



## ORIGINAL ARTICLE

# Chronic Sleep Deprivation as a Risk Factor for Cognitive Decline and Mood Disorders: Neurobiological Mechanisms and Therapeutic Implications

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## ABSTRAKT

Chronic sleep deprivation has emerged as a significant and modifiable risk factor for cognitive decline and mood disorders, with neurobiological consequences that extend far beyond transient impairment of alertness. Modern societies are characterised by widespread, voluntary curtailment of sleep, with surveys consistently indicating that large proportions of adults in industrialised nations sleep fewer than the recommended seven to nine hours per night (Watson et al., 2015). The consequences of this pervasive sleep deficit include measurable impairments in sustained attention, working memory, executive function, and decision-making, as well as substantially elevated risks of developing major depressive disorder, anxiety disorders, and bipolar spectrum conditions (Lim and Dinges, 2010; Baglioni et al., 2011). At the neurobiological level, sleep deprivation disrupts adenosine homeostasis, impairs prefrontal cortical function, dysregulates hypothalamic-pituitary-adrenal axis activity, elevates systemic inflammation, and suppresses hippocampal neurogenesis-mechanisms that collectively explain the breadth and severity of observed clinical effects (Xie et al., 2013; Irwin, 2015). This review synthesises current evidence on sleep physiology, the neurobiological basis of sleep deprivation effects, specific cognitive and mood consequences, vulnerable populations, diagnostic approaches, therapeutic strategies, and public health implications, with the goal of providing a comprehensive foundation for clinical recognition and intervention.

## KEYWORDS

sleep deprivation; cognitive decline; mood disorders; depression; anxiety; prefrontal cortex; adenosine; HPA axis; neuroinflammation; cognitive behavioural therapy for insomnia

## ARTICLE INFORMATION



Received: 23.06.2026



Revised: 29.08.2026



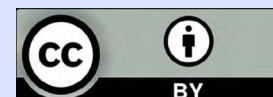
Accepted: 31.08.2026



Article type: Original Article

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## CITATION

Kzepka, H., Stępowski, P., Tajster, M. 2026. Chronic Sleep Deprivation as a Risk Factor for Cognitive Decline and Mood Disorders: Neurobiological Mechanisms and Therapeutic Implications..2026;95:73644. DOI: [DOI: 10.12775/JEHS.2026.95.73644](https://doi.org/10.12775/JEHS.2026.95.73644)

## Aim of the Work

This paper aims to provide a comprehensive, evidence-based review of the neurobiological mechanisms through which chronic sleep deprivation contributes to cognitive decline and mood disorders. Specific objectives include examining the physiology of normal sleep and circadian regulation, characterising the molecular and neural mechanisms disrupted by sleep loss, reviewing clinical evidence on cognitive and psychiatric consequences, identifying populations at heightened risk, and evaluating current diagnostic and therapeutic approaches. The paper also addresses the prevention of sleep-related cognitive and mood impairment from a public health perspective and identifies key gaps requiring further research.

## Methods and Materials

This narrative review was conducted following PRISMA guidelines for non-systematic reviews. A comprehensive search of PubMed, Scopus, and Web of Science was performed for the years 2000 to 2024, supplemented by landmark foundational studies from earlier decades. Search terms included sleep deprivation, sleep restriction, cognitive function, working memory, executive function, depression, anxiety, mood disorders, HPA axis, neuro-inflammation, glymphatic system, adenosine, circadian rhythm, CBT-I, and insomnia. Peer-reviewed original articles, systematic reviews, meta-analyses, randomised controlled trials, and official clinical guidelines were included. Sources were selected based on methodological quality, sample size, and relevance to the central questions of the review. Data were organised thematically across neurobiological, clinical, diagnostic, and therapeutic domains. Ethical approval was not required as no primary data were collected.

## Introduction

Sleep is a fundamental biological necessity, as universal across the animal kingdom as eating and reproduction, yet uniquely vulnerable to voluntary curtailment in the human species (Walker, 2017). Over the past century, average sleep duration in industrialised nations has declined by one to two hours, driven by artificial lighting, digital technology, shift work, and cultural attitudes that equate reduced sleep with productivity (Cappuccio et al., 2010). The consequences of this shift are now the subject of urgent scientific inquiry. Emerging evidence positions chronic sleep

deprivation not merely as an inconvenience but as a potent risk factor for conditions that impose substantial individual and societal burden, including cognitive decline, major depressive disorder, anxiety disorders, and neurodegenerative disease (Buysse, 2014).

Sleep deprivation is not a uniform state. It encompasses total sleep deprivation—the complete absence of sleep over one or more nights—and the far more common chronic partial sleep restriction, defined as habitually sleeping fewer hours than an individual requires for optimal functioning (Goel et al., 2009). The latter is particularly insidious because sleep-restricted individuals frequently underestimate their degree of impairment, believing subjective adaptation has occurred when objective cognitive performance continues to deteriorate (Van Dongen et al., 2003). This metacognitive blindspot renders partial sleep restriction especially dangerous in occupational settings where cognitive demands are high. The neurobiological consequences of sleep deprivation are broad and mechanistically interconnected. Adenosine accumulation during wakefulness, disruption of the glymphatic waste clearance system, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, elevation of pro-inflammatory cytokines, and suppression of hippocampal neurogenesis form a network of overlapping pathways through which sleep loss produces both cognitive impairment and psychiatric vulnerability (Xie et al., 2013; Irwin, 2015). Understanding these mechanisms is essential for appreciating why sleep deprivation produces consequences that range from impaired arithmetic to frank psychopathology, and for developing interventions that address root causes rather than symptoms.

