Nutritional Support for Sleep Quality and Anxiety Management

The Role of Magnesium, Taurine, and L-Theanine

With Considerations for Circadian Function, Sleep Architecture, and Recovery

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1. Introduction

Sleep quality and anxiety are deeply interconnected aspects of health. Poor sleep increases vulnerability to anxiety, while anxiety disrupts sleep onset and continuity, creating a cycle that can be difficult to break. Conventional pharmacological interventions, while effective in the short term, often carry risks of dependency, tolerance, and side effects that make them unsuitable for long-term use.

A growing body of evidence supports the use of specific nutritional compounds -- magnesium, taurine, and L-theanine -- as safe, well-tolerated interventions that target the neurobiological mechanisms underlying both sleep disturbance and anxiety. These compounds act through complementary pathways involving GABAergic neurotransmission, NMDA receptor modulation, and cortisol regulation, offering a multi-target approach that addresses the shared neurobiology of these conditions.

This report examines the current evidence for each compound, their mechanisms of action, practical dosing considerations, and the critical role of circadian function in sleep quality and mental health.

2. Magnesium

2.1 Overview and Prevalence of Deficiency

Magnesium is a cofactor in over 300 enzymatic reactions and plays an essential role in neuromuscular regulation, nervous system function, and energy metabolism. Despite its importance, subclinical magnesium deficiency is widespread, with estimates suggesting that 50-80% of the population in Western countries may have inadequate intake. This is particularly relevant because magnesium status is poorly reflected by standard serum blood tests, which measure only approximately 1% of total body magnesium. The remainder is stored in bone and soft tissue and used almost immediately upon absorption.

Athletes and physically active individuals face a compounded risk. Exercise increases magnesium loss primarily through elevated urinary excretion (20-30% above baseline), not through sweat as commonly assumed. The primary route of magnesium loss during exercise is renal, not dermal. Stress, whether physical or psychological, further depletes magnesium stores through HPA axis activation and increased cortisol output. Individuals managing high-stress occupations alongside high training volumes are therefore at particular risk of cumulative depletion.

The current RDA of approximately 410mg for adult males is based on data from the 1970s and assumes average activity levels and average occupational stress. For individuals training at high volumes while managing professional and personal demands, this figure is almost certainly insufficient. Total daily magnesium storage capacity is approximately 2 grams in the average male (compared with approximately 92 grams of calcium), meaning the body has very limited reserves and requires consistent daily intake.

2.2 Mechanisms of Action for Sleep and Anxiety

NMDA Receptor Antagonism

Magnesium ions function as natural voltage-dependent blockers of the NMDA receptor, a subtype of glutamate receptor. Under resting conditions, magnesium occupies the receptor pore and prevents excessive calcium influx. This is significant because glutamate is the brain's primary excitatory neurotransmitter, and overactivation of the NMDA receptor is implicated in neuronal hyperexcitability, anxiety, and insomnia. Oral magnesium supplementation has been shown to antagonise NMDA-mediated responses in a dose-dependent manner comparable to the channel blocker MK-801 (Decollogne et al., 1997). Magnesium deficiency, conversely, upregulates NMDA receptor expression and increases hypothalamic-pituitary-adrenal (HPA) axis activation, promoting anxiety-like behaviour in animal models (Sartori et al., 2012).

GABAergic Enhancement

Magnesium also exhibits agonist activity at GABA-A receptors, the primary inhibitory receptor system in the central nervous system. Research has demonstrated that magnesium's anxiolytic effects are partly mediated through benzodiazepine/GABA-A receptor pathways, with its calming effects being antagonised by the benzodiazepine receptor blocker flumazenil and synergistically enhanced by co-administration with benzodiazepines at sub-threshold doses (Poleszak, 2008). This dual mechanism -- reducing

excitatory glutamate signalling while enhancing inhibitory GABA activity -- makes magnesium a particularly effective compound for addressing both anxiety and sleep disturbance.

