Wired and Tired: Sleep Science from Neurobiology to Daily Struggles

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Abstract

Sleep is a complex biological process regulated by neural systems, circadian rhythms, environmental cues and more. Poor sleep quality has become a global public health concern, affecting nearly one-third of adults worldwide. This paper provides an overview of sleep biology, the causes of poor sleep, and its consequences for mental and physiological health. It outlines the structure and regulation of healthy sleep, followed by biological, behavioral, environmental, and genetic disruptions that can worsen sleep quality. It then examines common clinical sleep disorders - such as obstructive sleep apnea, insomnia, narcolepsy, hypersomnolence disorder and REM sleep behavior disorder - and reviews current medications. This paper aims to provide an interdisciplinary overview of sleep biology, explore the complex causes of poor sleep, and examine how its disruption affects nearly every aspect of human functioning.

Introduction

Sleep is a complex and essential biological process that sustains mental, emotional, and physiological health. Global surveys estimate that nearly one-third of adults experience sleep-related difficulties, making it a growing public health concern. While the importance of sleep is widely acknowledged, many questions remain about what defines healthy sleep, why it becomes disrupted, and how the body and brain respond when it does.

This paper explores these questions in three parts. First, it examines the biology of sleep, including its neural architecture, circadian regulation, and indicators of sleep quality. Second, it identifies biological, behavioral, environmental, and genetic factors that contribute to poor sleep. Finally, it investigates the wide-ranging consequences of sleep loss, along with five common sleep disorders and the medications used to manage them. Through this interdisciplinary lens, the paper aims to deepen understanding of sleep health and provide insight into how disrupted sleep can impact individual lives.

What is Sleep?

Sleep plays a critical role in cognitive functioning, mood regulation, metabolism, and overall well-being. In order to understand what makes poor or disrupted sleep, it is important to first understand what good-quality sleep looks like. This section outlines the architecture of sleep, how it is regulated by neural systems, its connection to circadian rhythms and sleep homeostasis, and the physiological and subjective markers of sleep quality.

Sleep has a highly structured and cyclical architecture that reflects the brain's dynamic activity during rest. The cycles are grouped into Non-Rapid Eye Movement (NREM) and Rapid Eye Movement (REM) stages, each with its own characteristics and function.

NREM sleep is divided into three stages:

- *N1*: the lightest stage, when "drifting off to sleep" occurs, dreams may spontaneously begin, and muscles may move in a jerky motion called hypnic jerks or sleep starts (Cuellar et al., 2015);

- *N2*: a deeper stage marked by sleep spindles and K-complexes (brief bursts of brainwave activity that occur during NREM sleep, specifically stage 2 sleep);
- *N3*: also known as slow-wave or deep sleep, characterized by delta waves (the slowest recorded brain waves in human beings; they are found most often in infants and young children, and are associated with the deepest levels of relaxation and restorative, healing sleep (Surawicz & Knilans, 2008)) on EEG.

These stages are essential for physical recovery, immune function, and metabolic regulation. REM sleep, on the other hand, is characterized by brain functioning very close to wakefulness. It is associated with dreaming, emotional regulation, and memory consolidation.

NREM and REM stages alternate in ~90-minute cycles throughout the night. Transitions between them are regulated by complex connections among brain regions like the thalamus, which controls sensory input and sleep stages, and the brainstem, which helps regulate transitions and autonomic functions like breathing and body temperature. Maintaining this cycle is essential for restorative sleep.

Neural Regulation of Sleep

Sleep is a complex process involving multiple brain systems working together. It is regulated by an interconnected network of brain regions, primarily the hypothalamus, brainstem, thalamus, and cerebral cortex (M. D. Schwartz & Kilduff, 2015). These regions interact to manage both the timing and architecture of sleep, including its various stages.

The hypothalamus, a regulator of homeostasis, is especially important for aligning sleep with the body's circadian rhythms. Without it, many functions, like body temperature, hunger, hormone release, and sleep timing, would be dysregulated (Moore, 2007). Within the hypothalamus, the suprachiasmatic nucleus (SCN) acts as the brain's clock. It aligns internal biological rhythms with external cues, known as zeitgebers, like light and temperature. The SCN receives input from specialized retinal ganglion cells, which allows the SCN to synchronize the sleep-wake cycle to the environment.