This review integrates findings from experimental sleep deprivation studies, epidemiological cohorts, and clinical trials to provide a mechanistic and clinically actionable account of sleep deprivation as a risk factor for cognitive decline and mood disorders. It addresses the full spectrum from normal sleep physiology to diagnostic and therapeutic approaches, with particular attention to the neurobiological mechanisms that link insufficient sleep to adverse cognitive and psychiatric outcomes.

## Chapter 1 – Physiology of Sleep and Circadian Regulation

Sleep is not a passive state of reduced activity but an active, highly organised process governed by two interacting biological systems: the circadian clock and

the sleep-wakehomeostatic drive (Borbély, 1982). The circadian system, seated in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus, generates approximately 24-hour rhythms in physiology and behaviour through interlocking transcription-translation feedback loops involving core clock genes including CLOCK, BMAL1, PER, and CRY. This system synchronises internal timing to the external light-dark cycle via retinal phototransduction and drives rhythmic release of melatonin from the pineal gland during darkness, signalling biological night (Czeisler and Gooley, 2007). The homeostatic drive, in contrast, reflects the progressive accumulation of sleep pressure during wakefulness, operationalised through the rise of adenosine—a byproduct of ATP catabolism during sustained neural activity—which binds to A1 and A2A receptors and promotes sleepiness (Porkka-Heiskanen et al., 1997).

Sleep architecture is organised into recurring 90-minute cycles, each comprising non-rapid eye movement (NREM) and rapid eye movement (REM) stages. NREM sleep is subdivided into three stages, of which stage N3—characterised by high-amplitude delta oscillations—is referred to as slow-wave sleep (SWS) and is considered the most restorative phase. SWS predominates in the first half of the night and is associated with memory consolidation, synaptic downscaling, growth hormone release, and immune regulation (Diekelmann and Born, 2010). REM sleep, characterised by desynchronised electroencephalographic activity, vivid dreaming, and atonia, predominates in the second half of the night and is critically involved in emotional memory processing, affective regulation, and complex cognitive integration (Walker and van der Helm, 2009).

The functional importance of each sleep stage becomes apparent when specific stages are selectively deprived. Suppression of SWS impairs declarative me-

memory consolidation and metabolic restoration, while REM deprivation disrupts emotional regulation and procedural learning (Diekelmann and Born, 2010). In practice, chronic partial sleep restriction compresses all sleep stages proportionally, with disproportionate loss of the later, REM-rich cycles. Additionally, both circadian misalignment—the disconnection between internal biological timing and social schedules, common in shift workers and adolescents—and sleep fragmentation independently impair cognitive and emotional function even when total sleep duration appears adequate (Buysse, 2014).

## Chapter 2 – Neurobiological Mechanisms of Sleep Deprivation

The neurobiological consequences of sleep deprivation are initiated by the unchecked accumulation of adenosine in the basal forebrain and other wake-promoting centres. Under normal conditions, sleep clears accumulated adenosine, restoring alertness upon waking. When sleep is curtailed, residual adenosine load compounds across days, progressively impairing the neural circuits that sustain attention and executive control (Porkka-Heiskanen et al., 1997). Caffeine exerts its alerting effect by competitively antagonising adenosine receptors, but cannot substitute for the restorative processes of sleep itself, as it blocks the subjective experience of sleepiness without preventing the accumulation of underlying neural debt (Walker, 2017). One of the most significant neurobiological discoveries of the past decade concerns the glymphatic system—a paravascular network of astrocyte-lined channels that facilitates the bulk flow of cerebrospinal fluid through brain parenchyma, clearing metabolic waste products including amyloid-beta and tau pro-

**Table 1 . Key Features of Sleep Architecture and Their Functional Significance**

| Sleep Stage            | EEG Characteristics           | Predominance in Night | Primary Functions                                                        | Consequences of Deprivation                                            |
|------------------------|-------------------------------|-----------------------|--------------------------------------------------------------------------|------------------------------------------------------------------------|
| N1 (Light NREM)        | Theta waves (4–8 Hz)          | Early cycles          | Sleep onset transition                                                   | Increased daytime sleepiness                                           |
| N2 (Intermediate NREM) | Sleep spindles, K-complexes   | Throughout night      | Memory consolidation; sensory gating                                     | Impaired learning and motor memory                                     |
| N3 (Slow-Wave Sleep)   | Delta waves (<2 Hz)           | First half of night   | Declarative memory; metabolic restoration; immune regulation; GH release | Impaired hippocampal memory; metabolic dysfunction                     |
| REM Sleep              | Desynchronised, low-amplitude | Second half of night  | Emotional memory processing; complex cognition; affective regulation     | Emotional dysregulation; impaired associative thinking; mood disorders |

teins that accumulate during wakefulness (Xie et al., 2013). Glymphatic activity increases approximately tenfold during sleep compared with wakefulness, driven by the expansion of the interstitial space that occurs as noradrenergic tone falls during NREM sleep. Chronic sleep deprivation suppresses this clearance system, allowing the progressive accumulation of neurotoxic proteins implicated in Alzheimer's disease and other neurodegenerative conditions (Xie et al., 2013). This mechanism provides a compelling neurobiological link between sleep insufficiency and long-term cognitive decline that extends well beyond acute impairment.

