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Optimizing Chronic Insomnia Care through Pharmacist Intervention, Laboratory Evaluation, and Administrative Coordination

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Abstract

Background: Chronic insomnia is a highly prevalent sleep disorder characterized by persistent difficulty initiating or maintaining sleep, leading to significant daytime impairment. It is a major public health concern with strong links to psychiatric and medical comorbidities, including depression, anxiety, and cardiovascular disease, and is driven by a complex pathophysiology involving hyperarousal and dysregulation of sleep-wake circuits.

Aim: This article aims to outline a comprehensive, interprofessional framework for managing chronic insomnia, emphasizing the synergistic roles of pharmacists, laboratory services, and health administrators in optimizing patient care from diagnosis through long-term treatment.

Methods: The proposed model integrates evidence-based interventions. First-line treatment is Cognitive Behavioral Therapy for Insomnia (CBT-I). Pharmacological options, including GABAergic agents, dual orexin receptor antagonists, and melatonin agonists, are used adjunctively. The framework leverages pharmacists for medication management and deprescribing, laboratory services for identifying underlying medical causes, and health administrators for implementing system-wide protocols and digital health solutions like internet-delivered CBT-I (iCBT-I).

Results: This collaborative approach facilitates accurate diagnosis, ensures guideline-concordant treatment, and improves patient adherence. It leads to reduced reliance on long-term pharmacotherapy, better management of comorbid conditions, and more efficient use of healthcare resources through streamlined workflows and improved access to first-line behavioral therapies.

Conclusion: Effective management of chronic insomnia requires a unified, systems-based strategy. By integrating the distinct expertise of clinicians, pharmacists, laboratory professionals, and administrators, healthcare systems can shift from a reactive, medication-focused model to a proactive, patient-centered approach that addresses the root causes of insomnia and improves long-term outcomes.

Keywords: Chronic Insomnia, Cognitive Behavioral Therapy for Insomnia (CBT-I), Hyperarousal, Interprofessional Collaboration, Pharmacist, Deprescribing, Healthcare Administration.

1. Introduction

Chronic insomnia represents the most common and persistent sleep disorder encountered in both community and clinical practice, affecting individuals across all age groups and socioeconomic backgrounds. It is not only a major complaint during primary care consultations but also a significant public health concern due to its profound impact on physical, mental, and social well-being. According to the American Academy of Sleep Medicine's International Classification of Sleep Disorders, Third Edition (ICSD-3), chronic insomnia is defined by difficulty initiating sleep, maintaining sleep, or achieving restorative sleep quality despite adequate opportunity and circumstances conducive to sleep. The diagnostic criteria specify that these symptoms must occur at least three nights per week and persist for a duration exceeding three months, ultimately leading to measurable daytime impairment in cognitive, emotional, or occupational functioning [1]. Unlike transient or short-term insomnia, which is often resolved with the removal of stressors or environmental changes, chronic insomnia is a sustained disorder requiring comprehensive evaluation and multidisciplinary management. Diagnosis remains primarily clinical, dependent on self-reported complaints of poor sleep and corroborating evidence from sleep diaries or questionnaires, as objective testing such as polysomnography is typically reserved for cases where other sleep disorders are suspected.

The burden of chronic insomnia extends beyond nocturnal symptoms, exerting far-reaching effects on multiple domains of health and quality of life. Persistent sleep disruption alters circadian rhythm regulation and neuroendocrine balance, leading to fatigue, irritability, impaired attention, reduced problem-solving ability, and emotional instability.

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These daytime consequences translate into tangible societal costs through diminished workplace productivity, higher rates of absenteeism, and an increased likelihood of motor vehicle and occupational accidents. In academic settings, students suffering from chronic insomnia frequently exhibit decreased concentration, impaired memory retention, and poorer academic performance. Over time, the cumulative cognitive and psychological strain contributes to maladaptive coping behaviors, such as increased caffeine or alcohol consumption, further perpetuating sleep disturbance and functional decline [2]. Moreover, chronic insomnia rarely occurs in isolation but is strongly associated with a broad range of psychiatric and medical comorbidities. Psychiatric disorders—most notably depression, anxiety, and posttraumatic stress disorder (PTSD)—frequently coexist with insomnia, forming bidirectional relationships wherein each condition exacerbates the other. From a physiological standpoint, the chronic activation of stress pathways, particularly the hypothalamic-pituitary-adrenal (HPA) axis, contributes to sympathetic overactivity and systemic inflammation. These alterations are implicated in the development or worsening of hypertension, cardiovascular disease, and metabolic dysregulation. In addition, chronic insomnia has been linked to numerous somatic conditions, including chronic pain syndromes, gastroesophageal reflux disease (GERD), chronic obstructive pulmonary disease (COPD), asthma, and benign prostatic hyperplasia (BPH), where nocturnal symptoms such as coughing, wheezing, or nocturia disrupt sleep continuity. Likewise, obstructive sleep apnea (OSA) often coexists with insomnia, creating a complex clinical picture known as "COMISA" (comorbid insomnia and sleep apnea), which is associated with poorer outcomes than either disorder alone. Vasomotor symptoms in postmenopausal women and substance use disorders also contribute to the perpetuation of insomnia through physiological arousal or withdrawal effects. The multidimensional nature of chronic insomnia—encompassing neurobiological, psychological, behavioral, and environmental components—necessitates an integrated approach to care that extends beyond symptomatic treatment. Early identification and intervention are critical to prevent chronicity, reduce the risk of comorbid psychiatric and medical conditions, and improve overall life satisfaction. Consequently, pharmacists, laboratory professionals, and health administrators each play a vital role within the multidisciplinary framework for managing chronic insomnia, contributing to prevention, diagnosis, and long-term therapeutic optimization [2].

Figure-1: Chronic Insomnia.

Etiology

Chronic insomnia arises from a multifactorial interplay of biological, psychological, and environmental factors that contribute to the persistence of disordered sleep. It can be categorized as either primary, occurring independently without an identifiable underlying condition, or secondary, when it develops as a symptom or complication of another medical, psychiatric, or behavioral disorder. Despite this distinction, both forms of insomnia share similar physiological and functional consequences, reinforcing the recognition of chronic insomnia as a distinct clinical disorder rather than a mere symptom of comorbidities. The pathophysiology of chronic insomnia remains incompletely understood, but current models emphasize the role of hyperarousal, stress reactivity, and dysregulation of circadian and homeostatic sleep mechanisms in its genesis [3]. The hyperarousal model posits that chronic insomnia is characterized by a persistent state of heightened physiological and cognitive activation that interferes with both sleep initiation and maintenance. This hyperarousal manifests as elevated metabolic rate, increased heart rate, higher core body temperature, and elevated secretion of stress hormones such as cortisol and adrenocorticotropic hormone (ACTH). However, whether hyperarousal acts as a causal factor, a secondary consequence, or an independent process remains uncertain. Psychological dimensions of the disorder—emotional reactivity, maladaptive cognitions about sleep, and personality traits such as perfectionism or neuroticism—further amplify and sustain this state of arousal. These cognitive and affective components can perpetuate a cycle of anxiety and rumination around sleep, often described as "sleep effort," in which the individual's preoccupation with obtaining adequate rest paradoxically prevents it [3].

