REVIEW ARTICLE

Fusion of Traditional and Contemporary Medicine in Treating Insomnia: An Exploration

Abhishek M Bulla[1], Puneet K Nagendra[2], Neerajraj Raj B[3], Gopal Das CM[4], Anupama V[5]

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Abstract

Introduction: Insomnia, a prevalent sleep disorder, significantly impacts quality of life and overall health. Contemporary medical approaches, such as pharmacotherapy and cognitive-behavioral therapy for insomnia, offer effective treatments with a strong evidence base. Traditional medicine, including the use of medicinal plants like valerian, magnolia, and artemisia, provides options that have been used for centuries. This article explores how the integration of traditional and contemporary medicine can enhance insomnia treatment outcomes.

Discussion: Contemporary medicine targets various neurotransmitter systems, and mindfulness methods address the behavioral and psychological aspects of insomnia. Traditional medicine leverages natural remedies that target GABAergic signaling to promote sleep.

Conclusion: By combining the strengths of both modalities, a more holistic and personalized treatment plan can be developed, potentially maximizing efficacy while minimizing adverse effects. Leveraging the benefits of natural remedies alongside modern therapeutic techniques can address the diverse needs of patients more comprehensively. Future research and clinical trials are essential to validate this integrative approach and establish standardized protocols for its implementation.

Keywords: Cognitive-behavioral therapy, Insomnia, Integrative treatment, Pharmacotherapy, Traditional medicine.

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Introduction

Insomnia is a disorder wherein patients tend to have frequent trouble falling or staying asleep, which leads to overall discontent with sleep despite having adequate opportunities for sleep. Additionally, the sleep problem is accompanied by anxiety over restless nights and/or impairment in a number of functional domains (such as defined by the American Academy of Sleep Medicine (AASM) (family, social, and vocational).[1] Over the course of medical classification development, insomnia has been defined in multiple ways: as a symptom of insomnia disorder. Categorization of insomnia as a symptom/indicator of an underlying psychiatric disorder to an independent disorder by itself is a challenge and debate on its own. One category for insomnia is short term if it lasts less than 3 months, and chronic if it lasts longer than 3 months and happens more than three times a week.[1]

Despite recent advancements, our comprehension of insomnia's nature, causes, and underlying mechanisms has been limited. It could be attributed to the diversity within insomnia, its high occurrence alongside other conditions, or variations in the analytical levels of models, ranging from observation to physiological study. Till now, there have been many theories about the pathophysiology. Let us explore some popular theories. In this review article, we also explore the mechanisms of sleep while considering various therapy modalities and their mechanisms of action. The evidence presented here is based on published literature and guidelines from various national and international organizations.[2,3]

Physiological Mechanisms of Sleep

Our brain's inherent 24 hour clock, known as the circadian rhythm, controls our cycles of arousal and somnolence in reaction to variations in ambient light. This circadian rhythm helps us to adapt to environmental changes. Without it, neither we could have predicted the variations in radiation, temperature, and food availability nor have had the ability to maximize energy use and sustain internal physiological processes. The suprachiasmatic system (SCN) houses the biological clock that regulates sleep and awake. Suprachiasmatic system have an inbuilt circuit that results in circadian oscillations in neuronal activity. It also receives afferent fibers from the retina to regulate the cycle.[4,5] Melatonin, a hormone produced by the pineal gland, is regulated by the photoperiod and plays a crucial role in managing body rhythms, such as sleep and temperature. It is released during darkness and decreases during daylight. The pineal gland's activity is governed by an internal rhythm generated by the SCN in the hypothalamus, with light having a dominant suppressive effect on this regulation. The pineal

[1,3–5]Department of Psychiatry, Dr. Chandramma Dayananda Sagar Institute of Medical Education & Research, Bengaluru, Karnataka, India

[2]Department of Respiratory Medicine, Dr. Chandramma Dayananda Sagar Institute of Medical Education & Research, Bengaluru, Karnataka, India

Corresponding Author: Abhishek M Bulla, Department of Psychiatry, Dr. Chandramma Dayananda Sagar Institute of Medical Education and Research, Bengaluru, Karnataka, India, Phone: +91 8088131293, e-mail: abhishekmbullaara@gmail.com

