face-to-face or via validated digital platforms, is the consistently recommended first-line treatment across all major current guidelines. Its effects are durable in a way that pharmacotherapy alone cannot replicate [2,8]. When pharmacological treatment is required, it should be short-term and dose-minimised. Agent selection should be guided by patient age, comorbidities, and dependence risk. Dual orexin receptor antagonists represent an option with a favourable dependence profile and European Medicines Agency (EMA) approval for chronic insomnia [1,3,4,8]. Structured deprescribing of chronically used hypnotics, supported by concurrent CBT-I, should be integrated into routine primary care practice [5].

KEYWORDS

Chronic Insomnia Disorder, Cognitive Behavioural Therapy for Insomnia, Primary Care, Pharmacotherapy, Deprescribing, Sleep Disorders

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I. Introduction

Chronic insomnia disorder is now understood as a condition in its own right, not simply a symptom of an underlying illness [8]. All three major classification systems- the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the third edition of the International Classification of Sleep Disorders (ICSD-3), and the eleventh revision of the International Classification of Diseases (ICD-11)- have removed the historical primary-secondary distinction. This reflects the current understanding that persistent, functionally impairing insomnia warrants independent treatment, regardless of any comorbid conditions [8].

Insomnia is one of the most common health problems in adults. In Europe, approximately one in ten adults meets diagnostic criteria for chronic insomnia disorder, though prevalence estimates vary widely- from 5.8% to 34.8%- depending on the classification system and country studied. Globally, the prevalence of chronic insomnia disorder is estimated at 6-23%. Risk is higher in women, older adults, shift workers, and those with comorbid medical or psychiatric illness [3].

Despite its high prevalence and well-documented impact on quality of life, occupational performance, and long-term health outcomes, chronic insomnia remains substantially underdiagnosed and undertreated across European primary care settings [12]. The general practitioner (GP) is consistently the first point of contact for patients seeking help with sleep difficulties [12]. Yet cognitive behavioural therapy for insomnia (CBT-I) - the evidence-based first-line treatment - is rarely offered in routine clinical practice. Instead, most patients across Europe receive sleep hygiene advice followed by pharmacotherapy, often continued well beyond the approved duration [12]. A survey of Swiss GPs illustrates this pattern: only 8% recommended CBT-I as the first-line treatment, while 49% prescribed antidepressants and 18% benzodiazepine receptor

agonists [12]. Similar findings have been reported across other European primary care settings [8,12]. This pattern reflects a broader structural deficit- insufficient sleep medicine education at undergraduate and postgraduate levels, limited access to behavioural specialists, and the absence of public funding frameworks for CBT-I delivery in most European health systems [8,12].

This narrative review summarises current diagnostic criteria, a structured differential evaluation framework, evidence-based treatment options, and guidance on specific patient populations in primary care. The recommendations are based on the most recent major guidelines [1,2,3,8] and are designed for direct use in routine primary care.

II. Definition and Diagnostic Criteria

Chronic insomnia disorder is defined consistently across three major classification systems - DSM-5, ICSD-3, and ICD-11[8]. Diagnosis requires three elements: a subjective sleep complaint, a minimum frequency and duration threshold, and clinically significant daytime impairment [3,8].

The patient must report at least one of the following: difficulty falling asleep, difficulty staying asleep, or waking too early, despite having adequate time and opportunity for sleep. These problems cannot be explained solely by environmental factors or an irregular sleep schedule [3,8]. Diagnosis is based on clinical assessment alone- a thorough sleep and medical history. It does not require objective measurements such as sleep latency or wake time after sleep onset. These values may serve as a reference, but are not required for diagnosis [8]. Symptoms must occur at least three nights per week and last for a minimum of three months [3,8].

The second required criterion is daytime impairment directly caused by nocturnal sleep disturbance. Recognised manifestations include fatigue, impaired attention or memory, reduced functioning at work or socially, mood disturbance, daytime sleepiness, reduced motivation, proneness to errors, and dissatisfaction with sleep [3,8]. Without demonstrable daytime impairment, the diagnosis cannot be made regardless of the severity of the nocturnal complaint[8].

Symptom severity can be assessed using the Insomnia Severity Index (ISI), a validated seven-item self-report questionnaire with a total score of 0-28 [3,8]. Scores of 8-14 indicate mild insomnia, 15-21 moderate, and 22-28 severe. A score of 10 is recommended cut-off for case identification in community settings [8]. Polysomnography is not required for a routine diagnosis. It should be reserved for cases where a comorbid sleep disorder - such as obstructive sleep apnoea (OSA) or periodic limb movement disorder- is clinically suspected [8,12].

