PATRZYKĄT, Klaudia Martyna, MARCINKOWSKA, Paula, NOWICKA, Ewelina, POPIELARSKA, Kinga, KRZYŻANIAK, Marta, BRYNCZKA, Izabela, PUZIO, Julia, GORZOCH-BURDUK, Zofia, JEZIERSKI, Michał and WRÓBLEWSKA, Kamila. Insomnia: A Narrative Review of Treatment Strategies For The Most Prevalent Sleep Disorder. Journal of Education, Health and Sport. 2025;84:66929. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.84.66929
https://apcz.umk.pl/JEHS/article/view/66929

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 27.11.2025. Revised: 06.12.2025. Accepted: 06.12.2025. Published: 06.12.2025.

Insomnia: A Narrative Review of Treatment Strategies For The Most Prevalent Sleep Disorder

Klaudia Martyna Patrzykąt, ORCID https://orcid.org/0009-0000-9440-5444
E-mail: patrzykat.klaudia@gmail.com
109 Military Hospital with Policlinic in Szczecin, Piotra Skargi 9-11 Street, 70-965 Szczecin, Poland

Paula Marcinkowska, ORCID https://orcid.org/0009-0000-6913-8831
E-mail: paulapuzio96@gmail.com
Medical University of Gdańsk, Marii Skłodowskiej-Curie 3a Street, 80-210 Gdańsk, Poland

Ewelina Nowicka, ORICD https://orcid.org/0009-0004-1782-0416
E-mail: ewelinanow553@onet.pl
University Clinical Hospital in Poznań, Przybyszewskiego 49 Street, 60-355 Poznań, Poland

Kinga Popielarska, ORCID https://orcid.org/0009-0009-7797-5301
E-mail: kingapopielarska@gmail.com
Medical University of Gdańsk, Marii Skłodowskiej-Curie 3a Street, 80-210 Gdańsk, Poland

Marta Krzyżaniak, ORCID https://orcid.org/0009-0001-4125-7553
E-mail: martakrzyzaniak11@gmail.com
PCK Maritime Hospital in Gdynia, Powstania Styczniowego 1 Street, 81-519 Gdynia, Poland

Izabela Brynczka, ORCID https://orcid.org/0009-0002-1527-5659
E-mail: iza.brynczka@gmail.com
Ludwik Rydygier Specialist Hospital, 1 Złota Jesień Street, 31-826 Kraków, Poland

Julia Puzio, ORCID https://orcid.org/0009-0001-9832-8527
E-mail: xjulia.puziox@gmail.com
Medical University of Gdańsk, Marii Skłodowskiej-Curie 3a Street, 80-210 Gdańsk, Poland

Zofia Gorzoch-Burduk, ORCID https://orcid.org/0009-0001-6457-4105
E-mail: zosia.gorzoch@icloud.com
University Clinical Center in Gdańsk, Dębinki 7 Street, 80-952 Gdańsk, Poland

Michał Jezierski, ORCID https://orcid.org/0009-0000-0185-4065
E-mail: jezierskim99@gmail.com
University Clinical Hospital in Poznań, Przybyszewskiego 49 Street, 60-355 Poznań, Poland

Kamila Wróblewska, ORCID https://orcid.org/0009-0002-8459-4792
E-mail: kuzmicz.kamila@wp.pl
University Clinical Hospital in Białystok, Marii Skłodowskiej-Curie 24a Street, 15-276 Białystok, Poland

Corresponding Author
Klaudia Martyna Patrzykąt, patrzykat.klaudia@gmail.com

Abstract

Background: Insomnia is the most prevalent sleep disorder, associated with impaired daytime functioning, increased risk of mental and physical illness, as well as social and economic burden.
Aim: This review summarizes the current state of knowledge on insomnia disorder and assesses the clinical value of scientifically proven treatment strategies, including both pharmacological and non-pharmacological methods.
Methods: A narrative literature review was conducted using PubMed/MEDLINE (January 2015- November 2025). Peer-reviewed human studies, clinical guidelines, systematic reviews, meta-analyses, and high-quality narrative reviews addressing definition and classification,

epidemiology, mechanisms, consequences, diagnostic tools, or treatment outcomes were included.

Results: Insomnia affects approximately 10% of the adult population and is more common in women, elderly people, individuals with chronic stress, and those with comorbid medical or psychiatric conditions. Evidence supports a multidimensional hyperarousal model as the core mechanism of insomnia pathophysiology. Insomnia is linked to a higher risk of anxiety, depression, cardiometabolic disease, cognitive impairment, accidents, and reduced quality of life. Diagnosis relies on clinical assessment supported by sleep diaries and validated questionnaires. Cognitive Behavioral Therapy for Insomnia (CBT-I) is the first-line treatment, with chronotherapy, mindfulness-based interventions, exercise, and complementary approaches as useful adjuncts. Pharmacological options, including benzodiazepines, "Z-drugs", orexin receptor antagonists, melatonin-based agents, sedating antidepressants, and antihistamines, may be considered, but with careful monitoring of safety and dependence.

Conclusions: Insomnia requires personalized care that prioritizes non-pharmacological strategies. Medications should be used with caution, for selected patients, and for short durations only. Early recognition, patient education, and wider access to CBT-I are essential to reduce the burden of insomnia.

Keywords

Sleep Initiation and Maintenance Disorders; Insomnia; Cognitive Behavioral Therapy; Chronotherapy; Hypnotics and Sedatives; Orexin Receptor Antagonists; Melatonin; Antidepressive Agents; Antihistamines; Complementary Therapies.