Cortisol and HPA Axis Regulation

Magnesium deficiency activates the HPA axis, leading to elevated cortisol secretion. Since cortisol is inherently arousing and its suppression is required for sleep onset, magnesium's ability to attenuate HPA axis hyperactivity directly supports both anxiety reduction and improved sleep initiation. Clinical trials have demonstrated that magnesium supplementation reduces cortisol levels and improves heart rate variability readiness, a marker of autonomic nervous system balance (Breus et al., 2024).

2.3 Clinical Evidence

A 2024 systematic review by Rawji et al., published in Cureus, examined the clinical evidence for magnesium supplementation in anxiety and sleep. The review found that while preclinical evidence for magnesium's role in both conditions is robust and mechanistically consistent, clinical trial data in humans remains limited in scale, with studies to date including relatively small sample sizes.

A randomised double-blind placebo-controlled crossover trial by Breus et al. (2024) found that magnesium supplementation (1g/day) produced significant improvements compared to placebo for sleep quality, mood, and activity outcomes, including sleep duration, deep sleep percentage, sleep efficiency, and HRV readiness. These effects were measured objectively using Oura Ring data as well as validated self-report questionnaires.

The largest placebo-controlled trial on magnesium and sleep to date (2025) examined magnesium bisglycinate supplementation in healthy adults reporting poor sleep. It found a statistically significant reduction in Insomnia Severity Index scores, with most improvements occurring within the first 14 days and being sustained thereafter. The study also confirmed excellent tolerability, with side effects occurring less frequently in the magnesium group than in the placebo group.

A meta-analysis by Moabedi et al. (2023) examining magnesium supplementation in adults with depressive disorder found a significant reduction in depression scores (SMD: -0.919, $p = 0.001$), further supporting magnesium's role in mood regulation.

2.4 Dosing and Form Considerations

For sleep and anxiety support, an evening dose of 200-400mg of elemental magnesium, taken 1-2 hours before bed, is well-supported by the literature. Total daily intake for active individuals may range from 400-600mg supplemental, divided across the day. Some individuals under acute stress may benefit from higher intakes (up to 900mg supplemental) for a limited period, though it is advisable to start at 300mg and increase gradually.

Form selection matters considerably:

Magnesium citrate offers 100% bioavailability, is inexpensive, and is well-tolerated gastrointestinally. This is the recommended default for most individuals.

Magnesium glycinate (bisglycinate) offers high bioavailability and the glycine component itself has calming properties. However, individuals with ADHD should exercise caution. Single nucleotide polymorphisms (SNPs) on the GAD1 gene, which are more prevalent in ADHD populations, can alter glycine metabolism such that it becomes glutamatergic rather than GABAergic. In these individuals, glycinate may paradoxically worsen insomnia and anxiety rather than improve it.

Magnesium taurinate combines magnesium with taurine and is an excellent alternative for individuals who cannot tolerate glycinate, providing the calming benefits of taurine alongside magnesium absorption.

Magnesium oxide should be avoided for therapeutic purposes. Its bioavailability is approximately 4-16%, meaning that a 375mg tablet may deliver as little as 15-60mg of usable magnesium. This is the form most commonly found in cheap high-street supplements and is largely ineffective for addressing deficiency or supporting neurological function.

3. Taurine

3.1 Overview

Taurine is a conditionally essential, sulphur-containing amino acid found in high concentrations in the brain, heart, and skeletal muscle. Unlike most amino acids, taurine is not incorporated into proteins but instead functions as a free intracellular compound with diverse physiological roles, including osmoregulation, membrane stabilisation, antioxidant protection, calcium handling, and critically, neuromodulation.

