Super Sleep: A Circadian Resynchronisation Formula Based on Multi-Mechanism Non-Sedative Sleep Architecture Support — Formulation Rationale, Target Population Guide, Flexible Dosing Architecture, and Safety Profile

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Abstract

Poor sleep quality is among the most prevalent functional complaints in modern Western populations, yet the dominant supplementation and pharmaceutical response — sedation — addresses a symptom while bypassing the underlying circadian biology. Super Sleep is an eight-ingredient, non-sedative evening formula designed as the night phase component of the EscapeMed 30D chronobiological supplement system. Its formulation logic rests on three biological arguments: sleep onset failure in the majority of healthy adults is not a sedation deficit but a failure of circadian resynchronisation; this failure is population-level in scale, affecting an estimated 60–80% of the modern working population through social jet lag and chronic circadian drift; and it can be addressed through GABAergic tone support, HPA axis normalisation, and tryptophan-melatonin pathway activation without sedation. Seven tables document the complete

formulation in logical sequence: global epidemiology of sleep insufficiency; sleep architecture phases with biological functions and formula coverage; ingredient architecture at both flexible dose levels; target population guide identifying who benefits most and why; symptom-to-mechanism mapping; comparison to conventional sleep aids; and a distinction between medical sleep disorders and biological misalignment. All ingredient doses are confirmed from the official product specification at both one-capsule and two-capsule levels. Super Sleep is, to the authors' knowledge, the first multi-mechanism, non-sedative sleep architecture formula to be documented in peer-reviewed literature within a defined chronobiological supplement system.

Keywords: sleep architecture; circadian resynchronisation; GABA-A modulation; melatonin; social jet lag; circadian drift; EscapeMed 30D; non-sedative sleep support; HPA axis; L-theanine; glycine; ashwagandha KSM-66; L-tryptophan; target population

1. The Scale of the Problem: Sleep Quality as a Global Public Health Issue

Most people who sleep poorly do not consider themselves to have a sleep problem. They consider themselves tired, stressed, or simply not a good sleeper. They do not realise that their subjective experience of poor sleep has a biological name, a well-documented population prevalence, measurable health consequences, and a correctable mechanism. Table 1 presents the global epidemiology of sleep insufficiency, circadian misalignment, and sleep-related health consequences. The final row is the most important for understanding what Super Sleep

addresses: the majority of people who sleep poorly have no diagnosable disorder. Their sleep problems reflect circadian misalignment, not pathology.

Table 1. Global epidemiology of sleep insufficiency and circadian misalignment.

Measure	Data	Source
Adults sleeping fewer than 7 hours per night	35–45% of adults in high-income countries; WHO classifies insufficient sleep as a global epidemic	Watson et al. 2015; WHO 2019
Adults reporting poor sleep quality (regardless of duration)	Approximately 45% of the world's population reports some form of sleep problem; 10–30% report chronic poor sleep quality	NSF Sleep in America 2020; Léger et al. 2008
Social jet lag: biological vs social schedule discrepancy ≥1 hour	59–80% of the working population in Western societies; most prevalent in adults aged 20–40 years	Roenneberg et al. 2012; Wittmann et al. 2006
Time spent indoors under artificial lighting with zero NIR	Modern humans spend approximately 93% of time indoors under artificial light providing zero near-infrared radiation — eliminating a major driver of extrapineal melatonin synthesis	Tan et al. 2023; Zimmerman and Reiter 2019
Economic cost of sleep deprivation	Estimated €35 billion annually in the EU from lost productivity and healthcare costs; US equivalent estimated at $411 billion per year	Hafner et al. 2017
Cardiovascular consequences	Adults sleeping fewer than 6 hours have 48% higher risk of coronary heart disease and 15% higher risk of stroke versus those sleeping 7–8 hours	Cappuccio et al. 2011
Metabolic consequences	Chronic sleep restriction increases type 2 diabetes risk by 28%; social jet lag of 1 hour increases obesity risk measurably; sleep deprivation produces insulin resistance within 3 days	Cappuccio et al. 2010; Roenneberg et al. 2012; Leproult and Van Cauter 2010
Cognitive and performance consequences	17–19 hours of wakefulness produces cognitive impairment equivalent to a blood alcohol concentration of 0.05%; chronic mild sleep restriction is subjectively imperceptible but objectively measurable	Williamson and Feyer 2000; Van Dongen et al. 2003
Adults using sleep medication regularly	8–10% of adults in OECD countries use prescription or OTC sleep aids	OECD Health Statistics 2021

	regularly; use increased 30–40% in the decade 2010–2020	
Sleep disorder vs biological misalignment	Approximately 10–15% of the population has a diagnosable sleep disorder. The remaining 45–65% with poor sleep have no diagnosable disorder — their sleep problems reflect circadian misalignment, not pathology	American Academy of Sleep Medicine 2014; Ohayon 2002

2. Sleep is Not a Sedation Problem

The prevailing therapeutic and supplementation response to poor sleep is pharmacological sedation: suppression of waking neurological activity through antihistamine blockade (diphenhydramine, doxylamine), GABA-A positive allosteric modulation at benzodiazepine binding sites (benzodiazepines, Z-drugs), or orexin receptor antagonism. These approaches share a common mechanistic error — they treat sleep onset failure as excess wakefulness to be suppressed rather than as a failure of the biological transition into sleep to be supported. Sedation reduces alertness without improving sleep architecture. It does not increase slow-wave sleep proportion or REM density; in many cases it actively suppresses both (Kripke 2016; Pagel and Parnes 2001). Waking from sedative sleep is characteristically impaired rather than restored.

Super Sleep contains no antihistamines, no benzodiazepines, no Z-drugs, no orexin antagonists, and no habit-forming compounds of any class. Its formulation rests on a different premise: sleep onset and sleep architecture failure in the majority of otherwise healthy adults results from correctable biological deficits — insufficient GABAergic tone at the sleep-wake transition, dysregulated HPA axis activity maintaining elevated evening cortisol, an under-supported tryptophan-serotonin-melatonin synthesis cascade, and NMDA receptor hyperexcitability that impairs deep sleep architecture. Super Sleep addresses all four simultaneously through eight

active ingredients selected for biological layer specificity and mutual mechanistic complementarity. The formulation rationale documented in this paper is intended to provide transparent scientific basis for each ingredient selection, dose, and timing decision.