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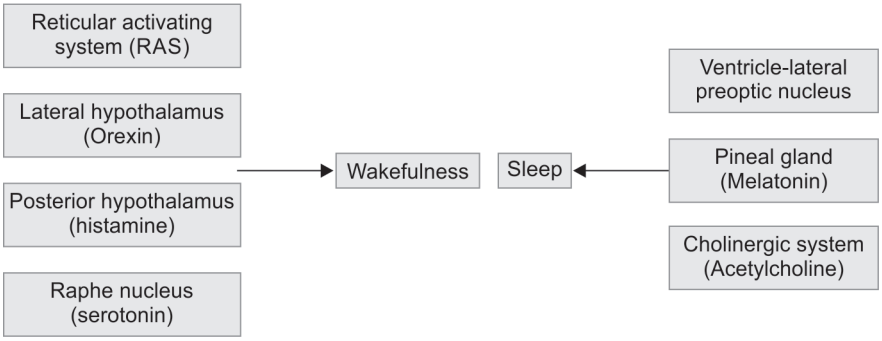

Fig. 1: Key brain centers influencing sleep and wakefulness

gland affects other biological processes by releasing melatonin into the bloodstream beats. In humans, melatonin promotes sleep and helps synchronize the sleep-wake cycle, making it effective in treating insomnia.[3,4]

The Spielman model of insomnia, also known as the "Three-Factor" or "Three P" model, includes three components: predisposing, precipitating, and perpetuating factors. Predisposing factors are inherent traits that increase an individual's vulnerability to insomnia, such as sleep reactivity, and personality characteristics like excessive worrying or rumination. Social factors, including mismatched sleep schedules between bed partners or societal pressures impacting sleep patterns, also play a role. The beginning of stressors is among the predisposing and triggering factors that frequently cause insomnia. Chronic insomnia can also persist due to behaviors like staying in bed later or earlier in an attempt to compensate for sleep deficits. These behaviors disrupt sleep homeostasis, causing insomnia to continue even after the initial trigger has been resolved or improved.[2]

Gamma-aminobutyric acid (GABA), a crucial inhibitory neurotransmitter, plays a central role in maintaining the balance between neuronal excitation and inhibition in the central nervous system and is essential for brain development and function. Approximately 20% of brain neurons are GABAergic. The regulation of sleep and arousal involves three types of GABA receptors: $GABA_A$, $GABA_B$, and $GABA_C$, although to varying degrees. Most commonly used hypnotics affect the GABA system, particularly by modulating the benzodiazepine site. Gamma-aminobutyric acid regulates the transition from rapid eye movement (REM) sleep to arousal or non-rapid eye movement (NREM) sleep by acting on REM-on and REM-off neurons in the brainstem and is considered the first line of treatment for insomnia. Additionally, numerous herbal medicines are believed to enhance GABAergic signaling, often by interacting with the $GABA_A$ receptor.[2,5]

Another major system involved in regulating sleep and wakefulness is the orexin neuropeptide signaling system. This system primarily maintains wakefulness throughout the day and plays a significant role in REM sleep. Derived from the precursor Pre-pro-orexin, two neuropeptides activate two post-synaptically localized G protein-coupled receptors, OX1R and OX2R. The start of REM sleep is suppressed by activating OX1R and OX2R, while non-REM sleep is likewise suppressed by activating OX2R. Thus, orexin agonists would promote arousal and antagonists would promote sleep. This mechanism has been used in the treatment of insomnia.[2,5]

Principles of Insomnia Therapy

Lack of sleep is detrimental to one's physical and mental well-being. Insomnia is often characterized by heightened arousal, involving increased activity in both the central (cortical) and peripheral (autonomic) nervous systems. This heightened arousal can manifest as increased cognitive and emotional activity, potentially contributing to both short-term and long-term insomnia (Fig. 1). Despite its significance in the literature, hyperarousal is not consistently defined. In many studies, hyperarousal is described as heightened physiological, cognitive, and emotional activity that disrupts the natural disengagement from daily activities necessary for sleep.[2]