The prefrontal cortex (PFC) is disproportionately sensitive to sleep loss, a finding consistently replicated across neuroimaging modalities. Functional MRI and PET studies demonstrate reduced metabolic activity in the PFC and thalamus after 24 hours of total sleep deprivation, with the magnitude of deactivation correlating with performance decrements on tasks of sustained attention and executive function (Krause et al., 2017). The PFC's vulnerability reflects its high metabolic demands and its dependence on adenosine-sensitive cholinergic innervation from the basal forebrain. Sleep-deprived individuals therefore exhibit not merely fatigue-induced slowing but a functional disconnection of the neural circuits mediating cognitive control, impulse regulation, and emotional modulation (Goel et al., 2009).

Sleep deprivation profoundly dysregulates the hypothalamic-pituitary-adrenal (HPA) axis, the principal mediator of the stress response. Acute sleep loss elevates evening cortisol concentrations, blunts the normal nocturnal cortisol nadir, and amplifies cortisol reactivity to stressors (Spiegel, Leproult and Van Cauter, 1999). Chronically elevated cortisol exerts neurotoxic effects on the hippocampus, a structure central to both episodic memory and emotional regulation: sustained glucocorticoid exposure suppresses hippocampal neurogenesis, reduces dendritic complexity, and impairs long-term potentiation—the cellular substrate of memory formation (McEwen, 2006). These HPA-mediated effects on hippocampal integrity provide a mechanistic pathway linking sleep deprivation to both memory impairment and vulnerability to depressive illness.

Sleep loss also triggers a systemic pro-inflammatory state. Experimental sleep restriction increases circulating concentrations of interleukin-6, tumour necrosis factor- $\alpha$ , and C-reactive protein within days of onset (Irwin, 2015). These cytokines cross the blood-brain barrier and modulate monoaminergic neurotransmission, reducing synaptic serotonin

and dopamine availability and contributing to the anhedonia, fatigue, and cognitive slowing characteristic of both sleep-deprived and depressed states (Irwin, 2015; Mullington et al., 2010). The bidirectionality of this relationship is clinically important: depression itself disrupts sleep architecture, and the resulting sleep impairment amplifies inflammatory activity, potentially establishing a self-perpetuating cycle of worsening mood and sleep quality.

## Chapter 3 – Effects on Cognitive Function

The cognitive consequences of sleep deprivation are broad in scope but not uniform in severity across domains. A landmark meta-analysis by Lim and Dinges (2010), synthesising results from 70 experimental studies, demonstrated that even short-term total sleep deprivation produces large effect sizes on measures of sustained attention and vigilance, moderate effects on working memory and complex attention, and smaller but significant effects on processing speed and reasoning. Critically, performance on the Psychomotor Vigilance Task—a validated measure of sustained attention sensitivity to sleep loss—declines in a dose-dependent manner with increasing sleep restriction, with individuals sleeping six hours per night for two weeks reaching impairment equivalent to one to two nights of total sleep deprivation while reporting only mild subjective sleepiness (Van Dongen et al., 2003).

Working memory—the capacity to hold and manipulate information over short intervals—is particularly impaired by sleep loss, with functional MRI studies demonstrating reduced activation of the dorsolateral PFC during working memory tasks in sleep-deprived individuals (Krause et al., 2017). This impairment has real-world consequences: reduced capacity to update mental representations, increased susceptibility to distraction, and diminished ability to follow complex instructions or manage multi-step tasks. Memory consolidation, which depends critically on NREM slow-wave sleep for hippocampus-dependent declarative memories and on REM sleep for procedural and emotional memories, is substantially impaired by sleep deprivation both before and after learning (Diekelmann and Born, 2010). Sleep-deprived students and workers therefore not only attend less effectively but encode and retain new information less efficiently.

Executive functions—encompassing planning, decision-making, cognitive flexibility, risk assessment, and impulse control—depend on prefrontal circuitry that is exquisitely sensitive to sleep loss (Goel et al., 2009).

Sleep-deprived individuals consistently demonstrate increased risk-taking behaviour, reduced inhibitory control, impaired moral reasoning, and atendency toward perseverative responding-continuing with ineffective strategies rather than adapting to changing task demands. Harrison and Home (2000) demonstrated that sleep-deprived subjects performing novel executive function tasks showed marked impairment that was not attributable simply to reduced alertness, implicating specific prefrontal vulnerability beyond general cognitive slowing. These executive impairments have documented consequences in clinical, aviation, transport, and military settings, where high-stakes decisions under conditions of sleep deprivation have been linked to serious adverse events (Walker, 2017).

The phenomenon of microsleeps-brief, involuntary episodes of sleep lasting two to fifteen seconds that occur during wakefulness in severely sleep-deprived individuals-represents one of the most dangerous cognitive consequences of sleep loss (Krause et al., 2017). During microsleeps, environmental monitoring ceases entirely, rendering the individual unable to respond to stimuli that emerge during the episode. Microsleeps are particularly hazardous in transport contexts: drowsy driving produces impairment comparable to legal intoxication, with a blood-alcohol equivalent of 0.05 to 0.08 percent after seventeen to nineteen hours of wakefulness (Williamson and Feyer, 2000). Despite this, subjective underestimation of impairment remains a consistent finding, as sleep-deprived individuals believe themselves more capable than objective measures confirm (Van Dongen et al., 2003).

## Chapter 4 – Sleep Deprivation and Mood Disorders

The relationship between sleep deprivation and mood disorders is bidirectional, dynamic, and supported by convergent evidence from epidemiology, experimental psychology, and clinical psychiatry. Insomnia-the most prevalent manifestation of chronic sleep insufficiency-is now recognised as both a symptom and an independent risk factor for depressive illness. A meta-analysis of 21 longitudinal epidemiological studies by Baglioni et al. (2011) demonstrated that non-depressed individuals with insomnia had approximately twice the risk of developing depression compared with those without sleep difficulties, independent of other known risk factors. This finding has been replicated and extended in subsequent meta-analyses, with pooled relative risks for incident depression in the range of 2.1 to 2.3 (Baglioni et al., 2011).