3. Sleep Architecture: Why Quality Matters as Much as Duration

3.1. The Two Phases Most People Do Not Know About

Most people evaluate their sleep by two parameters: how quickly they fell asleep, and whether they woke during the night. These are meaningful indicators, but they describe only the surface of a process that is biologically far more structured. Sleep is not a single state of unconsciousness. It is a cyclically organised programme, repeated four to six times per night in approximately 90-minute cycles, consisting of two fundamentally different biological phases: Non-Rapid Eye Movement (NREM) sleep and Rapid Eye Movement (REM) sleep. Each phase accomplishes specific, irreplaceable biological functions. When either phase is disrupted — by fragmentation, insufficient depth, or pharmacological suppression — those functions are lost regardless of whether the person was technically unconscious for seven or eight hours. This is why two people can both sleep eight hours and have completely different functional outcomes the following day.

The question most people never ask — but should — is not how long did I sleep, but what did my sleep accomplish. Table 2 answers that question and shows how each Super Sleep ingredient supports specific sleep phases.

Table 2. Sleep architecture phases: biological functions, disruption consequences, and Super Sleep ingredient coverage.

Phase	Duration per night	Biological functions accomplished	What disruption costs you	Super Sleep ingredients supporting this phase
SLEEP ONSETThe threshold event(N1 transition)	Normal: 10–20 minDelayed >30 min indicates circadian misalignment	Core body temperature drop ~1°C (essential prerequisite); GABAergic tone increase; sympathetic withdrawal; melatonin onset; adenosine threshold reached	Prolonged lying-awake frustration; clock-watching anxiety; conditioned arousal; elevated cortisol from frustration compounding the problem	Chamomile apigenin: GABA-A partial agonism at benzodiazepine siteL-theanine: GABA-A + alpha wave promotionGlycine: core temperature drop via peripheral vasodilationMelatonin 0.10 mg: SCN darkness signalAshwagandha: removes evening cortisol barrier to onset
PHASE 1: NREM SleepN2 (light sleep) and N3 (deep / slow-wave sleep, SWS)75–80% of total sleep time	N2: ~50% of night N3: 15–25 % of night (~60–100 min) Most N3 in first half of night	N2: Memory consolidation initiation; sleep spindle generation for memory processing; physical transition to deeper sleepN3 (SWS): Growth hormone secretion (90% occurs here); cellular repair and tissue regeneration; immune consolidation; glymphatic system waste clearance from the brain; long-term memory consolidation; HPA axis recovery and cortisol reset	N2 disruption: Frequent micro-awakenings; light sleep; unrefreshing outcomeN3 disruption: Impaired physical recovery; immune suppression; metabolic disruption; morning fatigue despite adequate hours in bed; no subjective sense of deep rest; impaired growth hormone release affecting body composition and recovery	Glycine: reduces NMDA hyperexcitability that prevents N3 entry; drives temperature drop required for SWSMagnesium bisglycinate: restores Mg^{2+} channel block allowing neural down-regulation for SWSL-theanine + chamomile apigenin: GABA-A modulation supporting transition through N2 into N3Ashwagandha KSM-66: normalises cortisol preventing premature arousal from N3
PHASE 2: REM SleepRapid Eye MovementActive brain; muscle atonia; dreaming20–25% of total sleep	~90–120 min per nightLongest REM periods in the final	Emotional memory processing and emotional regulation; procedural memory consolidation; creative problem-solving integration; synaptic pruning and neural plasticity; mood-related neurotransmitter restoration; prefrontal	Reduced emotional regulation; increased anxiety and emotional reactivity the following day; impaired creativity; poor stress tolerance; mood instability; progressive emotional dysregulation with chronic REM	Melatonin 0.10 mg: strengthens circadian timing of REM in final sleep cyclesGlycine: NMDA modulation promotes REM architecture (vivid dreams in early days of use are a marker of improving REM density)L-tryptophan + B6: serotonin-melatonin cascade supports the hormonal environment for REM

	cycles of the night (after 4–5 a.m.)	cortex executive function recovery	suppression; reduced empathy and social function	maintenanceAshwagandha: normalises cortisol preventing stress-induced REM suppression

The most important clinical insight from Table 2 is this: a person can fail at any or all three biological requirements simultaneously. They may struggle to fall asleep (onset failure), sleep without reaching adequate N3 depth (deep sleep failure), and wake before REM cycles complete (REM failure). These three failure modes have different biological causes but are all addressed by the multi-ingredient architecture of Super Sleep. This is why a formula targeting only one mechanism — a single melatonin dose, or a single sedative — produces partial benefit for some and none for others.

4. The Circadian Clock and Its Systematic Drift in Modern Life

4.1. The Intrinsic Period Problem

The human endogenous circadian clock, generated by the molecular CLOCK/BMAL1 feedback loop and entrained by the suprachiasmatic nucleus (SCN), has an intrinsic free-running period of approximately 24.2 hours — measurably longer than the solar day (Czeisler et al. 1999; Wright et al. 2013). Without daily zeitgeber (time-giver) signals, the biological clock drifts forward by approximately 12 to 24 minutes every 24 hours. In natural ancestral environments this drift was automatically corrected each morning by sunrise-driven photoentrainment via melanopsin-containing retinal ganglion cells, by environmental temperature dynamics, by consistent physical activity timing, and by regular meal timing. In the absence of these signals,

the biological clock and the social clock progressively desynchronise — the biological night extending later while social obligations remain fixed.

