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# THE EFFECTS OF ASHWAGANDHA (WITHANIA SOMNIFERA) ON INSOMNIA AND SLEEP QUALITY - A NARRATIVE REVIEW

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**ABSTRACT**

Insomnia is one of the most common sleep disorders in modern society and represents a major public health concern due to its wide-ranging impact on physical health, mental well-being, and overall quality of life. Due to the escalating prevalence of sleep disorders and the potential side effects associated with conventional pharmacotherapy, there is a growing clinical and public interest in natural substances that can safely support the treatment of insomnia.

The purpose of this review was to evaluate the current state of knowledge regarding the clinical and preclinical efficacy of Ashwagandha (*Withania somnifera*) in treating insomnia and sleep disturbances, while elucidating its underlying neurobiological mechanisms of action. A structured narrative literature review was conducted by searching major biomedical databases.

The analyzed studies demonstrated that ashwagandha supplementation leads to statistically significant improvements in sleep metrics. In patients diagnosed with insomnia, researchers reported a shortened sleep onset latency (SOL), reduced waking after sleep onset (WASO), and increased total sleep time (TST). The majority of findings consistently underscore that Ashwagandha's most profound impact on sleep architecture is driven by its interactions with the GABAergic pathway. Furthermore, supplementation correlated with a significant reduction in serum cortisol levels by a multi-pathway mechanism of action involving the hypothalamic-pituitary-adrenal (HPA) axis. The extracts were well-tolerated, with only mild and transient adverse effects reported.

Ashwagandha represents an effective and safe therapeutic alternative for managing insomnia and enhancing sleep quality, particularly in individuals experiencing chronic stress.

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**KEYWORDS**

Ashwagandha, Insomnia, Sleep Quality, Sleep Disturbances

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## 1. Introduction.

Insomnia is one of the most common sleep disorders in modern society and represents a major public health concern due to its wide-ranging impact on physical health, mental well-being, and overall quality of life. It is estimated that insomnia symptoms may affect up to 30% of the adult population [1]. Insomnia is defined as difficulty falling asleep, maintaining sleep, waking up too early, or insufficient sleep quality despite adequate restful conditions. If these problems occur at least three times per week for longer than three months, they lead to impaired daytime functioning, causing fatigue, drowsiness, difficulty concentrating, irritability, and decreased work and learning performance [2]. Modern lifestyles, chronic stress, excessive exposure to blue light emitted by electronic devices, irregular circadian rhythms, and the accelerating pace of life contribute to the increasing prevalence of sleep disorders [3]. Insomnia negatively impacts not only patients' quality of life but also cognitive functioning, mental and physical performance, and increases the risk of developing numerous chronic diseases [4]. Approximately 75%–90% of individuals with insomnia are at a higher risk of developing chronic diseases, including hypertension, obesity, type 2 diabetes, congestive heart failure, and myocardial infarction [5]. Persistent sleep disturbances may increase susceptibility to anxiety disorders and depressive relapses or be a significant risk factor for suicidal behavior [8]. Due to the increasing prevalence of sleep disorders, there is a growing interest in natural substances that can support the treatment of insomnia. One such example is ashwagandha, also known as *Withania somnifera*. Ashwagandha is a plant in the nightshade family (Solanaceae) and has been used in Ayurvedic medicine for centuries to improve stress resistance and support mental and physical recovery [10]. The species name "somnifera" means "sleep-inducing," suggesting its potential to improve sleep quality. This paper aims to present the current state of knowledge regarding the effects of Ashwagandha on insomnia and related sleep disturbances, discuss its potential mechanisms of action, and critically analyze the available evidence on its efficacy in improving sleep quality. In addition, the paper addresses the broader clinical and functional consequences of insomnia, including its impact on cognitive performance, emotional regulation, daytime functioning, and overall quality of life.

## 2. Methodology.

This review was conducted as a structured narrative literature review to evaluate the effects of *Withania somnifera* on insomnia and sleep-related outcomes. A comprehensive literature search was performed using major biomedical databases, including PubMed, Scopus, and Google Scholar. The search strategy included combinations of keywords such as “ashwagandha”, “*Withania somnifera*”, “insomnia”, “sleep quality”, “sleep onset latency”, “sleep disorders”, “sleep efficiency”, “total sleep time”, “sleep architecture”, “non-rapid eye movement sleep”, “REM sleep”, “sedative effect”, “GABAergic activity”, “histamine”, “H1 receptor”, “H3 receptor”, “neurotransmission”, and “adaptogens”. The review primarily included studies published between 2010 and 2026 to ensure inclusion of current evidence on the influence of ashwagandha on insomnia symptoms and sleep-related measures. Earlier studies were also included when considered relevant for understanding the neurobiological and endocrine mechanisms underlying insomnia, particularly those related to GABAergic and histaminergic neurotransmission, adenosine, hypothalamic–pituitary–adrenal (HPA) axis regulation, and cortisol-mediated stress responses, as well as broader neuroendocrine and inflammatory pathways implicated in sleep regulation. Only peer-reviewed articles published in English were considered. Eligible studies included randomized controlled trials, meta-analyses, preclinical animal studies, and systematic reviews investigating the effects of Ashwagandha or its bioactive compounds on sleep parameters. Studies were selected based on their relevance to sleep outcomes, methodological quality, and clarity of reported results. Data were extracted and synthesized narratively, with a focus on neurobiological mechanisms including GABAergic and histaminergic activity.