3.2 Mechanisms of Action for Sleep and Anxiety

GABA-A Receptor Activation in the Thalamus

The landmark discovery by Jia et al. (2008), published in the Journal of Neuroscience, identified that taurine is a potent activator of extrasynaptic GABA-A receptors in the thalamus. The thalamus functions as the brain's sensory relay centre, controlling transitions between sleep and wakefulness. During sleep, the thalamus discharges slowly and isolates the cortex from sensory input; during wakefulness, it permits sensory information to activate the cortex.

The researchers found that physiological concentrations of taurine (as low as 10-50 micromolar) were sufficient to reduce neuronal excitability in thalamocortical relay neurons, effectively supporting the transition from wakefulness to sleep. The lead researcher noted that taurine's neurological effect is more sedative than excitatory, and that it may contribute to the post-consumption 'crash' reported after energy drink consumption where taurine is combined with caffeine.

Neuroprotective and Antidepressant Effects

A 2022 study published in Cellular and Molecular Neurobiology demonstrated that taurine supplementation exerted antidepressive effects by protecting cortical neurons from dendritic spine loss and synaptic protein deficits in a chronic social defeat stress model. Reduced taurine levels have also been reported in populations of patients with anxiety (Strasser et al., 2019), suggesting that supplementation may help restore a neurochemical deficit rather than simply adding an exogenous compound.

Taurine's relationship with GABA receptors extends beyond simple receptor activation. Its affinity for GABA-A receptors varies depending on receptor subunit composition, meaning it has nuanced modulatory effects across different brain regions. This subunit-dependent interaction offers significant potential for targeted therapeutic benefit without the blunt sedation associated with pharmaceutical GABA agonists.

Melatonin Synthesis

Taurine is involved in melatonin synthesis in the pineal gland, providing an additional pathway through which it supports healthy sleep onset. This makes taurine particularly relevant as an evening supplement, supporting both the GABAergic and hormonal components of sleep initiation.

3.3 Performance in Heat

Beyond its sleep and anxiety applications, taurine has demonstrated remarkable effects on endurance performance in hot conditions. A dose-response study found that 4g of taurine (based on approximately 50mg/kg body weight) produced an 11% performance improvement during endurance cycling in hot and humid conditions. This is a substantially larger ergogenic effect than most established sports supplements, including sodium bicarbonate (typically 1-2%) and caffeine (2-5%). For individuals competing in warm-weather events, this represents a significant additional benefit of regular taurine supplementation.

3.4 Dosing

For sleep and anxiety support, 1-2g of taurine taken 60 minutes before bed is a reasonable starting point. For individuals seeking both daytime neuroprotective and evening sleep-supportive benefits, a total daily intake of 4-6g can be split across the day (e.g. 2g morning, 2g afternoon or pre-exercise, 2g evening). The European Food Safety Authority (EFSA) has confirmed the safety of taurine at supplemental levels, and no adverse effects have been reported at doses up to 6g/day in research settings. Taurine is available inexpensively as a bulk powder.

4. L-Theanine

4.1 Overview

L-theanine is a non-proteinogenic amino acid found predominantly in green tea (Camellia sinensis). It is well-established as an anxiolytic compound with proven relaxation benefits and is unique among calming supplements in that it promotes relaxation without sedation, making it suitable for both daytime anxiety management and evening sleep support.

4.2 Mechanisms of Action

GABA and Glutamate Modulation

L-theanine increases the release of GABA, the brain's primary inhibitory neurotransmitter, while simultaneously inhibiting the glutamine transporter, thereby reducing the formation of glutamate from glutamine. This dual action shifts the GABA:glutamate ratio towards inhibition, promoting a state of calm alertness during the day and facilitating sleep onset in the evening.

Alpha Brain Wave Enhancement

L-theanine has been consistently shown to increase alpha brain wave activity, which is associated with a state of relaxed alertness. This neurophysiological effect occurs within 30-60 minutes of ingestion and represents a measurable biomarker of the compound's calming action without cognitive impairment.