To conceptualize the development and persistence of chronic insomnia, a comprehensive tripartite model has been proposed, incorporating predisposing, precipitating, and perpetuating factors [4]. Predisposing factors include innate vulnerabilities such as genetically determined variations in stress reactivity, abnormalities in neurobiological pathways regulating arousal and circadian rhythm, and personality characteristics like heightened emotional

sensitivity or cognitive inflexibility. These traits do not cause insomnia directly but increase an individual's susceptibility when exposed to stress. Precipitating factors, such as psychosocial stressors—including bereavement, occupational strain, academic pressure, or major life transitions—often trigger the initial onset of sleep disturbance. Acute insomnia at this stage may resolve if the stressor abates, but in vulnerable individuals, maladaptive coping behaviors and conditioned arousal can transform transient sleeplessness into a chronic pattern. Perpetuating factors sustain the disorder long after the original trigger has disappeared. These include behavioral changes like irregular sleep schedules, extended time in bed, excessive daytime napping, or reliance on sedatives and alcohol; cognitive distortions such as catastrophizing about sleeplessness; and emotional dysregulation that reinforces hypervigilance and frustration at bedtime. The interaction of these three domains—predisposition, precipitation, and perpetuation—creates a self-reinforcing loop that perpetuates chronic insomnia. Over time, neuroplastic changes may occur within the brain's sleep–wake regulatory circuits, particularly in the hypothalamic-pituitary-adrenal (HPA) axis and limbic structures, further entrenching the disorder. Chronic activation of these pathways leads to persistent sympathetic nervous system stimulation and altered neurotransmitter balance, notably involving gamma-aminobutyric acid (GABA), serotonin, and dopamine, which are critical in sleep regulation and mood stability. These alterations not only sustain insomnia but also contribute to memory impairment, cognitive dysfunction, and psychopathology, including anxiety and depressive symptoms [4]. In summary, chronic insomnia emerges from a dynamic and interactive process involving biological predisposition, acute psychosocial stressors, and maladaptive perpetuating behaviors. Although the hyperarousal model provides a valuable framework, ongoing research into the neurological, endocrine, and genetic underpinnings of insomnia continues to expand understanding of its complexity. Recognizing these interdependent mechanisms underscores the importance of a multidisciplinary approach to management—addressing psychological, behavioral, and physiological dimensions simultaneously to restore healthy sleep and prevent long-term cognitive and emotional consequences.

Epidemiology

Insomnia, particularly its chronic form, represents one of the most prevalent and burdensome sleep disorders globally, exerting a substantial impact on population health and healthcare systems. Population-based surveys consistently reveal that approximately one-third of adults (30%–36%) report experiencing at least one symptom of insomnia, such as difficulty initiating sleep, maintaining sleep, or early morning awakening. However, when the more rigorous diagnostic standards established by the International Classification of Sleep Disorders, Third Edition (ICSD-3) or the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) are applied—criteria that require symptom persistence for at least three months with associated daytime impairment—the estimated prevalence declines to between 6% and 10% of the adult population [5]. Epidemiologic trends reveal clear demographic and socioeconomic gradients. Middle-aged and older adults are disproportionately affected due to physiological changes in circadian rhythm, increased prevalence of chronic medical conditions, and higher medication use. Women consistently demonstrate higher rates of insomnia than men, a difference attributed to hormonal fluctuations during menstruation, pregnancy, and menopause, as well as higher rates of mood and anxiety disorders. Shift workers and individuals with irregular sleep schedules experience circadian misalignment that predisposes them to persistent insomnia symptoms. Similarly, patients with comorbid medical or psychiatric illnesses—including depression, anxiety, chronic pain, and cardiopulmonary diseases—face greater risk, suggesting a bidirectional relationship between insomnia and these conditions [5]. Globally, the prevalence of insomnia exhibits substantial geographic variation, reflecting differences in cultural norms, lifestyle patterns, socioeconomic stressors, and healthcare access. Reported rates range as high as 79% in Brazil and as low as 23.2% in Western Europe, indicating that environmental and social determinants strongly influence sleep health across populations [6]. This wide variability underscores insomnia's complex etiology, shaped by intersecting biological, psychological, and contextual factors. Collectively, these data highlight insomnia as a major global health concern—one with profound implications for productivity, mental health, and quality of life.

Pathophysiology

The pathophysiology of chronic insomnia is multifactorial, encompassing genetic, neurobiological, psychological, and environmental mechanisms that interact within a complex, dynamic system regulating arousal and sleep. Predisposing factors for chronic insomnia include both genetic and epigenetic influences that modulate neural circuits involved in emotion regulation and stress response rather than those directly controlling sleep itself. Evidence from genome-wide association studies (GWAS) demonstrates that insomnia is a polygenic disorder, meaning that multiple genes, each exerting small effects, collectively contribute to susceptibility. These genetic factors overlap substantially with those implicated in restless legs syndrome, cardiometabolic disorders, and psychiatric conditions such as depression and anxiety, suggesting shared neurobiological pathways linking stress, mood, and arousal regulation [4]. Intriguingly, the neuronal cell

types expressing insomnia-associated genes are localized predominantly in emotion-regulating circuits—including the amygdala, anterior cingulate cortex, and prefrontal regions—rather than traditional sleep-promoting nuclei. This finding reinforces the hypothesis that chronic insomnia reflects a dysfunction of emotional and stress regulatory systems rather than a primary defect of the sleep-wake architecture itself [4]. A key neurophysiologic framework for understanding insomnia is the flip-flop switch model of sleep regulation. This model conceptualizes sleep-wake transitions as a bistable system in which sleep-promoting neurons and wake-promoting neurons exert reciprocal inhibition, maintaining stability in either state while permitting rapid transitions between them. Wakefulness is driven by neuronal populations in the posterior hypothalamus and brainstem reticular formation, including cholinergic, noradrenergic, serotonergic, and histaminergic systems that maintain cortical arousal. Sleep onset occurs when the ventrolateral preoptic nucleus (VLPO)—the primary sleep-promoting center—becomes active, releasing the inhibitory neurotransmitters gamma-aminobutyric acid (GABA) and galanin to suppress the wake-promoting nuclei. Conversely, wake-promoting systems release norepinephrine and serotonin, which inhibit the VLPO to maintain alertness. In healthy individuals, this reciprocal inhibition forms a stable switch that prevents simultaneous activation of both systems. However, in chronic insomnia, this switch mechanism becomes unstable, producing an imbalance between sleep-inducing and arousal-inducing processes. Persistent hyperactivity of the arousal networks—fueled by heightened sympathetic tone, cortical excitability, and stress hormone secretion—leads to difficulty initiating and maintaining sleep [4][7].