The brainstem, essential for basic survival, plays an important role in regulating basic physiological processes such as heart rate, breathing, and thermoregulation. It also helps initiate and maintain REM sleep by sending signals to the cerebral cortex and influencing transitions between sleep stages (Wang et al., 2021).

The cerebral cortex, responsible for high-level functions like perception, decision-making, and memory, becomes largely deactivated during slow-wave sleep (SWS) (Mitra et al., 2016). This "rest mode" allows for restorative processes to occur. Molecular processes in the cortex during sleep play a crucial role in memory consolidation and learning, which makes deep sleep a very important stage for cognitive restoration (Langille, 2019).

The thalamus acts as a relay center for sensory information. During sleep, thalamus filters incoming sensory signals - meaning it reduces the transmission of external stimuli to the cortex, helping a person stay asleep. It also generates sleep spindles, which are bursts of brain activity

seen during non-REM sleep (NREM) (Fernandez & Lüthi, 2020). They are crucial for memory consolidation.

Overall, sleep is controlled by an interconnected system of different brain regions. Because of this complexity, the regulation of sleep involves both circadian rhythms and the buildup of sleep pressure (homeostatic sleep need), which are further explored in the following sections.

Circadian Rhythms

Circadian rhythms are roughly 24-hour internal cycles that influence a wide range of physiological functions, such as hormone secretion, body temperature, and the sleep-wake cycle. CR helps prepare the body for sleep and wake and is regulated by an internal biological clock and external environmental cues.

A group of core clock genes, including PER, CRY, BMAL1, and CLOCK, are responsible for the internal biological circadian rhythm. These genes use transcriptional-translational feedback loops to create and control circadian rhythms. In this system, proteins produced by these genes regulate the timing of their own expression. For example, CLOCK and BMAL1 activate the transcription of PER and CRY, whose proteins eventually inhibit CLOCK/BMAL1 activity, creating a ~24-hour feedback cycle. This molecular process happens in nearly every cell of the body, but is coordinated by the suprachiasmatic nucleus (SCN). The SCN has a natural rhythm that is slightly longer than 24 hours - about 24.2 hours - so it requires regular resetting by zeitgebers, especially light, to stay in sync with the external environment. This synchronization ensures that physiological processes, such as sleep and alertness, occur at biologically advantageous times of day.

Sleep disorders can result from circadian rhythm disruptions of these environmental cues, such as those caused by work shifts or exposure to artificial light at night. Sleep disorders have been associated with various health issues, including metabolic disorders and cardiovascular diseases.

Homeostatic sleep need

Homeostatic sleep need refers to the body's internal drive to sleep. It builds up the longer a person stays awake. This process ensures that the body maintains a balance between time spent awake and time spent asleep. An important part of this process is the accumulation of adenosine. It is a neuromodulator that increases in the brain during wakefulness and promotes sleepiness by inhibiting arousal systems. During sleep, adenosine levels decrease, which reduces sleep pressure. After sleep deprivation, the homeostatic system increases both the intensity and duration of slow-wave sleep to compensate. Disruptions to this system, such as through caffeine intake (which blocks adenosine receptors), irregular sleep schedules, or chronic sleep restriction, can worsen the body's ability to recover and affect attention, memory, and mood.

Quality of Sleep

Quality sleep is defined based on several factors, including sleep duration, sleep continuity, and the subjective feeling of restfulness upon waking. High-quality sleep includes both objective physiological markers and subjective experiences (Table 1).

