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(REVIEW ARTICLE)

The Vital Role of Sleep in Human Health: Stages, Mechanisms and the Consequences of Sleep Disruption

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Abstract

Purpose: To summarize the biological significance of sleep, its regulatory mechanisms, and the health impacts of insufficient or disrupted sleep.

Methods: This review synthesizes current scientific understanding of sleep physiology, emphasizing the coordinated roles of neural, metabolic, hormonal, and immune systems, and outlining the distinct functions of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep stages.

Results: Sleep is an active, tightly regulated process essential for maintaining physical and cognitive health. NREM sleep supports tissue repair, growth hormone release, metabolic restoration, and declarative memory consolidation, whereas REM sleep underpins emotional regulation, neural plasticity, and procedural learning. Insufficient or disrupted sleep impairs cognition, mood, and alertness, and long-term deprivation contributes to metabolic dysfunction, hormonal imbalance, cardiovascular disease, immune suppression, psychiatric disorders, neurodegeneration, and increased mortality.

Conclusions: Sleep is a central pillar of overall health, comparable in importance to diet and exercise. Its wide-ranging physiological influence underscores the need for clinical and public health strategies that promote healthy sleep behaviors and mitigate the adverse effects of sleep disruption.

Keywords: Sleep physiology; NREM and REM stages; Memory consolidation; Hormonal regulation; Sleep deprivation

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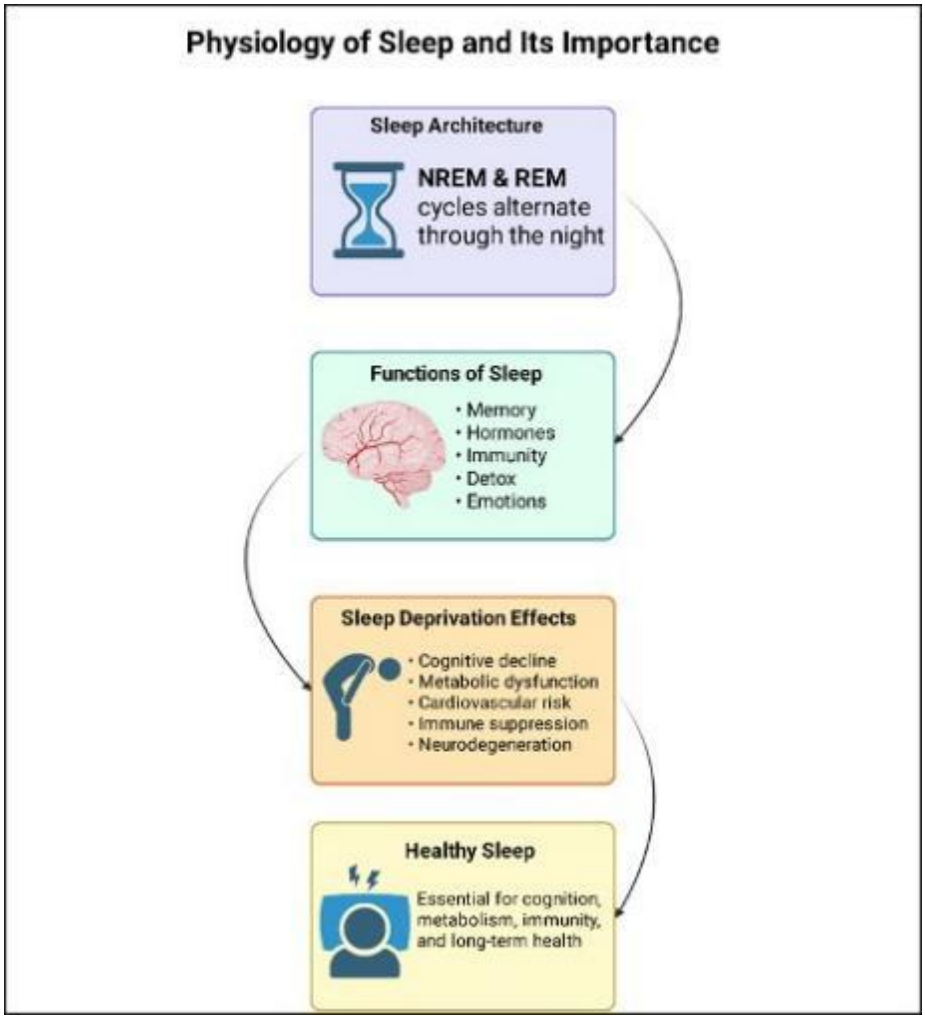
Graphical abstract