Although the circadian and homeostatic sleep drive mechanisms contribute to sleep regulation, evidence suggests they are not primary drivers of chronic insomnia. Instead, these processes interact with arousal systems, and dysregulation in their feedback loops may amplify the consequences of hyperarousal. Circadian misalignment—common in shift workers or individuals with irregular schedules—can exacerbate insomnia symptoms by desynchronizing internal sleep-wake rhythms. Similarly, deficits in sleep homeostasis, such as a reduced buildup of sleep pressure or increased tolerance to sleep deprivation, may perpetuate wakefulness even when sleep opportunity is available [4]. A third major contributor to chronic insomnia is emotional dysregulation, particularly the maladaptive response to stress. Stressful life events are among the most significant precipitants of insomnia, triggering transient disturbances that, in vulnerable individuals, evolve into chronic patterns through conditioning and hyperarousal. The hypothalamic-pituitary-adrenal (HPA) axis plays a central role in this process: increased cortisol secretion, heightened sympathetic activation, and elevated levels of corticotropin-releasing hormone (CRH) perpetuate physiological arousal incompatible with restful sleep. Furthermore, early-life adversity—including trauma or chronic stress exposure during childhood—has been shown to alter the epigenetic regulation of genes associated with stress responsiveness and emotional control, thereby increasing lifetime susceptibility to insomnia [4]. These findings emphasize the enduring effects of environmental influences on gene expression through epigenetic modifications such as DNA methylation and histone acetylation, which can modulate the function of arousal and emotion-related neural pathways.

Taken together, chronic insomnia emerges as a synergistic outcome of genetic predisposition, neurobiological dysregulation, and environmental stressors operating within an epigenetic framework. It is best conceptualized as a polygenic, stress-related disorder in which persistent hyperarousal disrupts the delicate equilibrium of sleep-wake regulatory networks. The interdependence of emotional, cognitive, and physiological systems explains the high comorbidity between insomnia and mood or anxiety disorders. Thus, chronic insomnia is not simply a symptom of poor sleep hygiene or psychological distress but a complex neurobehavioral condition reflecting maladaptive plasticity within brain circuits that mediate arousal, emotion, and stress response [8]. History and Physical A comprehensive evaluation of chronic insomnia begins with an in-depth clinical interview and a focused physical examination aimed at identifying predisposing, precipitating, and perpetuating factors contributing to the disorder. The patient history is the cornerstone of assessment, as the diagnosis is largely based on self-reported symptoms rather than objective testing. Clinicians must obtain a detailed account of the onset, duration, and course of sleep disturbances, including whether the difficulty lies in sleep initiation, maintenance, or early awakening. Understanding the timing of onset—for example, whether insomnia followed a stressful event, a medical diagnosis, or medication change—helps determine its precipitating causes and chronicity [1][9].

The medical history should address any comorbid conditions known to interfere with sleep, such as chronic pain syndromes, cardiovascular disease, asthma, gastroesophageal reflux disease (GERD), benign prostatic hyperplasia (BPH), or neurological disorders like Parkinson's disease. Because many substances alter sleep architecture, clinicians must inquire about caffeine, alcohol, nicotine, cannabis, prescription medications, and illicit drugs, as both dependence and withdrawal can exacerbate insomnia symptoms. Likewise, exercise habits and daytime activity levels should be reviewed, as sedentary lifestyles and excessive evening exercise may influence sleep quality. Given the high prevalence of psychiatric comorbidities, a focused

psychological assessment is essential, particularly for depression, anxiety, posttraumatic stress disorder, and stress-related conditions. The clinician should explore psychosocial stressors, including occupational, financial, legal, or interpersonal conflicts, that may perpetuate hyperarousal and rumination. For diagnostic accuracy, collateral information from the bed partner can be invaluable, especially regarding snoring, witnessed apneas, parasomnias, or periodic limb movements, which may suggest comorbid sleep disorders such as obstructive sleep apnea or restless legs syndrome [9]. Environmental and behavioral contributors must also be evaluated. Questions regarding shift or night work, sleep environment (including light exposure, noise, and temperature), and sleep hygiene behaviors—such as screen use, caffeine intake, or inconsistent bedtimes—help identify modifiable factors. Clinicians should assess sleep-wake patterns and daytime functioning, including fatigue, concentration difficulties, irritability, or reduced performance, to gauge the disorder's overall impact. Documentation of previous treatment attempts, including pharmacologic and nonpharmacologic interventions, provides valuable insight into prior response patterns and treatment resistance.

Objective tools can complement clinical assessment. Validated instruments such as the Insomnia Severity Index (ISI) [10] and the Sleep Condition Indicator (SCI) [11] provide standardized measures of insomnia severity and its functional consequences. Additionally, patients should maintain a sleep diary for 7 to 14 days to record bedtimes, wake times, naps, and perceived sleep quality, enabling clinicians to identify patterns, inconsistencies, and potential behavioral contributors. The physical examination should be directed by findings from the history—such as evaluating for upper airway obstruction, thyroid abnormalities, or neurological deficits—to exclude secondary causes. A thorough and structured history and physical examination thus form the foundation for accurate diagnosis, individualized treatment planning, and optimal long-term management of chronic insomnia [1][9].

Evaluation

The evaluation of chronic insomnia is a multidimensional process that relies primarily on a detailed clinical history rather than extensive laboratory or imaging studies. Diagnosis is established through an assessment of the individual's sleep history, along with a comprehensive review of medical, psychiatric, and substance use histories. Because insomnia is often a manifestation of multiple converging factors—ranging from behavioral and psychological to physiological—the clinician's primary goal during evaluation is to identify contributing causes, perpetuating factors, and comorbid conditions that might influence management strategies [9]. Initial assessment focuses on a structured clinical interview that explores sleep onset latency, frequency of nocturnal awakenings, early morning awakenings, total sleep time, and perceived sleep quality. Patients should be asked to describe their sleep environment, pre-sleep behaviors, and lifestyle patterns such as caffeine or alcohol consumption, nicotine use, and exercise habits. Psychiatric history is equally essential, as mood and anxiety disorders frequently coexist with insomnia and often intensify sleep difficulties. Likewise, medical comorbidities such as chronic pain syndromes, thyroid dysfunction, pulmonary disease, gastroesophageal reflux, and neurodegenerative disorders must be investigated since they may disrupt sleep continuity or architecture. A detailed medication history is vital, as certain agents—including corticosteroids, beta-blockers, stimulants, and some antidepressants—can provoke or worsen insomnia. Substance use, including over-the-counter sleep aids, herbal supplements, and recreational drugs, should also be explored to prevent misdiagnosis or inadvertent pharmacologic reinforcement of sleep disturbance [9]. Laboratory and imaging studies are not routinely required for the initial evaluation of chronic insomnia but may be considered when the history or physical examination suggests underlying systemic or neurologic disease. When warranted, testing may include a complete blood count (CBC) to assess for anemia or systemic inflammation, thyroid function tests to exclude hyperthyroidism, liver and renal function panels to identify metabolic derangements, and C-reactive protein to evaluate for systemic inflammatory processes. Additional specialized studies may include ferritin to assess for iron deficiency in patients with suspected restless legs syndrome, and vitamin B12 levels to rule out neuropathic contributors to disturbed sleep. In specific scenarios, electrocardiogram (ECG), electroencephalogram (EEG), or brain imaging may be appropriate to exclude structural or electrical abnormalities affecting arousal regulation. Research-based evaluations may also involve measurement of circadian phase markers, such as melatonin secretion profiles and core body temperature rhythms, particularly when a circadian rhythm sleep-wake disorder is suspected [9].