Subjectively, people describe quality sleep as waking up refreshed and alert. In research, this is often measured by cognitive performance tests, such as reaction time assessments and memory tasks (Di Muzio et al., 2020). Studies have shown that individuals who report high sleep quality tend to perform better on Psychomotor Vigilance Tasks (PVTs; Matsangas and Shattuck, 2020). PVTs are used in sleep research to assess attention and behavioral alertness. During these tests, participants are asked to respond as quickly as they can to a visual stimulus that appears on a screen at random intervals of time. The task allows researchers to detect variations in alertness and vigilance, particularly in the context of sleep deprivation or circadian rhythm disruptions (Basner et al., 2011). PVT performance is evaluated through metrics such as reaction time and false alarms. These indicators provide insight into the cognitive impairments associated with insufficient or poor-quality sleep. Studies have demonstrated that even a single night of sleep loss can result in measurable declines in PVT performance, including increased lapses (defined as a failure to react or any reaction exceeding 500 msec), making it a valuable tool in both laboratory and field settings (Basner & Dinges, 2011).

Sleep quality is not only shaped by circadian and homeostatic systems but is also influenced by brain activity and environmental/lifestyle factors. Understanding what contributes to high-quality sleep allows researchers to better identify and address poor sleep conditions, whether due to physiological, behavioral, or external causes.

Table 1. Sleep-Related Terms and Definitions

Terms	Definitions
Sleep duration	Total time spent asleep during a sleep episode
Sleep continuity	Degree to which sleep is undisturbed by awakenings or transitions
Sleep latency	Time it takes to fall asleep after going to bed
Circadian alignment Subjective sleep quality	Synchronization of sleep-wake cycle with internal biological rhythms
Subjective sleep quality	Self-reported satisfaction with restfulness, alertness, and sleep depth
Sleep efficiency	Ratio of time spent asleep to time spent in bed
Psychomotor vigilance	Performance on tasks measuring reaction time and sustained attention
Sleep spindles	Brief bursts of brain activity during NREM sleep associated

	with memory
Delta waves	Slow brain waves in deep sleep indicating restorative processes

Causes of Poor Sleep Quality

Now that we have outlined the components of high-quality sleep, the next question is: What prevents people from sleeping well? Poor sleep is a widespread issue, affecting nearly one-third of adults worldwide, according to global sleep health surveys (Scott et al., 2024a). Disruptions in biological, psychological, behavioral, or environmental factors can interfere with the body's natural sleep-wake rhythm, reduce sleep efficiency, and impair the restorative functions of sleep (Liu et al., 2021; Okun, 2011). By understanding these contributing factors more deeply, it becomes possible to identify effective strategies for improving sleep health and addressing chronic sleep difficulties.

This challenge is especially common among adolescents and shift workers, whose developmental stages or irregular schedules make it particularly difficult to maintain a stable circadian rhythm (Illingworth, 2020; Lee et al., no date).

Environmental light exposure

Environmental light exposure plays a major role in regulating sleep, largely through its effect on the circadian system. One of the most influential *zeitgebers* - a German term meaning "time-givers", or external cues that synchronize the internal body clock with the environment - is light. These cues help align the body's rhythms with the 24-hour day. For example, when a person travels across time zones, their internal clock adjusts gradually based on new light exposure and other zeitgebers, such as meal times and social activity (Roenneberg & Merrow, 2016). Nowadays, almost everyone is exposed to blue light (the type of light given off by phones, tablets, and LED lamps). Blue light exposure specifically has been shown to decrease the release of melatonin, which is a hormone responsible for signaling the beginning of sleep (West et al., 2011). Blue light affects photosensitive retinal ganglion cells (ipRGCs), which send signals to the SCN. When these cells are activated at night, melatonin secretion is delayed, causing a shift in the sleep-wake cycle and reducing sleep duration and quality (Ostrin et al., 2017). In addition to delaying the onset of sleep, prolonged exposure to artificial light in the evening may also decrease REM and slow-wave sleep (Münch et al., 2006), which are both critical for emotional control and cognitive recovery.

Diet

In addition to light exposure, dietary patterns significantly affect sleep quality. The brain requires nutrients such as magnesium, zinc, and vitamins B6 and D to synthesize neurotransmitters like serotonin and GABA, both of which facilitate sleep onset (Gallagher et al., 2024). Furthermore, diets high in fiber and unsaturated fats tend to promote deeper and more

restorative sleep, whereas diets high in sugar and saturated fats have been linked to lighter, more fragmented sleep (Frank et al., 2017). Certain foods like kiwis, cherries, and fish have been shown to promote sleep due to their omega-3 fatty acids (Patan et al., 2021), which are crucial in circadian regulation and serotonin synthesis. Meal timing is also crucial: eating large meals too close to bedtime can increase body temperature and gastrointestinal activity, potentially disrupting sleep. Irregular eating schedules(skipping breakfast or eating at inconsistent times), which many adolescents or shift workers often have, may disrupt the circadian rhythm, as feeding acts as a peripheral time cue for the body's internal clocks.