4.2. Modern Life Systematically Removes the Zeitgebers

Contemporary human environments eliminate or substantially attenuate the signals that prevent circadian drift. Tan and colleagues (2023) established that humans now spend approximately 93% of their time indoors under artificial lighting providing zero near-infrared (NIR) radiation — the largest reduction in solar spectral exposure in human history, with documented consequences for extrapineal melatonin synthesis and circadian amplitude. Evening blue light from screens activates melanopsin in retinal ganglion cells at wavelengths around 480 nm, actively suppressing pineal melatonin synthesis and delaying circadian phase (Czeisler 2013). Irregular meal timing eliminates the food-dependent zeitgeber. Climate-controlled indoor environments remove the temperature differential that normally reinforces morning activation and evening cooling. The consequence is a population whose biological clocks are chronically under-entrained.

4.3. Seasonal Time Changes Compound the Drift

Statutory daylight saving time transitions impose an acute one-hour forced shift in the socially imposed schedule relative to the biological clock. The SCN requires approximately one week to realign following a one-hour forced phase advance, during which measurable increases in cardiovascular events, metabolic disruption, and cognitive impairment have been documented (Manfredini et al. 2018; Roenneberg et al. 2019). For individuals already experiencing baseline circadian misalignment, seasonal time changes compound rather than correct the underlying

problem. Two such transitions per year impose two consecutive periods of acute circadian stress on an already chronically misaligned system.

5. Social Jet Lag: A Population-Level Circadian Mismatch

Social jet lag describes the discrepancy between an individual's biologically preferred sleep timing — their chronotype, determined by the molecular clock — and the socially imposed schedule of work, school, and daily obligations. Roenneberg and colleagues, using data from over 150,000 participants via the Munich Chronotype Questionnaire, established that social jet lag of one hour or more affects the majority of the working population in Western societies (Roenneberg et al. 2012). Estimates from multiple studies range from 59% to over 80% of working adults. Social jet lag produces physiological consequences equivalent to travelling across one to two time zones weekly without recovery: elevated cortisol upon forced early waking, suppressed melatonin onset due to evening light exposure, reduced slow-wave sleep proportion, impaired glucose regulation, and reduced cognitive performance on weekdays relative to weekends (Leproult, Holmbäck, and Van Cauter 2014; Wittmann et al. 2006).

These are not consequences of a sleep disorder. They are the consequences of a systematic mismatch between biological timing and social scheduling, experienced by the majority of the healthy working population. No pharmaceutical sedative addresses this mismatch. Sedation on weekday nights while the biological clock remains phase-delayed produces pharmacological unconsciousness in a biologically misaligned individual — with no correction of the underlying circadian drift and active suppression of the deep sleep architecture required for overnight repair. Super Sleep is designed for this population.

6. Medical Sleep Disorders vs Biological Misalignment: Who Super Sleep Is For

Super Sleep is not indicated for, and cannot address, the biological causes of diagnosable sleep disorders. The distinction between conditions requiring medical evaluation and the biological misalignment that Super Sleep addresses is clinically important. With the circadian biology from the preceding sections now established, Table 3 makes this distinction concrete.

Table 3. Medical sleep disorders versus biological misalignment: signs for medical evaluation versus signs Super Sleep addresses.

Signs that suggest medical evaluation is needed(Super Sleep is NOT the primary solution)	**Signs that suggest biological misalignment(Super Sleep is designed for this population)**
Loud snoring, gasping, or choking during sleep reported by a partner (suggests obstructive sleep apnea)	Difficulty falling asleep despite physical tiredness — the mind remains active and engaged
Waking unrefreshed every night despite apparently adequate duration, with significant daytime sleepiness that impairs function (may indicate sleep apnea or upper airway resistance syndrome)	Sleep problems that are noticeably worse on weekdays and better on weekends or holidays (the classic social jet lag pattern)
Irresistible urge to move legs in the evening or at rest, relieved by movement (restless leg syndrome)	Difficulty falling asleep that worsened gradually with changes in lifestyle, work schedule, or screen habits
Sudden episodes of muscle weakness triggered by strong emotion, or episodes of sleep paralysis (narcolepsy spectrum)	Sleep onset that is easier on weekends or holidays when the individual can choose their own timing
Sleep onset insomnia that has been present for more than 3 months with significant daytime functional impairment (clinical insomnia disorder requiring CBT-I and/or medical evaluation)	Waking feeling tired after 7–8 hours in bed — suggesting poor sleep architecture quality rather than insufficient duration
Sleepwalking, sleep terrors, or complex nocturnal behaviours (parasomnias requiring clinical evaluation)	Seasonal worsening of sleep quality correlating with time changes, reduced daylight hours, or increased work pressure
Sleep problems that began or worsened coincidentally with starting a new medication (medication-induced sleep disruption)	The ‘wired but tired’ pattern: physical fatigue combined with inability to stop thinking, particularly after stressful or demanding days
If in any doubt, consult a physician or sleep medicine specialist before initiating supplementation.	**Super Sleep was designed for this population — biologically healthy individuals whose sleep quality is degraded by circadian misalignment, HPA axis dysregulation, or tryptophan-melatonin pathway under-support, not by pathology.**

7. Super Sleep: Formula Architecture and Flexible Dosing

Super Sleep delivers eight active ingredients in a vegetarian HPMC capsule, targeting the biological failure points described in the preceding sections. Each capsule contains a half-dose of each active ingredient, enabling flexible dosing between one and two capsules per evening based on individual need, tolerance, and applicable regulatory context. The one-capsule dose allows gentle initiation and complies with the most conservative regulatory thresholds for each ingredient. The two-capsule dose delivers the full clinically targeted level as used in the EscapeMed 30D pilot study. The designation ‘30D’ refers to the 30-day pilot study protocol and does not imply a fixed consumption duration or dose. Super Sleep should be taken 30–60 minutes before intended sleep. All products are manufactured by a GMP-certified contract manufacturer in the European Union, in compliance with EU food supplement regulations, HACCP, and GMP quality standards. All ingredients are vegan, non-GMO, allergen-free, and gluten-free as confirmed in the product specification.

8. Ingredient-by-Ingredient Formulation Rationale

8.1. Melatonin (98%)

One capsule: 0.10 mg. Two capsules: 0.20 mg.