Experimental sleep deprivation provides mechanistic insight into this relationship. The amygdala-a limbic structure central to threat detection and emotional reactivity-shows markedly increased responsivity to negative emotional stimuli in sleep-deprived individuals, with functional MRI studies demonstrating sixty percent greater amygdala activation compared with well-rested controls (Yoo et al., 2007). Simultaneously, functional connectivity between the amygdala and the medial PFC, the circuit that normally provides top-down regulation of emotional responses, is substantially attenuated by sleep loss (Krause et al., 2017). This combined pattern-amplified limbic reactivity with weakened prefrontal control-mirrors the

**Table 2.** Cognitive Domains Affected by Chronic Sleep Deprivation and Mechanisms of Impairment

| Cognitive Domain                     | Degree of Impairment  | Primary Neural Substrate              | Key Mechanism                                         |
|--------------------------------------|-----------------------|---------------------------------------|-------------------------------------------------------|
| Sustained attention / vigilance      | Severe (large effect) | Thalamus, prefrontal cortex           | Adenosine accumulation; PFC deactivation              |
| Working memory                       | Moderate to severe    | Dorsolateral prefrontal cortex        | Reduced PFC activation; dopamine dysregulation        |
| Executive function / decision-making | Moderate to severe    | Prefrontal cortex, anterior cingulate | Prefrontal vulnerability; impaired inhibitory control |
| Declarative memory consolidation     | Moderate              | Hippocampus, neocortex                | Loss of SWS; reduced hippocampal replay               |
| Emotional memory processing          | Moderate              | Amygdala, prefrontal cortex           | REM deprivation; amygdala hyperreactivity             |
| Processing speed                     | Mild to moderate      | Widespread cortical networks          | General metabolic suppression                         |
| Creative and associative thinking    | Moderate              | Default mode network, PFC             | REM deprivation; disrupted network connectivity       |

neuroimaging signature of major depressive disorder and provides a mechanistic explanation for the irritability, emotional lability, and negativity bias consistently observed in sleep-deprived individuals.

The relationship between sleep and anxiety disorders follows similar patterns. Sleep deprivation heightens anticipatory anxiety, reduces emotional habituation to repeated aversive stimuli, and impairs extinction learning—the process by which conditioned fear responses are gradually attenuated through repeated unreinforced exposure (Harvey et al., 2011). These effects are particularly relevant to post-traumatic stress disorder, in which REM sleep disturbance is a cardinal feature and may perpetuate the failure of fear extinction that maintains trauma-related psychopathology. Longitudinal data indicate that sleep disturbance in the immediate aftermath of trauma is among the strongest predictors of subsequent PTSD development, suggesting that targeting sleep early in the post-traumatic period may offer preventive as well as therapeutic benefits (Harvey et al., 2011).

Bipolar disorder exhibits one of the most complex relationships with sleep. Disruptions to the circadian system—including delayed sleep phase, reduced total sleep time, and irregular sleep-wake schedules—are present during all phases of the illness, not merely during acute episodes (Harvey et al., 2011). Sleep deprivation can precipitate manic episodes in vulnerable individuals, a phenomenon exploited therapeutically in wake therapy but also reflecting the profound sensitivity of mood regulation circuitry to homeostatic sleep pressure. Conversely, hypersomnia characterises depressive episodes in a substantial minority of patients with bipolar depression, illustrating that both quantitative excess and deficiency of sleep can disrupt affective stability. Understanding the bidirectionality of these relationships is essential for clinical management: treating sleep disturbance is not merely a symptomatic adjunct but a potentially disease-modifying intervention (Harvey et al., 2011; Baglioni et al., 2011).

The neurochemical basis of sleep deprivation-induced mood disturbance centres on monoaminergic and inflammatory pathways. Serotonin synthesis and release are sleep-state dependent, with SWS promoting serotonergic activity and sleep deprivation reducing serotonin turnover in limbic circuits—an effect that parallels the neurochemical signature of depressive illness (Irwin, 2015). Dopaminergic reward signalling is similarly disrupted, producing blunted responses to pleasurable stimuli that manifest clinically as anhedonia. The inflammatory cytokines elevated by sleep loss—particularly interleukin-6 and tumour necrosis factor- $\alpha$ —further suppress monoamine availability through

induction of the enzyme indoleamine 2,3-dioxygenase, which diverts tryptophan away from serotonin synthesis toward the kynurenine pathway, generating neuroactive metabolites associated with depression and cognitive dysfunction (Mullington et al., 2010; Irwin, 2015).

## Chapter 5 – Special Populations and Risk Groups

While sleep deprivation produces cognitive and mood effects across all demographic groups, specific populations face disproportionate risk owing to biological, occupational, or social vulnerabilities. Adolescents represent a particularly important group: puberty is associated with a biological delay in circadian phase, causing a genuine shift toward later sleep and wake times that conflicts with early school start times in most educational systems (Carskadon, 2011). The resulting chronic sleep restriction during a critical period of prefrontal cortical maturation has documented consequences for academic performance, emotional regulation, risk-taking behaviour, and mental health, with large epidemiological studies linking short sleep duration in adolescents to increased rates of depression, anxiety, and suicidal ideation (Carskadon, 2011).