## 3. Results.

### 3.1. EEG Correlates of Sleep in Insomnia: Insights into the Effects of Ashwagandha on Sleep Architecture and Neurophysiological Activity.

Electroencephalographic (EEG) analysis demonstrated characteristic neurophysiological patterns across individual sleep stages, reflecting normal sleep architecture and variations in cortical activity during the sleep cycle. Wakefulness was associated with predominant alpha rhythm activity (8–13 Hz) observed during eye closure, whereas low-amplitude, mixed-frequency activity was present with the eyes open. This stage was additionally characterized by preserved muscle tone and voluntary eye movements. In stage N1 sleep, alpha activity disappeared, and theta waves (4–7 Hz) became more prominent, accompanied by slow eye movements. Stage N1 constituted approximately 5% of total sleep time. Stage N2 accounted for the largest proportion of total sleep, nearly 50% of the sleep cycle. EEG recordings during this stage revealed characteristic sleep spindles (11–16 Hz bursts) and K-complexes superimposed on a theta-wave background. Eye movements were absent, while muscle tone was reduced compared with wakefulness. Deep sleep (N3, slow-wave sleep) was characterized by high-amplitude delta waves (0.5–2 Hz,  $\geq 75 \mu\text{V}$ ). This stage accounted for approximately 20% of total sleep duration and was associated with markedly reduced muscle tone and absence of eye movements. Rapid eye movement (REM) sleep demonstrated low-amplitude mixed-frequency EEG activity resembling wakefulness, accompanied by characteristic sawtooth waves. Rapid eye movements occurring in bursts and complete muscle atonia were observed during this phase. REM sleep represented approximately 20–25% of total sleep time [26].

| Sleep Stage | EEG Characteristics                                                                               | Eye Movements and Muscle Tone                          | Proportion of Total Sleep |
|-------------|---------------------------------------------------------------------------------------------------|--------------------------------------------------------|---------------------------|
| Wakefulness | Alpha rhythm (8–13 Hz) when eyes closed; low-amplitude, mixed-frequency when eyes open.           | Eye blinks, reading eye movements; high muscle tone.   | —                         |
| N1          | Disappearance of alpha rhythm; low-amplitude, mixed-frequency (4–7 Hz theta); vertex sharp waves. | Slow rolling eye movements; moderate muscle tone.      | ~5%                       |
| N2          | Sleep spindles (11–16 Hz bursts) and K-complexes on theta background.                             | No eye movements; reduced muscle tone.                 | ~50%                      |
| N3          | High-amplitude delta waves (0.5–2 Hz, $\geq 75 \mu V$ ) comprising $\geq 20\%$ of the epoch.      | No eye movements; low muscle tone.                     | ~20%                      |
| REM         | Low-amplitude, mixed-frequency (resembles wakefulness); sawtooth waves.                           | Rapid eye movements in bursts; complete muscle atonia. | 20% to 25%                |

Consequently, electroencephalography (EEG) remains the clinical gold standard for mapping neural oscillations and macro-architectural shifts across distinct sleep stages, which was used in the first study evaluating the effects of different Ashwagandha leaf extracts on sleep regulation in mice after oral administration. These extracts were tested for their sleep-inducing properties, followed by electroencephalography and electromyography recordings. The alcoholic extract, despite containing high concentrations of active withanolides, did not demonstrate significant sleep-inducing activity. In contrast, the water extract, which contains triethylene glycol (TEG) as a major component, markedly promoted NREM sleep with only minimal alterations in REM sleep. The findings suggest that TEG is one of the main bioactive compounds responsible for the sleep-promoting effects of Ashwagandha leaves. The study confirmed the sleep-inducing effects of TEG in animal models, suggesting that this small molecule may have therapeutic effects in the management of insomnia [18].

### 3.2. GABAergic Modulation in Insomnia: The Role of GABA, Histamine, and Adenosine in Sleep Regulation.

At the neurobiological level, sleep regulation is a complex process involving the interplay of multiple neurotransmitter systems, with gamma-aminobutyric acid (GABA) playing a central role. Therefore, preclinical electrophysiological studies have demonstrated that *Withania somnifera* extracts exert GABA-mimetic effects, as evidenced by bicuculline-sensitive inward currents mediated via GABA A receptors. This suggests direct modulation of inhibitory neurotransmission that may contribute to its sedative and sleep-promoting properties [20].