1. Introduction

Sleep is a complex and dynamic biological process that helps the body maintain balance. Rather than being seen as a simple period of rest, it is now recognized as an active state that restores, regulates, and optimizes essential systems, including the nervous, metabolic, hormonal, and immune networks. As numerous experimental and clinical studies have demonstrated, sleep is essential to the processes of energy conservation, memory consolidation, tissue repair, and metabolic homeostasis, and it is indispensable for the maintenance of life and health in general [1]. Physiologically, sleep is a restorative process in which tissue growth, muscle regeneration, protein synthesis, and the secretion of hormones such as growth hormone and prolactin are carried out by the body. These functions are particularly prominent in slow-wave sleep (also known as deep non-rapid eye movement or NREM) sleep, that is, sleep with reduced brain metabolism and an increase in anabolic activity [2]. Sleep provides the brain with the opportunity to restore stores of adenosine triphosphate (ATP) that are depleted during wakefulness, and to decrease total energy expenditure compared with waking. Such a metabolic rebalancing is necessary to maintain neuronal homeostasis and activity [3].

Sleep itself is controlled in a two-process model of homeostatic sleep drive and circadian rhythms. Process S, the homeostatic process, reflects the accumulation of the pressure to sleep during wakefulness, which is mediated primarily by the increasing levels of adenosine in the forebrain. As the concentration of adenosine increases the sense of sleep increases to the point of causing sleep [4]. Process C is the circadian process regulated by the suprachiasmatic nucleus of the hypothalamus and controls the sleep-wake cycle in relation to the light-dark cycles of the environment. This system helps control daily rhythms such as hormone release, body temperature, and levels of alertness, keeping the body's internal functions in tune with external environmental signals [5]. The interaction between these two processes ensures that sleep takes place at the right time and in the right amount, maintaining the body's internal balance. Beyond its roles in restoration and regulation, sleep is now also recognized as vital for clearing harmful waste products from

the brain. During sleep, the glymphatic system becomes especially active, flushing out metabolic by-products such as amyloid-beta. This cleansing function helps protect brain health and is thought to play an important role in reducing the risk of neurodegenerative diseases [6]. Sleep also plays an important role in keeping the body's hormones and metabolism in balance. Circadian rhythms control the secretion of melatonin, cortisol, prolactin and growth hormone and influence the control of appetite, stress and cell repair. Not to mention, poor or inadequate sleep disrupts the hormonal cycles that contribute to metabolic failure, compromised immunity, and heart disease [7].

The fact that all species sleep is yet another reason why sleep must be evolutionarily and physiologically important. Sleep has been preserved as a biological necessity since the beginning of creation, which means it has been preserved throughout the most fundamental processes necessary to sustain life. Modern data demonstrates that sleep is not only energy-restoring and cognition-improving, but also immuno-protective, regulating genetic and metabolic processes, and maintaining homeostatic balance of the human systemic structure [8]. Sleep in this sense is best defined as a pillar of health as important as nutrition and physical activity. Its loss or disturbance has titanic effect on the short-term functioning and long-term health, and hence, the focus on the contribution of sleep to the homeostasis in humans [9].

We elaborate on the physiology of sleep in the current review, starting by providing a general overview of the stages of NREM and REM sleep and physiological changes that take place during each sleep stage. We then look at the key functions of sleep such as the ability to consolidate memories, maintain hormonal balance and immunity. The negative effects of sleep deprivation on cognitive, metabolic, and cardiovascular functions are also discussed, and a conclusion is made on the significance of healthy sleep habits in general health.