Caffeine Synergy

When combined with caffeine, L-theanine produces a state of focused, calm alertness that is greater than either compound alone. L-theanine preserves the cognitive and performance-enhancing effects of caffeine while eliminating the jitteriness, elevated heart rate, and anxiety that caffeine can produce in sensitive individuals. This combination is particularly valuable for individuals with ADHD, where the combination creates a 'tunnel vision' focus effect. For pre-exercise or pre-competition use, this combination can help maintain a controlled heart rate at the start of an event rather than the elevated, anxiety-driven heart rate that stress and adrenaline can produce.

4.3 Clinical Evidence for Sleep

A 2025 systematic review and meta-analysis published in Sleep Medicine Reviews examined 19 randomised controlled trials (N = 897 participants) and found that L-theanine significantly improved subjective sleep onset latency (SMD = 0.15, $p = 0.04$), subjective daytime dysfunction (SMD = 0.33, $p < 0.001$), and overall subjective sleep quality score (SMD = 0.43, $p = 0.03$).

A separate 2025 systematic review of dietary supplementation trials concluded that 200-450mg/day of L-theanine appears to be a safe and effective way to support healthy sleep in adults, based on 13 eligible trials (n = 550).

4.4 Clinical Evidence for Anxiety

A systematic review by Williams et al. (2020) found that L-theanine in its pure form at a daily dose of 200-400mg reduced stress and anxiety in people under acute stress. A 2024 randomised, double-blind, placebo-controlled trial found that 400mg/day of L-theanine for 28 days significantly decreased perceived stress by 17.98%, reduced light sleep duration, improved sleep quality, and enhanced cognitive attention (Moulin et al., 2024). No adverse effects at any dose have been identified to date, making L-theanine one of the safest supplemental interventions available.

4.5 Dosing

For anxiety management, 200-400mg daily is well-supported. For sleep support, 200-400mg taken 30-60 minutes before bed is consistent with the evidence. L-theanine can be taken up to 600mg/day, though there is likely a point of diminishing returns beyond this level. It can be taken alongside magnesium and taurine without negative interactions; the combination may in fact be synergistic given their complementary mechanisms. Available as both capsules (e.g. Viridian brand with lemon balm) and bulk powder.

5. Synergistic Mechanisms

The three compounds target overlapping but distinct neurobiological pathways, creating a complementary profile:

GABA enhancement: All three support GABAergic inhibition through different mechanisms. Magnesium acts as a positive allosteric modulator at GABA-A receptors, taurine is a direct agonist at extrasynaptic GABA-A receptors in the thalamus, and L-theanine increases GABA release. This convergent enhancement through different mechanisms reduces the risk of receptor desensitisation.

Glutamate/NMDA reduction: Magnesium blocks the NMDA receptor pore. L-theanine inhibits glutamine-to-glutamate conversion. Together, these actions restore the GABA:glutamate balance that is disrupted in states of anxiety and hyperarousal. This is particularly relevant for individuals with ADHD, who typically present with elevated glutamate and reduced GABA as a downstream consequence of dopaminergic dysfunction.

HPA axis and cortisol regulation: Magnesium attenuates HPA axis hyperactivity. L-theanine reduces perceived stress and modulates cortisol. Taurine protects cortical neurons from stress-induced damage. This multi-level approach supports both anxiety reduction and the cortisol suppression required for healthy sleep onset.

6. Circadian Function, Sleep, and Recovery

6.1 The Circadian System

Circadian rhythms are internal biological cycles of approximately 24 hours, governed by a master clock in the suprachiasmatic nucleus (SCN) of the hypothalamus. These rhythms regulate the sleep-wake cycle, hormone secretion (particularly melatonin and cortisol), core body temperature, immune function, and metabolic activity. The SCN is synchronised to the external environment primarily by light exposure detected by specialised melanopsin-containing retinal ganglion cells.