In this context, the first study was based on *in vitro* analyses to ascertain the hypothesis that Ashwagandha extract (AE) increases the expression levels of GABA and histamine receptors in treated cells in a dose-dependent manner. A pentobarbital sleep-induction model with Swiss albino mice and diazepam positive control, along with electroencephalography (EEG) brain waves, was studied to confirm that hypothesis. The use of EEG analysis provided a more comprehensive assessment of sleep quality and intensity beyond simple sleep induction models. The study reported that AE increased gene expression of the GABAA A $\alpha$ 1 and histamine H3 receptors at concentrations of 15 and 30  $\mu g/mL$  compared with the control. *In vivo*, administration of AE at doses of 10, 25, and 50 mg/kg significantly reduced sleep onset latency (SOL) and prolonged total sleep duration in the pentobarbital-induced sleep model. At the highest dose (50 mg/kg), sleep onset time decreased by 34%, while total sleep duration increased by 47%. EEG analysis further demonstrated dose-dependent modulation of brain activity, with significant changes across alpha, beta, theta, delta, and gamma frequency bands. Notably, delta wave activity increased by 30%, 46%, and 34% at 10, 25, and 50 mg/kg, respectively. Overall, these findings indicate that AE promotes sleep induction, enhances NREM sleep, and modulates slow-wave activity, supporting its sedative and sleep-quality-improving properties [19].

Similar findings are also reported in another preclinical study investigating EA, which was focused on the role of ashwagandha in sleep regulation via the GABAergic system. In this study, AE was evaluated in rodent models using both pentobarbital-induced sleep testing and EEG analysis to assess sleep-promoting activity and underlying mechanisms. In addition, the sleep-promoting mechanism of AE was elucidated by

analyzing the expression of sleep-related receptors. The results demonstrated that AE dose-dependently increased total sleep duration in the pentobarbital-induced sleep model. EEG recordings further showed significant increases in  $\delta$ -wave activity and non-rapid eye movement (NREM) sleep, indicating enhanced sleep depth and improved sleep quality. In addition, AE alleviated caffeine-induced insomnia symptoms, further confirming its sedative potential. The treatment significantly elevated GABA levels in the brain, increased the mRNA and protein expression of GABA and serotonin receptors compared with the control group. Notably, AE demonstrated sleep-promoting properties through interactions with multiple binding sites on GABA receptors. Overall, the study suggests that *Withania somnifera* promotes both sleep quantity and quality by modulating the GABAergic and serotonergic systems, highlighting its potential as a functional agent in managing sleep disturbances [22].

The next experimental study demonstrated that Ashwagandha extract containing withaferin A (WFA) may exert significant sleep-promoting effects through modulation of both GABAergic and adenosine signaling pathways. EEG analysis revealed reduced wakefulness, prolonged non-rapid eye movement sleep, and increased  $\delta$ -wave activity, suggesting improved sleep depth and quality. The extract also significantly prolonged sleep duration and alleviated caffeine-induced sleep disturbances in animal models. Importantly, the observed effects were mediated not only through GABA A receptor pathways, but also through adenosine A1 receptor signaling, indicating a broader neurochemical mechanism underlying the sleep-enhancing properties of Ashwagandha. Furthermore, chronic administration increased brain GABA levels and altered the expression of genes associated with GABAergic and serotonergic neurotransmission [31].

### 3.3. Neuroprotective and Stress-Reducing Effects of *Withania somnifera* in Sleep Deprivation and Stress-Related Sleep Disturbances.

Experimental evidence from animal models of sleep deprivation suggests that ashwagandha may exert beneficial neuroprotective effects not only on sleep-related processes and insomnia but also on the broader neurobiological consequences of sleep loss, including cognitive dysfunction and stress-related neuroendocrine dysregulation. Sleep deprivation has been shown to impair spatial memory, increase oxidative stress, and disrupt neuroendocrine regulation, including activation of the Hypothalamic-Pituitary-Adrenal axis (HPA) and reduced GABAergic signaling. Administration of ashwagandha extract across different doses led to a dose-dependent improvement in both neurochemical and behavioral outcomes. Higher-concentration formulations were particularly effective in reducing stress-related hormones, including corticosterone, Corticotropin-Releasing Hormone (CRH), and Adrenocorticotrophic Hormone (ACTH), suggesting attenuation of HPA axis overactivation. Additionally, treatment reduced oxidative stress markers (MDA) while enhancing endogenous antioxidant enzyme activity. Importantly, ashwagandha positively influenced neurotransmitter balance, increasing serotonin and dopamine levels, and improved markers of neuronal plasticity and GABA receptor function. These changes were accompanied by significant improvements in memory performance, indicating functional cognitive benefits. Overall, the findings suggest that ashwagandha may protect against stress- and sleep deprivation-induced cognitive impairment through a multimodal mechanism involving suppression of HPA axis hyperactivity, reduction of oxidative stress, and modulation of neurotransmitter systems [23].