III. Five-Domain Differential Evaluation

Missing a comorbid condition that is driving or maintaining insomnia - such as undiagnosed OSA or subclinical depression - can lead to ineffective treatment or, in the case of hypnotic prescribing in OSA, direct harm. Insomnia both worsens and is worsened by comorbid conditions. Current guidelines therefore recommend treating it concurrently and independently [8]. The following five-domain framework is designed to guide this assessment in primary care.

A. Mental health. Insomnia is more common across all major psychiatric disorders, including depression, anxiety, PTSD, bipolar disorder, and schizophrenia [8]. It is also a recognised independent risk factor for the onset and relapse of depressive episodes [8]. Persistent sleep disturbance has been linked to increased suicidal ideation and behaviour across multiple meta-analyses [3]. All patients presenting with chronic insomnia should be screened for depression using the Patient Health Questionnaire-9 (PHQ-9), a validated nine-item self-report tool widely used in primary care [13]. A score of ≥10 suggests at least moderate depression and warrants further clinical assessment. Scores of 15 or above indicate moderately severe to severe depression requiring targeted intervention [13]. The Generalised Anxiety Disorder-7 (GAD-7) serves the same function for anxiety disorders. In patients aged 65 and older, brief cognitive screening- using Mini-Mental State Examination (MMSE) or a comparable tool- is also warranted, as insomnia can both worsen and mask early cognitive decline [1].

B. Somatic conditions. Many chronic medical conditions are associated with disrupted sleep and may contribute or maintain insomnia [8]. These include cardiovascular disease, hypertension, chronic obstructive pulmonary disease (COPD), gastro-oesophageal reflux disease (GERD), chronic kidney disease, chronic pain syndromes, diabetes mellitus, and malignancy [8]. The relationship is bidirectional: insomnia has been shown to increase the long-term risk of cardiovascular events and depression [12]. When a somatic condition appears to be a primary driver of sleep disruption, it should be optimally treated. However, insomnia often develops

its own psychophysiological perpetuating cycle over time and may require concurrent, targeted management-independent of underlying conditions [8].

C. Medications and substances. A thorough medication review is an essential part of the insomnia assessment. Many commonly prescribed drugs can cause or worsen sleep disturbance. These include corticosteroids, beta-blockers, SSRIs, theophylline, and diuretics [8]. Stimulant substances - caffeine, nicotine, pseudoephedrine, and amphetamines - should also be specifically asked about. Alcohol is frequently overlooked. While commonly used by patients as a self-administered hypnotic, it disrupts sleep architecture and is an independent contributor to insomnia maintenance [8]. Screening questions about alcohol and substance use should be embedded within the broader sleep history, as patients frequently underreport use that they do not consider clinically relevant.

D. Lifestyle and environment. Irregular sleep scheduling, shift work, and excessive or variable daytime napping disrupt circadian rhythm and reinforce insomnia [8]. The bedroom environment should be assessed for noise, excessive light, inappropriate temperature, and the habitual use of screen-based devices at bedtime, all of which are modifiable. Sleep hygiene counselling alone is not sufficient to treat established chronic insomnia, but identifying these factors remains relevant to the overall management plan [2,8].

E. Primary sleep disorders - differential diagnosis. Three primary sleep disorders must be actively excluded, as each requires a different management approach. OSA co-occurs with insomnia in approximately 30-40% of insomnia patients, and insomnia is present in 30-50% of OSA patients [8] - a combination known as COMISA (comorbid insomnia and sleep apnoea). Identifying OSA has direct treatment implications, as benzodiazepines and Z-drugs are relatively contraindicated in this setting [8]. Screening should be performed using the STOP-BANG questionnaire (sensitivity 88%, specificity approximately 42% for moderate-to-severe OSA at a cut-off of ≥3) [3]. Its high sensitivity makes it useful for excluding OSA when negative. However, a positive result requires further confirmatory evaluation. Restless legs syndrome (RLS) is characterised by an irresistible urge to move the legs, predominantly in the evening and at rest. It can be identified through a single validated screening question or brief clinical interview. Confirmation of the diagnosis and initiation of treatment typically requires neurological referral [8].

Finally, circadian rhythm sleep-wake disorders - most commonly delayed sleep-wake phase disorder in younger adults and advanced sleep-wake phase disorder in older individuals - present with a mismatch between desired and actual sleep timing, rather than an inability to sleep. They are distinguished from insomnia disorder by careful history-taking [3].