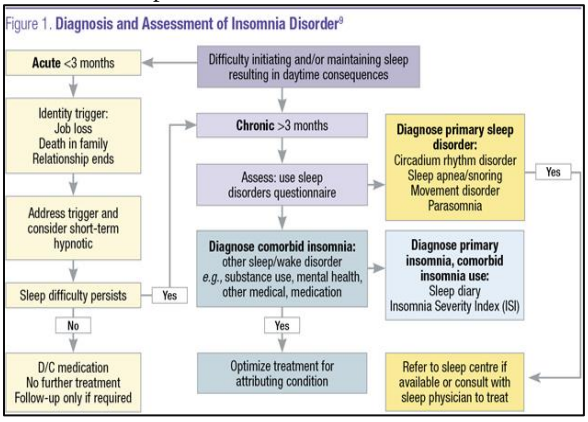

Figure-2: Diagnosis of Insomnia.

A key component of evaluation is the use of sleep diaries or sleep logs, which should be maintained for at least 7 to 14 days. These tools provide a practical, low-cost means of assessing an individual's habitual sleep-wake cycle, including variability in bedtime and wake time, nocturnal awakenings, total sleep time, and subjective sleep quality [9]. Diaries also document daytime naps, alcohol and caffeine intake, and bedtime activities, allowing clinicians to identify patterns and behaviors that may perpetuate insomnia. Parameters derived from sleep logs—such as sleep efficiency, sleep latency, and wakefulness after sleep onset (WASO)—help quantify sleep disturbance and circadian irregularities. Despite their usefulness, sleep diaries rely on patient self-reporting, and their validity and reliability may vary, particularly in individuals with poor insight or cognitive impairment [12]. Nevertheless, when used consistently, sleep logs remain among the most informative and accessible diagnostic tools for clinicians. To complement subjective data, validated scales provide standardized measures of insomnia severity and impact. The Insomnia Severity Index (ISI) [10] and the Sleep Condition Indicator (SCI) [11] are two widely endorsed instruments that assess sleep satisfaction, daytime function, and distress associated with poor sleep. Additional scales frequently used in clinical and research settings include the Epworth Sleepiness Scale (ESS), which quantifies daytime sleepiness and helps differentiate insomnia from hypersomnolence disorders, and the Pittsburgh Sleep Quality Index (PSQI), a comprehensive tool evaluating sleep quality and disturbances over a one-month interval [13][14]. Together, these validated questionnaires offer quantitative insight into sleep problems and provide baseline data for monitoring treatment outcomes over time.

Polysomnography (PSG), the gold standard for objective sleep assessment, is generally reserved for cases where coexisting sleep disorders are suspected rather than for primary insomnia itself. PSG records multiple physiological parameters during sleep—including electroencephalographic, electromyographic, electrocardiographic, respiratory, and oximetric data—and is indispensable in diagnosing sleep-related breathing disorders such as obstructive sleep apnea (OSA), or sleep-related movement disorders such as restless legs syndrome (RLS) or periodic limb movement disorder (PLMD) [8]. However, routine use of PSG in patients with uncomplicated chronic insomnia is not recommended, as findings often fail to correlate with subjective symptoms and may not alter management [15]. Instead, PSG is indicated only when history, examination, or treatment resistance suggests another underlying sleep pathology. When circadian rhythm disturbances are suspected—such as delayed sleep-wake phase disorder or irregular sleep-wake rhythm disorder—actigraphy can serve as a valuable adjunctive tool. Actigraphy involves wearing a wrist-mounted accelerometer that continuously measures motor activity to infer sleep and wake periods over days to weeks. The data provide objective estimates of total sleep time, sleep latency, wake after sleep onset, and daytime naps, enabling clinicians to identify misalignment between the patient's internal circadian rhythm and external schedules [16]. While actigraphy offers convenience and ecological validity, it cannot detect respiratory events, periodic limb movements, or abnormal behaviors during sleep, and therefore should not replace polysomnography when such disorders are suspected [8]. Ultimately, the evaluation of chronic insomnia is comprehensive and integrative, combining detailed history-taking, behavioral and environmental assessment, subjective sleep logging, and selective objective testing. The emphasis remains on identifying reversible causes and contributing factors while distinguishing primary insomnia from secondary or comorbid sleep disorders. A thoughtful, patient-centered approach—rooted in clinical observation rather than routine laboratory investigation—ensures accurate diagnosis, avoids unnecessary testing, and forms the foundation for individualized, evidence-based management of chronic insomnia.

Treatment / Management

Nonpharmacological Management

International guidelines converge in recommending nonpharmacological therapy—most prominently cognitive behavioral therapy for insomnia (CBT-I)—as the first-line intervention for chronic insomnia across clinical contexts, given robust benefits for nocturnal symptoms, daytime functioning, and common comorbidities [8]. CBT-I is a manualized, skills-based treatment typically delivered by trained clinicians in individual or group formats, though hybrid and digital adaptations have expanded reach in recent years. Conceptually, CBT-I is grounded in the three-factor model of chronic insomnia, which frames symptom persistence as the interplay of predisposing, precipitating, and perpetuating influences; treatment targets the behavioral and cognitive processes that maintain hyperarousal and conditioned wakefulness long after initial triggers abate [17][18] (A1). Core elements include a behavioral arm—dominated by sleep restriction therapy and stimulus control—and a cognitive arm that uses restructuring and metacognitive strategies to recalibrate beliefs about sleep, mitigate catastrophic appraisal of short-term sleep loss, and reduce sleep effort. Together, these components realign the sleep–wake system by restoring homeostatic pressure, re-associating the bed and bedroom with rapid sleep onset, and dampening psychophysiological arousal common to chronic insomnia [8][18]. Sleep restriction therapy is purposefully counterintuitive: rather than extending time in bed to "catch up" on lost sleep, clinicians deliberately limit sleep opportunity to match the individual's empirically observed sleep ability, as tracked over a baseline diary period [18]. The process

begins with a sleep log to estimate average total sleep time and variability. A prescribed sleep window is then set so that time in bed equals average sleep time, anchored to a consistent wake time that is maintained seven days a week. Weekly review of the diary guides titration: if sleep becomes consolidated and sleep efficiency improves, the time-in-bed window is lengthened in small increments; if fragmentation persists, the window is held or reduced. Over several weeks, this controlled restriction increases homeostatic sleep drive, shortens sleep latency, reduces wake after sleep onset, and builds confidence in the capacity to sleep, thereby reversing maladaptive patterns of compensatory napping and long, unproductive nights in bed [18].