Similar findings have also been reported in human populations, further supporting its potential role in managing stress-associated sleep disturbances. In this context, recent randomized controlled trials have evaluated standardized ashwagandha formulations to better understand their impact on perceived stress, anxiety, mood, and sleep quality in adult populations. This randomized, double-blind, placebo-controlled clinical trial suggests that the standardized ashwagandha formulation (Zenroot™) may effectively reduce perceived stress and improve related psychological and sleep outcomes in adults with mild-to-moderate stress. Supplementation with 125 mg daily for 84 days resulted in significant improvements in subjective measures, including perceived stress (PSS), anxiety (BAI), mood states (POMS), and sleep quality (PSQI), indicating a broad beneficial effect on stress-related symptoms. In addition, the intervention showed favorable effects on physiological stress-related markers, including improvements in heart rate variability parameters (RMSSD and SDNN) and skin conductance response, suggesting enhanced autonomic nervous system balance and reduced sympathetic arousal. However, no significant changes were observed in biochemical stress biomarkers such as serum cortisol and salivary alpha-amylase, which may indicate either a more complex mechanism of action or limited sensitivity of these markers in populations with mild to moderate stress. Overall, Zenroot™ was well tolerated, with no safety concerns reported, supporting its short-term safety profile. The findings indicate that ashwagandha may improve psychological resilience and sleep quality primarily through modulation of stress perception and autonomic function rather than direct, measurable reductions in circulating cortisol [24].

### **3.4. Dose-Dependent Effects of *Withania somnifera* on Sleep Quality and Insomnia Symptoms.**

The available evidence suggests that the effects of *Withania somnifera* on insomnia are dose-dependent, with higher doses and longer treatment duration being associated with greater improvements in sleep outcomes. In this context, a systematic review and meta-analysis evaluated the efficacy of *Withania somnifera* extract on sleep outcomes in adults based on five randomized controlled trials, involving 400 participants. The pooled analysis demonstrated a statistically significant but modest improvement in overall sleep quality compared with placebo, indicating moderate heterogeneity between studies. Subgroup analyses revealed that the beneficial effects of ashwagandha were more pronounced in individuals with diagnosed insomnia, as well as in studies administering higher doses ( $\geq 600$  mg/day) and longer treatment durations ( $\geq 8$  weeks). Improvements were observed across multiple sleep domains, including sleep onset latency, total sleep time, wake after sleep onset, and sleep efficiency, although effects on the Pittsburgh Sleep Quality Index (PSQI) and total time in bed were not statistically significant. In addition to sleep parameters, ashwagandha supplementation significantly improved secondary outcomes, including reduced anxiety levels and enhanced mental alertness upon waking, while no meaningful improvement in quality of life was detected. Across all included trials, no serious adverse events were reported, although safety data remained limited, particularly regarding long-term use. Overall, the authors concluded that ashwagandha may have a beneficial effect on sleep in adults, particularly among individuals with insomnia [21].

The next randomized, double-blind, placebo-controlled trial demonstrates that standardized Ashwagandha root extract may be an effective and well-tolerated intervention for patients suffering from insomnia and anxiety. Administration of 300 mg twice daily was associated with improvements in sleep quality in individuals with insomnia. Compared with the control group, Ashwagandha demonstrated clear therapeutic efficacy by substantially reducing sleep onset latency, optimizing overall sleep efficiency, and improving patient-reported sleep quality across validated scales. Additionally, participants receiving ashwagandha showed a significant reduction in anxiety symptoms, suggesting a beneficial effect on hyperarousal states that often contribute to sleep disturbances. Although some improvement in sleep parameters was also observed in the placebo group, the effects were consistently greater in the treatment group, supporting a true therapeutic benefit of ashwagandha. Overall, the findings suggest that ashwagandha root extract has sleep-promoting and anxiolytic properties and may serve as a potential alternative or adjunctive treatment for insomnia [17].

### **3.5. Effects of *Withania somnifera* in Individuals with Non-Restorative Sleep (NRS).**

Nonrestorative sleep (NRS) is characterized by a subjective sense of insufficiently refreshing sleep, even when sleep duration and conventional objective sleep parameters appear normal [27]. The randomized, double-blind, placebo-controlled trial demonstrates that standardized *Withania somnifera* extract significantly improves sleep quality in individuals with non-restorative sleep (NRS). Supplementation with 120 mg daily for six weeks led to a marked improvement in both subjective and objective sleep parameters compared with placebo. Participants receiving ashwagandha demonstrated improvements in sleep efficiency, total sleep time, sleep onset latency, and wake after sleep onset. Self-reported sleep quality increased by 72% in the treatment group, compared with 29% in the placebo group. In addition, ashwagandha supplementation was associated with significant improvements in overall quality of life, including physical, psychological, and environmental domains. Importantly, the intervention was well tolerated, with no treatment-related adverse events reported. Overall, these findings suggest that ashwagandha may be an effective and safe intervention for improving sleep quality and reducing symptoms of non-restorative sleep in individuals [16].