Treating insomnia involves various methods, from established medications to herbal remedies and newer adjunct therapies. In this discussion, we aim to thoroughly examine these treatment options, looking at how effective they are, how safe they are, and how they might work together to treat this widespread sleep disorder. By exploring each approach and observing how they affect sleep, we hope to shed light on the complexities of managing insomnia. Our goal is to discuss the integration of medication, herbal remedies, and other interventions, providing a fair assessment of how they can help people sleep better and improve their overall health.[6]

The primary recommendation for addressing insomnia, regardless of known causes, is cognitive behavioral therapy for insomnia (CBT-I) and other adjuvant therapies. Presently, there is no universal agreement on the most effective or balanced pharmacological approach. Cognitive behavioral therapy for insomnia and pharmacological methods are believed to produce comparable immediate results, yet only CBT-I has demonstrated sustained benefits even after treatment cessation. Combining CBT-I with medication may expedite treatment response but could compromise the lasting efficacy of CBT-I's positive effects.[5,7–9]

For centuries, plants or plant-derived materials have been utilized to provide therapeutic benefits for sleep disorders. Notable herbs include valerian, passionflower, lemon balm, and Californian poppy. Recent research has concentrated on analyzing the components of natural sleep aids and identifying the compounds responsible for their sedative effects. Various mechanisms of action have been proposed, including enhancing GABAergic signaling, often through interaction with $GABA_A$ receptors. This therapy has been attempted for the treatment of insomnia, and herbal medicines have shown an exceptional safety record, particularly when compared to pharmacotherapy for insomnia. Additionally, they are widely accepted among patients.[10]

In ancient texts, sleep, known as Nidra, is recognized as one of the essential components for overall well-being. "Yoga Nidra," a guided meditation technique, has gained attention in the last 20 years as a therapeutic approach for conditions like stress, mental health issues, and insomnia. It is distinct from traditional yoga as it doesn't involve physical poses. Yoga Nidra offers a promising method to induce a deep state of relaxation akin to natural deep sleep, aiming for complete emotional, physical,

Table 1: Hypnotic class drugs, their mechanisms of action, and their effects on sleep[7,9]

Name	*Mechanism of action*	*Effect on sleep*
Benzodiazepines		
Estazolam	γ-aminobutyric acid ($GABA_A$) receptor in neurons	Sleep maintenance
Flurazepam		Sleep maintenance
Quazepam		Sleep maintenance
Temazepam		Sleep maintenance and onset
Triazolam		Sleep onset
Z-drugs (non-benzodiazepine)		
Eszopiclone	Selective subunits of $GABA_A$ receptors	Sleep onset and maintenance
Zaleplon		Sleep onset
Zolpidem		Sleep onset and maintenance
Zolpidem, extended release		Sleep onset and maintenance
Zolpidem, sublingual		Night awakening
Melatonin agonist		
Ramelteon	Acts on MT1 and MT2 receptors	Sleep onset
Orexin receptor antagonist		
Suvorexant	Acts on OX1R and OX2R receptors	Sleep onset and maintenance
Lemborexant		Sleep maintenance
Daridorexant		
Tricyclic/tetracyclic antidepressants		
Amitriptyline	The mechanism of action is not known but is likely related to antagonism of the histamine H1 receptor	Limited use
Doxepin		Sleep maintenance
Nortriptyline		Limited use
Antipsychotics		
Olanzapine	Blocking D2 dopamine receptors and serotonin receptor antagonist	All of them have limited use
Quetiapine		
Risperidone		
Anticonvulsants		
Gabapentin	Reduces relapse of monoamine neurotransmitter	Limited use
Pregabalin		

and mental relaxation. Regular morning practice at home may alleviate anxiety and discomfort associated with waking up early, potentially enhancing sleep quality, quantity, and overall wellness. This non-pharmacological approach holds therapeutic promise for enhancing well-being.[10,11]

Pharmacotherapy for Insomnia

According to guidelines from the American Psychological Association (APA), it's recommended to involve patients in decisions about using medication for insomnia. This involves discussing the benefits, risks, and costs of pharmacologic therapy. The American College of Physicians (ACP) treatment guidelines list pharmaceuticals that have been approved by the United States food and Drug Administration (USFDA) to treat insomnia, including first-generation histamine antagonists like doxepin, benzodiazepines (BZDs), non-BZD hypnotics (often referred to as "*Z*-drugs"), Dual Orexin Receptor Antagonists like Suvorexant (DORA), and melatonin receptor agonists like Ramelteon. Additionally, other medications may be used off-label. While the guidelines list medications for insomnia, they don't recommend specific ones due to limited evidence of their effectiveness and risks.[5]