IV. Red Flags: When to Refer to a Specialist

Chronic insomnia disorder can and should be diagnosed and managed in primary care. Certain presentations, however, require referral to the appropriate specialist. The primary care clinician should remain alert to features suggesting a primary sleep disorder, a serious psychiatric condition, or a clinical situation in which primary care management is no longer sufficient. The principal indications for referral are summarised in Table 1 [3,8,14,15].

Table 1. Indications for specialist referral in patients presenting with insomnia complaints.

Clinical situation	Recommended referral
Positive OSA screening (STOP-BANG ≥3 or STOP ≥2); witnessed apnoeas; clinical features suggestive of OSA (obesity, retrognathia, treatment-resistant hypertension)	Pulmonology / Sleep medicine
Symptoms consistent with RLS (uncomfortable evening/nocturnal leg sensations relieved by movement); confirmed or suspected PLMD	Neurology
Circadian rhythm disorder (stable shifted sleep-wake timing incompatible with social/occupational demands)	Sleep medicine / Psychiatry

Clinical situation	Recommended referral
REM sleep behaviour disorder (dream enactment, nocturnal vocalisation, injury to self or bed partner)	Neurology / Sleep medicine
Excessive daytime sleepiness disproportionate to nocturnal sleep disruption; suspected narcolepsy or idiopathic hypersomnia	Sleep medicine / Neurology
NREM parasomnias (sleepwalking, sleep eating, confusional arousals) with safety concerns or high frequency	Sleep medicine / Neurology
Active suicidal ideation	Psychiatry (urgent / emergency referral)
PHQ-9 ≥15 (moderately severe to severe depression)	Psychiatry (routine referral)
Chronic insomnia with suspected or established hypnotic dependence; failure of deprescribing attempts in primary care	Psychiatry / Addiction medicine
Treatment-refractory insomnia after adequate trial of CBT-I and pharmacotherapy	Sleep medicine / Psychiatry

V. Treatment - Management Algorithm

A. Non-pharmacological treatment - first line

CBT-I is the recommended first-line treatment for chronic insomnia disorder across all major current guidelines, including the AASM 2021, the VA/DoD CPG 2025, the European Insomnia Guideline 2023, and the Polish recommendations of the PTBS, PTMR, and PTP 2023 [1,2,3,8]. Its benefits have been demonstrated consistently across comorbid presentations. There is no evidence to support withholding it on the basis of psychiatric or somatic comorbidity [2,8]. CBT-I effects are also more durable than those of pharmacotherapy. Studies have shown durable improvements at long-term follow-up, with one randomised controlled trial reporting sustained gains at ten-year follow-up [16]. In contrast, sleep disturbance frequently returns after pharmacological discontinuation [8].

CBT-I is a structured multicomponent intervention typically delivered over four to eight individual or group sessions by a trained clinician. Its components are summarised in Table 2 [2,8]. Sleep restriction therapy and stimulus control appear to be the most effective individual elements based on network meta-analytic evidence, though the full multicomponent format remains the standard of care [8]. Sleep hygiene advice delivered as a standalone intervention is not an evidence-based treatment for chronic insomnia and should not be offered in isolation [2,8].

A major practical barrier to face-to-face CBT-I is the shortage of trained therapists across Europe [8,12]. In most European healthcare systems, CBT-I is not routinely offered in primary care- primarily due to limited access and the absence of funding frameworks [12]. Digital CBT-I (dCBT-I), delivered via automated or guided web- and app-based platforms, offers a clinically effective alternative where therapist access is limited [11]. Meta-analyses show that dCBT-I produces clinically meaningful improvements in insomnia severity, though effect sizes are moderate and somewhat smaller than those seen with face-to-face CBT-I [8,11]. NICE has recommended Sleepio — a platform supported by a clinical evidence base of 28 studies, including 12 randomised controlled trials — as a treatment option for patients who would otherwise receive only sleep hygiene advice or pharmacotherapy [10] Polish-speaking patients can also access Goodsleeper, a Polish- and Ukrainian-language application offering a full self-directed sleep programme, as well as the internationally available CBT-I Coach [1].

Table 2. Core components of CBT-I.