Melatonin is the primary chemical signal of darkness — the molecule through which the SCN communicates the transition from the active phase to the rest phase (Reiter 1991). Its endogenous synthesis in the pineal gland peaks between 2:00 and 4:00 a.m. in healthy adults with normal

circadian entrainment and is undetectable during most of the waking day. The dose in Super Sleep — 0.10 mg per capsule, 0.20 mg at two capsules — is deliberately in the physiological signal range. Brzezinski and colleagues (2005) established that 0.1 to 0.3 mg raises plasma melatonin to the normal physiological nocturnal range with equivalent or superior phase-shifting effects compared to pharmacological doses of 1 to 10 mg. The EU authorized health claims (0.5 mg for jet lag alleviation; 1 mg for sleep onset reduction) represent minimum doses at which regulatory efficacy evidence was assessed — not the minimum effective dose. Super Sleep provides a physiological darkness primer while tryptophan and B6 simultaneously support the body's own melatonin synthesis cascade from within.

Experienced benefit: signal to the SCN that darkness has arrived; supports faster sleep onset without sedation; no morning residual drowsiness.

8.2. L-Theanine (from Green Tea Extract, 40% standardisation)

One capsule: 30 mg (from 75 mg extract). Two capsules: 60 mg (from 150 mg extract).

L-theanine increases alpha brain wave power — the 8–14 Hz frequency associated with relaxed alertness, the cognitive state between active wakefulness and sleep preparation — without inducing sedation (Nobre, Rao, and Owen 2008). It additionally modulates GABA-A receptor activity through positive allosteric interaction, supporting the inhibitory neurotransmitter tone required for the N1/N2 sleep onset transition (Kimura et al. 2007). Combined with chamomile apigenin, it provides additive GABAergic support through two pharmacologically independent pathways.

Experienced benefit: calmer cognitive state before sleep; reduced mental rumination; easier transition into rest without grogginess.

8.3. L-Tryptophan (vegan fermented, 98%)

One capsule: 73.5 mg. Two capsules: 147 mg.

L-tryptophan is the obligate dietary precursor to serotonin and melatonin. The gastrointestinal tract contains approximately 400 times the melatonin content of the pineal gland, with enterochromaffin cells producing melatonin from tryptophan in quantities that substantially influence circulating concentrations (Bubenik 2002; Tan et al. 2023). Hajak and colleagues (1991) demonstrated that tryptophan administration significantly elevated plasma melatonin in human subjects. In Super Sleep, tryptophan is co-delivered with its enzymatic cofactor vitamin B6, ensuring that substrate and cofactor arrive at the synthesis site together. The vegan fermented form achieves 98% purity without animal-derived precursors.

Experienced benefit: supports the body's own melatonin synthesis from within; earlier melatonin onset; an internal circadian resetting effect distinct from exogenous melatonin administration.

8.4. Glycine (98.5%)

One capsule: 167.45 mg. Two capsules: 334.9 mg.

Glycine is the largest active ingredient by mass in Super Sleep, reflecting its dual indispensable roles. First, as NMDA receptor co-agonist, it reduces the cortical hyperexcitability that prevents deep NREM sleep. Second, it promotes core body temperature reduction through peripheral vasodilation, documented by Kawai and colleagues (2015) to significantly improve subjective and objective sleep quality in a controlled human study. Core body temperature must fall by approximately 1°C to trigger full sleep onset and maintain deep sleep; glycine-driven vasodilation redistributes heat from the body core to the periphery, accelerating this essential

biological prerequisite. Glycine has an excellent tolerability profile with no habit-forming potential and no next-day impairment.

Experienced benefit: faster sleep onset via temperature reduction; improved deep sleep; fresh waking without grogginess.

8.5. Ashwagandha KSM-66 Organic Extract (5% withanolides)

One capsule: 120 mg extract / 6 mg withanolides. Two capsules: 240 mg extract / 12 mg withanolides.

Withanolides modulate glucocorticoid receptor sensitivity and attenuate stress-induced HPA axis activation, reducing elevated evening cortisol. Chandrasekhar, Kapoor, and Anishetty (2012) demonstrated in a double-blind, placebo-controlled randomised trial that ashwagandha root extract significantly reduced serum cortisol and validated stress scores. Priyanka and colleagues (2020) confirmed significant improvements in sleep quality, onset latency, and total sleep time. KSM-66 is the most extensively studied full-spectrum ashwagandha root extract, standardised to minimum 5% withanolides from root only, with no hepatotoxicity signals reported in published clinical studies at doses up to 600 mg extract per day. Ashwagandha does not produce sedation. It removes a physiological barrier — elevated evening cortisol — that prevents the body's own sleep onset mechanisms from functioning.

Experienced benefit: calmer evening nervous system state; reduced physical tension and cognitive activation after demanding days; the transition from work to rest that chronic stress makes difficult.

8.6. Chamomile Extract 4:1 (1.2% apigenin)

One capsule: 150 mg extract / 1.80 mg apigenin. Two capsules: 300 mg extract / 3.60 mg apigenin.

Apigenin is a partial agonist at the benzodiazepine binding site of GABA-A receptors, producing anxiolytic and sleep-facilitative effects without the respiratory depression, tolerance development, or dependence liability of pharmaceutical benzodiazepines (Viola et al. 1995; Srivastava, Shankar, and Gupta 2010). Combined with L-theanine's GABA-A modulation through a pharmacologically distinct molecular interaction, Super Sleep achieves convergent GABAergic support through two independent pathways simultaneously.

Experienced benefit: reduced pre-sleep physical tension and anxiety; gentler sleep onset without sedation or next-day impairment.

8.7. Magnesium Bisglycinate (20% elemental Magnesium)

One capsule: 100 mg bisglycinate / 20 mg elemental Mg. Two capsules: 200 mg bisglycinate / 40 mg elemental Mg.

Magnesium bisglycinate restores the voltage-dependent Mg^{2+} block of the NMDA receptor channel, reducing cortical excitability and enabling the neural down-regulation required for deep NREM sleep. The bisglycinate form is selected for superior bioavailability via the amino acid transporter pathway and excellent pre-sleep tolerability. At 20 mg elemental Mg per capsule this is a targeted NMDA modulation contribution rather than a standalone magnesium repletion strategy. Total system magnesium when Super Sleep is used alongside Magnesium PM is documented in the companion EscapeMed 30D magnesium formulation paper (Samarin 2026a).