Stress and Environment

Chronic stress and an individual's psychosocial environment also play an important role in the development of sleep disturbances. Physiologically, stress triggers the hypothalamic-pituitary-adrenal (HPA) axis, which raises cortisol secretion, delays the beginning of sleep, and reduces REM sleep (Chu et al., 2025; Mbiydzenyuy & Qulu, 2024). When the home environment is a source of constant tension - whether due to strained family relationships or noise pollution - it can reinforce a state of increased sensory sensitivity. Studies have found a strong correlation between psychological safety and sleep health, with adolescents reporting higher levels of sleep disturbance when they experience more family-related conflicts (Çiçek & Yıldırım, 2025; Kelly et al., 2024). The body's ability to initiate and maintain sleep can also be disrupted by environmental factors like uncomfortable room temperatures, artificial light from urban surroundings, and excessive noise (such as traffic or train tracks). These environmental cues affect sleep architecture by fragmenting sleep and increasing nighttime awakenings, thus lowering perceived sleep quality.

These environmental and psychological disruptions are especially relevant for shift workers, who often experience both chronic stress and irregular environments due to rotating schedules, bright nighttime exposure, and the difficulty of achieving restorative sleep during the day.

Behavioral habits

Behavioral habits around sleep, particularly one's pre-sleep routine, are central to regulating circadian alignment and sleep efficiency. Sleep latency and quality are improved by regular sleep and wake times, which support the body's natural circadian rhythm (Murray et al., 2019). On the other hand, irregular schedules, which are frequently brought on by social or academic obligations, disrupt internal biological clocks and may cause a delayed sleep phase.

Physical activity

Physical activity also has an influence on sleep quality. Moderate exercise during the day has been shown to increase slow-wave sleep, reduce sleep latency, and lower nighttime awakenings (Kredlow et al., 2015; Park et al., 2021). This is explained by improvements in mood, decreased anxiety, and thermoregulation. However, high-intensity workouts done right before bed may raise core body temperature and adrenaline levels, delaying sleep onset (Alkhaldi et al., n.d.). Thus, through effects on the SCN, the timing, intensity, and consistency of physical activity influence the sleep-wake cycle.

Genetic factors

Lastly, genetic factors affect circadian rhythms and vulnerability to sleep disorders, thus affecting sleep quality. Numerous genes have been connected to the control of the circadian rhythm, including PER1, PER2, and CLOCK (Abel et al., 2015). These genes determine an individual's chronotype and responsiveness to light cues. Mutations in these circadian clock genes can lead to sleep disorders.

For example, mutations in the PER2 gene are commonly responsible for Familial Advanced Sleep Phase Syndrome (FASPS), in which individuals fall asleep and wake up much earlier than typical. Interestingly, people with FASPS generally do not feel tired or sleep-deprived, because their total sleep duration and architecture remain intact, they are just misaligned with the typical day-night schedule. Thus, the quality of their sleep is usually normal, even though their schedule is shifted. In contrast, those with Delayed Sleep Phase Disorder (DSPD) tend to fall asleep very late at night and wake up late in the morning (Sack et al., 2007). While they may also experience normal sleep architecture, many are forced to wake up early for school or work. This mismatch can lead to sleep deprivation, daytime fatigue, and impaired cognitive functioning, despite no problems with sleep quality itself. In both cases, the issue lies not in the sleep itself but in the misalignment between the person’s internal clock and external demands, which can indirectly reduce sleep quality and well-being.

Knowing the genetic foundation of sleep helps explain why some people may experience trouble sleeping even when they follow healthy routines.