### **3.6. Effects of Ashwagandha Root Extract on Chronic Stress, Anxiety, and Sleep Quality.**

The next review reported a strong clinical association between anxiety disorders and sleep disturbances, particularly insomnia. Sleep complaints were highly prevalent among patients with anxiety, and the two conditions frequently occurred and appeared to reinforce each other mutually. The review identified hyperarousal as the central mechanism linking anxiety and insomnia. This state was characterized by increased activity of stress-related systems, including the HPA axis, CRH, and noradrenergic pathways [28].

In this prospective, randomized, double-blind, placebo-controlled trial, 64 adult participants with a history of chronic stress received *Withania somnifera* root extract (300 mg twice daily) or placebo for 60 days. The ashwagandha-treated group demonstrated a statistically significant reduction in stress and anxiety-related outcomes compared with placebo. All assessed stress scales showed marked improvement in the intervention group, with reductions reaching up to approximately 44% on the Perceived Stress Scale (PSS), whereas the placebo group showed only minimal changes. In addition, serum cortisol levels were significantly decreased in the ashwagandha group compared with baseline and placebo, indicating a measurable effect on the physiological stress response. Improvements were also observed in anxiety-related subscales, including reductions in scores reflecting anxiety and insomnia symptoms. Tolerability was comparable between groups, with only mild adverse effects reported and no serious adverse events observed throughout the study period. Overall, the findings demonstrate that ashwagandha supplementation significantly reduces perceived stress and anxiety levels and lowers cortisol concentrations in chronically stressed adults [29].

The next study aimed to evaluate the pharmacological effect of Ashwagandha root extract on sleep in healthy subjects and in subjects with insomnia. It demonstrated that 8-week supplementation with ashwagandha root extract significantly improved multiple sleep parameters in both healthy participants and patients with insomnia. The effects were more pronounced in subjects with insomnia. Significant improvements were observed in sleep onset latency (SOL), sleep quality, mental alertness upon waking, and anxiety scores assessed using the Hamilton Anxiety Rating Scale (HAM-A). The group also demonstrated improvements in sleep efficiency and overall subjective sleep quality compared with placebo, proving it can be used to treat insomnia [15].

### **3.7. Insomnia in the Elderly Population, the Potential Role of *Withania somnifera* in Management.**

A prospective, randomized, double-blind, placebo-controlled study evaluated the efficacy and tolerability of *Withania somnifera* root extract in elderly individuals with sleep disturbances and reduced general well-being. This 12-week trial indicated that Ashwagandha root extract may significantly improve quality of life (QoL), sleep quality, and mental alertness in elderly individuals aged 65–80 years. Compared with placebo, participants receiving ashwagandha demonstrated marked improvements in overall QoL scores as measured by the WHOQOL-BREF questionnaire, with beneficial effects observed across all domains. Furthermore, significant improvements in sleep quality and morning alertness were reported, suggesting enhanced restorative sleep and reduced subjective fatigue upon waking. The intervention was also well tolerated, with no major safety concerns reported, supporting its suitability for use in an older population. Overall, these findings suggest that ashwagandha supplementation may be a safe and effective strategy to enhance general well-being, sleep parameters, and cognitive alertness in the elderly. However, further, larger-scale studies are warranted to confirm these effects [25].

### **3.8. Efficacy of Ashwagandha and Tryptophan Co-Administration.**

As an essential amino acid, L-tryptophan serves as a crucial precursor for serotonin and melatonin synthesis, thereby playing a fundamental role in regulating the sleep–wake cycle. Enhances sleep quality and reduces the duration of night-time awakenings [34]. Given the well-documented independent benefits of both ashwagandha extracts and dietary tryptophan in mitigating sleep disturbances, Pérez-Piñero et al. (2024) designed a randomized, double-blind, controlled trial to evaluate a novel intervention. The study tested the efficacy of lactose-free skimmed milk enriched with either ashwagandha alone (at 250 mg or 600 mg) or a combination of ashwagandha (250 mg) and tryptophan (175 mg). The investigators hypothesized that combining these two ingredients would provide a synergistic effect, offering superior therapeutic outcomes

compared with ashwagandha monotherapy. The study revealed that while general Pittsburgh Sleep Quality Index (PSQI) variations between groups did not reach statistical significance, individual subjective measures—such as the Visual Analogue Scale (VAS) for sleep quality and the Insomnia Severity Index (ISI) showed more favorable outcomes in the ashwagandha-treated groups compared to the placebo. Daily intake of the ashwagandha-enriched products leads to a significant decrease in the ISI. It reduces daytime drowsiness across all experimental groups, with the 600 mg ashwagandha dose showing particularly pronounced benefits regarding insomnia reduction. Contrary to the researchers' expectations, the co-administration of tryptophan did not exert a significant synergistic effect, indicating that ashwagandha alone drives the primary sleep-promoting benefits. The synergistic effect was not statistically significant, as ashwagandha alone proved highly effective. Beyond that, the study highlighted the safety profile of long-term (90 days) ashwagandha intake and had no negative impact on body composition [35].