The AASM provides recommendations on specific medications for treating different types of insomnia—sleep onset and sleep maintenance. Certain medications, such as certain BZD receptor agonists and BZDs, are suggested for both types of insomnia. Zaleplon, Triazolam, and Ramelteon are recommended for sleep-onset insomnia, while suvorexant and doxepin are suggested for sleep-maintenance insomnia. However, medications like Trazodone, Tiagabine, Diphenhydramine, Melatonin, L-tryptophan, and Valerian are not recommended (Table 1).

These recommendations are considered "weak" due to limitations in confidence regarding efficacy data, quality, risks, and patient preferences, rather than because the medications are ineffective for treating insomnia.[7,8]

Herbs Used in the Treatment for Insomnia

Traditional ayurvedic practices emphasize a comprehensive approach to addressing sleep disorders, incorporating various techniques such as physical treatments, psychological interventions, medicinal remedies, dietary adjustments, prayer, and yoga. While classical ancient literature outlines herbal formulations for sleep disorders, contemporary markets offer additional formulations not documented in ancient texts. Numerous studies have explored the use of herbal extracts to reduce sleep latency, increase sleep duration, and improve sleep quality. However, clinical evidence supporting their effectiveness in treating insomnia is currently lacking. Nevertheless, research has indicated that herbal medicines used for insomnia treatment may work through various mechanisms of action.[6]

For example, components of Ginseng (*Withania somnifera L.*), Ginkgo biloba L., and St John's Wort (*Hypericum perforatum L.*) have

Table 2: Medicinal plants with sleep-inducing properties used for the treatment of insomnia: Targeting GABAergic signaling for action and their effect on sleep[6]

Scientific name with chemical composition (Indian name)	*Mechanism of action*	*Effect on sleep*
Valeriana officinalis L.		
Alkaloids, terpenes, organic acids and their derivatives, valepotriates, and flavones (Valerian)	$GABA_A$ receptor	Reduces sleep latency, improves subjective measures
Magnolia sp		
Magnolol and honokiol (Hima Champa)	$GABA_A$ receptor	Promotes REM sleep
Artemisia sp		
Benzodiazepines (Nagdona)	$GABA_A$ receptor	Reduces sleep latency
Nelumbo nucifera Gaertn		
Nuciferine, alkaloids (Lotus)	$GABA_A$ receptor	Promotes sleep
Moringa oleifera Lam		
Oleic acid, β-Sitosterol, and Stigmasterol (Drumstick tree)	$GABA_A$ receptor	Increases sleep quality
Piper methysticum L.		
Kavapyrones (Kava-kava)	$GABA_A$ receptor (not benzodiazepine site)	Decreases sleep latency; no effect on NREM sleep
Zizyphus jujube Sanjoinine A, suanzaorentang (Jujube, or red date)	$GABA_A$ receptor activation of GABA synthesis through enhanced expression of GAD; serotonin receptors	Improves sleep quality, prolonging sleep time and increasing NREM sleep
Passiflora incarnata		
Apigenin, alkaloids, flavones (Passionflower)	$GABA_A$ and $GABA_B$ receptor and possibly $GABA_C$ receptor	Reduces sleep latency, increases sleep duration
Withania somnifera L.		
Withanolide A, withaferin A (Indian ginseng)	$GABA_A$ and $GABA_C$ receptor	Reduces sleep latency, improves sleep quality
Eschscholzia californica Cham.		
Alkaloids (Californian poppy)	$GABA_A$ receptor and serotonin receptors	Improves sleep latency and duration
Polygala tenuifolia Willd		
Tenufolin (Field milkworts)	Increases the levels of GABA and GABA transporter I	Increases sleep duration
Melissa officinalis L.		
Rosmarinic acid (Lemon balm)	Decreases the level of GABA transaminase	Improves sleep quality
Ginkgo biloba L.		
Ginkgotoxin, flavonoids, terpenoids (Ginkgo)	Inhibition of GAD activity	Improves subjective sleep quality measures
Hypericum perforatum L.		
Hypericin, pseudohypericin, hyperoside, among others (St John's Wort)	Inhibition of GAD and GABA transporter activity	Increases REM latency and deep sleep
Citrus aurantium L.		Increases sleep duration
Limonene, β-myrcene (bitter orange)	Serotonergic system; proposed interaction with GABA receptor binders, such as diazepam	