Component	Description
Sleep restriction therapy	Time in bed is initially limited to the patient's actual average sleep duration; progressively extended as sleep efficiency improves (target ≥85%)
Stimulus control	Reassociates the bed and bedroom with sleep; key instructions include going to bed only when sleepy, leaving bed if unable to sleep, and maintaining a fixed wake time
Cognitive restructuring	Identification and systematic challenge of dysfunctional beliefs and catastrophic thoughts about sleep using Socratic dialogue and behavioural experiments
Relaxation training	Progressive muscle relaxation (Jacobson), autogenic training, diaphragmatic breathing, or guided imagery to reduce somatic and cognitive arousal
Paradoxical intention	Patient is instructed to remain passively awake, thereby reducing performance anxiety and sleep effort
Psychoeducation / sleep hygiene	Foundational information on sleep physiology, circadian regulation, and modifiable behavioural factors; delivered as an integral element of CBT-I, not as a standalone therapy

B. Pharmacotherapy - second line

Pharmacotherapy should be considered as a second-line option when CBT-I has proven insufficient, is not accessible, or when the clinical situation requires rapid symptom relief as a bridge to behavioural treatment. The core principles of pharmacological management are: use of the lowest effective dose, the shortest clinically necessary duration (generally no longer than four weeks for most agents), active shared decision-making with the patient regarding risks and benefits, and a pre-agreed deprescribing plan established at the outset of treatment [3,4,8].

Prolonged-release melatonin (PR melatonin, 2 mg) is the preferred first pharmacological option in patients aged 55 years and older. It holds EMA approval for this indication for up to 13 weeks [8]. It does not cause dependence or rebound insomnia and is not contraindicated in OSA [8].

Daridorexant, a dual orexin receptor antagonist (DORA), is the only agent in this class to hold EMA authorisation for chronic insomnia disorder in adults. It is indicated specifically for patients with insomnia of at least three months' duration that significantly impacts daytime functioning [8]. By selectively blocking orexin receptors OX1R and OX2R, it promotes sleep by reducing wakefulness drive rather than causing generalised CNS depression. This results in no next-morning sedation, no rebound insomnia on discontinuation, and no evidence of dependence in studies of up to one year [8]. Phase III data demonstrated significant improvements in wake after sleep onset and sleep latency at doses of 25 mg and 50 mg; improvement in daytime functioning was demonstrated only at the 50 mg dose [17]. The recommended dose is 25 or 50 mg taken at bedtime [17]. Daridorexant is contraindicated in narcolepsy, with strong CYP3A4 inhibitors, and in severe hepatic impairment; caution is advised in older adults due to the risk of falls. It should be considered when CBT-I is inadequate or is inaccessible and when Z-drugs or benzodiazepines are contraindicated or poorly tolerated - particularly in patients with OSA, in whom it carries no respiratory depressant effect [8]. In Poland, daridorexant has been recently evaluated by the Agency for Health Technology Assessment and Tariff System (AOTMiT) for the indication of non-organic insomnia in patients with inadequate response to or intolerance of available pharmacological alternatives [9].

Z-drugs (non-benzodiazepine benzodiazepine receptor agonists: zolpidem, zopiclone, zaleplon, eszopiclone) act as positive allosteric modulators of the GABA-A receptor and are effective for short-term treatment of sleep onset and sleep maintenance insomnia [4,8]. Their use is approved for up to four weeks. Prolonged use carries risks of tolerance, dependence, rebound insomnia, complex sleep behaviours, and -

particularly in older adults - falls and cognitive impairment [8,12]. They are relatively contraindicated in OSA and should be used with caution in patients with a history of substance use disorder [3,8].

Benzodiazepines (lormetazepam, nitrazepam, temazepam) share the mechanism of action and risk profile of Z-drugs and are likewise approved for short-term use only [8]. They should generally be avoided in patients aged 65 years and older, in whom they are listed in the American Geriatrics Society Beers Criteria as potentially inappropriate medications [1].

Sedating antidepressants are used off-label for insomnia in the absence of comorbid depression, typically at doses lower than those employed in the treatment of affective disorders [8]. Trazodone and low-dose doxepin have the most robust evidence base among this group, with small to moderate effects on sleep maintenance and total sleep time [8]. Mirtazapine, mianserin, and agomelatine are also used in clinical practice, though their evidence base for insomnia as the primary indication remains limited [8,12]. These agents are preferred when insomnia co-occurs with depressive or anxiety symptoms [8].