Effects of Poor Sleep

Now that we have defined sleep and described the causes of poor sleep quality, we can evaluate the consequences that arise from low-quality sleep and discuss approaches to resolving these. Affecting nearly one-third of adults worldwide, poor sleep is increasingly recognized as a global health crisis (Scott et al., 2024). In this section, we will explore the general consequences of insufficient or disturbed sleep, examine several specific sleep disorders, and evaluate the efficacy and limitations of current medical treatments.

General Consequences of Sleep Loss

Poor sleep quality impairs cognitive function on multiple levels. As described previously, studies using Psychomotor Vigilance Tasks (PVTs) have shown that sleep-deprived individuals demonstrate slowed reaction times and increased lapses in attention (Basner et al., 2018; Hansen et al., 2019) Executive functions such as working memory, planning, and decision-making also deteriorate after just one night of poor sleep (Killgore, 2010). Over time, chronic sleep deprivation disrupts memory consolidation processes that occur during slow-wave and REM sleep, making it harder to retain new information (Walker, 2009). In school or work, poor sleep can grow into difficulty learning new concepts, maintaining focus during tasks, or making accurate and timely decisions.

Behaviorally, poor sleep is linked to increased emotional reactivity, impulsivity, and poor behavioral regulation (Demichelis et al., 2023). Amygdala is a brain region involved in memory,

emotion, and fear conditioning. Functional MRI studies have shown that sleep-deprived individuals exhibit heightened amygdala responses (Krause et al., 2017; Shao et al., 2014). These patients exhibited more emotional dysregulation. Sleep-deprived individuals also show weaker prefrontal regulation, contributing to emotional instability, which demonstrates the importance of sleep in stabilizing emotions (Yoo et al., 2007). This dysregulation can manifest as irritability, increased risk-taking, and even symptoms of anxiety and depression. On a social level, individuals experiencing poor sleep often report lower satisfaction in social relationships (Sell et al., 2023), which may stem from a reduced capacity for empathy and emotion regulation.

Sleep quality also has a direct impact on physiological health. Hormonal regulation is disrupted following sleep deprivation, particularly in cortisol, a stress hormone that, when chronically elevated, contributes to inflammation and cardiovascular risk (Pan et al., 2023). These hormonal changes are thought to result from activation of the HPA axis and circadian misalignment. Elevated cortisol during sleep deprivation promotes disruptions in hormones associated with appetite and stress. Ghrelin (a hormone that stimulates hunger) levels rise while leptin (a hormone that regulates energy) decreases, leading to increased hunger and poorer food choices (Spiegel et al., 2004; Taheri et al., 2004). Even athletic performance is affected - sleep-deprived individuals often show decreased physical endurance, slower reaction times, and poorer motor coordination (Gong et al., 2024).

Altogether, these cognitive, emotional, and physiological impairments demonstrate the importance of sleep for long-term physical health as well as mental health, social well-being, and academic/work satisfaction. As sleep disturbances accumulate, their effects become more widespread and more difficult to reverse.

Sleep Disorders and Treatments

While occasional poor sleep, whether due to stress, environmental noise, or other temporary factors, is a normal and reversible experience, some individuals face more persistent and severe sleep difficulties. In these cases, the problem often stems from an underlying clinical sleep disorder. Five common sleep disorders – obstructive sleep apnea (OSA), insomnia, narcolepsy (types 1 and 2), hypersomnolence disorder, and REM sleep behavior disorder – will be discussed in more detail in the section that follows. Each will be examined through their neurobiological mechanisms and treatments.

Obstructive Sleep Apnea (OSA)

OSA is caused by repeated collapse of the upper airway during sleep, causing a person to repeatedly wake up. It is frequently associated with anatomical abnormalities (enlarged tonsils, a recessed jaw, thick neck circumference), excess body weight, and decreased pharyngeal muscle tone (Peppard et al., 2000). These obstructions lead to hypoxia (low levels of oxygen in your body tissues), arousals, and fragmented sleep architecture (A. R. Schwartz et al., 2008). OSA affects approximately 1 billion people worldwide, with 49% of men and 23% of women between the ages of 30 and 70 having moderate to severe forms (Benjafield et al., 2019). Up to 70% of people with OSA are also obese (Wolk et al., 2003). Symptoms include loud snoring, gasping, excessive daytime sleepiness, morning headaches, and impaired memory. Sleep fragmentation causes a reduction in deep sleep (N3) and frequent transitions out of REM. According to studies, having severe OSA increases the risk of cardiovascular disease

by 79% and stroke by more than 100% (Lin et al., 2012). Information further links OSA to a 58% increase in risk for heart failure (Shahar et al., 2001).