1. Introduction

Insomnia is a common sleep disorder that causes people to experience difficulties with both initial sleep onset and maintaining sleep throughout the night, including early morning wakefulness. The disrupted sleep patterns lead to significant daytime problems, which cause fatigue, mental performance decline, and emotional state changes. Clinical guidelines recognize two insomnia categories based on their duration, which include acute insomnia that lasts less than three months and chronic insomnia that persists longer than three months of sleep opportunity.[1,3]

Insomnia stands as the leading sleep disorder, which affects a substantial number of people. Adults worldwide experience insomnia at a rate of approximately 10% according to global

statistics.[1,2] Multiple factors contribute to increasing the risk of insomnia.[1,4] The combination of inherited traits and major life transitions (including other medical conditions) can trigger new insomnia symptoms or make existing ones worse.[4,5] Insomnia is age-related; it is common in middle-aged and older adults, who may be especially at risk due to age-related health changes.[2,6,7] Women are affected at higher rates than men.[1,2]

Scientists have not established the exact mechanisms of insomnia.[2,8] It is mainly depicted as a hyperarousal disorder that causes patients to experience continuous cognitive and physical arousal during both day and night.[2,8] Recent studies of neurobiological mechanisms reveal that brain circuits related to arousal, such as the locus coeruleus, maintain hyperactivity, which prevents people with insomnia from resting during their sleep.[8]

Patients suffering from insomnia experience multiple negative health effects. The condition causes difficulties in daily activities and reduces life quality. Research indicates that insomnia leads to mental health problems, as it increases the risk of anxiety and depression.[9,10] Patients who experience insomnia develop a higher chances of having suicidal thoughts and starting drug use.[11,12] Insomnia often leads to the worsening of existing physical conditions, including hypertension and obesity.[13,14] The severe consequences of insomnia create a major public health problem due to its negative impact on performing daily responsibilities.[1,15]

The treatment of insomnia requires a unique approach, focusing on non-pharmacological methods with the short-term addition of pharmacotherapy.[3,16,27] This review will examine scientifically proven methods in insomnia treatment and assess their value in clinical practice.

2. Methods

This article is a narrative literature review on insomnia epidemiology, risk factors, pathophysiological mechanisms, health consequences, diagnostic criteria and tools, as well as the efficacy and safety of both pharmacological and non-pharmacological treatment strategies. A structured search of the PubMed/MEDLINE database was conducted for articles published between January 2015 and November 2025. We used MeSH terms and free-text keywords related to insomnia and its management (e.g. “insomnia”, “sleep initiation and maintenance disorders”, “chronic insomnia”, “acute insomnia”, “cognitive behavioral therapy for insomnia”, “CBT-I”, “chronotherapy”, “benzodiazepines”, “Z-drugs”, “orexin receptor antagonists”, “melatonin”, “melatonin agonists”, “sedating antidepressants”, “antihistamines”), combined with Boolean operators. Reference lists of key clinical guidelines, systematic reviews, and original studies were manually screened to identify additional relevant publications. Grey literature, conference abstracts, and non-peer-reviewed sources were not included.

We included peer-reviewed human studies and reviews on insomnia that addressed: (1) definition and classification; (2) epidemiology and risk factors; (3) neurobiological, psychological, or behavioral mechanisms; (4) psychiatric, cardiometabolic, or neurocognitive consequences; (5) diagnostic criteria and assessment tools; or (6) outcomes of pharmacological and non-pharmacological treatments. Eligible designs were randomized controlled trials, observational studies, clinical practice guidelines, systematic reviews, meta-analyses, and high-quality narrative reviews. We excluded publications focused exclusively on pediatric populations, other primary sleep disorders without separate analysis of insomnia, purely preclinical or animal studies, isolated case reports or very small case series, and non-peer-reviewed opinion pieces or letters without primary data.

3. Research results

3.1. General Information on Insomnia

3.1.1. Definition and Clinical Characteristics

Sleep is an essential biological process that determines human health and functioning. The brain uses sleep time to perform maintenance and reorganization of neural circuits, which enables synaptic plasticity and memory consolidation that wakefulness cannot achieve. The body uses sleep and wakefulness to sort incompatible physical processes, which would otherwise interfere with each other. The REM sleep neuromodulatory environment produces near-total noradrenergic suppression, which allows synaptic reorganization to occur. Sleep directly affects how alert we stay and how well we think, which both decline when we do not get enough rest.[1,17]

The medical definition of insomnia describes a sleep disorder that causes people to struggle with both starting sleep and staying asleep throughout the night or waking up too early. The diagnostic process for insomnia requires patients to experience sleep disturbances that occur at least three times a week for an extended period of time, and they must also show daytime functional problems. The medical field recognizes short-term insomnia as a condition that lasts less than three months and chronic insomnia, which continues for three months or longer. The distinction between short-term insomnia and normal occasional poor sleep matters, as insomnia disorder persists continuously and causes substantial daytime interference.[3,18,19]

3.1.2. Epidemiology and Affected Populations

Insomnia occurs frequently in the population. Approximately 10-15% of adults meet diagnostic criteria at any given time, making insomnia one of the most common sleep

disorders.[1,2] The presence of short-term or light sleep disturbances is even more widespread. The majority of people experience brief insomnia symptoms, but only a few cases develop into persistent insomnia. The prevalence of insomnia disorders affects women more than men, as they experience insomnia at approximately 1.4 times the rate. People who experience stressful life events, have chronic illnesses, or psychiatric disorders become more likely to develop insomnia. Modern society faces increasing stress levels, which lead to higher insomnia rates that have turned into a significant public health concern.[2,4,15]

3.1.3. Risk Factors

Multiple elements in a person's background make them more likely to be affected by insomnia. Twin studies reveal that genetic elements play a role in insomnia development to the extent of approximately 50%, while a family history of sleep problems increases the risk of insomnia in individuals.[5] People who naturally experience high anxiety levels and stress reactions during life transitions are more susceptible to this disorder.[4,10] Insomnia often occurs with chronic medical conditions, such as post-stroke condition, depression, respiratory diseases, heart failure, and pain-related illnesses, including cancer.[9,13,23] People who suffer from ongoing psychiatric conditions, especially depression, face a high risk of developing persistent insomnia.[9,12]