GABA, gamma-aminobutyric acid; GAD, generalized anxiety disorder; NREM, non-rapid eye movement; REM, rapid eye movement

been shown to influence the re-uptake of neurotransmitters like norepinephrine, dopamine, and serotonin. Additionally, extracts of Jujube seeds and Valerian (*Valeriana officinalis L.*) directly interact with serotonin receptors, and Griffonia simplicifolia Baill. contains 5-hydroxytryptophan, a natural precursor of serotonin. L-theanine, found in green tea, has been found to potentiate GABA, dopamine, and serotonin receptors and inhibit glutamate reuptake (Table 2). Active components of lavender (Lavandula angustifolia Miller) can bind to glutamate N-methyl-D-aspartate receptors and serotonin transporters. Furthermore, several herbal substances may interact with glutamic acid decarboxylase or modulate GABA and serotonin receptors.[6]

Adjuvant Therapies for Insomnia

Cognitive-behavioral therapy and pharmacotherapy stand out as the only two treatments with robust evidence supporting their efficacy in managing insomnia. While numerous clinical trials have independently assessed the effectiveness of CBT and medication, few have directly compared their individual and combined effects on insomnia. Overall, the limited available evidence suggests that both modalities yield short-term effectiveness, with medication offering rapid symptom relief, albeit often without sustained benefits post-treatment. In contrast, CBT may require more time to produce results initially but tends to deliver longer-lasting benefits. Combining CBT with medication seems to offer some advantages

Table 3: Adjuvant treatment modalities, their description, and effect on sleep[7]

Treatment modalities	*Treatment description*	*Effect on sleep*
Cognitive behavioral therapy for insomnia (CBT-I)	CBT-I combines one or more of the cognitive therapy strategies with education about sleep regulation plus stimulus control instructions, sleep hygiene education, relaxation training, and counter-arousal methods. Information typically gathered with sleep diaries completed by the patient throughout the course of treatment (typically 4–8 sessions).	CBT-I is preferred because it has superior long-term effectiveness and improvement in symptoms with minimal side effects as compared to control conditions.
Brief therapies for insomnia (BTIs)	BTIs include abbreviated versions of CBT-I (typically 1–4 sessions) emphasizing the behavioral components. It has treatment approaches along with a tailored behavioral prescription based on stimulus control and sleep restriction therapy information derived from a pretreatment sleep diary.	A meta-analyses study showed clinically significant improvements in remission rate, responder rate, and sleep quality in patients practicing BTIs.
Stimulus control	Set of instructions designed to extinguish the association between the bed/bedroom and wakefulness to restore the association of bed/bedroom with sleep; and establish a consistent wake-time.	It helps in regulating the sleep-wake cycle with a set of designated activities to follow.
Sleep restriction therapy	Initially, time in bed matches the average sleep duration and is then adjusted based on sleep efficiency thresholds until satisfactory sleep duration and quality are reached.	It improves sleep quality by adjusting time spent in bed according to the patient's actual sleep duration, as recorded in daily diaries.
Relaxation therapy	Structured exercises intended to alleviate somatic tension (such as abdominal breathing, progressive muscle relaxation, and autogenic training) and cognitive arousal (such as guided imagery training and meditation) can contribute to ongoing sleep issues.	Improves the onset and maintenance of sleep.
Sleep hygiene	Sleep hygiene may include some education about what constitutes "normal" sleep and changes in sleep patterns with aging.	A set of general recommendations about lifestyle (e.g., diet, exercise, substance use) and environmental factors (e.g., light, noise, temperature) that may promote or interfere with sleep is improved.
Cognitive therapy	Strategies include structured psycho education, Socratic questioning, the use of thought records, and behavioral experiments designed to identify and modify unhelpful beliefs about sleep.	It may support sleep-disruptive habits and/or raise performance anxiety about sleeping.
Biofeedback	Variant of relaxation training that employs a device capable of monitoring and providing ongoing feedback on some aspect of the patient's physiology.	Monitoring of frontalis electromyography (EMG) activity to assess the overall level of muscle tension. The biofeedback device produces an ongoing auditory tone to train the patient to relax by learning how to alter the auditory feedback tone in the desired direction (e.g., reduced muscle tone).
Paradoxical intention	Patients are instructed to remain awake as long as possible after getting into bed. The patient is instructed to purposefully engage in the feared activity (staying awake) in order to reduce performance anxiety and conscious intent to sleep that confound associated goal-directed behavior (falling asleep).	This method alleviates both the patient's excessive focus on sleep and anxiety over not sleeping; as a result, sleep becomes less difficult to initiate.
Intensive sleep retraining	After a night with 5 h in bed, the treatment involves a 24-h lab test, where the patient tries to sleep every 30 min. If they doze off, they're woken up after 3 min and stay awake until the next 30-min trial. They get feedback on each attempt.	This treatment is designed to markedly enhance homeostatic sleep drive in order to reduce both sleep onset difficulties and sleep misperception.
Mindfulness therapies	Mindfulness involves meditation, focusing on being aware of thoughts and feelings without judgment. It's often done in group sessions.	It's applied to people with insomnia and is often combined with other insomnia therapies such as stimulus control, sleep restriction therapy, and sleep hygiene.