Stimulus control complements sleep restriction by dismantling the conditioned association between the sleep environment and wakeful arousal. Patients are instructed to lie down only when subjectively sleepy; if sleep does not occur within roughly 15 to 20 minutes, they leave the bed and engage in a quiet, non-stimulating activity under low light, returning only when drowsy—repeating the cycle as needed through the night [18]. Fixed wake times, elimination of daytime naps, and confining the bed to sleep (and sexual activity) are emphasized so that the bed again signals rapid onset of sleep rather than rumination and frustration. Although sleep hygiene alone is insufficient as a stand-alone therapy, it remains an essential adjunct within CBT-I for addressing modifiable environmental and lifestyle contributors, including reducing evening caffeine and alcohol, establishing an ergonomically comfortable sleep setting, implementing a wind-down routine, and scheduling exercise while avoiding vigorous late-evening activity [18]. Collectively, these practices support the behavioral reconditioning that underpins the efficacy of CBT-I. Access to trained therapists has historically constrained dissemination of CBT-I, and financing mechanisms can be inconsistent. However, pragmatic trials demonstrate that internet-delivered CBT-I (iCBT-I) implemented within routine care pathways reduces insomnia severity relative to usual care, offering a scalable, cost-effective avenue for health systems to meet demand [19][20] (A1). Hybrid models—brief clinician contact paired with digital modules—can preserve fidelity while enhancing adherence through coaching, troubleshooting, and personalization. Beyond CBT-I, relaxation-based techniques such as diaphragmatic breathing, mindfulness meditation, progressive muscle relaxation, and gentle yoga can attenuate physiological arousal and intrusive cognitions at bedtime; when practiced regularly, these strategies improve sleep continuity and complement behavioral prescriptions [18]. For patients with prominent cognitive hyperarousal, brief psychotherapeutic interventions that emphasize attentional retraining and acceptance may further enhance outcomes when integrated with core CBT-I components [8][18].

Pharmacological Management

Pharmacotherapy is best conceptualized as an adjunct to, not a replacement for, CBT-I. Medication selection should be individualized to the patient's predominant complaint (sleep onset vs maintenance), age, comorbidities, concurrent medications, safety profile, and personal preferences, with periodic reassessment to minimize dose, duration, and adverse effects. Broadly, classes include gamma-aminobutyric acid-A ($GABA_A$) receptor agonists (benzodiazepines and benzodiazepine receptor agonists), dual orexin receptor antagonists, agents acting on melatonin pathways, sedating antidepressants (notably doxepin at low doses), selected anticonvulsants in comorbid conditions, and sedating antihistamines—though the latter have limited evidentiary support and notable anticholinergic liabilities [1][9][21][22]. $GABA_A$ receptor agonists encompass traditional benzodiazepines and the so-called "Z-drugs." While all benzodiazepines display relatively broad affinity across alpha subunits of the $GABA_A$ receptor, non-benzodiazepine agonists differ in subtype selectivity: zolpidem and zaleplon preferentially bind the alpha-1 subunit implicated in sedative–hypnotic effects, whereas eszopiclone exhibits higher affinity at alpha-2 and alpha-3 subunits associated with anxiolysis and myorelaxation [1][21]. Clinically, both benzodiazepines and benzodiazepine receptor agonists can improve sleep onset and, depending on half-life and formulation, sleep maintenance. However, they carry important risks: next-day sedation and psychomotor slowing, cognitive impairment, anterograde amnesia, parasomnias and complex sleep-related behaviors, rebound insomnia upon discontinuation, as well as longer-term concerns about tolerance, dependence, falls, fractures, depression, and possibly dementia in observational data [21][22]. Reflecting these vulnerabilities, the Beers Criteria recommend avoiding these agents in adults over 65 when possible. In the United States, the FDA has approved temazepam, triazolam, flurazepam, and estazolam among benzodiazepines, and eszopiclone, zaleplon, and zolpidem (including extended-release and sublingual formulations) among non-benzodiazepine receptor agonists for insomnia (A1).

Dual orexin receptor antagonists—suvorexant, lemborexant, and daridorexant—represent a mechanistically distinct option that counters orexin/hypocretin-mediated wakefulness by preventing orexin A and B from binding OX1 and OX2 receptors in arousal pathways. They can benefit sleep onset and maintenance without GABAergic effects, but higher doses are discouraged because of safety signals related to next-day driving impairment, residual somnolence, and rare narcolepsy-like phenomena such as hypnagogic/hypnopompic

hallucinations, cataplexy-like events, and vivid dreams. Given their pharmacodynamic action, these drugs are contraindicated in narcolepsy [1]. Agents targeting the melatonin system include over-the-counter melatonin and prescription melatonin receptor agonists. Endogenous melatonin, secreted by the pineal gland under suprachiasmatic nucleus control, facilitates sleep initiation and circadian entrainment; exogenous melatonin is available without prescription and is commonly used in older adults, though food delays absorption and product quality can vary. Ramelteon, a high-affinity MT1/MT2 agonist acting at the SCN, reduces sleep latency without GABAergic adverse effects and is well tolerated, while tasimelteon is especially useful for non-24-hour sleep–wake disorder and has demonstrated efficacy in certain syndromic contexts such as Smith–Magenis syndrome. Class effects include somnolence, fatigue, and dizziness [1].

Among antidepressants, low-dose doxepin functions essentially as a selective H1 antagonist and has evidence for sleep maintenance benefits—improving total sleep time, wake after sleep onset, and sleep efficiency—with a relatively benign adverse-effect profile at 3–6 mg (headache and somnolence predominate) [1]. Other antidepressants frequently used off-label for insomnia because of antihistaminic properties include trazodone, mirtazapine, amitriptyline, and nortriptyline; while they may help selected patients—particularly when comorbid depression or anxiety is present—risks of anticholinergic burden, orthostasis, weight gain, and next-day sedation warrant judicious use and periodic attempts to taper when clinically feasible [9]. Selected anticonvulsants can be considered in specific comorbidities. Gabapentin may increase sleep efficiency and reduce wake after sleep onset, and it can support recovery in alcohol dependence where sleep disruption fuels relapse risk; pregabalin has shown benefit in generalized anxiety disorder and fibromyalgia with co-occurring insomnia [23]. By contrast, sedating antihistamines (e.g., diphenhydramine, hydroxyzine) are widely available but have limited evidence for chronic insomnia and carry anticholinergic adverse effects including xerostomia, constipation, urinary retention, delirium risk, and cognitive impairment—particularly problematic in older adults [9]. For these reasons, routine use is discouraged in long-term management. Guideline-based dosing recommendations from the 2017 American Academy of Sleep Medicine guideline underscore that pharmacologic evidence is generally moderate-to-low in quality and recommendations are correspondingly weak; nonetheless, the statement provides practical starting doses aligned with symptom targets [24]. For sleep-onset insomnia, options include eszopiclone 2–3 mg at bedtime, ramelteon 8 mg at bedtime, temazepam 15 mg at bedtime, triazolam 0.25 mg at bedtime, zaleplon 5–10 mg at bedtime, or zolpidem 10 mg at bedtime. For sleep-maintenance insomnia, suggested regimens include doxepin 3–6 mg at bedtime, eszopiclone 2–3 mg at bedtime, temazepam 15 mg at bedtime, suvorexant 10, 15, or 20 mg at bedtime, or zolpidem 10 mg at bedtime (A1). Doses should be individualized with attention to hepatic or renal impairment, drug–drug interactions (e.g., CNS depressants), and patient-specific vulnerabilities (falls, cognitive effects, occupational driving). Periodic attempts at tapering and deprescribing are prudent once CBT-I has improved sleep continuity and resilience [24].