Shift workers constitute a large and systematically sleep-deprived occupational group. Rotation through night, evening, and day shifts disrupts circadian entrainment, fragments sleep, and reduces total sleep time. Chronic circadian misalignment is associated not only with acute cognitive impairment and elevated accident risk but with long-term increased incidence of depressive disorders, metabolic syndrome, cardiovascular disease, and certain cancers (Cappuccio et al., 2010). Medical personnel, a group particularly prone to extended wakefulness through on-call duties, show marked impairment in clinical decision-making, procedural accuracy, and empathic communication following sleep deprivation, with consequences for patient safety that have driven regulatory reforms in resident working hours in many countries (Walker, 2017).

Older adults experience significant changes in sleep architecture with ageing, including reduced SWS, increased sleep fragmentation, earlier circadian phase, and diminished sleep efficiency (Buysse, 2014). These age-related changes amplify vulnerability to the cognitive consequences of additional sleep disruption. Importantly, impaired glymphatic clearance of amyloid- $\beta$  and tau proteins—which depends on the interstitial space expansion that occurs during SWS—may partially explain the association between sleep

disturbance and accelerated cognitive decline in older adults (Xie et al., 2013). Longitudinal cohort studies consistently demonstrate that self-reported short sleep duration and poor sleep quality in midlife predict higher rates of cognitive impairment and dementia in later decades (Yaffe et al., 2014).

Individuals with pre-existing psychiatric conditions are at compounded risk. Insomnia is present in approximately eighty percent of patients with major depressive disorder, seventy percent of those with anxiety disorders, and nearly all patients with bipolar disorder during acute episodes (Harvey et al., 2011). In these individuals, sleep disturbance both reflects and amplifies the underlying psychopathology, reducing treatment responsiveness and increasing relapse risk. Pregnant women face elevated rates of sleep disruption due to physical discomfort, nocturnal awakening, and hormonal changes, with gestational sleep disturbance associated with higher rates of perinatal depression and impaired cognitive performance (Baglioni et al., 2011). Children with neurodevelopmental conditions including ADHD and autism spectrum disorder also exhibit substantially higher rates of sleep problems, with bidirectional relationships between sleep quality and the core features of these conditions.

## Chapter 6 – Assessment and Diagnosis

Accurate assessment of sleep deprivation and its cognitive and mood consequences requires integration of

subjective and objective measures, as self-report alone is systematically unreliable in sleep-deprived populations (Van Dongen et al., 2003). The clinical interview remains fundamental and should routinely address habitual sleep duration, sleep quality, sleep onset latency, nocturnal awakenings, daytime sleepiness, and functional impairment. Standardised questionnaires provide validated and reproducible estimates of sleep quantity and quality: the Pittsburgh Sleep Quality Index (PSQI) assesses seven components of sleep over the preceding month and has excellent psychometric properties in both clinical and research settings (Buysse et al., 1989). The Epworth Sleepiness Scale quantifies the tendency to fall asleep in everyday situations and provides a sensitive measure of pathological daytime sleepiness (Johns, 1991).

For insomnia specifically, the Insomnia Severity Index offers a brief seven-item measure that is sensitive to treatment change and widely used in clinical trials. Mood screening instruments are equally essential given the high comorbidity between sleep disorders and psychiatric conditions: the Patient Health Questionnaire-9 (PHQ-9) for depression and the Generalized Anxiety Disorder-7 (GAD-7) for anxiety provide validated clinical cut-offs that guide further evaluation. When sleep-disordered breathing is suspected as a contributing or primary cause—particularly in patients who report non-restorative sleep, witnessed apnoeas, or excessive daytime sleepiness despite adequate time in bed—polysomnography or home sleep apnoea testing is indicated (Sateia, 2014).

**Table 3.** Principal Assessment Tools for Sleep, Cognitive Function, and Mood in Clinical Practice

| Tool                                  | Domain Assessed                                            | Format                        | Clinical Use                                              |
|---------------------------------------|------------------------------------------------------------|-------------------------------|-----------------------------------------------------------|
| Pittsburgh Sleep Quality Index (PSQI) | Sleep quality, latency, duration, efficiency, disturbances | Self-report, 19 items         | Screening and monitoring across clinical populations      |
| Epworth Sleepiness Scale (ESS)        | Daytime sleepiness                                         | Self-report, 8 items          | Screening for pathological sleepiness; sleep apnea triage |
| Insomnia Severity Index (ISI)         | Insomnia symptoms and impact                               | Self-report, 7 items          | Insomnia screening; treatment response monitoring         |
| Polysomnography (PSG)                 | Sleep architecture, respiratory events, limb movements     | Objective, laboratory-based   | Definitive diagnosis of sleep disorders                   |
| Actigraphy                            | Sleep-wake patterns over days to weeks                     | Objective, ambulatory         | Real-world sleep assessment; circadian pattern evaluation |
| Psychomotor Vigilance Task (PVT)      | Sustained attention, reaction time                         | Computerised performance test | Objective cognitive assessment; drowsiness quantification |
| PHQ-9 / GAD-7                         | Depression / anxiety severity                              | Self-report, 9 / 7 items      | Psychiatric comorbidity screening and monitoring          |

Actigraphy, which uses wrist-worn accelerometry to estimate sleep-wake patterns over days to weeks, provides objective data on sleep timing, duration, and regularity that complements polysomnographic assessment and captures variability across real-world conditions (Buysse, 2014). Cognitive assessment should accompany sleep evaluation when clinically indicated, as the pattern of impairment—particularly deficits in sustained attention and working memory disproportionate to general intellectual function—can help attribute cognitive symptoms to sleep deprivation versus primary neurodegenerative or psychiatric causes. Neuropsychological batteries including the Psychomotor Vigilance Task, the Trail Making Test, and digit span tasks provide sensitive and reproducible measures of the cognitive domains most affected by sleep loss (Goel et al., 2009).

