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The Current State of Knowledge on Magnesium's Influence on Sleep Health: A Narrative Review

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Abstract

Background: Sleep is a fundamental biological process essential for physical and mental health. Adequate sleep supports metabolic balance, immune function, cognitive performance, and emotional regulation. Chronic sleep insufficiency, however, is a major health burden. There is accumulating evidence to suggest that low magnesium intake and subclinical magnesium deficiency may be associated with poor sleep quality and an increased risk of insomnia.

Aim: The aim of this study was to analyze the mechanisms of magnesium action in the body that may influence our sleep health and summarise the available scientific literature on the subject.

Materials and methods: A narrative review was performed using PubMed and Google Scholar databases. In total, 29 publications were included, focusing on systematic reviews, meta-analyses, and randomized controlled trials.

Results: The reviewed studies show an association between higher magnesium levels and better sleep efficiency. Clinical trials reported modest improvements in sleep quality among groups that either supplemented or had higher dietary intake of Mg compared to placebo. The limitations of most presented trials include relatively small populations and short durations.

Conclusions: The reviewed evidence indicates that low magnesium status is associated with fragmented or nonrestorative sleep, while magnesium supplementation may provide modest improvements in sleep outcomes. Future work should clarify optimal dosages and formulations and identify which populations (e.g. older adults, individuals experiencing stress, or those with inflammatory conditions) benefit the most from Mg supplementation.

Keywords: sleep; insomnia; sleep quality; circadian rhythm; magnesium; Mg; magnesium supplementation

1. Introduction

One-third of an average person's life is spent asleep. Sleep quality is a crucial factor for maintaining optimal physical and mental health. Night rest allows the brain to consolidate memories, process information, repair neural connections, and eliminate waste [1]. It plays an essential role in metabolic homeostasis, immune function, cognitive performance, and emotional regulation. Insufficient sleep is associated with an increased risk of various medical conditions, such as diabetes, obesity, hypertension, cardiovascular diseases, depression, reduced cognitive function, and negative mood [1-3,7].

The most common sleep disorder is insomnia, which may include difficulty falling asleep, early wakening, or feeling unrefreshed on waking. The prevalence of insomnia in all population groups is between 10% and 48% and increases with age. It is especially problematic for older patients because they are at a higher risk of falling, have weaker physical function, cognitive impairment, and a higher mortality rate [4]. Since sleep deprivation is associated with a higher mortality, it is considered a heavy burden on our healthcare and socioeconomic systems [5-7]. Given the impact of insomnia on health, it is particularly important to identify modifiable factors that influence sleep quality, in order to prevent physical and mental health disorders [8,13].

In recent years, the role of nutritional factors and micronutrients in regulating sleep has received increasing attention. Research suggests that the quality of our sleep is significantly affected by the composition of our diet, in terms of both macronutrients and micronutrients [1,5,9]. Among these, magnesium (Mg) has emerged as a potentially important modulator of sleep physiology. Magnesium is an essential micronutrient involved in more than 300 enzymatic reactions, including those related to energy metabolism, protein synthesis, and neuronal function [4,5,19-21]. It plays a critical role in sleep regulation, mostly due to its action as an antagonist of N-Methyl-D-Aspartic Acid (NMDA) and an agonist of γ -Aminobutyric Acid (GABA) [4,10]. By binding to GABA receptors, Mg activates GABA, thereby reducing neuronal excitability [3]. In contrast, by inhibiting the NMDA receptors, it suppresses the intracellular calcium (Ca) concentration in muscle cells, promoting muscle relaxation [11]. Magnesium has also been implicated in increasing serum levels of renin and melatonin, while decreasing cortisol levels, both of which have a positive influence on restful sleep [4,11,14].

There is accumulating evidence to suggest that low magnesium intake and subclinical magnesium deficiency may be associated with poor sleep quality and an increased risk of

insomnia. Several clinical studies have investigated the effects of magnesium supplementation on sleep parameters, with some demonstrating improvements in sleep efficiency, sleep time, and subjective sleep quality, particularly in older adults [4,22-28]. However, the available evidence remains heterogeneous, and the underlying mechanisms are not yet fully understood. This work examines the relationship between magnesium and sleep, focusing particularly on neurobiological mechanisms and clinical outcomes.