EU authorized health claim: magnesium contributes to normal functioning of the nervous system and normal muscle function.

Experienced benefit: reduced nocturnal muscle cramping; neuromuscular relaxation supporting the rest state; improved deep sleep quality.

8.8. Vitamin B6 Pyridoxine HCL (98%)

One capsule: 1.47 mg (105% NRV). Two capsules: 2.94 mg (210% NRV).

Vitamin B6 is the required cofactor for AANAT — the rate-limiting enzyme converting serotonin to N-acetylserotonin in the melatonin synthesis pathway. Without adequate B6, the tryptophan-melatonin cascade stalls regardless of substrate availability. B6 is co-delivered with tryptophan in Super Sleep, ensuring that substrate and enzymatic cofactor arrive together. This evening B6 function is categorically different from morning B6 (which drives dopamine and serotonin synthesis for activation): evening B6 converts the day's serotonin pool into the melatonin needed for sleep onset. The two-capsule dose of 2.94 mg is 12% of the EFSA Tolerable Upper Intake Level of 25 mg per day, providing a substantial safety margin.

EU authorized health claim: vitamin B6 contributes to normal psychological function, normal functioning of the nervous system, and the regulation of hormonal activity.

Experienced benefit: complete enzymatic support for the body's own melatonin production from the tryptophan co-delivered in the same formula.

9. Formula Tables

Table 4. Super Sleep ingredient architecture: confirmed doses at 1-capsule and 2-capsule levels, sleep phase, and mechanism.

Ingredient	1 capsule	2 capsules	Sleep phase supported	Primary mechanism
Melatonin (98%)	0.10 mg	0.20 mg	Onset + REM timing	Physiological SCN darkness signal below EU claim thresholds; primes endogenous melatonin cascade while tryptophan + B6 drive synthesis from within
L-Theanine (green tea 40%)	30 mg	60 mg	Onset (N1/N2)	GABA-A positive allosteric modulation via distinct site from apigenin; alpha wave promotion without sedation
L-Tryptophan (vegan fermented 98%)	73.5 mg	147 mg	REM + circadian cascade	Obligate melatonin precursor; stimulates gastrointestinal melatonin synthesis; co-delivered with B6 cofactor
Glycine (98.5%)	167.45 mg	334.9 mg	Onset + N3 deep sleep	NMDA co-agonist reducing cortical hyperexcitability; peripheral vasodilation drives core temperature drop required for sleep onset and N3 entry
Ashwagandha KSM-66(5% withanolides)	120 mg / 6 mg withanolides	240 mg / 12 mg withanolides	Onset + N3 + REM	HPA axis: reduces elevated evening cortisol that blocks melatonin synthesis, GABA-A transition, and all sleep architecture phases
Chamomile 4:1(1.2% apigenin)	150 mg / 1.80 mg apigenin	300 mg / 3.60 mg apigenin	Onset (N1/N2)	Apigenin partial agonist at benzodiazepine site of GABA-A receptor; no tolerance, no dependence, no respiratory depression at dietary supplement doses
Magnesium Bisglycinate (20% Mg)	100 mg / 20 mg Mg	200 mg / 40 mg Mg	N3 deep sleep	Restores NMDA Mg^{2+} voltage-dependent channel block allowing neural down-regulation for SWS; neuromuscular relaxation
Vitamin B6 (98%)	1.47 mg (105% NRV)	2.94 mg (210% NRV)	REM + circadian cascade	AANAT enzyme cofactor; rate-limiting step in serotonin-to-melatonin conversion; co-delivered with tryptophan substrate

Table 5. Target population guide: who benefits most from Super Sleep, primary sleep disruption mechanism, expected benefit, starting dose recommendation, and monitoring.

Population	Primary sleep disruption mechanism	Key benefit from Super Sleep	Most relevant ingredients	Starting dose	Monitoring
Chronic stress / high cognitive load	HPA axis dysregulation; elevated evening cortisol blocking sleep onset and N3; cortisol-driven circadian amplitude reduction	Ashwagandha normalises evening cortisol; GABA-A support facilitates the mental-to-physical transition; tryptophan cascade	Ashwagandha KSM-66; chamomile apigenin; L-theanine	1 cap; increase to 2 caps if insufficient onset improvement at 2 weeks	Salivary cortisol evening reading; sleep onset time self-report

		restores melatonin amplitude			
Shift workers / frequent travellers	Acute and chronic circadian phase misalignment; irregular melatonin onset; cumulative social jet lag	Melatonin at destination timing as phase-setting signal; tryptophan + B6 for endogenous synthesis resynchronisation; glycine accelerates adaptation via temperature mechanism	Melatonin; L-tryptophan + B6; glycine	2 caps at destination bedtime for jet lag; 1 cap for maintenance on regular shift nights	Sleep onset time at destination; subjective alertness recovery
Perimenopausal and postmenopausal women	Oestrogen decline reduces melatonin amplitude and progesterone-GABA signalling; hot flashes fragment N3; elevated nocturnal cortisol from HPA sensitisation	GABA-A support (apigenin + theanine) replaces declining progesterone-GABA contribution; melatonin + tryptophan cascade compensates for reduced melatonin amplitude; ashwagandha supports HPA axis	Chamomile apigenin; L-theanine; ashwagandha; melatonin + tryptophan	1 cap to start; 2 caps if sleep fragmentation persists at 2 weeks	Sleep fragmentation frequency; hot flash timing relative to waking episodes
Adults over 50	Age-related melatonin amplitude decline; sleep architecture fragmentation with shorter N3 cycles; reduced GABA-A receptor sensitivity	Physiological melatonin dose compensates for pineal age-related decline; tryptophan + B6 support endogenous synthesis; glycine + bisglycinate restore N3 depth via NMDA modulation	Melatonin; tryptophan + B6; glycine; magnesium bisglycinate	1 cap; increase to 2 caps if N3 disruption (unrefreshing sleep) persists	Sleep quality self-report; morning freshness rating; dream recall as REM proxy
Late chronotype with social jet lag	Chronic biological phase delay 1–3 hours later than social obligations; forced early waking on weekdays; cortisol awakening response insufficient for early schedule	Melatonin at an earlier clock time progressively resets SCN phase; consistent timing reinforces earlier phase; ashwagandha reduces cortisol spike from forced early waking	Melatonin (taken 30 min earlier than typical sleep time); ashwagandha; L-theanine	1 cap on weekdays at target bedtime; consistent timing essential	Sleep onset time on weekdays vs weekends over 4–8 weeks
Athletes in high training load phases	Training-induced cortisol elevation persisting into the	Ashwagandha KSM-66 reduces training cortisol	Ashwagandha; glycine;	2 caps during high training load; 1 cap	Subjective recovery quality;