## Chapter 7 – Management and Therapeutic Strategies

The treatment of sleep deprivation-related cognitive and mood impairment requires a stratified approach that begins with addressing the underlying sleep disorder or behavioural sleep restriction, and extends to managing the cognitive and psychiatric sequelae where they have become clinically established. Across all treatment settings, adequate sleep is itself a therapeutic target: no pharmacological or psychological intervention for cognitive impairment or mood disorders is likely to achieve its full efficacy in the presence of ongoing significant sleep loss (Walker, 2017; Harvey et al., 2011).

Cognitive Behavioural Therapy for Insomnia (CBT-I) is the first-line treatment for chronic insomnia according to guidelines from the American Academy of Sleep Medicine, the European Sleep Research Society, and the National Institute for Health and Care Excellence (Riemann et al., 2017). CBT-I combines several evidence-based components: sleep restriction therapy, which consolidates sleep by initially limiting time in bed to match actual sleep time; stimulus control, which re-establishes the bed as a cue for sleep rather than wakefulness; sleep hygiene education; relaxation techniques; and cognitive restructuring to address maladaptive beliefs about sleep. Meta-analyses of randomised controlled trials consistently demonstrate that CBT-I produces durable improvements in sleep onset latency, wake after sleep onset, and sleep efficiency superior to pharmacological treatments, with benefits that are maintained at long-term follow-up and extend to comorbid depression and anxiety (Rie-

mann et al., 2017; Morin et al., 2006).

The effect of successfully treating insomnia on comorbid depression is clinically significant and has been established in randomised controlled trials. Treating insomnia with CBT-I in patients with comorbid depression produces greater antidepressant response and higher remission rates than treating depression alone, and reduces depressive relapse rates during follow-up (Manber et al., 2008). This finding shifts the clinical paradigm from viewing insomnia as a mere symptom of depression to treating it as an independent therapeutic target with disease-modifying potential. Similarly, CBT-I has demonstrated reductions in anxiety symptoms, improvements in quality of life, and modest reductions in PTSD symptom severity when delivered as an adjunct to trauma-focused therapies (Harvey et al., 2011).

Pharmacological sleep aids are indicated in specific circumstances but carry important limitations. Benzodiazepine receptor agonists—including the Z-drugs zopiclone, zolpidem, and zaleplon—reduce sleep onset latency and nocturnal awakenings through GABA-A receptor potentiation, but do not reliably enhance the restorative slow-wave and REM sleep stages most critical for cognitive and emotional restoration, and carry risks of tolerance, dependence, rebound insomnia, and cognitive side effects particularly in older adults (Sateia, 2014). Melatonin receptor agonists, including melatonin itself and ramelteon, are better suited for circadian-related insomnia and sleep phase disorders. Low-dose doxepin, a histamine H1 antagonist, has demonstrated efficacy for sleep maintenance insomnia with a favourable safety profile. Orexin receptor antagonists—including suvorexant and lemborexant—represent a mechanistically novel class that selectively suppresses wakefulness-promoting orexin signalling and preserves natural sleep architecture more closely than GABA-A agonists (Sateia, 2014).

Light therapy is an evidence-based intervention for circadian sleep disorders, seasonal affective disorder, and non-seasonal depression, particularly in contexts where circadian misalignment contributes to mood disturbance. Morning bright light exposure (2,500–10,000 lux for 20–30 minutes) advances circadian phase, suppresses melatonin, and exerts antidepressant effects through serotonergic pathways, making it particularly useful for delayed sleep phase syndrome and winter depression (Golden et al., 2005). For shift workers and travellers with circadian disruption, structured light exposure protocols combined with strategic melatonin administration can accelerate circadian re-entrainment and partially mitigate cognitive impairment.

Physical activity deserves recognition as a non-pharmacological adjunct with bidirectional benefits for sleep and mood. Regular aerobic exercise increases SWS duration, reduces sleep onset latency, and improves subjective sleep quality through multiple mechanisms including core body temperature regulation, adenosine metabolism, and HPA axis modulation (Kredlow et al., 2015). The antidepressant effect of regular exercise, comparable to that of antidepressant medication in mild to moderate depression, synergises with sleep improvement to produce comprehensive mood benefits. Mindfulness-based stress reduction programmes have similarly demonstrated efficacy in reducing insomnia severity and depressive symptoms, operating through attentional and emotional regulation pathways that overlap substantially with the mechanisms disrupted by sleep loss.

## Chapter 8 – Prevention and Public Health Implications

The scale of sleep deprivation as a public health challenge is substantial. Population surveys across Europe and North America indicate that between thirty and forty percent of adults habitually sleep fewer than seven hours per night, below the minimum recommended by a joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society (Watson et al., 2015). The Centers for Disease Control and Prevention in the United States has declared insufficient sleep a public health epidemic, and comparable concerns have been articulated by health authorities in the United Kingdom and across the European Union (Cappuccio et al., 2010). The economic burden attributable to sleep disorders and inadequate sleep—through reduced productivity, workplace errors, traffic accidents, healthcare utilisation, and premature mortality—has been estimated in the hundreds of billions of dollars annually in major economies.