Treatments for OSA focus on mechanical and pharmacological approaches. The main treatment is CPAP (Continuous Positive Airway Pressure), which physically keeps the airway open during sleep. However, alternative pharmacological options are being explored for patients who cannot tolerate CPAP. These include drugs like acetazolamide and modafinil. An overview of these treatments, their mechanisms, and limitations is provided in Table 2.

Insomnia

Insomnia is a disorder characterized by persistent difficulty falling asleep, staying asleep, or waking up too early despite adequate sleep. It is frequently maintained by a state of hyperarousal that is both physiological (increased cortisol and sympathetic nervous system activity) and psychological (stress, anxiety, intrusive thoughts) (Riemann et al., 2010). It disrupts the normal sleep-wake cycle and alters the balance of sleep-promoting and arousal-promoting neurotransmitters. Chronic insomnia affects 10–15% of adults globally, and about half of those who have it also exhibit signs of anxiety or depression (Riemann et al., 2010b). According to neuroendocrine studies, insomnia is linked to elevated cortisol levels throughout the 24-hour cycle and chronic HPA axis activation, indicating a state of sustained physiological arousal (Vgontzas et al., 2001). However, this remains debated: while some studies report consistently higher cortisol in people with insomnia, others suggest variability depending on insomnia subtype (Buckley & Schatzberg, 2005; Vgontzas et al., 2001). More research is needed to clarify whether elevated cortisol is a cause of insomnia, a consequence of it, or both.

There are several subtypes of insomnia, which can differ based on their timing, duration, and underlying causes. Difficulty falling asleep in the morning is known as sleep-onset insomnia, whereas sleep-maintenance involves frequent awakenings or trouble staying asleep. When someone wakes up too early and is unable to fall back asleep, it is known as early-morning awakening insomnia. Another way to categorize insomnia is as acute (lasting days to weeks, typically brought on by stress or life events) or chronic (lasting at least three months, frequently associated with physiological or psychological hyperarousal).

Because insomnia affects multiple brain systems, medications to treat insomnia target various pathways: some suppress arousal signals (e.g. the orexin system), while others aim to restore circadian rhythm (e.g. melatonin agonists). An overview of the medications used to treat insomnia and other sleep disorders is summarized in the table below (Table 2).

Narcolepsy

Narcolepsy is a neurological disorder caused by autoimmune destruction of hypocretin-producing neurons in the lateral hypothalamus (Mahlios et al., 2013). Arousal stability, REM sleep, and wakefulness are all regulated by hypocretin (also called orexin). It affects 1 in 2,000 people globally (Scammell, 2015). According to the National Institute of Neurological Disorders and Stroke, narcoleptics enter REM sleep almost immediately after falling asleep, disrupting the natural sleep cycle. Consistent findings across multiple studies confirm that over 95% of narcolepsy Type 1 cases involve severe hypocretin deficiency, which supports the autoimmune hypothesis (Mahoney et al., 2019). Type 1 narcolepsy is defined by the presence of cataplexy. Cataplexy is muscular weakness which can range from a barely

perceptible slackening of the facial muscles to complete muscle paralysis with postural collapse (Mirabile & Sharma, 2025). Type 1 narcolepsy is associated with extremely low hypocretin levels in the cerebrospinal fluid (approximately 90% of patients with cataplexy have undetectable hypocretin in cerebrospinal fluid (Singh et al., 2013). Type 2 narcolepsy is characterized by excessive daytime sleepiness and disturbed night-time sleep. Individuals may experience symptoms such as sleep paralysis or hypnagogic hallucinations, but without the sudden muscle weakness that defines cataplexy.