2. Methods

The aim of this narrative review is to summarise current evidence on the role of magnesium in sleep regulation, emphasizing the most recent and relevant studies published in the field. A literature search was performed using electronic databases, including PubMed and Google Scholar, and it was limited to articles published in English. Publications from 2012 to 2026 were considered, focusing on the latest evidence from 2023-2026. Search terms included combinations of the keywords: “sleep”, “insomnia”, “magnesium”, “Mg”, “sleep quality”, “circadian rhythm” and “magnesium supplementation”. Eligible studies included systematic reviews, meta-analyses, and clinical studies, particularly randomized controlled trials (RCT). Articles were selected based on their relevance to the topic, methodological quality and contribution to understanding the relationship between magnesium and sleep.

3. Research results

3.1 Physiology of sleep

Sleep is a highly dynamic and regulated neurobiological process involving coordinated activity across multiple brain regions, neurotransmitter systems, and hormonal pathways [1-3]. For maintaining optimal health and daytime performance, it is recommended to achieve a minimum of 7 hours of total sleep per night, with a sleep efficiency of 85% [3,12]. Sleep efficiency is defined as the percentage of time spent asleep while in bed [9].

Normal sleep is composed of two main states: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. NREM sleep is further divided into three stages (N1, N2, and N3), with N3 representing slow-wave sleep (SWS), which is considered the deepest and most restorative phase [13]. REM sleep, characterized by rapid eye movements, vivid

dreaming, and muscle atonia, plays a crucial role in emotional regulation and memory consolidation. Sleep occurs in cycles lasting approximately 90-110 minutes, alternating between NREM and REM stages. Proper sleep architecture depends on the stability of these cycles and the balance between restorative slow-wave sleep and REM sleep [3,17].

Sleep is regulated by the interaction of two principal regulatory processes: the circadian rhythm and the homeostatic process [3]. The circadian rhythm is governed by the suprachiasmatic nucleus (SCN) of the hypothalamus, which synchronizes sleep-wake cycles with environmental light-dark cues. The circadian system regulates the timing of sleep propensity through hormonal signals, most notably melatonin, which is secreted by the pineal gland in response to darkness and helps to initiate sleep [3,14]. Meanwhile, the homeostatic process reflects the accumulation of sleep pressure during wakeful hours, which increases gradually with time [3]. Disruption of these systems may lead to impaired sleep architecture and contribute to the development of sleep disorders.

Sleep and wakefulness are regulated by a complex balance between excitatory and inhibitory neurotransmitter systems. Wakefulness is maintained by a network of neurons releasing neurotransmitters like norepinephrine, serotonin, acetylcholine, histamine, and orexin. These systems promote cortical activation and behavioral arousal. In contrast, sleep initiation and maintenance depend on inhibitory signaling, including GABA, adenosine, and nitric oxide [12,15]. Equally important is the regulation of excitatory neurotransmission, mediated by glutamate, particularly through NMDA receptors. It stimulates orexinergic neurons, promoting wakefulness, and inhibits both NREM and REM sleep stages [12]. The balance between GABAergic inhibition and glutamatergic excitation plays a central role in maintaining sleep stability. Disruption of this balance has been implicated in insomnia and other sleep disorders [3,8,15].

Sleep is closely linked to neuroendocrine function, particularly the hypothalamic-pituitary-adrenal (HPA) axis [3,16]. Under normal conditions, cortisol follows a circadian rhythm, decreasing in the evening and rising before awakening. Elevated nocturnal cortisol levels are associated with impaired sleep initiation and sleep fragmentation [16]. Melatonin, secreted by the pineal gland, is a key regulator of circadian rhythms and sleep onset. Its secretion is influenced by both environmental light exposure and internal neurochemical pathways [14]. Chronic activation of the HPA axis and increased sympathetic activity contribute to hyperarousal and are commonly observed in insomnia.