Primary prevention of sleep deprivation requires addressing its structural and behavioural determinants. School start time policies represent a high-impact, evidence-based public health intervention: delaying high school start times by 30 to 60 minutes produces measurable improvements in sleep duration, academic performance, mental health indicators, and road traffic accident rates among adolescents, with modelling studies suggesting highly favourable cost-benefit ratios (Carskadon, 2011). Workplace policies

that limit shift work duration, mandate adequate rest periods between shifts, and restrict overnight working hours reduce both occupational accident risk and the long-term health burden of circadian disruption (Cappuccio et al., 2010). Regulatory reform of medical training hours, while controversial in its effects on clinical experience, has demonstrably reduced the frequency of serious medical errors attributable to sleep-deprived physicians.

Digital technology use in the hours preceding sleep is a modifiable contributor to sleep delay and curtailment across age groups. Blue-spectrum light emitted by screens suppresses melatonin secretion and delays circadian phase, while the cognitively and emotionally stimulating content of social media, news, and entertainment platforms maintains arousal incompatible with sleep onset (Buysse, 2014). Public health messaging that addresses screen hygiene—including device-free bedrooms, night mode settings, and defined screen-free periods before bed—represents a cost-effective population-level intervention that can be delivered through schools, workplaces, and healthcare settings.

Within healthcare systems, routine enquiry about sleep duration and quality during primary care consultations remains uncommon despite the well-established associations between sleep insufficiency and major chronic disease (Buysse, 2014). Integrating validated brief sleep screening—such as the PSQI or single-item sleep duration questions—into standard health assessments would enable earlier identification of at-risk individuals and timely referral to sleep medicine or psychological services. Given that effective treatments for insomnia—particularly CBT-I—are underutilised relative to their evidence base, expansion of digital CBT-I platforms represents a scalable approach to extending access to first-line care beyond the capacity of specialist sleep services (Riemann et al., 2017).

Future public health approaches to sleep deprivation must grapple with the social and occupational determinants that render adequate sleep difficult for many individuals regardless of individual intention. These include unsociable working hours, long commuting times, caregiving responsibilities, financial stress, and neighbourhood factors such as noise and light pollution that impair sleep in lower socio-economic communities. A comprehensive public health framework therefore requires not only individual behaviour change strategies but structural policies that create conditions in which adequate sleep is socially and economically feasible for all population groups.

(Cappuccio et al., 2010; Buysse, 2014).

## Conclusion

Chronic sleep deprivation constitutes a pervasive and largely preventable risk factor for cognitive decline and mood disorders, acting through neurobiological mechanisms of considerable depth and clinical consequence. The progressive accumulation of adenosine, the suppression of glymphatic waste clearance, the dysregulation of the HPA axis, the elevation of neuro-inflammatory cytokines, and the functional deactivation of the prefrontal cortex collectively explain how insufficient sleep produces impairments spanning attention, memory, executive function, emotional regulation, and psychiatric vulnerability (Xie et al., 2013; Lim and Dinges, 2010; Irwin, 2015).

The epidemiological evidence linking sleep deprivation to major depressive disorder and anxiety disorders is robust and consistent, with meta-analytic data establishing that insomnia approximately doubles the risk of incident depression independent of other risk factors (Baglioni et al., 2011). These associations are not merely correlational: experimental and clinical evidence demonstrates that improving sleep quality through CBT-I produces clinically meaningful reductions in depressive and anxiety symptoms, repositioning sleep as a modifiable treatment target rather than a passive symptom of psychiatric illness (Riemann et al., 2017; Manber et al., 2008).

Certain populations—adolescents, shift workers, older adults, pregnant women, and individuals with pre-existing psychiatric conditions—bear a disproportionate burden of sleep deprivation and its consequences, warranting targeted screening and intervention strategies. The diagnostic toolkit available to clinicians is comprehensive, spanning validated questionnaires, polysomnography, actigraphy, and cognitive assessment, and its routine integration into primary care could substantially reduce the time between sleep disorder onset and effective intervention (Sateia, 2014; Buysse et al., 1989).

At the public health level, the challenge is substantial but not intractable. Evidence-based structural interventions including delayed school start times, regulated shift work practices, and expanded access to digital CBT-I have demonstrated measurable benefits in real-world settings and offer favourable cost-benefit profiles (Carskadon, 2011; Riemann et al., 2017). Future research priorities include elucidating the precise threshold of sleep deprivation at which irreversible neurobiological change occurs, characterising the

genetic determinants of inter-individual variability in sleep deprivation susceptibility, and developing biomarkers of glymphatic dysfunction that might enable early identification of those at highest risk for sleep-deprivation-related neurodegeneration. Recognising adequate sleep not as a luxury but as a foundational pillar of cognitive and mental health is the essential first step toward addressing one of the most consequential and under-recognised public health challenges of contemporary society.