2. Stages of sleep

2.1. Overview of Sleep Architecture

The structure of human sleep is called sleep architecture, and it reflects the cyclical proportion of NREM and rapid eye movement (REM) sleep throughout the night. The total sleep cycle is between 90 and 110 minutes and in an average night of sleep, the cycle is repeated four to six times [10]. The NREM sleep, which includes stages N1, N2, and N3, comprises approximately 75 to 80 percent of the total sleep time, with the remaining 20 to 25 percent of the sleep time being occupied by the REM sleep [11]. These phases are defined by typical electroencephalographic (EEG) activities, muscle activity, movement of the eyes, and specific physiological alterations in the autonomic and endocrine systems. Sleep starts with NREM and moves in a sequential order starting with stage N1 up to stage N3 and then to REM sleep. This cycling goes on all night, but the relative amounts of NREM and REM stages change in subsequent cycles. The predominant sleep type throughout the first part of the night is slow-wave sleep (stage N3), which promotes restorative and anabolic activities, with episodes of REM sleep increasing in length in the second half of the night, indicating its importance in the cognitive and emotional processes [12]. This time chart highlights the fact that there are different physiological and psychological processes which are prioritized at different times during the sleep cycle. Sleep architecture is dynamic and may be influenced by developmental stage, circadian timing, and pathology. Incidentally, infants spend approximately fifty percent of the total sleep duration in REM, compared to adults who show more consolidated patterns and have fewer awakenings and relatively constant percentages of NREM and REM phases [13]. Sleep architecture change, including a decrease in slow-wave sleep or the shortening of REM latency, is a common finding in people with psychiatric, metabolic, or neurodegenerative conditions, again highlighting the clinical importance of a healthy sleep architecture [14]. In general, sleep architecture is the dynamic equilibrium of NREM and REM sleep that allows the body and brain to perform the restorative, cognitive, and homeostatic functions of sleep. Before delving into each stage in more detail, it is important to understand this architecture. The stages of sleep and their physiological characteristics are shown in **Table 1** and **Figure 1.**

Table 1 Stages of Sleep and Their Physiological Characteristics

Stage / Aspect	EEG & Neural Features	Physiological Changes	Functional Role
Overview of sleep architecture	Cycles of NREM and REM lasting 90–110 min; 4–6 cycles per night	Alternating distribution of NREM (75–80%) and REM (20–25%)	Sequential alternation ensures balance between restorative and cognitive functions
NREM Stage N1 (light sleep)	Transition; disappearance of alpha, appearance of theta waves	Reduced HR and muscle tone; slow eye movements	Gateway to deeper sleep; ~5–10% of total sleep

NREM Stage N2 (intermediate/light sleep)	Sleep spindles and K-complexes	Further decline in HR, respiration, and temperature	Sleep stabilization, memory consolidation, protection from arousal; ~45–55% of total sleep
NREM Stage N3 (deep/slow-wave sleep)	Dominant delta waves; high amplitude, low frequency	Growth hormone peak, parasympathetic dominance, reduced BP and HR	Tissue repair, protein synthesis, immune strengthening, declarative memory consolidation
REM sleep	EEG resembles wakefulness; theta activity; muscle atonia	Autonomic instability, irregular HR & BP, suspended thermoregulation	Emotional memory consolidation, neural plasticity, dreaming, creativity
Physiological changes across stages	NREM = synchronized slow activity; REM = desynchronized active EEG	NREM = stable HR/respiration; REM = variability, irregularity	NREM provides stability and restoration; REM supports cognitive/emotional functions
Developmental & age-related changes	Infants: ~50% REM; adults: ~20–25% REM; elderly: reduced N3	Aging = more awakenings, reduced sleep efficiency	Development: brain growth, synaptogenesis; Aging: cognitive decline, reduced restoration