3.1.4. Health Consequences

The ongoing nature of insomnia leads to multiple adverse effects that negatively impact physical and emotional health and deteriorate life quality. People with insomnia experience constant fatigue, along with concentration and memory issues, which make it difficult to perform their daily responsibilities and work tasks. This condition creates a higher risk of anxiety and depressive disorders.[9,10] It also contributes to various physical health issues, including obesity, hypertension, and Alzheimer’s disease.[13,14,21] The public health system encounters more difficulties as insomnia causes people to become more accident-prone, for example, traffic accidents due to drowsiness, while also requiring extensive medical care and resulting in economic losses from work absence.[1,15]

3.1.5. Pathophysiology

1. Neurobiological Mechanisms

Research into insomnia neurobiology reveals that patients experience ongoing hyperarousal, which leads to their sleep disturbances. People with insomnia show signs of dysregulated stress

physiology, as their sympathetic nervous system remains active throughout the night, and their hypothalamic-pituitary-adrenal axis produces increased output during nighttime hours. The brain circuits that control arousal and emotional responses, including noradrenergic locus coeruleus pathways, continue to function at elevated levels during sleep, according to neuroimaging and neurophysiology studies. The proposed model, as shown by Van Someren, explains how arousal circuits create a self-reinforcing cycle of hyperarousal throughout the night, which makes the brain feel like it remains awake.[8] EEG recordings show that insomnia patients have abnormal brain function and reveal increased cortical activity, which leads to higher beta frequency power and lower slow-wave power during non-REM sleep. The brain fails to achieve its normal deep slow-wave synchronization pattern during rest. Levenson and his team explain that the pathophysiology of insomnia needs to be studied through multiple levels, starting from genetic and molecular aspects to neural circuits and behavioral elements, while hyperarousal stands as the main concept. Thus, insomnia involves a neurochemical imbalance, for example, decreased GABAergic inhibition and increased arousal-promoting neurotransmitters, which keeps the central nervous system in a persistent active state.[2,8,20]

2. Psychological and Behavioral Mechanisms

The research on insomnia investigates psychological and behavioral elements that demonstrate how mental operations and habits maintain elevated arousal states. Cognitive models show that people with insomnia not only develop excessive worry and ruminations but also form false negative beliefs about their sleeping patterns. The main focus of cognitive therapy, according to some studies, should be on treating unhelpful sleep-related thoughts that people experience during insomnia.[27,28] Moreover, the brain learns to associate the bed with wakefulness instead of sleep through poor sleep practices, including long periods of bed rest, irregular sleep schedules, caffeine consumption, and daytime napping. Over time, the maladaptive behaviors and beliefs create patterns that keep the insomnia going, and even after the initial trigger is gone, the person stays watchful at night.[19,27]

3. Conceptual Models

Conceptual models of insomnia try to integrate both biological and cognitive-behavioral aspects. The classic Spielman “3-P” model shows insomnia as having predisposing factors, such as genetic predisposition, precipitating events, such as stress, illness, or environmental change, and perpetuating factors, such as distortions and bad sleep habits. Other models emphasize neurocognitive circuits or psychobiological processes, but they all generally acknowledge a web of interlocking influences. In short, the pathophysiology of insomnia involves neurobiological arousal (with EEG patterns and hormone changes) and learned

psychological-behavioral patterns. Conceptual models link these pieces by showing how pre-existing sensitivity and sudden stress can lead to a cycle that keeps the brain on alert and creates long-term insomnia.[4,19,34]

3.1.6. Diagnostic Criteria and Definitions

The diagnosis of insomnia depends on a clinical evaluation, which assesses ongoing sleep problems and their effects on daily activities. Major nosological systems use specific diagnostic criteria to identify insomnia in terms of experiencing sleep onset difficulties, sleep maintenance problems, and early morning wakefulness or significant daytime performance impairment. Chronic insomnia, as defined by the third edition of the International Classification of Sleep Disorders (ICSD-3), or persistent insomnia, according to the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), is diagnosed when sleep problems occur at least three nights a week and last for more than three months. Short-term insomnia (ICSD-3) or episodic insomnia (DSM-5) can be diagnosed when the symptoms last less than three months. The definition in all schemes insists that sleep difficulties cannot be explained by insufficient sleep time or any other medical condition. However, if the insomnia symptoms are severe enough to require medical assistance, insomnia should be seen as a separate comorbid condition, irrespective of any other disorder.[3,18,19]

3.1.7. Short-term and Chronic Insomnia

Insomnia is classified into two types based on its duration: short-term (acute) and chronic. Short-term insomnia lasts for days to multiple weeks before it ends, usually due to identifiable life stressors that trigger its occurrence. The duration of chronic insomnia extends beyond several months, with patients developing sustaining factors that include maladaptive sleep habits, conditioned arousal, and medical conditions. In practice, persistent insomnia lasts for more than three months, according to DSM-5 guidance, and involves more ingrained sleep and behavioral patterns. The resolution of acute insomnia depends on the disappearance of its triggering factor, but chronic insomnia maintains itself through body arousal and negative associations with the sleep environment. Research indicates that insomnia affects numerous people, as surveys reveal that approximately 30-40% of individuals experience insomnia symptoms at some point, and 10% progress to chronic insomnia.[1,2,18]