in the early stages of treatment, but the long-term outcomes vary among patients.[5,8]

American Academy of Sleep Medicine guidelines on the psychological and behavioral treatments of insomnia recommend non-pharmacological and adjuvant therapies with medicines. These recommendations are based on a systematic evaluation of clinical trials, and the evidence is evaluated using the Grading of Recommendations Assessment Development and Evaluation (GRADE) process.[7,10] Help clinicians treat patients with chronic insomnia disorder (Table 3).

The interventions suggested in this guideline are designed to fulfill the needs of the majority of patients in a range of circumstances. Like a quality metric, a "strong" suggestion denotes advice that physicians should almost always follow (Fig. 2).

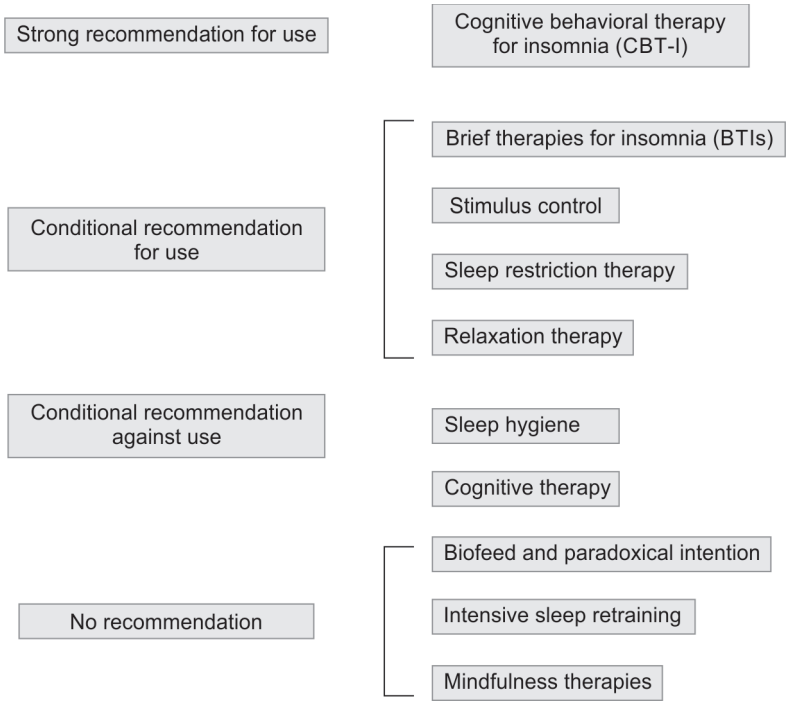

Fig. 2: AASM clinical practice guidelines implication if strong conditional recommendations for treatment of insomnia

Mindfulness therapies have also been elaborated in Indian studies, which involve meditation and yoga and their effects on resolving factors related to sleep disorders, maintenance, and onset of sleep. Many mindfulness therapies, involving meditation and yoga, have been developed for treating insomnia. However, the available studies and evidence are insufficient. Therefore, larger-scale studies and trials should be conducted to generate more robust evidence in this area.