Emerging evidence suggests that inflammation and oxidative stress play a role in sleep regulation. Elevated levels of pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor- α , have been associated with poor sleep quality and altered sleep architecture [3,17]. These mediators may affect both central nervous system function and peripheral physiological processes involved in sleep regulation.

Additionally, increased neuromuscular excitability, as observed in conditions such as restless legs syndrome or nocturnal muscle cramps, can disrupt sleep continuity [18]. Regulation of muscle tone and peripheral nervous system activity is, therefore, an important component of sleep physiology.

These mechanisms are highly interdependent and collectively determine sleep quality, latency, and architecture. Importantly, many of these pathways have been identified as targets of magnesium action [12]. Understanding these physiological processes provides a necessary framework for interpreting how magnesium may influence sleep at both molecular and systemic levels.

3.2 Magnesium metabolism

Magnesium is the fourth most abundant cation in the human body, and the second most abundant intracellular cation [21]. Approximately 50-60% of total body magnesium is stored in bones, while the remainder is distributed in soft tissues, particularly in muscles and the central nervous system. Less than 1% is present in extracellular fluid [20]. Mg homeostasis is tightly regulated through intestinal absorption, renal excretion, and exchange with bone stores. The kidneys play a central role in maintaining magnesium balance by adjusting urinary excretion according to physiological need [19,21]. The bioavailability of magnesium depends on several factors, including dietary composition, gastrointestinal function, and the chemical form of magnesium [20,21].

At the cellular level, magnesium is essential for energy metabolism and enzymatic activity. It forms complexes with ATP, enabling efficient energy transfer, stabilizes cell membranes, regulates ion channels, modulates calcium influx, and participates in protein and nucleic acid synthesis [20,21]. Importantly, magnesium keeps ionic balance through interactions with calcium, potassium, and sodium, which is essential for multiple processes that are directly relevant to sleep regulation [12]. At the systemic level, Mg contributes to physiological processes such as neuromuscular transmission, cardiovascular function, immune regulation,

and metabolic homeostasis. This makes magnesium a modifiable factor that may also influence many chronic diseases [20].

3.3 Magnesium mechanisms in sleep

In October 2025, He et al. published a review summarising the latest findings on the mechanisms of magnesium's action on sleep health [12]. Based on this review, and after our own analysis of the sources, we would like to outline the mechanisms discussed in the research, and then refer to new studies on the topic.

Magnesium may influence sleep through several converging biological pathways, rather than through a single, dominant mechanism. Recent PubMed-indexed literature suggests that its relevance to sleep stems from its broad role in neuronal signaling, intracellular energy metabolism, circadian regulation, endocrine function, autonomic balance, and inflammatory control. Accordingly, magnesium should be viewed not as a classical hypnotic agent, but as a physiological modulator of processes that shape sleep initiation, sleep continuity, and daytime restoration. [4,5,10-12,19-21]

Magnesium binds to GABA receptors and enhances GABAergic activity by acting as a positive modulator of GABA_A receptors, thereby promoting neuronal inhibition and facilitating sleep initiation. Increased GABAergic tone is associated with reduced sleep latency and improved sleep continuity. Conversely, magnesium deficiency may impair inhibitory neurotransmission, leading to heightened neuronal excitability and difficulty initiating sleep. At the same time, Mg acts as a blocker of NMDA-type glutamate channels. At normal resting membrane potentials, Mg²⁺ ions sit in the NMDA receptor channel pore and prevent calcium (Ca²⁺) influx. This voltage-dependent block is relieved only upon strong depolarization. By occupying the NMDA channel, Mg²⁺ reduces glutamate-mediated excitatory transmission. In muscle cells, this NMDA blockade suppresses intracellular Ca²⁺ buildup, promoting muscle relaxation and lower body temperature, both conducive to sleep onset. When magnesium is deficient, unblocked NMDA channels lead to heightened neuronal firing and elevated neuromuscular excitability, which can disrupt sleep. The dual action of magnesium as an NMDA antagonist and a GABA agonist creates a synergistic sleep-promoting effect. [7,10-12,29]