Narcolepsy treatments are drugs that target different aspects of the disorder, such as excessive daytime sleepiness and cataplexy. Sodium oxybate is considered highly effective. Pitolisant promotes wakefulness but is often limited by side effects. These therapies are outlined in Table 2.

Hypersomnolence Disorder

Prolonged sleep at night and trouble waking up are both indicators for idiopathic hypersomnia, a disorder causing excess sleep. Although the precise mechanism is unknown, hypotheses include dysregulation of the central nervous system, low arousal signaling, or irregular circadian input (Trotti, 2017). Onset of idiopathic hypersomnia typically is in adolescence or early adulthood (*Idiopathic Hypersomnia*, n.d.). Patients report persistent daytime sleepiness that is not relieved by naps, sleep inertia (feeling "drunk" upon waking), and sleeping more than 9 to 10 hours every night (Thorpy et al., 2024). Some studies suggest altered GABAA signaling in the thalamus and cortex as a possible mechanism (Rye et al., 2012). An overview of the medications used to treat hypersomnolence disorder and other sleep disorders is summarized in the table below (Table 2).

REM Sleep Behavior Disorder (RBD)

People with RBD physically enact their dreams, sometimes violently, because the muscle atonia that typically accompanies REM sleep is disrupted. It’s considered a prodromal (an early stage of a disease when symptoms first appear, but before full diagnosis is possible) symptom of neurodegenerative disorders. Longitudinal research shows that up to 80% of people with idiopathic RBD go on to develop other conditions like Parkinson's disease or Lewy Body dementia (Iranzo et al., 2006, 2009; Postuma et al., 2012). Symptoms include vocalizations, limb movements, or falling out of bed during REM sleep. Patients often remember vivid, action-filled dreams. The REM atonia circuitry in the pons and medulla is disrupted, although the exact pathological trigger is still unknown.

Treatment for RBD aims to reduce abnormal motor activity during REM sleep. The most commonly prescribed drug is clonazepam, which is effective in suppressing muscle activity. Melatonin is a safer alternative, especially for older adults, with milder side effects. An overview of these treatments and their limitations is presented in Table 2.

Table 2. Treatments for Sleep Disorders: Mechanisms and Drawbacks

sleep disorder	medication	mechanism	downside
insomnia	Benzodiazepines (e.g. temazepam). Z-drugs (e.g. zolpidem) are preferred for short-term use due to a better side-effect profile	Enhance GABA activity, promote sedation	Dependence risk, REM suppression
	Orexin receptor antagonists (e.g. suvorexant)	Block wakefulness-promoting signaling, preserving sleep architecture more effectively.	May cause next-day drowsiness
	Melatonin receptor agonists (ramelteon)	Used for circadian-related insomnia and have low abuse potential.	Less effective in severe cases, mild effects
OSA	First-line treatment like CPAP (Continuous Positive Airway Pressure)	Keeps the airway open mechanically	May be uncomfortable for some
	Acetazolamide	Stabilizes breathing via CO2 sensitivity in specific OSA cases	Not widely used, limited scope
	Modafinil	Inhibits dopamine reuptake, increases wakefulness, enhances alertness	Headaches, anxiety, variable effectiveness
Narcolepsy	Sodium oxybate	Deepens slow-wave sleep, treats cataplexy & daytime sleepiness	Strict control due to abuse potential
	Pitolisant	Histamine H3 antagonist, enhances wakefulness	Headache, limited access
Hypersomnolence Disorder	Solriamfetol and pitolisant	Increase dopamine/norepinephrine to reduce sleepiness	Hypertension risk, limited data
RBD	Clonazepam	Reduces motor activity in REM	Drowsiness, dependence, cognitive dulling
	Melatonin	Reduces REM activity, safer in elderly	Milder effects than clonazepam

Discussion

The paper has outlined the biological mechanisms, influences, and consequences of poor sleep. While many aspects of sleep are well established, several open questions remain. Firstly, although experts typically recommend adults to sleep 7–9 hours per night, sleep need is not universal. Genetic research suggests that some individuals may naturally require less sleep without noticeable cognitive deficits (Chen et al., 2025). However, this remains debated – do the "short sleepers" genuinely need less sleep, or do they simply compensate more efficiently? Understanding these individual differences could help change the sleep guidelines and help individuals understand their own needs better in the future.