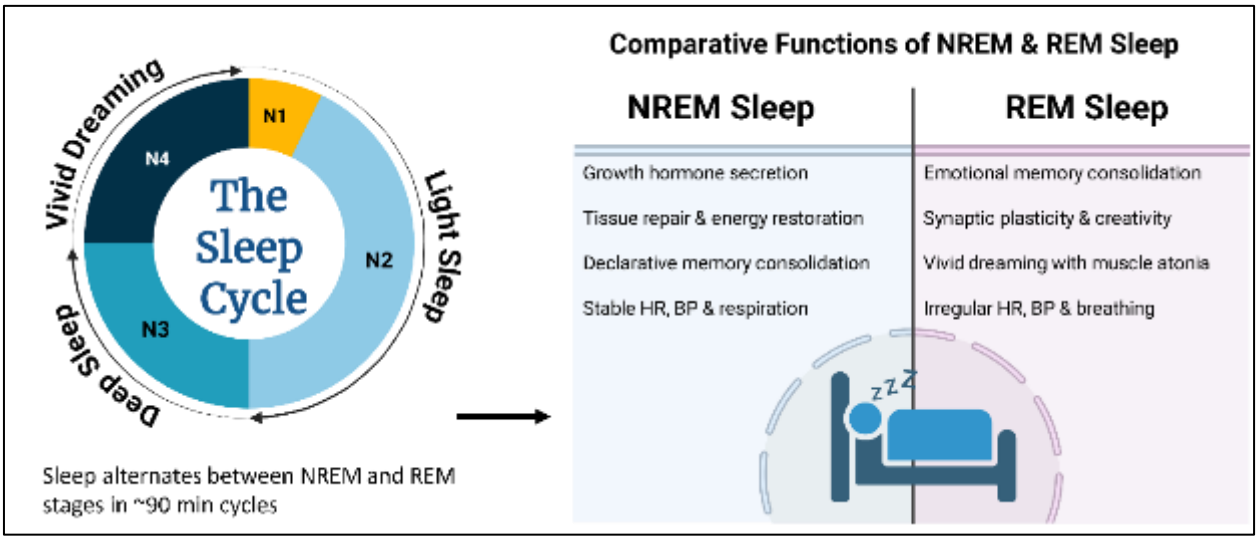


Figure 1 The Sleep Cycle and Comparative functions of NREM & REM

2.2. Non-Rapid Eye Movement (NREM) Sleep

The most common constituent of human sleep is the NREM which occupies nearly three quarters of total sleep-in healthy adults. It is divided into three stages, i.e., N1, N2 and N3, which are defined by different electroencephalographic (EEG) activity and the degree of sleep, respectively. NREM sleep is so important in systemic homeostasis that it is thought to be essential to physical rest, immune regulation, and metabolic equilibrium [15]. Stage N1 is the transition between waking and sleeping and is commonly referred to as light sleep. EEGs reveal the loss of alpha waves (8-12 Hz) and the appearance of low amplitude theta waves (4-7 Hz). Physiologically, the stage is characterized by low muscle tone, slow eye movements and slow heart rate. Though stage N1 represents only 5-10 percent of total sleep, it plays a critical role in the descent into deeper stages of sleep [16]. The most common stage of sleep is stage N2, which constitutes approximately 45 to 55 percent of total sleep time. It is characterized by the occurrence of sleep spindles (12-14 Hz bursts of activity) and K-complexes that are thought to have protective functions against arousals and are involved in memory consolidation. At this phase, the body temperature drops, the muscle activity decreases further, and the heart rate and respiratory rate slow down. Notably, N2 sleep is regarded as a gatekeeper phase that stabilizes the sleep and prepares the shift to a deeper slow-wave sleep [2].