When circadian function is robust, the body produces melatonin in the evening to initiate sleep, suppresses cortisol to permit sleep maintenance, regulates body temperature to optimise sleep architecture, and coordinates tissue repair and immune function during overnight recovery. When circadian rhythms are disrupted, all of these processes are compromised.

6.2 Circadian Disruption, Anxiety, and Depression

A bidirectional relationship exists between circadian rhythm disruption and psychiatric symptoms. A comprehensive review published in Translational Psychiatry (Walker et al., 2020) established that disruption of circadian rhythms via jet lag, night-shift work, or exposure to artificial light at night can precipitate or exacerbate affective symptoms in susceptible individuals. Anxiety disorders are among the most common psychiatric conditions associated with circadian disruption.

The mechanisms linking circadian disruption to anxiety and depression include impaired neurotransmitter release (particularly serotonin and GABA), disrupted melatonin and cortisol rhythms, metabolic dysfunction, neuroinflammation, and neural apoptosis. Animal studies have shown that housing rodents in constant light eliminates diurnal rhythms in activity, melatonin, and corticosterone, with concurrent increases in depressive and anxiety-like behaviour. Restoring circadian hormonal rhythms reverses these changes.

A 2024 review in PNAS described sleep-circadian disturbance as a 'transdiagnostic contributor' to psychiatric disorders, noting that insomnia is highly comorbid with virtually all psychiatric conditions and may play a causal role in their development and maintenance.

6.3 Light Exposure

Morning Light

Exposure to bright light upon waking is the single most powerful zeitgeber (time-giver) for the circadian system. Morning sunlight (or a 10,000 lux therapy light in winter months) suppresses melatonin, initiates the cortisol awakening response, and sets the timing of the entire 24-hour hormonal cascade. A minimum of 10-20 minutes of outdoor light exposure within the first hour of waking is recommended. For individuals with ADHD, recent research (2026) suggests that shifting the wake time earlier may improve daytime function, as the groggy period between 5-6am gives way to high alertness by the time work demands begin.

Midday and Sunset Light

Additional outdoor light exposure during midday reinforces the circadian signal. Exposure to the changing light spectrum at sunset provides a secondary timing cue that calibrates the evening transition towards melatonin production. Training outdoors during these windows achieves both exercise and circadian benefit simultaneously.

Evening Light Restriction

Even modest light exposure at night (as low as 3 lux) can suppress melatonin onset and shorten melatonin secretion duration (Gooley et al., 2011). Practical measures include reducing blue light and bright light exposure in the 4-5 hours before bed, using red-spectrum filters on screens, dimming household lighting after sunset, and using a sleep mask if the bedroom cannot be made fully dark. For individuals who coach or communicate with clients via phone in the evening, this is particularly important, as screen use signals 'daytime' to the SCN regardless of the actual hour.

6.4 Meal Timing and Sleep

Avoid Large Meals Before Bed

The digestive system has its own peripheral circadian clocks. Late-night eating, particularly large meals or meals high in protein or fat, elevates core body temperature through the thermic effect of feeding. Sleep onset requires a drop in core body temperature; if internal temperature is elevated, the body cannot initiate this process effectively, regardless of how cool the room is. A cool room with a warm body can paradoxically trigger heat production as the body senses cold skin and attempts to warm itself. The goal is a cool body in a cool room. Avoid large meals 2-3 hours before bed.

Strategic Carbohydrate Intake Before Bed

A small carbohydrate-based snack approximately 1-2 hours before bed can actually improve sleep onset. This is not due to a 'blood sugar crash' (healthy, active individuals do not experience clinically significant reactive hypoglycaemia). Rather, glucose stimulates POMC neurons in the hypothalamus, which reduce cortisol almost instantaneously. Since cortisol must decrease and melatonin must increase for sleep to occur -- and these two hormones are antagonistic -- anything that lowers cortisol in the evening supports melatonin's rise. A bowl of cereal, a couple of pieces of fruit, or similar simple carbohydrate sources are sufficient.