### **3.9. Comparative Efficacy of Ashwagandha Root Extract and Melatonin on Sleep Parameters.**

Melatonin (MLT) is an endogenous hormone synthesized and secreted primarily by the pineal gland in response to darkness, serving as the master regulator of the body's biological clock and the circadian sleep–wake cycle. Clinically, exogenous melatonin supplementation is widely recognized as the traditional gold standard therapy for circadian disruptions, jet lag, and primary insomnia due to its well-documented ability to decrease sleep onset latency and shift circadian phases [37]. Building upon this established framework, Movva et al. (2026) conducted an innovative clinical trial to determine whether a natural adaptogen could offer comparable therapeutic benefits. Their prospective, randomized, double-blind, placebo-controlled study evaluated the clinical efficacy and safety of Ashwagandha root extract (ARE) in direct comparison to melatonin and a placebo among adults experiencing sleep disturbances. This study enrolled 200 male and female participants (aged 18–50 years), who were equally allocated into one of four distinct intervention arms for an 8-week duration: ARE monotherapy (300 mg administered twice daily), MLT monotherapy (3 mg daily), a combined regimen of ARE (600 mg/day) and MLT (3 mg/day), or a placebo. The primary clinical parameter evaluated was the change in actigraphy-verified sleep onset latency (SOL) from baseline to week 8. Secondary variables comprised actigraphy-derived measures of total sleep time (TST), wake after sleep onset (WASO), and sleep efficiency (SE), alongside subjective clinical tools including the Pittsburgh Sleep Quality Index (PSQI) and the Hamilton Anxiety Scale (HAM-A). By week 8, inferential statistical analyses demonstrated highly significant advancements across all objective and subjective sleep metrics. While both individual therapies offered moderate and clinically comparable improvements over the placebo, the co-administration of ARE and MLT triggered the most robust reductions in SOL, WASO, and daytime anxiety, while maximizing TST and overall SE. Safety and tolerability matrices indicated a balanced distribution of only minor adverse events across all treatment arms, revealing no clinically meaningful differences between groups [36].

## **4. Discussion.**

The present review indicates that *Withania somnifera* may represent a multi-target intervention for sleep disturbances, with evidence supporting its beneficial effects on sleep quality, sleep onset latency, and overall sleep efficiency across both clinical and preclinical studies. Ashwagandha not only improves sleep quality but also has a beneficial effect on patients' overall mental and physical health, reducing anxiety and mental tension in individuals using preparations containing Ashwagandha extract [15,28,29]. Moreover, it is important to acknowledge that recent years have seen a rapid increase in the number of studies evaluating the effectiveness of ashwagandha in the treatment of insomnia and stress-related disorders.

#### **4.1. The GABAergic and Histaminergic Neurotransmission.**

The cited studies indicate that Ashwagandha's effects involve multiple neurobiological mechanisms underlying sleep regulation; however, a central and consistently proposed mechanism is its modulatory influence on GABA receptors [13]. Findings suggest that histaminergic and GABAergic neurotransmission play crucial roles in the regulation of the sleep–wake cycle. Histamine is a neurotransmitter that is responsible for maintaining the awake state, histaminergic neurons are also capable of releasing  $\gamma$ -aminobutyric acid. In contrast, GABA is recognized as one of the principal neurotransmitters promoting sleep, with GABAergic neuronal populations being strongly associated with sleep induction and maintenance. The GABA system is the primary inhibitory system in the central nervous system and is responsible for reducing neuronal excitability. Many hypnotics and anxiolytics, such as benzodiazepines, act through GABA receptors. Experimental studies have shown that histamine signaling through H1 and H3 receptors plays a key role in maintaining the normal sleep–wake cycle. Some studies suggest that compounds in ashwagandha may exhibit GABA-mimetic effects, including increased GABA levels, upregulation of GABA receptor expression, and direct interaction with GABA A<sub>A</sub> receptor sites [19,20,22]. These effects are associated with enhanced non-rapid eye movement sleep and increased slow-wave activity, which are considered markers of deep and restorative sleep, which may explain its sedative and sleep-promoting properties [18]. The balance between these neurotransmitter systems appears to be essential for normal sleep physiology and may represent an important therapeutic target in sleep disorders such as insomnia.