3.1.8. Assessment Tools and Methods

The evaluation of insomnia requires both individualized medical interviews and standardized tracking systems. Patients often keep sleep diaries or logs for one to two weeks, recording bedtime, wake time, the number and duration of awakenings, napping, and subjective sleep quality. The Insomnia Severity Index (ISI) and Pittsburgh Sleep Quality Index (PSQI) function as standardized questionnaires, which help evaluate the severity of insomnia symptoms and their impact on daily activities. This self-report instrument helps healthcare providers to diagnose insomnia and monitor treatment responses. The standard evaluation for insomnia does not require polysomnography (overnight laboratory sleep study), as patients suffering from insomnia experience sleep problems even when their sleep environment remains normal. Healthcare providers may perform this test to diagnose other sleep disorders, for example, if snoring or restless legs symptoms suggest sleep apnea.[19,22,34]

3.1.9. Differential Diagnosis and Comorbidity

The assessment of insomnia requires healthcare providers to rule out possible factors that lead to sleep disturbances.[1,3] Medical conditions, including heart failure, pulmonary disease, and psychiatric disorders such as depression, anxiety, or PTSD symptoms, frequently disrupt sleep patterns and make insomnia symptoms worse.[9,13,25] Doctors need to identify any substances or medications that cause side effects resulting in poor sleep quality.[3,33] Core insomnia symptoms require sleep-directed interventions, but healthcare providers should assess all related conditions.[12,24,26]

3.2. Non-pharmacological Treatment of Insomnia

3.2.1. Cognitive Behavioral Therapy for Insomnia (CBT-I)

Cognitive Behavioral Therapy for Insomnia (CBT-I) involves behavioral methods that enable patients to address their insomnia-related thoughts and habits. The fundamental elements of CBT-I include: stimulus control, which teaches patients to use their bed for sleep and procreation only and leave their bed when unable to sleep; sleep restriction, which starts with matching bed time to actual sleep duration before gradually increasing it to enhance sleep quality; cognitive therapy, which helps patients identify and change their negative sleep-related beliefs; relaxation training, which teaches progressive muscle relaxation and deep breathing techniques to reduce bedtime physical tension; and sleep hygiene education, which instructs

patients to establish consistent sleep patterns and create an optimal sleeping environment with avoiding caffeine and electronic devices before bedtime.[27,28]

A trained therapist provides CBT-I through multiple weekly sessions, which introduce new components at each stage. Research studies, including controlled trials and meta-analyses, have shown that CBT-I leads to faster sleep onset, reduced nighttime awakenings, better sleep efficiency (the percentage of time in bed actually spent asleep), and improved sleep satisfaction. The treatment effects of CBT-I continue to benefit patients after the completion of their therapy sessions.[27,28,31]

CBT-I leads patients to experience not only better sleep quality but also reduced daytime fatigue and improved emotional well-being. In comparison to sleep medication, CBT-I produces longer-lasting benefits while avoiding all drug-related side effects. Clinical guidelines recommend CBT-I as the primary treatment for patients who suffer from chronic insomnia, whether their condition stands alone or develops from other disorders, such as depression. Digital and group-based CBT-I programs enable adults without access to specialist care to achieve major sleep quality enhancements.[3,29,30]

3.2.2. Chronotherapy

The practice of chronotherapy involves medical treatments that help patients match their body rhythms to achieve better sleep patterns. Bright light therapy stands as a primary approach, which involves patients receiving high-intensity light (5,000–10,000 lux) at particular times of the day. Morning bright light can advance the circadian clock (making it easier to fall asleep earlier and wake up earlier), while evening light can delay the clock when needed. In practice, a person with insomnia who has delayed sleep patterns should use a bright light box for 30 minutes after their morning wake-up. After multiple days of therapy, the treatment leads to melatonin production at earlier times, which results in better sleep patterns. Medical evidence shows that bright light interventions lead to significant improvements in sleep initiation and quality for people with circadian insomnia and night shift workers.

The timed administration of melatonin serves as another chronotherapeutic method. The body receives a signal about an approaching night through the consumption of low-dose melatonin, which should be taken several hours before bedtime. Patients who have to fall asleep late at night can use melatonin in the early evening to shift their sleep schedule. Studies show that properly timed melatonin treatment helps people fall asleep faster and leads to slightly better sleep quality among adults whose circadian rhythms are out of sync.

Practical sleep scheduling is especially important for shift workers. Their body can adjust to non-standard work hours through three strategies, which include setting a regular core sleep period (sleeping at the same time each day), taking scheduled naps, and controlling light conditions (bright light during night shifts, blackout curtains for daytime sleep). These specific techniques enable shift workers to sleep for a longer duration while reducing their daytime sleepiness.

The combination of chronotherapy techniques, which include bright light therapy at specific times, melatonin administration, and sleep schedule planning, helps people with circadian-related insomnia to get better sleep and improved daytime functioning.[1,3,27]

3.2.3. Mindfulness and Relaxation-Based Therapies

Mindfulness and relaxation-based therapies enable patients to control their elevated arousal and anxiety, which causes ongoing sleep disturbances. The Mindfulness-Based Stress Reduction (MBSR) program provides sessions that teach patients how to practice meditation techniques, breathing methods, and perform gentle physical exercises. Mindfulness practice helps to monitor intrusive thoughts and worries without criticism, which reduces performance anxiety related to sleep initiation and pre-sleep cognitive arousal, thus creating better sleep onset conditions. The combination of progressive muscle relaxation and guided imagery serves as a bedtime technique to calm the mind and achieve physical relaxation. Research studies show that these interventions lead to improvements in sleep quality and duration through their ability to reduce mental hyperarousal symptoms, which affect people with chronic insomnia.[27,30]

3.2.4. Exercise and Lifestyle Interventions

Regular exercise, combined with lifestyle modifications, plays a significant role in improving sleep in patients with insomnia. Regular aerobic or resistance exercise sessions throughout the week help individuals fall asleep faster and achieve better sleep quality. Exercise enables people to reach deeper stages of slow-wave sleep, stabilize circadian rhythms, and decrease stress and depression symptoms. The most effective exercise schedule occurs in the morning or afternoon, as evening high-intensity workouts can lead to increased arousal and delay sleep onset. People who exercise daily while maintaining a regular sleep pattern will achieve major improvements in sleep quality.[16,27]

3.2.5. Complementary Therapies

Multiple complementary therapies, including yoga, acupuncture, and aromatherapy, have been studied as additional treatments for insomnia. Yoga practice includes physical exercises, breathing methods, and mindfulness meditation, which work together to create relaxation that can reduce insomnia symptoms. Research shows that practicing yoga daily results in minor improvements in sleep quality. The confirmation for acupuncture remains unclear; while certain trials suggest it may shorten sleep onset time and extend sleep duration, the overall evidence is limited by small sample sizes and difficulty ruling out placebo effects. Aromatherapy, for example, using lavender oil before sleep, creates a soothing bedtime routine, but sleep benefits are not strongly supported by clinical data.