Magnesium maintains the electrochemical stability of neurons by modulating ion fluxes, particularly of calcium and potassium, and by stabilizing membrane potentials. Mg serves as a natural Ca antagonist, limiting excitatory signaling and preventing excessive Ca²⁺ influx in

neurons. Conversely, Mg deficiency leads to unchecked Ca^{2+} entry and neuronal overactivity, which can manifest as insomnia or restless muscle contractions. Magnesium also supports the Na^+/K^+ -ATPase pump and K^+ channel conductance, stabilizing neuronal membrane potential. Notably, clinical data show that poor sleepers often have low serum Mg and Ca, and that the calcium-to-magnesium ratio can influence sleep duration. [11-12,20]

Low Mg is linked to chronic inflammation and oxidative stress. Deficiency elevates reactive oxygen species (ROS) and pro-inflammatory cytokines (e.g. IL-6, TNF- α). Elevated C-reactive protein (CRP), a marker of inflammation, is correlated with both low dietary magnesium and poor sleep. Chronic inflammation itself disrupts sleep, and conversely, poor sleep can increase inflammatory markers. Supplementing magnesium can reduce these markers and oxidative damage, which might indirectly promote deeper, more restorative sleep. [10-12,17]

Magnesium affects several sleep-related hormones and neuromodulators. It is involved in melatonin synthesis through serotonin pathways. Because melatonin is the main hormone signaling darkness and initiating sleep, suboptimal Mg could blunt this sleep-triggering signal. Magnesium also influences serotonin conversion to melatonin by enhancing serotonin-N-acetyltransferase activity. Separately, Mg supplementation has been shown to lower serum cortisol and calm the HPA axis. Since elevated cortisol disrupts sleep onset and continuity, the ability of Mg to reduce cortisol entry into the brain likely contributes to improved sleep quality. Thus, magnesium's relaxation of the central nervous system may stem both from neurotransmitter effects and from dampening stress hormone signals. [10-12,14]

Emerging research suggests Mg also participates in circadian regulation. Intracellular free Mg^{2+} levels cycle in a roughly 24-hour pattern in cells, helping to coordinate cellular energy balance with day-night cycles. Fluctuations in intracellular Mg are thought to influence ATP-dependent processes across the day. Although human data are limited, this indicates that systemic Mg levels could reinforce the body's biological clock and thus stabilize sleep timing. [12,24]

3.4 Recent findings

In the past few years, several studies have examined magnesium's impact on sleep outcomes. These include randomized trials and observational analyses, which collectively reinforce the link between magnesium status and sleep quality.

Several RCTs report sleep benefits of magnesium supplements:

Breus et al. (2024, USA) conducted a double-blind crossover pilot (N=31, *M* age = 46.01) comparing 1 g/day of Magnesium Condition vs. Placebo Condition for 2 weeks. The Magnesium Condition significantly improved sleep outcomes: deep sleep, sleep duration, sleep efficiency, activity balance, and heart rate variability readiness (all $p < 0.05$). In mood and stress measures there were nonsignificant improvements ($p > 0.05$). Adherence was 100%, and no adverse events were noted. Limitations: small sample, short duration, and nonclinical insomnia subjects (pilot study). [22]

Hausenblas et al. (2024, USA) randomized 80 adults (age 35-55) to 1 g/day of magnesium L-threonate (MgT) or placebo for 21 days. The MgT group maintained better sleep quality: objective Oura ring measures showed significantly increased deep sleep score, REM sleep score, light sleep time, and activity and readiness parameters ($p < 0.05$), whereas the placebo group declined. Subjectively, MgT participants reported improved behavior upon awakening, energy and daytime productivity, grouchiness, mood, and mental alertness (all $p < 0.05$). This trial's strengths include objective sleep monitoring. Limitations are the short intervention (3 weeks) and specific Mg form. [27]