Sedating antipsychotics (quetiapine, chlorprothixene, levomepromazine) are used off-label in some clinical settings, particularly in patients with comorbid psychiatric conditions, but current guidelines do not recommend them for insomnia without a primary psychiatric indication [3,8]. Their use is associated with significant risks including metabolic effects, extrapyramidal symptoms, QT prolongation, and - in elderly patients with dementia - substantially increased mortality.

The main pharmacological options are summarised in Table 3 [1,3,4,8].

Table 3. Summary of pharmacological agents used in the management of chronic insomnia disorder.

Drug class	Representative agents	Approved duration	Key risks	Notes
Prolonged-release melatonin	Melatonin PR 2 mg	Up to 13 weeks	Minimal; no dependence	First choice ≥55 years; safe in OSA
Dual orexin receptor antagonist (DORA)	Daridorexant 25-50 mg	Up to 3 months; reassess every 3 months	Falls risk in elderly; contraindicated in narcolepsy and with strong CYP3A4 inhibitors	EMA-approved for chronic insomnia; no dependence, no rebound; safe in OSA
Z-drugs (BzRA)	Zolpidem, zopiclone, zaleplon, eszopiclone	≤4 weeks	Dependence, rebound, falls, complex sleep behaviours	Avoid in OSA; caution in substance use disorder
Benzodiazepines	Lormetazepam, nitrazepam, temazepam	≤4 weeks	Dependence, rebound, cognitive impairment, falls	Avoid in ≥65 years; Beers Criteria
Sedating antidepressants (off-label)	Trazodone, doxepin, mirtazapine, mianserin, agomelatine	No approved indication (off-label)	Variable by agent; suicidal ideation warning	Preferred when comorbid depression/anxiety present
Sedating antipsychotics (off-label)	Quetiapine, chlorprothixene, levomepromazine	Not recommended for routine use	Metabolic risk, QT prolongation, mortality increased in elderly	Last resort; only with comorbid psychiatric indication

Off-label use is defined with reference to current European Medicines Agency (EMA) marketing authorisations. National approval status may vary.

VI. Special Populations

Patients aged ≥65 years

In older adults, the risks of pharmacotherapy for insomnia - including falls, cognitive impairment, and dependence - substantially outweigh the benefits. CBT-I remains the first-line treatment of choice [1,2]. When pharmacotherapy is necessary, prolonged-release melatonin 2 mg is the preferred agent in patients aged 55 years and older, and particularly in those aged ≥65 years. It carries no risk of dependence, no clinically meaningful next-morning sedation, no impairment of psychomotor performance, and no increased risk of falls [1]. It should be taken 1-2 hours before the intended sleep time, combined with behavioural interventions, for up to 13 weeks [1,8].

If Z-drugs and benzodiazepines are considered unavoidable, they should be used at half the standard adult dose in patients aged ≥65 years, given the age-related reduction in hepatic metabolism and renal clearance [3,4]. Both drug classes are listed in the American Geriatrics Society Beers Criteria as potentially inappropriate medications in older adults, due to their well-documented association with falls, fractures, cognitive impairment, motor vehicle accidents; for benzodiazepines - a risk-benefit ratio that worsens with advancing age [1,4]. Sedating antipsychotics carry a black-box warning for increased mortality in elderly patients with dementia-related psychosis and should be avoided in this age group except when a primary psychiatric indication exists [3,8].

Insomnia comorbid with depression or anxiety

When insomnia co-occurs with a depressive or anxiety disorder, treatment of the psychiatric condition takes priority, as effective management of the underlying disorder typically improves sleep as well [8]. However, insomnia should also be targeted independently, since residual sleep disturbance is a recognised predictor of depressive relapse [3,8]. In this clinical context, sedating antidepressants - particularly trazodone, mirtazapine, and low-dose doxepin - are preferred over Z-drugs, as they address both the psychiatric and the sleep complaint [8]. CBT-I is effective regardless of the presence of depression or anxiety, and can be safely combined with antidepressant treatment [8].

Insomnia comorbid with obstructive sleep apnoea (COMISA)

In patients with confirmed or suspected OSA, prolonged-release melatonin is the safest pharmacological option for the management of coexisting insomnia, as it carries no respiratory depressant effect [1,8]. Benzodiazepines and Z-drugs are relatively contraindicated in this setting due to their potential to suppress upper airway muscle tone and worsen apnoea severity [3,8]. CBT-I can be safely delivered in patients with COMISA and may itself improve OSA-related outcomes by reducing time in bed and increasing sleep drive [8].