Sleep quality is important, as discussed in this review. Being able to identify low sleep quality prior to the negative effects on behavior could open therapeutic opportunities for better sleep treatment. However, there are currently no standard physiological markers of sleep quality. A promising one is the sleep spindle, a burst of brain activity during NREM stage 2 sleep. While spindles are associated with memory consolidation, their exact function and individual variability remain unclear. Further research on spindle characteristics, such as frequency or cortical location, could reveal more about how they support sleep-dependent cognitive functions and whether they can serve as biomarkers of sleep quality.

A major gap in the sleep field is understanding how certain symptoms of sleep disorders are connected to sleep loss. For example, how do specific sleep disorders contribute to cardiovascular diseases? Is the impact on heart health simply because sleep deprivation can increase obesity or is more prevalent with age? Or does it directly impact heart health? Similar questions could be asked about diet. Further questions include: Is elevated cortisol a key component of insomnia, or does it differ across subtypes? How does hypocretin loss produce the diverse symptoms of narcolepsy? What biological mechanisms explain the excessive sleepiness?

For example, OSA leads to sleep fragmentation, reduced deep sleep, and oxygen deprivation - all of which significantly raise the risk of heart disease and stroke. Understanding how different sleep disorders influence cardiovascular diseases is an interesting topic for interdisciplinary research. As another example, chronic insomnia is associated with elevated cortisol levels. However, research is divided on whether elevated cortisol is a consistent feature or if it varies by insomnia subtype. Regardless, elevation of this chronic stress hormone has potential implications for cardiovascular health. Additionally, narcolepsy symptoms such as hallucinations and sleep paralysis may be linked to hypocretin loss, but exactly how this single neuropeptide system leads to such a broad range of symptoms remains unclear. Similarly, in hypersomnolence disorder, the core mechanism is still unknown - proposed explanations include low arousal signaling or CNS dysregulation, but current findings are not conclusive.

The role of diet in shaping sleep quality is also not well-understood. For example, eating large meals before bed has been shown to delay sleep by raising body temperature and activating digestion. Yet, heavy meals can also induce sleepiness, likely due to blood being redirected from the brain to the digestive tract. This contradiction raises questions about the balance between physiological arousal and subjective sleepiness after eating, which future research should address.

While a range of pharmacological treatments exists for various sleep disorders, many of these medications focus on the symptoms rather than the underlying causes. For instance, stimulant medications used in narcolepsy, such as modafinil or pitolisant, aim to promote wakefulness without targeting the autoimmune destruction of hypocretin-producing neurons that drives the disorder. Similarly, treatments for idiopathic hypersomnia and other forms of excessive daytime sleepiness focus on increasing alertness but do not resolve the core dysregulations in sleep-wake mechanisms. This means that current understanding of the neurobiological processes that regulate sleep disorders remains limited. As a result, there is a significant gap between symptom management and curative therapy.

Finally, RBD is one of the most clinically significant yet biologically puzzling conditions. While up to 80% of individuals with RBD go on to develop Parkinson’s or related neurodegenerative diseases, researchers still don’t clearly understand the cause of this relationship. The REM atonia system in the pons and medulla is clearly disrupted, but the precise pathological trigger remains unknown. As such, RBD represents both a potential early warning sign and a critical opportunity for future research into neurodegenerative progression.

Conclusion

As this paper has shown, sleep plays a crucial role in one’s health. Sleep is shaped by neural systems, biological rhythms, and external signals, all working together to maintain health and cognitive function. In our society, a wide range of influences, from artificial light to stress, interfere with the body’s natural sleep regulation, leading to widespread disruptions. When sleep quality declines, the effects extend across nearly every system in the body, impairing memory, emotional regulation, and metabolic stability. These impairments become especially serious when they develop into clinical sleep disorders. Understanding the science behind both healthy and disrupted sleep offers a critical foundation for treatment, prevention and education.

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