Schuster et al. (2025, Germany) performed a larger RCT (N=155, 18-65 years) with 250 mg/day magnesium bisglycinate supplementation vs. placebo for 4 weeks. Insomnia Severity Index (ISI) decreased significantly more in the Mg group: mean ISI change was -3.9 vs. -2.3 in placebo ($p = 0.049$). The effect size was small (Cohen's $d = 0.2$), indicating a modest clinical benefit. Exploratory analyses suggested that participants with lower baseline dietary Mg intake may have experienced larger improvements. Other sleep measures showed nonsignificant trends favoring Mg. Strengths include the relatively large sample and dietary intake assessment. Limitations: effect was modest, largely subjective sleep scales, and short follow-up. [23]

Large cohort analyses consistently link higher Mg to better sleep metrics:

Alsheikh et al. (2025, USA) analyzed NHANES (2005–2016, N=10,486) to study Circadian Syndrome (CircS), which includes metabolic syndrome (MetS) plus circadian rhythm disturbances (short sleep) and depression. They found that each increasing quartile of dietary Mg intake was associated with lower odds of Circadian Syndrome. In the fully adjusted model, adults in the highest Mg quartile had up to 35.2% reduced odds of circadian syndrome compared to the lowest (p for trend < 0.001). This suggests dietary magnesium intake supports circadian health. Limitations: cross-sectional design prevents causal inference, and sleep measures were self-reported. [24]

In Saudi populations, two large surveys examined Mg and sleep. Al-Musharaf et al. (2026, Saudi Arabia) conducted a cross-sectional study of 1041 adults. Using biochemical serum Mg and dietary intake data, they found serum Mg deficiency (<1.8 mg/dL) was associated with a 30-80% increase in odds of poor sleep quality (PSQI) in crude and adjusted models. Dietary Mg below the recommended allowance also independently predicted poor sleep. These associations were stronger in men. Limitations: cross-sectional, use of self-reported sleep index, and regional homogeneity limit generalizability. [25]

Alshammari et al. (2025, Saudi Arabia) surveyed 305 university students. Higher monthly dietary Mg intake (assessed by magnesium food frequency questionnaire - MgFFQ) was significantly associated with better sleep: students with greater Mg intake reported significantly longer sleep duration and less daytime dysfunction ($p < 0.01$ for both). Overall PSQI scores were lower (better) in the high-Mg group. Females had worse sleep quality than males, highlighting potential sex differences. Limitations: young healthy sample, self-report measures, and relatively small cohort. [26]

All these studies are summarised in **Table 1** below. Despite variations in design, these recent studies collectively suggest that magnesium, whether measured as dietary intake or supplemented, tends to correlate with improved both sleep architecture (more deep/REM sleep) and sleep-related symptoms. Importantly, these trials used different forms of magnesium (bisglycinate, threonate, etc.), which vary in bioavailability, yet all reported some positive effect. However, note that RCT effect sizes were generally moderate, and many studies relied on subjective sleep scales. Risk of bias varies: pilot trials and cross-sectional surveys carry higher bias than large RCTs. Sample sizes ranged from ~30 to >10,000; the smallest studies ($N \sim 30-80$) are underpowered, while large cohorts offer statistical power but lower causal certainty. Most trials lasted only two to four weeks, leaving long-term efficacy untested.

Table 1. Representative studies on magnesium status and sleep metrics.

Study (year)	Design & Population	Magnesium Dose/Measurement	Key Findings	Citation
Breus <i>et al.</i> (2024)	RCT, crossover (N=31 adults)	1 g/day Magnesium Condition vs Placebo Condition (2 weeks)	Mg improved sleep metrics: $\uparrow$ total sleep time, $\uparrow$ deep sleep %,	[22]