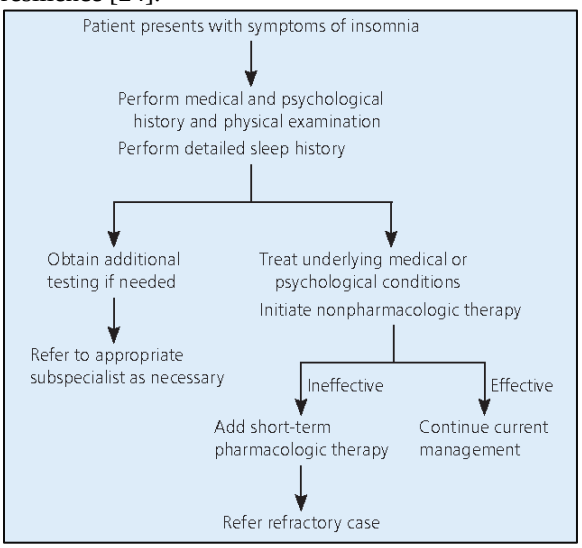

Figure-3: Management and Treatment of Insomnia.

Other Considerations

The European Insomnia Guideline (2023) reiterates CBT-I as first-line and provides a pragmatic pharmacologic framework that emphasizes short-term use, structured reassessment, and explicit risk–benefit dialogue [9]. Benzodiazepines and benzodiazepine receptor agonists may be used for short-term treatment of up to four weeks, with thorough counseling regarding next-day impairment, tolerance, and dependence; any consideration of longer-term therapy should be undertaken cautiously and only after failure or inaccessibility of CBT-I, with a clear plan for monitoring and periodic taper attempts [9]. Sedating antidepressants at low doses can be used short-term after contraindications are reviewed, with similar caution regarding extension beyond initial courses; clinicians should continually weigh antihistaminic benefit against anticholinergic and cardiometabolic risks. Orexin receptor antagonists may be used for up to three months, and the pros and cons of longer courses should be discussed candidly in shared decision-making conversations, particularly around driving safety and rare dissociative phenomena [9]. In contrast, antihistamines, antipsychotics, fast-release melatonin, and herbal remedies lack sufficient evidence for chronic insomnia and carry potential disadvantages that can outweigh uncertain benefits; routine use is therefore not advised [9]. For adults older than 55, extended-release melatonin may be

considered for up to three months, aligning with age-related declines in endogenous melatonin secretion and a favorable safety profile in this group [9]. Across all modalities, a stepped-care, multimodal strategy improves outcomes. Most patients should begin with CBT-I; when pharmacotherapy is indicated—because of severe distress, urgent functional demands, or partial CBT-I response—short-term, targeted medication can provide symptomatic relief while behavioral mechanisms consolidate more durable gains [8][24]. Close follow-up using sleep diaries and validated scales such as the Insomnia Severity Index and Sleep Condition Indicator provides objective anchors for dose adjustments, tapering decisions, and the timing of booster CBT-I sessions [10][11]. For circadian contributions—common in shift workers or adolescents—strategic light exposure, regularizing wake times, and, when appropriate, time-locked melatonin can be layered onto CBT-I to resynchronize rhythms without defaulting to long-term hypnotics [9].

Risk mitigation is integral to pharmacologic care. In older adults and those with fall risk, cognitive impairment, or polypharmacy, clinicians should preferentially use agents with minimal next-day sedation (e.g., low-dose doxepin for maintenance, ramelteon for onset) and avoid benzodiazepines and Z-drugs when possible, consistent with Beers recommendations [21][22]. In patients with comorbid obstructive sleep apnea or chronic respiratory disease, sedatives that depress ventilatory drive warrant caution; when insomnia coexists with OSA (COMISA), parallel treatment of sleep-disordered breathing (e.g., CPAP) alongside CBT-I often yields superior outcomes compared to either modality alone [8]. In individuals with substance use disorders, non-GABAergic options and behavioral therapies should be prioritized to reduce misuse risk; gabapentin can be considered selectively in alcohol use disorder under careful monitoring [23]. For women who are pregnant or lactating, nonpharmacologic therapies are preferred, and any medication use requires individualized risk–benefit assessment with obstetric input [9]. Finally, translating evidence into practice hinges on health-system design. Embedding brief insomnia screening into primary care, building referral pathways to CBT-I (including digital programs), training pharmacists to reinforce sleep-compatible medication schedules and counsel on caffeine, alcohol, and nicotine, and deploying registries to track outcomes can close the implementation gap. Laboratory services play supportive roles when iron deficiency, thyroid disease, or inflammatory conditions are suspected contributors (e.g., ferritin, thyroid studies, C-reactive protein), while administrators can underwrite access to iCBT-I and ensure equitable coverage for first-line behavioral care [9][19][20]. With this coordinated approach, nonpharmacologic and pharmacologic tools operate synergistically: CBT-I provides durable, mechanism-level change; targeted medications bridge acute distress and address specific phenotypes; and system supports sustain adherence, monitor safety, and promote deprescribing when appropriate. The result is a patient-centered, evidence-aligned management plan that addresses chronic insomnia's multidimensional drivers and improves both sleep and daytime functioning over the long term [8][18][24].

Differential Diagnosis

The differential diagnosis of chronic insomnia encompasses a broad spectrum of medical, psychiatric, neurological, and sleep-related conditions, as well as the effects of various substances and medications that may either cause or exacerbate disturbed sleep. Because chronic insomnia can occur as both an independent primary disorder and as a symptom of another underlying condition, careful differentiation through clinical history and focused assessment is essential to guide appropriate management [9]. From a psychiatric standpoint, chronic insomnia frequently coexists with mood and anxiety disorders. Conditions such as major depressive disorder, bipolar disorder, generalized anxiety disorder, posttraumatic stress disorder (PTSD), and schizophrenia may present with persistent sleep disturbance as a core or prodromal feature. For instance, insomnia often precedes depressive episodes and can predict recurrence, while anxiety disorders are associated with heightened physiological arousal and rumination that prevent sleep initiation. In bipolar disorder, decreased need for sleep may signal the onset of mania or hypomania. Borderline personality disorder and trauma-related disorders are also strongly associated with insomnia, reflecting dysregulation in emotional and stress-response systems.