#### **4.2. The serotonergic modulation.**

Furthermore, ashwagandha may influence the serotonergic system and the regulation of circadian rhythms, which may further improve the quality and duration of sleep [9]. Based on the evidence, the serotonergic system plays an important modulatory role in circadian timing. Serotonergic afferents influence the suprachiasmatic nucleus (SCN) activity by regulating its responsiveness to environmental light cues, including inhibition of light-induced phase shifts and induction of phase adjustments under non-photoc conditions. These findings support the concept that serotonin is not only involved in sleep–wake regulation but also acts as an integral component of the circadian timing system. Consequently, serotonergic modulation may contribute to the synchronization of behavioral and physiological rhythms with environmental cycles, and disturbances in this pathway could potentially impair circadian stability and sleep–wake regulation [33].

#### **4.3. The adenosine signaling.**

It has also been demonstrated that sleep regulation is closely linked to adenosine signaling. Previous studies have demonstrated that extracellular adenosine accumulates during prolonged wakefulness and promotes sleep by inhibiting wake-promoting neuronal activity. In particular, activation of adenosine A1 receptors has been associated with increased slow-wave activity and enhanced NREM sleep, suggesting an important role in the regulation of sleep pressure and sleep recovery. Furthermore, antagonism of adenosine receptors by substances such as caffeine has been shown to promote wakefulness, further supporting the physiological importance of adenosinergic signaling in sleep regulation. Therefore, the sleep-enhancing effects of Ashwagandha observed in experimental studies may be mediated not only through modulation of GABAergic neurotransmission, but also through activation of adenosine A1 receptor pathways [32].

#### **4.4. The Hypothalamic-Pituitary-Adrenal Axis.**

The main biologically active substances in ashwagandha are withanolides, alkaloids, sitoindosides, and flavonoids. These compounds have been shown to affect numerous neurotransmitter and hormonal systems [11]. One of the most important mechanisms of ashwagandha's action is modulation of the HPA axis and reduction of cortisol levels, commonly known as the "stress hormone." Chronically elevated cortisol levels lead to excessive arousal, make it difficult to fall asleep, and impair sleep quality. Research suggests that supplementing with ashwagandha may reduce the body's response to stress and promote a calming effect on the nervous system [12]. Neurobiological mechanisms responsible for cognitive impairment in insomnia may involve dysfunction of the prefrontal cortex, chronic activation of the HPA axis, and disturbances in synaptic plasticity. Overactivation of the HPA axis, increased cortisol levels, and increased sympathetic nervous system activity play a significant role in the pathogenesis of insomnia [6]. Moreover, insomnia is considered a condition associated with persistent hyperarousal occurring throughout the day. This state may manifest as increased daytime alertness and hypervigilance, as well as difficulties with falling asleep and maintaining sleep during the night [7]. A growing body of research also points to the involvement of neurotransmitters such as

gamma-aminobutyric acid, serotonin, dopamine, and melatonin, which are responsible for regulating the sleep-wake cycle [9]. Clinical and experimental evidence suggests reductions in stress-related hormones such as cortisol, corticosterone, CRH, and ACTH, indicating attenuation of hyperarousal states commonly observed in insomnia. This is particularly relevant given the strong association between chronic stress, HPA axis dysregulation, and sleep disturbances [23,24].

#### **4.5. Oxidative stress.**

Ashwagandha demonstrates antioxidant and neuroprotective properties, reducing oxidative stress and enhancing endogenous antioxidant defenses. These effects may be especially important in the context of sleep deprivation, where increased oxidative stress contributes to cognitive impairment and neuronal dysfunction. Preclinical studies also suggest improvements in neurotransmitter balance, including serotonin and dopamine modulation, as well as enhancement of neuronal plasticity, which may further support cognitive and emotional stability during sleep disruption. Chronic inflammation and oxidative stress are currently recognized as factors that may contribute to the pathogenesis of sleep disorders. Withanolides present in ashwagandha may reduce the production of proinflammatory cytokines and limit cellular damage induced by free radicals [14].

#### **4.6. Elderly population.**

Clinical evidence, including randomized controlled trials and meta-analyses, supports these mechanistic findings by demonstrating improvements in subjective sleep quality, sleep onset latency, and psychological well-being in both general and elderly populations. Insomnia is highly prevalent in the elderly population and represents one of the most common sleep disorders in older adults. Its frequency increases with age due to age-related changes in sleep architecture, comorbid medical and psychiatric conditions, polypharmacy, and dysregulation of neuroendocrine and circadian systems. As a result, sleep disturbances in this group are often chronic and associated with reduced quality of life, impaired cognitive function, and increased risk of physical and mental health complications [30]. In this context, Ashwagandha may be considered a promising adjunctive approach in the management of insomnia in older adults, given its potential to modulate several neurobiological pathways involved in sleep regulation.