These alternative treatments function as additional resources and are generally safe, but they do not provide complete solutions for insomnia and should be combined with primary treatments, such as cognitive behavioral therapy for insomnia (CBT-I).[16,32]

3.3. Pharmacological Treatment of Insomnia

Non-pharmacological treatment, especially Cognitive Behavioral Therapy for Insomnia (CBT-I), remains the primary approach for managing insomnia.[3,16,27] However, when these methods become insufficient or patients require immediate symptom relief, pharmacotherapy might be needed.[3,22,31]

3.3.1. Benzodiazepines

Benzodiazepines (BZs) function as sedative-hypnotics, which activate GABA-A receptors through positive allosteric modulation to shorten sleep latency and extend the total duration of sleep. The most common medications used are temazepam and triazolam. Temazepam (5-30 mg before bedtime) belongs to the intermediate-acting drugs (approximately 8-10 hours) and helps to maintain sleep throughout the night. Triazolam (0.125-0.25 mg) is a very short-acting medication (approximately 2-5 hours) and is usually prescribed for sleep-onset insomnia or early awakenings, without causing daytime drowsiness.

BZs treatment should not continue for more than four weeks. Longer use increases the risk of tolerance and dependence development, which may lead to rebound insomnia or withdrawal symptoms when patients discontinue the medication. The use of benzodiazepines often results in sedation, memory fog, and impaired coordination during the following day, which increases the danger of accidents and falls. The side effects of BZs are especially noticeable with long-

acting benzodiazepines and in elderly patients, so doctors must prescribe the smallest amount needed for the shortest treatment period.[1,3,33]

3.3.2. 'Z-drugs'

Non-benzodiazepine hypnotics known as “Z-drugs” bind to GABA-A receptors at the same benzodiazepine site through their unique chemical structures. The drugs target GABA-A receptors containing α1 subunits, resulting in strong sleep-inducing effects with relatively less anxiolytic or muscle-relaxant activity.

The three non-benzodiazepine hypnotics include zolpidem, zaleplon, and zopiclone. Zolpidem (5-10 mg) and zaleplon (5-10 mg) belong to fast-acting drugs with short elimination times, lasting between 2-3 hours and 1 hour, respectively. They are used to improve sleep onset without causing drowsiness the following day. Zopiclone has a moderate half-life duration (approximately 5 hours), and the 7.5 mg dosage helps with both sleep initiation and staying asleep throughout the night.

Similar to benzodiazepines, these medications help patients achieve better sleep quality through short-term improvements in both sleep onset and duration. However, doctors should only prescribe them for brief use (2-4 weeks), as extended treatment periods can cause tolerance, dependence, and make insomnia symptoms even more severe. Z-drug users may experience unusual sleep-related behaviors, including sleepwalking and 'sleep-driving,' according to reports. Sedation that persists into the following day often occurs when patients take the medication late at night or use high dosages.[3,22,33]

3.3.3. Orexin Receptor Antagonists

The new class of hypnotics known as orexin receptor antagonists (dual orexin receptor antagonists, DORAs) works by blocking wake-promoting orexin-A and -B neuropeptides from binding to their OX1R and OX2R receptors. The blockade of orexin-mediated arousal pathways enables patients to fall asleep faster and maintain sleep throughout the night without altering their typical sleep patterns.

The three approved DORA medications for insomnia treatment include suvorexant, lemborexant, and daridorexant. The recommended bedtime dosage for suvorexant ranges from 10 to 20 mg, while lemborexant requires 5 to 10 mg, and daridorexant needs 25 to 50 mg. The three medications have different elimination times, which range from 12 hours for suvorexant to 18 hours for lemborexant and 8 hours for daridorexant, with the shortest duration potentially minimizing daytime drowsiness. The clinical research shows that DORAs improve sleep

quality by reducing sleep latency and extending total sleep time by several minutes to a half an hour.[1,22]

The medications are generally well tolerated, even with long-term use. The most common adverse effects include somnolence the next day, fatigue, headaches, and dry mouth. Dangerous adverse effects, such as sleep paralysis, hypnagogic hallucinations, or cataplexy-like episodes, are rarely reported. The extended use of DORAs for insomnia treatment remains safe, as these medications do not lead to dependence or cognitive issues, which benzodiazepines and "Z-drug" sedatives cause.[1,22]

3.3.4. Melatonin and Melatonin Agonists

The body's natural sleep cycle can be targeted through the use of melatonin and melatonin receptor agonists. The pineal gland produces melatonin, which uses MT1 and MT2 receptors to induce sleepiness; patients can use melatonin supplements in the evening (commonly 1-5 mg about 30 minutes before bed) to help with sleep-onset insomnia and reset their body clock for jet lag and shift work disorders. Because endogenous melatonin declines with age, the 2 mg prolonged-release melatonin received approval for elderly insomnia treatment in particular geographic regions. The short half-life of melatonin (less than 1 hour) makes it effective, particularly for sleep initiation. The medication delivers small sleep benefits by shortening the time needed to fall asleep (about 10-15 minutes) and slightly extending overall sleep duration, but it remains completely safe for extended use.[1,3,34]