Several medical conditions contribute to or mimic chronic insomnia. Cardiovascular diseases such as heart failure or hypertension, endocrine disorders including diabetes mellitus and hyperthyroidism, and chronic kidney disease can disrupt sleep through nocturia, dyspnea, or metabolic imbalance. Chronic pain syndromes—particularly those associated with rheumatic diseases, fibromyalgia, and malignancies—interfere with the ability to maintain restful sleep due to nociceptive activation and nighttime discomfort. Pulmonary conditions, including chronic obstructive pulmonary disease (COPD) and asthma, often lead to nocturnal symptoms such as coughing, wheezing, and dyspnea, all of which fragment sleep architecture [9]. A number of neurological disorders are also implicated in secondary insomnia. Neurodegenerative diseases such as Alzheimer's and Parkinson's disease disrupt circadian regulation and alter REM and non-REM sleep distribution. Cerebrovascular disease and traumatic brain injury can impair sleep–wake control through damage to hypothalamic or brainstem structures. Multiple sclerosis, restless legs syndrome (RLS), and periodic limb movement disorder (PLMD) are additional causes of disrupted sleep continuity,

while the extremely rare fatal familial insomnia, a prion-related disorder, results in progressive and irreversible insomnia culminating in dementia and death. Primary sleep disorders must also be distinguished from chronic insomnia. Obstructive sleep apnea (OSA) and related sleep-related breathing disorders can mimic or coexist with insomnia, especially when patients complain primarily of sleep maintenance difficulties or nonrestorative sleep. In such cases, polysomnography is essential for diagnosis. Circadian rhythm sleep–wake disorders, such as delayed or advanced sleep phase syndrome, may masquerade as insomnia when misalignment between the internal circadian clock and external social schedule causes difficulty initiating or maintaining sleep. Finally, substance use, and medication effects represent critical yet often underrecognized contributors. Caffeine, nicotine, and alcohol are among the most common substances affecting sleep: caffeine and nicotine delay sleep onset and reduce slow-wave sleep, while alcohol may initially promote drowsiness but leads to fragmentation later in the night. Prescription medications such as stimulants, glucocorticoids, selective serotonin reuptake inhibitors (SSRIs), beta-blockers, and certain decongestants can also provoke insomnia. Withdrawal from sedatives, opioids, or alcohol can further compound sleep disruption. In clinical practice, a thorough history focusing on medical, psychiatric, and substance use factors is indispensable for distinguishing primary chronic insomnia from secondary causes. Comprehensive evaluation ensures that coexisting or causative disorders—ranging from depression and pain syndromes to respiratory or neurological disease—are appropriately managed, enabling targeted treatment of the insomnia itself and improving overall patient outcomes [9].

Ongoing Trials

Clinical guidance for chronic insomnia converges on cognitive behavioral therapy for insomnia (CBT-I) as the preferred first-line treatment across diverse clinical settings. Yet the availability of trained therapists remains a major access bottleneck, motivating implementation research that tests scalable delivery models within a stepped-care framework. In stepped care, individuals with milder or less complex insomnia receive lower-intensity, broadly accessible interventions, while those with greater chronicity, comorbidity, or treatment resistance are triaged upward to more specialized care. This approach conserves scarce specialist resources and can shorten waiting times without compromising outcomes for most patients [9]. A central thrust of ongoing trials is the evaluation of internet-delivered CBT-I programs, including structured platforms such as SHUTi and Sleepio, which operationalize core components of stimulus control, sleep restriction, and cognitive restructuring through modular lessons, automated feedback, and diary-driven titration. Although these programs reproducibly reduce insomnia severity and daytime dysfunction, implementation studies note higher attrition relative to therapist-delivered CBT-I, highlighting the importance of engagement strategies, motivational supports, and hybrid designs that incorporate brief clinician contact to sustain adherence [25][26]. Beyond web platforms, investigators are testing smartphone applications engineered to embed CBT-I techniques into daily routines. Examples under evaluation include Sleep Ninja, pro-Act-S, UnMind Night, and Calm, each leveraging push notifications, in-app sleep diaries, and micro-interventions for pre-sleep arousal. Early trials suggest that CBT-based apps can reduce insomnia symptoms and improve sleep efficiency, particularly when used with a fixed wake time and sleep restriction algorithms, but head-to-head comparisons with therapist-led CBT-I and pragmatic trials in safety-net settings remain priorities for the evidence base [25][26]. A complementary research agenda targets personalization: by identifying stable insomnia phenotypes—such as sleep-onset predominance, maintenance insomnia with prolonged wake after sleep onset, circadian misalignment, and comorbid insomnia with sleep apnea (COMISA)—studies aim to match patients to the most efficient treatment packages. Methodological standardization across laboratories, expansion of home-based assessments using actigraphy and digital diaries, and the development of feasible hyperarousal assays are additional priorities. Parallel pharmacologic work continues on improved hypnotics with better next-day safety, while mechanistic studies probe psychoneurobiological circuits linking stress, emotion regulation, and cortical excitability to persistent wakefulness [4]. Finally, prevention trials that prioritize early detection and mitigation of mental disorders seek to reduce incident insomnia by addressing upstream determinants of hyperarousal and affective dysregulation [4].

Prognosis

The natural history of chronic insomnia illustrates both its incidence and tenacity. In a longitudinal naturalistic cohort, 13.9% of initially good sleepers developed an insomnia syndrome over five years, and 37.5% of participants with insomnia reported persistent symptoms at every annual follow-up across the same period, underscoring that once insomnia crosses syndromic thresholds, it tends to endure [27]. Demographic analyses reveal a higher likelihood of persistence among women and older adults, consistent with hormonal transitions, medical comorbidities, and age-related circadian shifts that together amplify arousal and fragment sleep. Despite the proliferation of behavioral and pharmacologic options, there are currently no externally validated prediction models that reliably estimate individual treatment response, a gap that ongoing trials seek to close through phenotyping and digital biomarkers [28]. Prognostically, timely access to CBT-I improves symptomatic trajectories and reduces downstream

healthcare utilization, while unrecognized or undertreated insomnia increases risks of chronicity, incident psychiatric morbidity, and cardiometabolic complications [27].

Complications

Chronic insomnia is often underdiagnosed and undertreated in primary care, where patients may normalize sleep disturbance or present with nonspecific daytime complaints. The disorder is a robust risk factor for future insomnia episodes and portends a persistent symptom course. Untreated insomnia increases the risk of several adverse outcomes, including hypertension, depression, anxiety, absenteeism, and both all-cause and cardiopulmonary mortality [27][29][27]. Mechanistic links include sustained sympathetic activation and systemic inflammation; elevated circulating C-reactive protein—an accessible inflammatory biomarker—has been independently associated with heightened long-term cardiovascular risk and death, offering a plausible pathway connecting chronic sleep disturbance with incident cardiometabolic disease [30]. Health-economic impacts are substantial: reduced productivity, occupational accidents, and greater medical utilization contribute to societal costs that exceed those of many other chronic conditions. For patients and caregivers, functional consequences range from cognitive inefficiency and mood dysregulation to impaired social and family functioning, reinforcing the urgency of early, effective intervention.