Slow-wave sleep (SWS) or stage N3 is a sleep stage in which the EEG is dominated by high-amplitude, low-frequency delta waves (less than 4 Hz). This phase usually takes up 15-25 percent of total sleep and is especially plentiful in the first half of the night. Physiologically N3 sleep is characterized by maximum parasympathetic nervous system dominance, low sympathetic activity and a deep reduction in blood pressure and heart rate. Secretion of growth hormone is the highest in this phase and it is associated with repair of tissues, protein production and improvement of immune system [17]. Slow-wave sleep that leads to the consolidation of declarative memory and synaptic plasticity is also correlated with hippocampal-neocortical communication [18]. The impact of the disruption of NREM sleep helps highlight the restorative effect of this sleep. Disruption or suppression of slow-wave sleep has been associated with the loss of glucose tolerance, low pain thresholds and predisposition to hypertension and heart disease [19]. In addition, ageing is characterized by reduced N3 sleep and this has been linked to cognitive impairment, hence its pivotal role in physical and mental well-being [20].

Overall, NREM sleep is a continuation of light sleep to deep restful states, and each has its part to play in the preservation of homeostasis. Stage N1 helps to transition into sleep, stage N2 helps to stabilize the sleep process and promotes memory, and stage N3 is the most restorative to the body and brain.

2.3. Rapid Eye Movement (REM) Sleep

REM sleep comprises about 20-25% of total sleep-in healthy adults and is characterized by a paradoxical state of cortical activation and muscular paralysis. Brain wave recordings during REM sleep look like those seen in wakefulness, showing low-amplitude, mixed-frequency activity largely driven by theta rhythms. At the same time, the brainstem suppresses signals to the spinal motor neurons, causing deep muscle paralysis that stops the body from acting out dreams [21]. REM sleep is especially important for memory, thinking, and emotional stability. Research shows that it supports the consolidation of procedural and emotional memories, as well as neural plasticity. During REM, limbic regions such as the amygdala and hippocampus exhibit heightened activity, whereas prefrontal cortical areas remain relatively suppressed. This imbalance helps explain both the emotional intensity and the reduced logical coherence of REM-associated dreams [22]. Physiological changes in this stage are unique compared to NREM sleep. Autonomic instability is a hallmark, with fluctuating heart rate, variable blood pressure, and irregular respiratory patterns. In addition, thermoregulation is suspended, leaving the body more sensitive to ambient temperature. These features highlight the specialized role of REM in neural and psychological functions rather than in purely restorative processes [23]. Developmental evidence further underscores the importance of REM sleep. Infants spend nearly half of their total sleep time in REM, a proportion considered vital for synaptic development and brain maturation. With age, REM sleep consolidates into the latter half of the night, while in older adults both the percentage and continuity of REM decline. Such reductions have been linked to impairments in cognition and emotional regulation in later life [24]. In summary, REM sleep complements the restorative benefits of NREM by supporting memory consolidation, emotional stability, and neurodevelopment. Its disruption is closely associated with mood disorders, cognitive deficits, and neurodegenerative conditions, reflecting its indispensable role in human health.

2.4. Physiological Changes Across Sleep Stages

The transition from wakefulness into the various stages of sleep is accompanied by profound changes across virtually every physiological system. These shifts reflect the brain's dynamic regulation of sleep states and highlight the distinctive functions associated with NREM and REM sleep. From a central nervous system perspective, the electroencephalogram (EEG) undergoes predictable alterations across stages. During stage N1, low-voltage theta activity predominates, while stage N2 introduces sleep spindles and K-complexes, which are thought to protect sleep and facilitate memory consolidation [25]. Stage N3, or slow-wave sleep, is characterized by high-amplitude delta activity and reduced neuronal firing, representing the deepest state of unconsciousness and the stage most closely linked to restorative processes [20]. By contrast, REM sleep displays EEG patterns similar to wakefulness, with desynchronized cortical activity, vivid dreaming, and muscle atonia [21]. Cardiovascular function also varies systematically across sleep stages. NREM sleep, particularly slow-wave sleep, is associated with parasympathetic dominance, reduced heart rate, and decreased blood pressure. These nocturnal reductions provide cardiovascular rest and are thought to be protective for long-term heart health [7]. In REM sleep, however, autonomic instability emerges, producing irregular heart rate and fluctuations in blood pressure. These oscillations may explain why cardiac events such as arrhythmias and myocardial ischemia are sometimes reported during REM periods [17]. Respiratory physiology follows a similar pattern. During NREM sleep, ventilation becomes slower and more regular, with a decline in both respiratory rate and minute ventilation. This stability is lost in REM sleep, where breathing becomes irregular and susceptible to apnea events, particularly in individuals with sleep-disordered breathing [26]. Endocrine regulation is tightly coupled to sleep stages. Growth hormone secretion peaks during slow-wave sleep, underlining its restorative and anabolic role. Cortisol levels, on the other hand, begin to rise during the second half of the night, reflecting circadian influences and preparing the body for wakefulness [27]. Collectively, these systemic changes demonstrate that sleep is not a passive state but a