The FDA approved ramelteon (8 mg before bedtime), a selective MT1/MT2 receptor agonist, as a treatment for people who experience sleep-onset difficulty. Ramelteon works similarly to melatonin by reducing sleep latency by approximately 10-15 minutes and lacks any potential for abuse or tolerance development, which makes it completely safe for extended use. Another melatonin agonist, tasimelteon (20 mg nightly), serves as a treatment for non-24-hour sleep-wake disorder in totally blind patients to establish their sleep-wake pattern at 24 hours. The medication tasimelteon does not lead to dependence or withdrawal symptoms.[1,3,34]

3.3.5. Antidepressants and Other Off-Label Agents

Sedating antidepressants, including low-dose doxepin, trazodone, and mirtazapine, serve as off-label treatments for insomnia, especially in patients with comorbid depression. The low doses of doxepin (originally a tricyclic antidepressant) between 3-6 mg taken before bedtime act by selectively blocking histamine H1 receptors. The medication begins to work within 30 minutes and results in reduced awakenings and prolonged sleep duration. The recommended

dose for insomnia is much lower than for depression, resulting in minimal anticholinergic side effects, primarily next-day sedation and dry mouth. Trazodone, a serotonin receptor antagonist and reuptake inhibitor, is recommended for insomnia at a dosage of 50-150 mg taken nightly. This medication is particularly useful in depressed patients as it helps to improve both mood and sleep maintenance. The hypnotic properties of trazodone result from its primary mechanism of blocking 5-HT2 receptors (with additional mild H1 and alpha-1 blockade), which increases slow-wave (deep) sleep and improves sleep quality. The drug needs to be taken about one hour before bedtime. The main side effects of trazodone include drowsiness that persists after waking, dizziness, and orthostatic hypotension; weight gain can rarely occur. Mirtazapine is another sedating antidepressant, often used at a dosage of 7.5-15 mg at bedtime. The medication produces strong sedative effects through its ability to block H1 receptors and its antagonism of 5-HT2/5-HT3 receptors. Mirtazapine improves both initial sleep onset and duration, serving as an effective treatment for depressed patients who experience insomnia. The medication remains active throughout the entire night, but may lead to next-day drowsiness. Other common side effects include increased appetite and weight gain.[22,31,33]

3.3.6. Antihistamines

The first-generation antihistamines diphenhydramine and doxylamine are widely used as sleep medications due to their sedative properties. They penetrate the blood-brain barrier and block central H1 receptors, which cause sedation and strong anticholinergic activity. The recommended dosage consists of 25-50 mg of diphenhydramine or 25 mg of doxylamine taken before bedtime to induce drowsiness and shorten sleep latency. Antihistamines help users to fall asleep faster, but they do not prove to be effective for maintaining sleep throughout the night, and users tend to build up tolerance within several days. The anticholinergic adverse effects, such as dizziness and cognitive decline, can lead to confusion and falls in older patients. Other side effects, including an increased heart rate, blurred vision, dry mouth, constipation, and urinary retention, can also be troublesome. Due to these limitations, antihistamines should only be used for brief periods of time.[3,31,34]

4. Discussion

This review shows that insomnia exists as a common, multidimensional condition that creates substantial health problems.[1,2] Ongoing sleep difficulties that affect sleep onset, sleep maintenance, or early morning awakenings lead to daytime fatigue, cognitive decline, mood disturbances,[1,9,17] increased accident risk, and deteriorating medical conditions.[13,14,21]

The evidence demonstrates that insomnia should be perceived as an independent medical condition rather than a secondary symptom.[12]

Research findings from neurobiological and psychological studies reveal that hyperarousal is the primary mechanism of insomnia.[2,4,8] Night-time activity in arousal circuits, combined with worry, rumination, and maladaptive sleep habits, results in ongoing insomnia.[4,8,19] Diagnostic systems, such as the third edition of the International Classification of Sleep Disorders (ICSD-3) and the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), transform these mechanisms into specific clinical guidelines by defining symptom frequency, duration, and separating short-term from chronic insomnia, with required daytime impairment.[3,18,19]

In everyday practice, healthcare providers track sleep disorders and their effects on patients through ongoing assessment, which includes sleep diaries and questionnaires, such as the Insomnia Severity Index and the Pittsburgh Sleep Quality Index.[17,19,22] In addition, careful differential diagnosis, including the selective use of polysomnography, helps clinicians distinguish primary insomnia from other conditions and monitor treatment response over time.[1,22,26]

The evaluation of treatment methods shows that non-pharmacological interventions should be used as the main therapeutic approach.[3,16,27] Cognitive Behavioral Therapy for Insomnia (CBT-I) offers tremendous improvements in sleep quality and daytime performance, without causing major adverse effects.[27–29] The combination of chronotherapy[1,3,27] with mindfulness-based interventions [27,30], exercise [16,27], and specific complementary methods provides additional benefits to patients who experience circadian rhythm problems or high arousal levels.[16,27,32] Pharmacological options, including benzodiazepines and non-benzodiazepine "Z-drugs," should be prescribed when behavioral methods are insufficient and for brief durations only.[3,22,31] Their potential for tolerance, dependence, and sedative effects during the next day requires careful use.[1,3,33]

Off-label medications, such as sedating antidepressants and first-generation antihistamines, should be reserved for selected patients as they cause multiple side effects, including daytime drowsiness.[3,31,33] The new treatment methods, such as dual orexin receptor antagonists and melatonin receptor agonists, work on reducing sleep latency and circadian system disruptions while providing improved safety characteristics for long-term use; however, scientists need to conduct more studies to determine their effectiveness for treating chronic insomnia.[1,22,34]

The best treatment plan for insomnia requires personalized, step-by-step care that integrates behavioral, circadian, and medication strategies, while considering existing medical and

psychiatric conditions.[3,18,22] Future research should focus on high-quality and long-term clinical trials to identify the best combinations of individual treatment approaches and expand access to CBT-I (including digital formats) in order to improve the quality of life for the many people affected by this common condition.[27–29]

5. Conclusions

Insomnia is the most prevalent sleep disorder, which goes beyond just having a bad night's sleep. It is closely linked to impaired daytime functioning, an increased risk of mental and physical illness, and substantial social and economic costs. The condition involves difficulties with sleep onset, maintenance problems, and waking up too early, which derive from neurobiological, psychological, and behavioral mechanisms, with hyperarousal at its core. Research indicates that women, elderly people, and those who experience chronic stress or have comorbid medical conditions develop insomnia at higher rates.