Consultations

Given its frequent comorbidity and shared pathophysiology with other illnesses, chronic insomnia often warrants interprofessional consultation. Depending on the clinical picture, referrals may include sleep medicine for differential diagnosis and consideration of polysomnography or actigraphy; psychiatry for comorbid mood, anxiety, trauma-related, or substance use disorders; neurology for neurodegenerative disease, cerebrovascular sequelae, traumatic brain injury, or movement disorders; and subspecialties such as cardiology, oncology, rheumatology, pulmonology, nephrology, endocrinology, or addiction medicine when systemic disease or iatrogenic factors contribute to insomnia. Coordinated assessment enables targeted treatment of contributory conditions—obstructive sleep apnea, chronic pain, hyperthyroidism, nocturnal dyspnea, reflux, nocturia—while aligning insomnia therapy with the broader care plan to minimize conflicting recommendations and polypharmacy.

Patient Education

Patient education remains the cornerstone of deterrence and long-term self-management. Individuals should learn that most adults need seven to nine hours of sleep nightly and that regularity—consistent wake time seven days a week—is the strongest behavioral cue for stabilizing sleep. Counseling emphasizes minimizing daytime napping, restricting evening caffeine and nicotine, and avoiding alcohol as a sleep aid given its rebound-arousal and fragmentation effects. Patients benefit from developing wind-down routines that reduce cognitive arousal, optimizing sleep environments for darkness, quiet, and cool temperatures, and scheduling vigorous exercise earlier in the day to prevent late-evening activation. Education also normalizes the bidirectional association between insomnia and other conditions: chronic insomnia can be a stand-alone disorder or a manifestation of medical, psychiatric, and neurological problems; stress, shift work, and travel across time zones can destabilize rhythms, requiring anticipatory strategies such as time-cued light exposure or short-term melatonin for circadian realignment. Engagement with CBT-I—whether in person or through validated digital programs—is reinforced as first-line care, with clinicians setting expectations about the structured, skills-based nature of therapy, the rationale for sleep restriction and stimulus control, and the likely time course to improvement.

Other Issues

Several clinical considerations guide real-world decision-making. Chronic insomnia is common, complex, and frequently underrecognized, often co-occurring with medical, psychiatric, and neurological disorders as well as substance use; careful history remains indispensable to distinguish primary from secondary presentations. Insomnia-related distress, occupational underperformance, and disability are prevalent and should be explicitly assessed to inform treatment urgency. CBT-I—delivered face-to-face or via evidence-based digital programs—remains the first-line therapy, conferring durable benefit on nocturnal symptoms and daytime function. When needed, pharmacotherapy can be layered to address acute distress or specific phenotypes, drawing on benzodiazepine receptor agonists, dual orexin receptor antagonists, and low-dose doxepin, with mindful attention to age, comorbid conditions, and fall or cognitive risks. Evidence for over-the-counter antihistamines, antipsychotics, and other sedatives in chronic insomnia is limited, and routine use is discouraged due to unfavorable risk–benefit profiles. Clinicians should routinely reassess medication need, attempt deprescribing once CBT-I gains consolidate, and avoid chronic sedative hypnotic use in older adults whenever possible.

Enhancing Healthcare Team Outcomes

Improving outcomes in chronic insomnia depends on early identification, timely access to first-line treatment, and sustained, coordinated follow-up. An effective interprofessional team draws on the complementary expertise of neurologists, psychiatrists, sleep medicine physicians, advanced practitioners, nurses, pharmacists, respiratory therapists when sleep-disordered breathing is present,

and behavioral sleep specialists. Foundational skills include recognizing the diverse clinical presentations of insomnia, conducting structured assessments that incorporate validated instruments, and understanding the indications and limitations of polysomnography and actigraphy. Care pathways should routinize screening in primary care and behavioral health, incorporate sleep diaries into intake workflows, and enable rapid referral to CBT-I. Where therapist capacity is constrained, systems can deploy stepped-care models with digital CBT-I as a first step for suitable patients, reserving therapist-delivered care for complex or refractory cases [9]. Pharmacists play a pivotal role by reconciling medications that impair sleep, educating patients about caffeine, nicotine, and alcohol timing, and counseling on appropriate hypnotic use and tapering strategies. Nurses and advanced practitioners reinforce sleep-compatible routines, deliver brief behavioral coaching between visits, and monitor side effects, while behavioral health clinicians integrate insomnia treatment with care for comorbid anxiety, depression, PTSD, and substance use disorders. Sleep specialists coordinate evaluation for coexisting obstructive sleep apnea or movement disorders and co-manage COMISA, where concurrent therapy for sleep-disordered breathing and insomnia yields superior outcomes. Administrators and quality leaders can strengthen performance through registries that track wait times, CBT-I completion, insomnia severity trajectories, and medication deprescribing rates, while financing digital therapeutics to improve equity of access.

Ethical and strategic considerations also matter. Shared decision-making respects patient autonomy regarding therapy modality and medication trials; transparent discussion of benefits, risks, and alternatives promotes adherence and trust. Clear role delineation across the team ensures accountability for follow-up, minimizes duplication, and supports seamless handoffs between primary and specialty care. Finally, care coordination—through standardized communication, closed-loop referrals, and telehealth touchpoints—reduces fragmentation and safety risks. By embedding these practices, interprofessional teams can transform insomnia care from episodic, pharmacocentric management to sustained, skills-based treatment that addresses root mechanisms of hyperarousal and conditioned wakefulness, thereby improving sleep, daytime functioning, and long-term health trajectories [27][29][30][28][25][26][9][4].

Conclusion:

In conclusion, chronic insomnia is a complex, multifactorial disorder that demands a paradigm shift from symptomatic, short-term pharmacological management to a comprehensive, systems-based approach. The evidence firmly establishes Cognitive Behavioral Therapy for Insomnia (CBT-I) as the foundational and first-line treatment, offering durable benefits by addressing the maladaptive thoughts and behaviors that perpetuate the condition. Pharmacotherapy, while a valuable adjunct for acute relief or specific cases, must be used judiciously, with a clear plan for periodic reassessment and deprescribing to mitigate long-term risks. The optimal management model is inherently interprofessional, leveraging the unique skills of each team member. Clinicians provide diagnosis and lead treatment planning, pharmacists optimize and deprescribe medication regimens, laboratory services identify and monitor contributing medical conditions, and health administrators create the necessary infrastructure through standardized pathways and digital health solutions like iCBT-I. This collaborative framework ensures that care is continuous, patient-centered, and guideline-concordant. By embedding these principles into practice, healthcare systems can effectively tackle the significant burden of chronic insomnia, leading to improved sleep outcomes, enhanced daytime functioning, and a reduction in the associated psychiatric and medical morbidity. Ultimately, a coordinated, team-based strategy is essential for transforming the care of this pervasive disorder.

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