	evening; physical fatigue without sleep quality (common in overreaching phases); N3 deficit impairs growth hormone release	elevation; glycine supports N3 depth for growth hormone release; magnesium bisglycinate supports neuromuscular relaxation after training	magnesium bisglycinate	during taper or recovery phases	morning readiness score; training load periodisation

Table 6. Symptom-to-mechanism mapping: lived experience, biological target, ingredient, dosing, and expected onset.

Symptom / Experience	Underlying mechanism	Relevant ingredient and action	Dose	Expected onset
Difficulty falling asleep despite tiredness	Insufficient GABAergic tone; elevated evening cortisol blocking the sleep-wake switch	Chamomile apigenin (GABA-A); L-theanine (GABA-A + alpha waves); ashwagandha (cortisol reduction)	Start 1 cap; increase to 2 if insufficient at 1 week	1–2 weeks
Wired mind at bedtime (‘can’t switch off’)	HPA axis dysregulation; elevated cortisol maintaining sympathetic activation despite physical fatigue	Ashwagandha KSM-66 (cortisol normalisation); L-theanine (alpha wave cognitive calm)	1–2 caps based on stress level	1–2 weeks
Waking at 3–4 a.m. unable to return to sleep	Cortisol nadir disruption; NMDA hyperexcitability during light sleep; insufficient N3 depth for sustained sleep	Glycine + magnesium bisglycinate (NMDA Mg^{2+} block for N3); melatonin (circadian signal strengthening for final sleep cycles)	2-cap dose recommended for sleep maintenance	2–4 weeks
Waking tired after 7–8 hours (unrefreshing sleep)	Insufficient N3 slow-wave sleep; inadequate core temperature reduction; cortisol-driven N3 disruption	Glycine (temperature reduction + NMDA); magnesium bisglycinate (NMDA Mg^{2+} block); ashwagandha (prevents cortisol-driven N3 disruption)	Start 1 cap; increase based on response	1–2 weeks
Jet lag or shift work disruption	Acute circadian phase misalignment; SCN desynchrony from imposed schedule change	Melatonin (phase-setting signal at destination time); tryptophan + B6 (endogenous synthesis resynchronisation)	1–2 caps at destination bedtime	2–5 days
Vivid dreams / improved dream recall	Increased REM density during circadian resynchronisation; early adaptation signature	Glycine + bisglycinate (NMDA modulation supporting REM architecture); melatonin cascade strengthening	Either dose level	Days 3–5 of use

Muscle tension at bedtime preventing sleep	Calcium-magnesium imbalance at sarcoplasmic reticulum; insufficient neuromuscular inhibitory tone	Magnesium bisglycinate (neuromuscular relaxation); glycine (glycine receptor inhibition at spinal cord)	2-cap dose for muscle tension	1–2 weeks
Social jet lag pattern (tired weekdays, awake weekends)	Chronic biological phase delay relative to social obligations; cumulative HPA axis desynchrony	Melatonin taken earlier on weekdays to shift the biological clock; ashwagandha to reduce forced-waking cortisol; consistent timing to reinforce SCN resetting	2 caps weekdays at target time; 1 cap maintenance weekends	4–8 weeks for chronotype adjustment
Seasonal sleep disruption (autumn/winter)	Reduced NIR and full-spectrum light reducing extrapineal melatonin; longer dark period; time change acute misalignment	Tryptophan + B6 + melatonin primer compensating for reduced endogenous synthesis from NIR light deficit documented by Tan et al. (2023)	2-cap dose during winter months	1–2 weeks

Table 7. Super Sleep versus conventional sleep aids: comparative analysis.

Dimension	**Antihistamine OTC(e.g. diphenhydramine)**	**Benzodiazepines / Z-drugs(prescription)**	**Melatonin 1–10 mg(standard OTC)**	**Super Sleep(EscapeMed 30D)**
Primary mechanism	H1 receptor blockade (sedation)	GABA-A full agonist at benzodiazepine site	Supraphysiological melatonin replacement	Multi-mechanism: GABA-A partial agonism + HPA axis + tryptophan-melatonin + NMDA modulation
Improves N3 deep sleep	No — suppresses	No — suppresses	Minimal	Yes — glycine + NMDA modulation + temperature reduction
Improves REM sleep	No — suppresses	No — suppresses	Partial at physiological doses	Yes — melatonin + tryptophan cascade + NMDA
Addresses evening cortisol (HPA axis)	No	No	No	Yes — ashwagandha KSM-66 withanolides
Addresses circadian phase drift	No	No	Partial — phase signal only, no endogenous cascade support	Yes — physiological melatonin signal + tryptophan + B6 for

				endogenous synthesis
Morning grogginess / residual sedation	Significant next-day impairment common	Significant, half-life dependent	Possible at high doses	Absent — non-sedative mechanism throughout
Tolerance / dependence risk	Yes — antihistamine tolerance within days	Yes — established dependence liability	Low at physiological doses	None — explicitly excluded by formulation design
Supports endogenous melatonin synthesis	No	No	No — replaces rather than supports	Yes — tryptophan + B6 drive the body's own production
Appropriate for long-term use	No	No — short-term only	Uncertain at high doses	Yes — supports biological function rather than suppressing it

10. Safety Profile

10.1. Melatonin at 0.10 mg: The Physiological Dose Rationale

The melatonin dose in Super Sleep — 0.10 mg per capsule, 0.20 mg at two capsules — is deliberately in the physiological signal range. Brzezinski and colleagues (2005) established that 0.1 to 0.3 mg raises plasma melatonin to the normal physiological nocturnal range with equivalent or superior phase-shifting effects to pharmacological doses. The EU authorized health claims (0.5 mg for jet lag alleviation; 1 mg for sleep onset reduction) represent minimum doses at which regulatory efficacy evidence was assessed — not the minimum effective dose. The Super Sleep dose is below both thresholds at either capsule count, positioning the formula within the most conservative regulatory classification across all EU member states. EFSA does not establish a Tolerable Upper Intake Level for melatonin at these doses. No adverse effects have been reported in the published literature at 0.10 to 0.20 mg.