The discussed pathophysiological models show that hyperarousal functions as the main mechanism, involving ongoing active arousal circuits, irregular cortical activity, and disrupted stress system regulation. Psychological factors such as worry, negative beliefs about sleep, and maladaptive habits keep the disorder going. The cognitive and "3-P" models demonstrate how predisposing, precipitating, and perpetuating factors interact with each other. Healthcare providers use the third edition of the International Classification of Sleep Disorders (ICSD-3) and the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) frameworks, along with clinical interviews, sleep diaries, and questionnaires, to distinguish short-term from chronic insomnia and to monitor change over time.

In most cases, the treatment of insomnia should begin with non-pharmacological methods, including Cognitive Behavioral Therapy for Insomnia (CBT-I) as the first-line option. Chronotherapy, mindfulness-based approaches, exercise, and selected complementary methods can be added to achieve better results that match individual patients' circadian rhythms and arousal states. Pharmacological treatment, including benzodiazepines, “Z-drugs”, orexin receptor antagonists, melatonin-based drugs, antidepressants, and antihistamines, should be used in particular cases with caution and only for limited periods of time.

The most effective treatment for insomnia involves non-pharmacological methods as the foundation, with carefully selected pharmacological options when necessary. Personalized care, which addresses all contributing factors and comorbid conditions, is the key to reducing the overall impact of the disorder.

Disclosure

Author's Contribution

Conceptualization: Klaudia Martyna Patrzykąt

Methodology: Klaudia Martyna Patrzykąt, Ewelina Nowicka, Kinga Popielarska, Paula Marcinkowska, Kamila Wróblewska

Formal analysis: Klaudia Martyna Patrzykąt, Izabela Brynczka, Julia Puzio, Zofia Gorzoch-Burduk

Investigation: Michał Jezierski, Paula Marcinkowska, Ewelina Nowicka, Kinga Popielarska, Kamila Wróblewska

Writing-rough preparation: Izabela Brynczka, Julia Puzio, Zofia Gorzoch-Burduk

Writing-review and editing: Klaudia Martyna Patrzykąt, Marta Krzyżaniak, Michał Jezierski

Supervision: Klaudia Martyna Patrzykąt

All authors have read and agreed with the published version of the manuscript.

Funding statement

No funding was received for the Authors.

Institutional Review and Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflict of Interest Statement

Authors declare no conflicts of interests.

References

1. Dopheide JA. Insomnia overview: epidemiology, pathophysiology, diagnosis and monitoring, and nonpharmacologic therapy. American Journal of Managed Care. 2020;26(4 Suppl):S76-S84. doi: https://doi.org/10.37765/ajmc.2020.42769.

2. Naha S, Sivaraman M, Sahota P. Insomnia: A Current Review. Missouri Medicine. 2024;121(1):44-51.
3. Qaseem A, Kansagara D, Forciea MA, Cooke M, Denberg TD, Clinical Guidelines Committee of the American College of Physicians. Management of Chronic Insomnia Disorder in Adults: A Clinical Practice Guideline From the American College of Physicians. Annals of Internal Medicine. 2016;165(2):125-133. doi: https://doi.org/10.7326/M15-2175.
4. Kalmbach DA, Anderson JR, Drake CL. The impact of stress on sleep: Pathogenic sleep reactivity as a vulnerability to insomnia and circadian disorders. Journal of Sleep Research. 2018;27(6):e12710. doi: https://doi.org/10.1111/jsr.12710.
5. Byrne EM. The relationship between insomnia and complex diseases—insights from genetic data. Genome Medicine. 2019;11(1):57. doi: https://doi.org/10.1186/s13073-019-0668-0.
6. Patel D, Steinberg J, Patel P. Insomnia in the Elderly: A Review. Journal of Clinical Sleep Medicine. 2018;14(6):1017-1024. doi: https://doi.org/10.5664/jcsm.7172.
7. León-Barriera R, Chaplin MM, Kaur J, Modesto-Lowe V. Insomnia in older adults: a review of treatment options. Cleveland Clinic Journal of Medicine. 2025;92(1):43-50. doi: https://doi.org/10.3949/ccjm.92a.24073.
8. Van Someren EJW. Brain mechanisms of insomnia: new perspectives on causes and consequences. Physiological Reviews. 2021;101(3):995-1046. doi: https://doi.org/10.1152/physrev.00046.2019.
9. Riemann D, Dressle RJ, Benz F, Spiegelhalder K, Johann AF, Nissen C, et al. Chronic insomnia, REM sleep instability and emotional dysregulation: A pathway to anxiety and depression? Journal of Sleep Research. 2025;34(2):e14252. doi: https://doi.org/10.1111/jsr.14252.
10. Irwin MR. Insomnia and Inflammation Conspire to Heighten Depression Risk: Implications for Treatment and Prevention of Mood Disorders. Biological Psychiatry. 2025;98(11):819-829. doi: https://doi.org/10.1016/j.biopsych.2025.04.018.
11. Berro LF. Insomnia as a Risk Factor for Substance Use Disorders in Women. Sleep Medicine Clinics. 2023;18(4):511-520. doi: https://doi.org/10.1016/j.jsmc.2023.06.010.
12. Benca RM, Buysse DJ. Reconsidering Insomnia as a Disorder Rather Than Just a Symptom in Psychiatric Practice. Journal of Clinical Psychiatry. 2018;79(1):me17008ah1c. doi: https://doi.org/10.4088/JCP.me17008ah1c.