Insomnia comorbid with restless legs syndrome

In patients with RLS-related insomnia, the first clinical step is measurement of serum ferritin and transferrin saturation. If ferritin is ≤75 ng/mL or transferrin saturation is <20%, oral or intravenous iron supplementation should be initiated, as iron repletion alone can substantially reduce RLS symptoms and improve sleep [7]. For pharmacological treatment of RLS, gabapentin and pregabalin are the current first-line agents based on strong AASM recommendations [7]. Dopamine agonists (pramipexole, ropinirole) are not recommended for routine use given the risk of augmentation with long-term dopaminergic therapy, and should be reserved only for patients who do not tolerate or respond to alpha-2-delta ligands after careful risk-benefit discussion [7]. Mirtazapine and antipsychotic medications should be avoided in patients with RLS. Mirtazapine has been reported to exacerbate or precipitate RLS symptoms, likely through its antihistaminergic and possible antidopaminergic effects, while antipsychotics worsen RLS via dopamine receptor blockade [7].

VII. Deprescribing Hypnotic Medications

A substantial proportion of patients presenting to primary care with chronic insomnia are long-term users of benzodiazepines or Z-drugs, often having exceeded the approved treatment duration by months or years. In some European surveys, more than 40% of hypnotic users report continuous use exceeding 12 months [5,12]. Chronic hypnotic use is associated with progressive loss of efficacy and physical dependence [5,8]. Cognitive impairment and falls are well-documented risks [8,12]. Discontinuation is frequently complicated by rebound insomnia and conditioned fear of sleep without medication [5,8].

The 2024 European neuropsychopharmacology and sleep expert consensus on switching and deprescribing hypnotic medications provides the most current framework for this clinical challenge [5]. The recommended approach involves a gradual dose reduction of 10-25% per week, titrated to the patient's tolerance [5]. Concurrent initiation of CBT-I - delivered face-to-face or via a validated digital platform - substantially improves the likelihood of successful discontinuation. It provides patients with alternative

strategies for managing sleep and addressing the cognitive perpetuating factors that pharmacotherapy leaves untreated [5,8].

Where direct tapering is poorly tolerated, a cross-tapering strategy may be used. The current hypnotic is gradually replaced with prolonged-release melatonin 2 mg or - where available - eszopiclone, both of which have been shown to facilitate benzodiazepine and Z-drug discontinuation within a structured cross-taper programme [5]. Daridorexant 50 mg is an additional cross-tapering option supported by naturalistic evidence [5]. Patients should be explicitly counselled before initiating deprescribing that rebound insomnia is an expected and time-limited consequence of hypnotic withdrawal, not a sign of treatment failure or a return of the underlying disorder [5].

VIII. Conclusions

The evidence reviewed supports five actionable conclusions for primary care clinicians managing chronic insomnia disorder.

First, chronic insomnia is a disorder in its own right and should be treated as such regardless of comorbidities. The removal of the primary-secondary distinction from DSM-5, ICSD-3, and ICD-11 reflects the contemporary understanding that insomnia perpetuates itself through psychophysiological mechanisms largely independent of any precipitating cause. Targeted treatment is warranted in all cases [8].

Second, CBT-I is the consistently recommended first-line treatment, with durable effects that pharmacotherapy cannot replicate. Its efficacy extends across comorbid conditions and patient age groups. Where face-to-face delivery is unavailable, validated digital CBT-I platforms offer a clinically effective and scalable alternative [1,2,8,10,11].

Third, pharmacotherapy is a short-term adjunct- not a substitute for behavioural treatment. When indicated, it should be prescribed at the lowest effective dose for the shortest necessary duration, with a deprescribing plan agreed upon at the outset. Prolonged-release melatonin is the preferred agent in patients aged 55 years and older. Daridorexant represents an evidence-based alternative with EMA approval for chronic insomnia where first-line agents are contraindicated or poorly tolerated. Z-drugs and benzodiazepines should not be used long-term in any age group, and are particularly hazardous in older adults [1,3,4,8,17].

Fourth, the primary care consultation is the principal setting for identifying and managing comorbid conditions that perpetuate insomnia - including depression [13], OSA [3,8], and RLS [7]- and for recognising the clinical presentations that require specialist referral, including central hypersomnolence disorders [14] and REM sleep behaviour disorder [15].

Fifth, the large number of primary care patients on long-term hypnotics is a problem that is both prevalent and addressable within routine practice. Structured deprescribing, guided by the 2024 European expert consensus, supported by concurrent CBT-I, and accompanied by patient education about rebound insomnia, should be integrated into routine primary care [5,8].

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