#### **4.7. Dose-Dependent Management.**

Notably, benefits appear to be dose- and duration-dependent, with higher doses ( $\geq 600$  mg/day) and longer interventions ( $\geq 8$  weeks) associated with more pronounced effects. Importantly, most studies report a favorable safety profile, with no serious adverse events, although long-term data remain limited [21].

#### **4.8. The Co-Administration of L-Tryptophan and Ashwagandha Root Extract.**

When exploring natural alternatives for the management of sleep disturbances, contemporary research increasingly focuses on multi-ingredient formulations. While monotherapies targeting a single physiological pathway are common, exploring the co-administration of various natural compounds with distinct mechanisms of action represents a highly promising avenue of clinical research. Investigating the potential synergistic effects between adaptogens, such as Ashwagandha and other sleep-promoting substances, could unlock more holistic therapeutic strategies, simultaneously addressing stress, sleep architecture, and neurotransmitter balance. Among these compounds, L-tryptophan holds a prominent position.

To check this, the next trial checked the influence of Ashwagandha alone (at 250 mg or 600 mg) or a combination of Ashwagandha (250 mg) and tryptophan (175 mg). The investigators originally hypothesized that blending these two evidence-based ingredients would exert a robust synergistic effect, yielding superior therapeutic outcomes compared to Ashwagandha monotherapy [35]. Contrary to the researchers' initial expectations, the co-administration of tryptophan did not produce a statistically significant synergistic or additive effect. This lack of superiority over monotherapy indicates that Ashwagandha extract alone drives the primary sleep-promoting benefits observed in the study, proving highly effective even without additional amino acid support. This finding is particularly valuable for formulation strategies, suggesting that optimizing the dose of Ashwagandha may be clinically more relevant than combining it with sub-gram dietary doses of tryptophan. These insights underscore the potent, independent role of Ashwagandha as a standalone functional ingredient for managing stress-related or primary sleep problems.

#### **4.9. The Co-Administration of Melatonin and Ashwagandha Root Extract.**

In contrast to the findings regarding tryptophan co-administration, recent clinical evidence suggests that combining Ashwagandha with melatonin may indeed offer the hypothesized therapeutic synergy. This was clearly demonstrated in the study, which investigated the comparative and collective efficacy of Ashwagandha root extract (ARE) and melatonin (MLT) [36]. The interaction between ARE and MLT revealed a true complementary relationship. However, the most compelling finding within this discussion is that the concurrent administration of ARE (600 mg/day) and MLT (3 mg/day) triggered the most robust therapeutic outcomes. In conclusion, these findings establish that while standalone supplementation with either Ashwagandha or melatonin effectively mitigates sleep problems, their combination provides a superior, highly consistent, and dual-action therapeutic response by simultaneously addressing chronobiological and stress-induced pathogenic mechanisms. Consequently, this multi-targeted, non-pharmacological regimen represents a compelling alternative for adults suffering from primary or stress-related sleep disorders, successfully optimizing both objective sleep architecture and subjective quality of life. Nonetheless, subsequent large-scale clinical trials with extended longitudinal follow-ups are warranted to further substantiate these outcomes and guide their integration into mainstream clinical practice.

#### **4.10. Limitations.**

Despite these promising findings, several limitations should be acknowledged. The available clinical evidence is characterized by heterogeneity in study design, extract standardization, dosing regimens, and outcome measures, which limits direct comparability between studies. However, the strength of the evidence is limited by heterogeneity across studies, including variability in study design, populations, extract standardization, dosage, and treatment duration. A key limitation of this review is the heterogeneity of the included evidence. While the primary focus was on clinical studies in humans, part of the available literature also includes preclinical experimental studies conducted in animal models, particularly rodents. This introduces variability in study design, outcomes, and translational relevance, as findings from animal studies may not be directly generalizable to human populations. Consequently, the mixed nature of the evidence base may reduce the overall comparability and strength of the conclusions regarding the effects of *Withania somnifera* on insomnia and sleep-related outcomes. Additionally, many trials rely primarily on subjective sleep assessments, with fewer incorporating objective measures such as polysomnography or actigraphy. Further large-scale, long-term randomized controlled trials are needed to confirm efficacy, establish optimal dosing strategies, and better define the safety profile of ashwagandha in the management of insomnia.

### **5. Conclusions**

The available clinical evidence suggests that Ashwagandha is a promising adjunctive intervention for improving sleep-related outcomes, particularly in individuals with insomnia or stress-related sleep disturbances. Across randomized controlled trials and meta-analyses, supplementation has been associated with improvements in sleep quality, sleep onset latency, and overall subjective sleep parameters, but also reductions in anxiety and perceived stress. In conclusion, *Withania somnifera* appears to be a generally safe and potentially effective herbal intervention for stress-related insomnia, but further high-quality, large-scale randomized controlled trials are required to confirm its efficacy, optimize dosing strategies, and better define its role in the management of insomnia.

**Conflicts of Interest:** No conflicts of interest to declare.

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