13. Tsuchihashi-Makaya M, Matsuoka S. Insomnia in Heart Failure. Circulation Journal. 2016;80(7):1525-1526. doi: https://doi.org/10.1253/circj.CJ-16-0501.
14. Allison KC, Parnarouskis L, Moore MD, Minnick AM. Insomnia, Short Sleep, and Their Treatments: Review of Their Associations with Weight. Current Obesity Reports. 2024;13(2):203-213. doi: https://doi.org/10.1007/s13679-024-00570-3.
15. Sánchez-Cárdenas AG, Navarro-Gerrard C, Nellen-Hummel H, Halley-Castillo E. [Insomnia. A severe health care problem]. Revista Médica Del Instituto Mexicano Del Seguro Social. 2016;54(6):760-769.
16. Maness DL, Khan M. Nonpharmacologic Management of Chronic Insomnia. American Family Physician. 2015;92(12):1058-1064.
17. Bollu PC, Kaur H. Sleep Medicine: Insomnia and Sleep. Missouri Medicine. 2019;116(1):68-75.
18. Ferini-Strambi L. Insomnia disorder. Minerva Medica. 2025;116(4):309-322. doi: https://doi.org/10.23736/S0026-4806.25.09690-9.
19. Grima NA, Bei B, Mansfield D. Insomnia theory and assessment. Australian Journal of General Practice. 2019;48(4):193-197. doi: https://doi.org/10.31128/AJGP-12-18-4780.
20. Humer E, Pieh C, Brandmayr G. Metabolomics in Sleep, Insomnia and Sleep Apnea. International Journal of Molecular Sciences. 2020;21(19):7244. doi: https://doi.org/10.3390/ijms21197244.
21. Sadeghmousavi S, Eskian M, Rahmani F, Rezaei N. The effect of insomnia on development of Alzheimer's disease. Journal of Neuroinflammation. 2020;17(1):289. doi: https://doi.org/10.1186/s12974-020-01960-9.
22. Porwal A, Yadav YC, Pathak K, Yadav R. An Update on Assessment, Therapeutic Management, and Patents on Insomnia. BioMed Research International. 2021;2021:6068952. doi: https://doi.org/10.1155/2021/6068952.
23. Baylan S, Griffiths S, Grant N, Broomfield NM, Evans JJ, Gardner B, et al. Incidence and prevalence of post-stroke insomnia: A systematic review and meta-analysis. Sleep Medicine Reviews. 2020;49:101222. doi: https://doi.org/10.1016/j.smrv.2019.101222.
24. Pacheco AP, Cedernaes J, Benedict C. Insomnia, OSA, and Mood Disorders: The Gut Connection. Current Psychiatry Reports. 2024;26(12):703-711. doi: https://doi.org/10.1007/s11920-024-01546-9.
25. Avidan A, Burke A. Identifying and Treating Insomnia in Patients Living With Alzheimer's Disease. Journal of Clinical Psychiatry. 2020;81(3):ME19014WC1C. doi: https://doi.org/10.4088/JCP.ME19014WC1C.

26. Sweetman A, Frank O, Stocks N, Mukherjee S, Lack L. General practitioner management of comorbid insomnia and sleep apnoea. Australian Journal of General Practice. 2023;52(9):607-612. doi: https://doi.org/10.31128/AJGP-12-22-6648.
27. Chan NY, Chan JWY, Li SX, Wing YK. Non-pharmacological Approaches for Management of Insomnia. Neurotherapeutics. 2021;18(1):32-43. doi: https://doi.org/10.1007/s13311-021-01029-2.
28. Cheung JMY, Ji X, Morin CM. Cognitive Behavioral Therapies for Insomnia and Hypnotic Medications: Considerations and Controversies. Sleep Medicine Clinics. 2019;14(2):253-265. doi: https://doi.org/10.1016/j.jsmc.2019.01.006.
29. Asarnow LD, Manber R. Cognitive Behavioral Therapy for Insomnia in Depression. Sleep Medicine Clinics. 2019;14(2):177-184. doi: https://doi.org/10.1016/j.jsmc.2019.01.009.
30. Furukawa Y, Nagaoka D, Sato S, Toyomoto R, Takashina HN, Kobayashi S, et al. Cognitive behavioral therapy for insomnia to treat major depressive disorder with comorbid insomnia: A systematic review and meta-analysis. Journal of Affective Disorders. 2024;367:359-366. doi: https://doi.org/10.1016/j.jad.2024.09.017.
31. Salisbury-Afshar E. Management of Insomnia Disorder in Adults. American Family Physician. 2018;98(5):319-322.
32. Wang WL, Chen KH, Pan YC, Yang SN, Chan YY. The effect of yoga on sleep quality and insomnia in women with sleep problems: a systematic review and meta-analysis. BMC Psychiatry. 2020;20(1):195. doi: https://doi.org/10.1186/s12888-020-02566-4.
33. Wichniak A, Wierzbicka AE, Jarema M. Treatment of insomnia – effect of trazodone and hypnotics on sleep. Psychiatria Polska. 2021;55(4):743-755. doi: https://doi.org/10.12740/PP/125650.
34. Mansfield D, Grima NA, Bei B. Insomnia management. Australian Journal of General Practice. 2019;48(4):198-202. doi: https://doi.org/10.3316/informit.275978642121787