highly active process in which different physiological functions are prioritized across NREM and REM stages. NREM promotes stability, restoration, and anabolic processes, whereas REM emphasizes cognitive, emotional, and developmental roles, each contributing to the maintenance of homeostasis and long-term health.

2.5. Developmental and Age-Related Changes

There are dramatic changes in the architecture and quality of sleep throughout the human lifespan, which are both developmental and age-related. During early childhood, sleep is disordered, polyphasic and biased towards REM sleep. Infants can also spend as much as 50 percent of their total sleep time in REM, a percentage that is thought to aid brain development, synaptic growth, and the maturation of sensory processing systems [24]. A number of age-related variations in the total sleep duration, nocturnal sleep consolidation, and percentage of slow-wave sleep (N3) also demonstrate its role in physical growth and rest [13]. Additional alterations are observed during adolescence when circadian rhythms begin to shift to later wake and later sleep-onset, and this has also been linked to hormonal and social mechanisms. Despite the fact that total sleep needs are high at this stage, it is common to observe chronic sleep restriction due to lifestyle and behavioral changes that have been associated with impaired learning, mood fluctuations, and metabolic imbalance [12]. During adulthood, sleep patterns become rather stable, and 7-9 hours of sleep are considered to be the best. The ratio between the NREM and the REM is relatively stable, but the quality and duration can be affected by lifestyle, working hours, and pathological factors. Senescence creates significant changes in sleep structure. Older people have a decrease in slow-wave sleep, SLEs, more night awakenings, and sleep fragmentation. The result of these changes is usually a loss in overall sleep efficiency and a transition to the lighter sleep stages. Reduced N3 sleep specifically has been linked to memory impairment, compromised immunity and susceptibility to neurodegenerative conditions like Alzheimer [20]. In addition, decreases in REM continuity have been associated with mood swings and affective disorders during late life [21]. Therefore, although sleep is critical at all ages, the structure and role of sleep is not fixed. During the developmental stages in the neural growth process, REM is targeted, and during the developmental stages in the physical growth process, N3 is targeted, although aging is characterized by the progressive decrease in the mentioned restorative factors. These changes are important to understand both the normal patterns of sleep development and pathological deviations that are associated with risk of disease.

3. Functions of Sleep

3.1. Memory Consolidation and Learning

The role of sleep-in memory consolidation and learning is one of the most widely researched functions of sleep. Neurophysiological, behavioral and neuroimaging studies have shown consistent evidence that both NREM and REM sleep play a unique role in stabilizing and integrating information that has just been obtained. Instead of sleep being a passive consequence of fatigue, sleep is an active biological process that helps to transfer fragile, short-term memory traces to more stable long-term representations [12]. The deepest form of NREM, slow-wave sleep (SWS), has been closely linked to declarative memory consolidation, including facts, vocabulary and episodic memories. During SWS, coordinated oscillatory activity including hippocampal sharp-wave ripples, thalamocortical spindles, and cortical slow oscillations supports the transfer of memory representations from the hippocampus to the neocortex [18]. This hippocampal–neocortical dialogue is thought to underpin the gradual integration of new information into existing cortical networks, thereby strengthening memory storage [28]. Experimental studies show that subjects deprived of slow-wave sleep demonstrate significant impairments in declarative memory recall, underscoring the functional necessity of this stage [29].