## References

- Baglioni, C. et al. (2011) 'Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies,' *Journal of Affective Disorders*, 135(1–3), pp. 10–19.
- Borbély, A.A. (1982) 'A two process model of sleep regulation,' *Human Neurobiology*, 1(3), pp. 195–204.
- Buysse, D.J. (2014) 'Sleep health: can we define it? Does it matter?' *Sleep*, 37(1), pp. 9–17.
- Buysse, D.J. et al. (1989) 'The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research,' *Psychiatry Research*, 28(2), pp. 193–213.
- Cappuccio, F.P. et al. (2010) 'Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies,' *Sleep*, 33(5), pp. 585–592.
- Carskadon, M.A. (2011) 'Sleep in adolescents: the perfect storm,' *Pediatric Clinics of North America*, 58(3), pp. 637–647.
- Czeisler, C.A. & Gooley, J.J. (2007) 'Sleep and circadian rhythms in humans,' *Cold Spring Harbor Symposia on Quantitative Biology*, 72, pp. 579–597.
- Diekelmann, S. & Born, J. (2010) 'The memory function of sleep,' *Nature Reviews Neuroscience*, 11(2), pp. 114–126.
- Goel, N. et al. (2009) 'Neurocognitive consequences of sleep deprivation,' *Seminars in Neurology*, 29(4), pp. 320–339.
- Golden, R.N. et al. (2005) 'The efficacy of light therapy in the treatment of mood disorders: a review and meta-analysis of the evidence,' *American Journal of Psychiatry*, 162(4), pp. 656–662.
- Harrison, Y. & Horne, J.A. (2000) 'The impact of sleep deprivation on decision making: a review,' *Journal of Experimental Psychology: Applied*, 6(3), pp. 236–249.
- Harvey, A.G. et al. (2011) 'Sleep and circadian functioning: critical mechanisms in the mood disorders?' *Annual Review of Clinical Psychology*, 7, pp. 297–319.
- Irwin, M.R. (2015) 'Why sleep is important for health: a psychoneuroimmunology perspective,' *Annual Review of Psychology*, 66, pp. 143–172.

- Johns, M.W. (1991) ,A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale,' *Sleep*, 14(6), pp. 540–545.
- Kredlow, M.A. et al. (2015) ,The effects of physical activity on sleep: a meta-analytic review,' *Journal of Behavioral Medicine*, 38(3), pp. 427–449.
- Krause, A.J. et al. (2017) ,The sleep-deprived human brain,' *Nature Reviews Neuroscience*, 18(7), pp. 404–418.
- Lim, J. & Dinges, D.F. (2010) ,A meta-analysis of the impact of short-term sleep deprivation on cognitive variables,' *Psychological Bulletin*, 136(3), pp. 375–389.
- Manber, R. et al. (2008) ,Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia,' *Sleep*, 31(4), pp. 489–495.
- McEwen, B.S. (2006) ,Sleep deprivation as a neurobiologic and physiologic stressor: allostasis and allostatic load,' *Metabolism*, 55(10 Suppl 2), pp. S20–S23.
- Morin, C.M. et al. (2006) ,Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004),' *Sleep*, 29(11), pp. 1398–1414.
- Mullington, J.M. et al. (2010) ,Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation,' *Progress in Cardiovascular Diseases*, 51(4), pp. 294–302.
- Porkka-Heiskanen, T. et al. (1997) ,Adenosine: a mediator of the sleep-inducing effects of prolonged wakefulness,' *Science*, 276(5316), pp. 1265–1268.
- Riemann, D. et al. (2017) ,European guideline for the diagnosis and treatment of insomnia,' *Journal of Sleep Research*, 26(6), pp. 675–700.
- Sateia, M.J. (2014) ,International Classification of Sleep Disorders: highlights and modifications,' *Chest*, 146(5), pp. 1387–1394.
- Spiegel, K., Leproult, R. & Van Cauter, E. (1999) ,Impact of sleep debt on metabolic and endocrine function,' *The Lancet*, 354(9188), pp. 1435–1439.
- Van Dongen, H.P.A. et al. (2003) ,The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation,' *Sleep*, 26(2), pp. 117–126.
- Walker, M.P. (2017) *Why We Sleep: Unlocking the Power of Sleep and Dreams*. New York: Scribner.
- Walker, M.P. & van der Helm, E. (2009) ,Overnight therapy? The role of sleep in emotional brain processing,' *Psychological Bulletin*, 135(5), pp. 731–748.
- Watson, N.F. et al. (2015) ,Recommended amount of sleep for a healthy adult: a joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society,' *Sleep*, 38(6), pp. 843–844.
- Williamson, A.M. & Feyer, A.M. (2000) ,Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication,' *Occupational and Environmental Medicine*, 57(10), pp. 649–655.
- Xie, L. et al. (2013) ,Sleep drives metabolite clearance from the adult brain,' *Science*, 342(6156), pp. 373–377.
- Yaffe, K. et al. (2014) ,Sleep-disordered breathing, hypoxia, and risk of mild cognitive impairment and dementia in older women,' *JAMA*, 306(6), pp. 613–619.

**Author's Contribution:****Conceptualization:** HRZ,PS,MT**Methodology:** HRZ,PS,MT**Formal analysis:** HRZ,PS,MT**Investigation:** HRZ,PS,MT**Supervision:** HRZ,PS,MT**Writing-rough preparation:** HRZ,PS,MT**Writing review and editing:** HRZ,PS,MT**Receiving funding:** not applicable

All authors have read and agreed with the published version of the manuscript.

**Funding:** The article did not receive any funding.**Institutional Review and Board Statement:** Not applicable.**Informed Consent Statement:** Not applicable.**Data Availability Statement:** Not applicable.**Conflict of Interest Statement:** Not applicable.

**AI Disclosure:** Artificial intelligence tools were utilized in this study in three distinct roles. First, they supported the analysis of clinical reasoning narratives by identifying recurring linguistic patterns associated with specific logical fallacies. Second, they contributed to improving the manuscript's academic language, enhancing clarity, precision, and adherence to established scientific writing standards. Third, they assisted in organizing and preprocessing the data, thereby streamlining the overall analytical workflow. Throughout the study, AI tools functioned exclusively as supportive instruments under continuous human supervision. The interpretation of findings, the identification and classification of reasoning errors, and the development of conclusions were carried out solely by experts in clinical medicine and formal logic. AI was used primarily to increase efficiency in tasks such as data management, pattern recognition, and language refinement, and it did not replace human judgment at any stage of the research.