Study (year)	Design & Population	Magnesium Dose/Measurement	Key Findings	Citation
	with poor sleep)		↑efficiency; improved mood/activity measures ($p < 0.05$).	
Schuster <i>et al.</i> (2025)	RCT, parallel (N=155 healthy adults)	250 mg/day Magnesium bisglycinate vs placebo (4 weeks)	Small but significant reduction in Insomnia Severity Index (ISI): −3.9 vs −2.3 ($p =$ 0.049). Effect size $d=0.2$ (modest clinical benefit).	[23]
Hausenblas <i>et al.</i> (2024)	RCT, parallel (N=80 adults, ages 35–55)	1 g/day Magnesium L- threonate vs placebo (21 days)	MgT group maintained better sleep: significantly higher deep and REM sleep (Oura ring); improved awakening alertness, energy, mood ($p <$ 0.05).	[27]
Alsheikh <i>et al.</i> (2025)	Cross- sectional (NHANES, N=10,486 US adults)	Dietary Magnesium (24h recall) quartiles	Higher Mg intake linked to lower “Circadian Syndrome” prevalence: highest quartile had 35.2% lower odds of CircS vs lowest ($p < 0.001$), after multivariable adjustment.	[24]
Al-Musharaf <i>et al.</i> (2026)	Cross- sectional	Serum Magnesium (<1.8 mg/dL	Serum Mg deficiency increased odds of poor	[25]

Study (year)	Design & Population	Magnesium Dose/Measurement	Key Findings	Citation
	(N=1041 Saudi adults)	deficiency) and dietary Magnesium	sleep by >30%; dietary Mg below RDA independently predicted poor sleep after full adjustment ($p < 0.01$).	
Alshammari <i>et al.</i> (2025)	Cross- sectional (N=305 Saudi students)	Dietary Magnesium (MgFFQ)	Higher Mg intake associated with better sleep: longer duration and less daytime dysfunction ($p \leq$ 0.009). Females had poorer sleep vs males, highlighting a sex disparity in Mg–sleep link.	[26]

4. Discussion

This review provides a synthesis of current evidence regarding the role of magnesium in sleep regulation, integrating mechanistic insights with recent clinical and observational studies. Overall, the available data suggest that Mg exerts a multimodal and context-dependent effect on sleep, primarily through modulation of neurobiological pathways rather than a single, direct hypnotic action [12,28].

Magnesium influences several core systems involved in sleep physiology, including GABAergic and glutamatergic neurotransmission, circadian rhythm regulation, neuroendocrine stress response, and inflammatory signaling [4,10-12,19-21]. The most central mechanism appears to be its influence on GABA and NMDA receptors. Mg directly promotes neural conditions favorable for sleep induction and continuity by enhancing inhibitory neurotransmission and reducing excitatory signaling [10,29]. Moreover, magnesium facilitates melatonin production and interacts with circadian clock mechanism, aligning sleep timing

[4,10-12,24]. It also mitigates the stress response and inflammation, two major non-neuronal factors that disrupt sleep [10-12,24].

This is further supported by both preclinical evidence and positive outcomes in supplementation trials [4,12,22-27]. Meta-analyses and systematic reviews consistently show that Mg supplementation produces modest improvements in sleep parameters, such as sleep latency and total sleep time [5,9-12,28]. However, clinical evidence remains moderate and heterogeneous, often limited to specific populations or small sample sizes [4,11,22-27]. Doses, forms, and durations used in trials vary widely, making it difficult to determine optimal regimens. The relative contribution of co-supplemented nutrients, like melatonin or zinc, in some trials complicates the isolation of magnesium's role [24,25]. Also, evidence supporting Mg use in severe or chronic insomnia remains limited, which suggests that it may function more effectively as a supportive or adjunctive intervention, rather than a primary treatment modality. Given the limitations of included trials it occurs that magnesium cannot be perceived as a standalone treatment of insomnia but rather as a part of multimodal approach.

5. Conclusions

Reviewed evidence indicates that low magnesium status is associated with fragmented or nonrestorative sleep [9-12]. Recent large trials and cohort studies (2023–2026) generally support the view that addressing suboptimal magnesium intake may benefit sleep, especially in individuals with insomnia symptoms or low baseline Mg. However, the effect sizes are moderate, and not everyone responds equally [4,11,22-27].

Given the limited results of published trials, future studies should aim to create well-designed RCTs that include larger and more diverse groups of participants. The goal is to clarify optimal dosages and formulations of supplemented magnesium, as well as to identify subpopulations that benefit the most from supplementation. In the meantime, ensuring adequate dietary magnesium through foods such as green leafy vegetables, nuts, legumes, and whole grains remains a simple, low-risk strategy that may improve sleep health as part of a holistic approach to wellness [5,9,24].

Disclosure

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