Front-Loading Daily Nutrition

Eating the majority of daily calories earlier in the day and tapering intake towards the evening aligns with circadian rhythms of metabolic function. The thermic effect of feeding is higher in the morning than in the evening due to circadian rhythms of liver metabolism, meaning the same meal produces less heat and is processed more efficiently when consumed earlier. For individuals with ADHD, this front-loaded eating pattern also supports more stable energy and focus throughout the working day.

6.5 Exercise Timing

Exercise is a significant physiological stressor, regardless of how psychologically beneficial it may feel. Even easy runs require increased adrenaline, cortisol, and heart rate. Training late

in the evening (after 7pm) can delay cortisol clearance and elevate core body temperature at a time when both should be declining, impairing sleep onset and quality.

Passive heat exposure (saunas, hot baths) should also be considered in terms of nervous system load. A hot bath can elevate heart rate to 110bpm at rest and 155bpm upon standing, essentially equivalent to a moderate-intensity training session. When added to easy training days, these modalities can unintentionally eliminate recovery days by keeping the nervous system in a stressed state. If heat adaptation is a goal, it is better to schedule these sessions earlier in the day and avoid them within 3-4 hours of bed. Hot showers, interestingly, do not have the same negative effect on sleep as hot baths, likely due to the shorter duration and lower total thermal load.

6.6 Sleep, Recovery, and Performance

Sleep is the body's primary recovery state. During sleep, the glymphatic system clears metabolic waste from the brain, emotional memories are processed and consolidated, growth hormone supports tissue repair, and the immune system undergoes maintenance. When sleep quality is compromised, recovery is impaired across all domains.

For athletes, the consequences are direct and measurable. Poor sleep elevates inflammatory markers, impairs glycogen resynthesis, reduces pain tolerance, increases perceived effort, and compromises emotional regulation. An elevated resting heart rate during a taper period is a reliable indicator that the nervous system has not recovered, often driven by poor sleep, unresolved stress, or both. In these circumstances, an inappropriate adrenaline response on race morning can manifest as an elevated heart rate from the start line, making it impossible to sustain the planned pace.

The nutritional interventions described in this report work most effectively when paired with good circadian hygiene. Magnesium's role in melatonin synthesis, taurine's involvement in pineal gland function, and L-theanine's cortisol-modulating effects all operate within the framework of the circadian system. Addressing circadian disruption alongside targeted supplementation creates a complete approach to improving sleep quality, reducing anxiety, and optimising recovery.

7. Practical Recommendations

Magnesium citrate (or taurinate): Start at 300mg/day supplemental on top of dietary intake. Can increase to 600-900mg/day under periods of high stress. Take divided throughout the day or with the evening dose 1-2 hours before bed. Avoid magnesium oxide (poor bioavailability) and exercise caution with glycinate if ADHD is present (GAD1 gene SNPs can make glycine glutamatergic).

Taurine: 1-2g taken 60 minutes before bed for sleep support. Can be split 2g morning / 2g evening for combined benefits. For competition in warm conditions, 4g (approximately 50mg/kg) taken 60 minutes before has demonstrated significant performance benefits in the heat.

L-Theanine: 200-400mg daily. Can be taken before bed for sleep, during the day for anxiety, or combined with caffeine pre-exercise for focused calm. Start at 200mg and assess response. Available as capsules or bulk powder.

Circadian hygiene: Morning light within 1 hour of waking (10-20 min minimum). Blue light restriction 4-5 hours before bed. No large meals 2-3 hours before bed. Small carbohydrate snack 1-2 hours before bed. Consistent sleep-wake timing. Avoid late evening exercise and hot baths close to bedtime.

Note: These recommendations are for informational purposes. Individual responses vary. Individuals taking medication, particularly benzodiazepines, SSRIs, or other CNS-active drugs, should consult their prescribing physician before commencing supplementation.

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