10.2. Ashwagandha KSM-66: Dose Rationale and Regulatory Context

Ashwagandha is under regulatory review in certain EU jurisdictions following case reports of liver toxicity at high doses, primarily from products using root-and-leaf combined extracts or non-standardised materials. KSM-66 is a full-spectrum root-only extract standardised by specific ethanolic-water extraction to minimum 5% withanolides, with over 24 published clinical studies reporting no hepatotoxicity signals at doses up to 600 mg extract per day. The Super Sleep dose of 120 mg (6 mg withanolides) per capsule is among the most conservative available in any commercial ashwagandha product. If regulatory developments in any EU jurisdiction require removal, the formula architecture accommodates reformulation without affecting the primary GABA-A, tryptophan-melatonin, NMDA, and temperature-reduction mechanisms of the remaining seven ingredients.

10.3. General Safety and Precautionary Note

At the doses used in Super Sleep — melatonin 0.10 to 0.20 mg, L-theanine 30 to 60 mg, L-tryptophan 73.5 to 147 mg, glycine 167 to 335 mg, ashwagandha KSM-66 120 to 240 mg, chamomile extract 150 to 300 mg, magnesium bisglycinate 100 to 200 mg, and vitamin B6 1.47 to 2.94 mg — the formula is considered safe for the general healthy adult population. These are dietary supplement doses, substantially below the thresholds at which pharmacological interactions have been documented in the literature. As a general precaution applicable to all dietary supplements, individuals taking prescription immunosuppressants, monoamine oxidase inhibitors (MAOIs), or anticoagulants should consult their physician before initiating any new supplement regimen. Individuals with significant renal impairment should not self-supplement magnesium from any source without medical supervision. Super Sleep is not intended for use during pregnancy or breastfeeding without medical guidance.

11. Super Sleep and Sleep Hygiene: Complementary, Not Competing

Super Sleep is not a replacement for sleep hygiene. It is a biological support for the transitions that good sleep hygiene makes possible. The specific practices that most directly interact with the formula's mechanisms: evening light reduction removes the primary suppressor of endogenous melatonin synthesis, allowing the tryptophan-B6-melatonin cascade to function without being blocked at the pineal level; bedroom temperature reduction to approximately 18–19°C amplifies the glycine-driven peripheral vasodilation that reduces core body temperature; consistent sleep timing — taking Super Sleep at the same clock time each evening — reinforces the circadian phase-setting signal of the melatonin dose and progressively resets the SCN; avoiding caffeine after 2:00 p.m. allows natural adenosine sleep pressure to build alongside the formula's GABAergic and melatonin signals; and reducing evening meal size avoids the thermogenic temperature elevation that counteracts glycine's temperature reduction mechanism.

Many people seeking supplemental sleep support do so precisely because consistent sleep hygiene is difficult in their current life context. Super Sleep provides meaningful biological support even without perfect sleep hygiene compliance. The combination of formula and basic sleep hygiene measures is likely to produce substantially superior outcomes to either approach alone, because both reinforce the same biological mechanisms from complementary directions.

12. Preliminary Observational Support and Ongoing Study

A systematic 30-day observational study of the complete EscapeMed 30D four-formula system — which includes Super Sleep as the night phase component — is currently under peer review (Samarin 2026b). In that pilot study (N=20, two-capsule dose), 75% of participants reported improvement in sleep quality after 30 days, with overall wellbeing improvement in 90% of cases. An adaptation phase signature was informally observed in approximately 50% of participants during days 3–5: increased dream vividness and transient afternoon fatigue, followed by improved sleep efficiency, reduced total sleep duration, stable daytime energy, and absence of morning grogginess. This pattern is mechanistically consistent with glycine-driven REM density increase, progressive melatonin cascade strengthening, and ashwagandha-mediated HPA axis normalisation.

A Super Sleep-specific observational study is currently underway. Informal observations from clinical practice preceding the structured study include: faster sleep onset, described by participants as falling asleep within 15 to 20 minutes compared to 45 to 90 minutes previously; consistent absence of morning grogginess, qualitatively different from the impairment reported with previous OTC or prescription sleep aid use; resolution of the chronic 3 a.m. waking pattern in multiple participants; and the observation that sleep quality improved in a way that had not been achievable through previous interventions including prescribed medications, sleep hygiene measures applied alone, or other commercially available supplements. These observations are informal and not statistically evaluated. They are reported here because they are mechanistically consistent with the formulation's biological rationale and because they informed the design of the ongoing systematic study. Systematic results will be reported in a dedicated publication upon completion.

13. Why This Formula Cannot Be Replicated Through Individual Supplement Purchase

13.1. The Melatonin Problem: 0.10 mg is Not Commercially Available

The most fundamental barrier to DIY replication is the melatonin dose. Super Sleep uses 0.10 mg per capsule — selected for precision in the physiological circadian signalling range. Commercially available melatonin supplements in European markets are sold almost exclusively at doses of 0.5 mg, 1 mg, 1.9 mg, or higher. A dose of 0.10 mg is not commercially available as a standalone supplement in any standard European pharmacy, health food store, or online supplement retailer known to the author. An individual attempting to replicate this dose would need to split a 0.5 mg tablet into five equal pieces — imprecise, impractical, and not dosimetrically equivalent to an encapsulated powder — or to source raw melatonin powder and self-encapsulate at milligram-fraction quantities, a process requiring laboratory equipment and analytical scales entirely beyond consumer reach. The 0.10 mg dose is not a minor product detail. It is the pharmacological and regulatory precision of the formula. Replacing it with a 1 mg commercially available tablet delivers five times the intended dose, crossing from the physiological signal range into the pharmacological replacement range.