Cyclic meditation (CM) is a technique combining yoga postures interspersed with supine rest. The combination is based on ancient Indian texts. During the practice, participants maintained closed eyes and followed pre-recorded instructions emphasizing a slow, mindful approach with relaxation. The session commences with reciting a verse from the yoga text *Mandukya Upanisad* for 40s, followed by isometric muscle contractions throughout the body, culminating in supine rest for 1 min. Participants then transition to standing in *tadasana*, centering their weight evenly for 2 min. This is followed by specific postures, including bending to the right (*Ardhakaticakrasana*) for 80 s, a 70-s pause in *tadasana* for relaxation and awareness, bending to the left (*Ardhakaticakrasana*) for 80s, another 70s pause, forward bending (*Padahastasana*) for 80s, another 70s pause, and backward bending (*Ardhacakrasana*) for 80s. The session concluded with a slow descent into a supine posture, with instructions to sequentially relax different body parts over 10 min. Postures are performed slowly, with a heightened awareness of bodily sensations. The total duration of the practice is 22 minutes and 30 seconds. *Cyclic meditation* appeared to increase slow-wave sleep and lower the percentage of time in rapid-eye-movement sleep. Also, the number of awakenings per hour is less. The practice of cyclic meditation twice a day appears to improve the objective and subjective quality of sleep the following night.[12]

Yoga practice has demonstrated a significant reduction in work stress, anxiety, muscle fatigue, and emotional exhaustion while enhancing sleep quality, self-care, coping skills, and overall well-being. Previous research indicates that yoga decreases sympathetic nervous system activity, increases parasympathetic activity, and balances autonomic nervous system reactivity. Moreover, it has been shown to boost the secretion of GABA, a neurotransmitter associated with relaxation. These findings suggest that the increase in parasympathetic activity, decrease in sympathetic activity, and heightened GABA secretion following yoga practice may contribute to stress reduction and improved sleep quality. Additionally, the elevation in positive affect and self-compassion, along with the suppression of the posterior hypothalamus and salivary cortisol levels post-yoga, may also play crucial roles in stress reduction. However, while these mechanisms offer insights, a comprehensive understanding of how yoga intervention reduces stress and enhances sleep quality remains elusive. Yoga can be used and can be adopted as a cost-effective, community-based, non-pharmacological tool for the treatment of insomnia.[10,11]

The review has explored various maintenance strategies and the potential for alternative treatment sequences to improve outcomes. For instance, patients who don't respond adequately or achieve remission with initial therapy might benefit from a second-level intervention, which could involve either adding to or switching their treatment approach. A patient initially treated with CBT and other adjuvant therapies might transition to medication, while someone initially treated with medication might switch to CBT or herbal treatments. In making clinical decisions about initial treatment, practical factors such as the availability and acceptability of treatment options, along with clinical considerations like the nature of insomnia (acute vs chronic), previous treatment experiences, comorbidities, and evidence of efficacy, should also be considered.

Conclusion

Various modalities of insomnia therapy have unique approaches, with evidence demonstrating their practices and benefits.

Currently, treating insomnia involves a variety of methods, and over time, it should be noted that integrating different treatment methodologies can yield better patient outcomes. The literature review indicates that insomnia treatment should not be confined to one modality; exploring all possible approaches, including pharmacotherapy, herbal therapy, and adjuvant therapy, is essential for treating chronic insomnia. Given the limited evidence available for an integrated approach, combining different treatments may lead to better outcomes and benefits for patients. Therefore, a study is needed to assess the various treatment modalities and approaches to generate evidence in these areas.

Orcid

Abhishek M Bulla https://orcid.org/0009-0008-2361-3842
Puneet K Nagendra https://orcid.org/0000-0002-9887-3699

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