13.2. Standardisation and Form Specificity of Key Ingredients

KSM-66 is a trademarked full-spectrum root-only ashwagandha extract with a specific certification and standardisation profile. Generic ashwagandha supplements vary substantially in withanolide content, extraction method, plant parts used, and organic certification. The chamomile extract in Super Sleep is standardised to 1.2% apigenin in a 4:1 concentration — not available in standard pharmacy chamomile products, which are typically unstandardised with

highly variable and generally low apigenin content. The vegan fermented L-tryptophan at 98% purity is a pharmaceutical-grade amino acid different from the generic 500 mg tryptophan capsules in health food stores; replicating the 73.5 mg dose would require splitting such a capsule into approximately one-seventh — impractical and imprecise.

13.3. Cost and Complexity

An individual attempting to replicate all eight Super Sleep ingredients as standalone products at equivalent specifications from reputable European suppliers would require custom-encapsulated sub-milligram melatonin (unavailable commercially), standardised green tea L-theanine extract, vegan fermented pharmaceutical-grade L-tryptophan, glycine at food supplement grade, KSM-66 specifically, chamomile standardised to 1.2% apigenin, magnesium bisglycinate, and pharmaceutical-grade vitamin B6. The combined monthly cost approximates €120 to €200 per month from specialist European nutraceutical suppliers — substantially exceeding the cost of the integrated formula. Beyond cost, the consumer would face the challenge of determining the correct dose of each ingredient, the correct timing of co-administration, and the logic of the tryptophan-B6 co-delivery and three-pathway GABA-A convergence — none of which existed in documented form prior to this publication.

14. Super Sleep in the EscapeMed 30D System

Super Sleep is designed to function both as a standalone evening formula and as the night phase component of the EscapeMed 30D four-formula system. For individuals using it alongside Magnesium PM, the synergistic relationship is clinically significant: Magnesium PM delivers taurine via magnesium taurate, activating thalamic GABA-A receptors through a fourth

independent pathway beyond the three present in Super Sleep alone; Magnesium PM bisglycinate (110 mg elemental Mg at two-capsule dose) extends the NMDA Mg^{2+} block initiated by Super Sleep's bisglycinate; and the evening B6 in Magnesium PM adds a second cofactor delivery for the AANAT melatonin synthesis step. The L-tryptophan in Super Sleep provides the serotonin precursor that both formulas' evening B6 can convert toward melatonin. The formulas are synergistic by design.

For individuals new to the EscapeMed 30D system, Super Sleep is the recommended entry point for those whose primary complaint is sleep quality. It can be used alone indefinitely or as the gateway to the complete four-formula system, which additionally covers morning energy activation (Magnesium AM) and daytime cellular protection (Skin Renewal Complex).

15. Future Research Directions

The primary testable hypothesis is that multi-mechanism, non-sedative sleep architecture support targeting GABAergic tone, HPA axis, and the tryptophan-melatonin pathway simultaneously produces superior sleep quality outcomes compared to single-ingredient approaches at equivalent dose levels. The ideal study design is a four-arm randomised controlled trial: (1) Super Sleep at two-capsule dose; (2) melatonin 1 mg alone; (3) L-theanine 200 mg alone; (4) placebo. Primary outcomes: sleep onset latency and total sleep time via actigraphy; subjective quality via Pittsburgh Sleep Quality Index; morning alertness via validated cognitive battery. Secondary outcomes: salivary cortisol diurnal slope; plasma melatonin onset time; nocturnal HRV. Minimum study duration: 8 weeks. Recommended sample size: 50 participants per arm.

Subgroup studies in the six target population groups identified in Table 5 are planned and in preparation, with outcome measures tailored to each group's specific depletion mechanism. The Super Sleep-specific observational study currently underway will provide the first systematic N-of-many dataset on one-capsule versus two-capsule dosing response and population subgroup differences.

16. Conclusions

Super Sleep is, to the authors' knowledge, the first dietary supplement formula to be documented in peer-reviewed literature as a multi-mechanism, non-sedative sleep architecture support system grounded explicitly in circadian biology and social jet lag science. Its eight-ingredient formulation addresses the three primary biological failure points responsible for the majority of uncomplicated sleep problems in the modern working population — GABAergic insufficiency, HPA axis dysregulation, and tryptophan-melatonin cascade under-support — plus NMDA receptor modulation and core body temperature reduction that directly improves sleep architecture quality independently of the other three mechanisms.

The formula's foundational argument is populational: sleep onset failure is not, for the majority of affected adults, a disease requiring pharmaceutical sedation. It is a civilisational mismatch between biological timing and modern environments, experienced by an estimated 60 to 80% of the working population. Seven tables provided in this paper build this case in logical sequence: the epidemiology table establishes the population-level scale; the sleep architecture table connects the formula's mechanisms to specific biological functions of each sleep phase; the ingredient architecture table documents confirmed doses at both flexible dose levels; the target

population table identifies the six specific groups who benefit most and why; the symptom-to-mechanism table connects lived experience to biological targets; the comparative analysis documents the mechanistic distinction from conventional sleep aids; and the medical versus misalignment table clarifies who the formula serves and who requires clinical evaluation. This paper enters the permanent scientific record as the first description of its kind.

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Conflicts of Interest

The author is the founder and principal formulator of the EscapeMed supplement system investigated in this paper. The products are manufactured and distributed by EscapeMed d.o.o. This represents a potential conflict of interest, declared in full transparency in accordance with journal editorial policy. Escape Protocol Research is an independent research initiative. The author receives no research funding from EscapeMed d.o.o. or any commercial entity. All research is conducted independently.

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