By contrast, REM sleep has been implicated in the consolidation of procedural and emotional memories. Unlike SWS which stabilizes factual information, REM appears to maximize skills, habits and affective learning. In neuroimaging studies, increased limbic system activity in the amygdala and hippocampus during the REM state has been observed and may be implicated in the processing of emotionally-charged experiences [22]. The stage also favors the creative synthesis of memories by reactivation and recombination of already existing neural representations which are generally expressed in the vivid and sometimes bizarre dream content of REM sleep [30]. The complementary character of NREM and REM phases implies a sequential memory processing paradigm whereby the SWS induces the memory consolidation process through reactivation and transfer of memory traces and the following episodes of REM refines and consolidates the traces by incorporating them into long-term memory. This sequential relationship explains why completing full sleep cycles, rather than experiencing isolated stages, is necessary for optimal learning and memory retention [31]. Overall, sleep provides the biological foundation that strengthens mental functions. By coordinating brain activity patterns across both NREM and REM stages, sleep not only consolidates new information but also fosters creativity, supports emotional stability, and improves decision-making skills. When this process is disturbed by either insufficient sleep or frequent awakenings, learning ability declines and mental resilience is weakened.

3.2. Hormonal Regulation and Metabolic Balance

Sleep has a strong effect on the body's hormone system, helping regulate hormone release, align circadian rhythms, and maintain metabolic balance. Both the structure and timing of sleep directly shape the secretion patterns of hormones that are vital for growth, energy regulation, and stress response. When sleep is shortened or fragmented, it is consistently linked with metabolic problems and greater risk of chronic illness [32]. One of the best-known hormonal changes during sleep is the nighttime release of growth hormone, which peaks mainly during the early cycles of slow-wave sleep, a deep and restorative stage of NREM sleep. Growth hormone supports protein production, tissue repair, and cell regeneration, making it essential for recovery and physical balance [17]. This pulsatile release becomes significantly reduced in people exposed to ongoing sleep disruption or deprivation, weakening the body's ability to heal and grow [33]. Cortisol, the body's main stress hormone, follows a circadian rhythm, being lowest during sleep and rising steadily toward morning. This rhythm not only reflects circadian regulation of behavior but also aligns with transitions between sleep stages. Disturbed sleep or insomnia interferes with this pattern, leading to elevated nighttime cortisol levels, which are tied to poor glucose control, insulin resistance, and higher cardiovascular risk [34]. Melatonin, secreted by the pineal gland, is another key signal for circadian timing. Its production is stimulated by darkness and suppressed by light, thereby promoting the onset of sleep and regulating the sleep–wake cycle. In addition to being a sleep-promoting hormone, melatonin is involved in antioxidant protection and immunological regulation. Circadian misalignment and metabolic dysregulation have been linked to disruptions in melatonin secretion which is frequently caused by exposure to artificial light at night [35].

Sleep also has a powerful regulatory impact on appetite and energy metabolism by affecting leptin and ghrelin, two hormones that are part of the satiety and hunger signal. Sleep deprivation reduces leptin and elevates ghrelin, which establishes a hormone environment that promotes a higher appetite and caloric intake. These changes provide a physiological explanation for the established link between short sleep duration and obesity risk [36]. Moreover, chronic sleep curtailment reduces insulin sensitivity, predisposing individuals to type 2 diabetes and metabolic syndrome [37]. Together, these findings demonstrate that sleep is a critical modulator of endocrine function. Deep NREM stages promote anabolic hormone release, circadian-regulated cortisol patterns prepare the body for daily activity, melatonin synchronizes biological rhythms, and appetite-regulating hormones balance energy intake. Disturbances to these processes not only impair immediate physiological stability but also increase vulnerability to long-term metabolic and cardiovascular disorders.

3.3. Immune Function and Repair

Sleep is increasingly recognized as a fundamental regulator of immune function, with both innate and adaptive responses strongly influenced by sleep duration and quality. The bidirectional relationship between sleep and immunity is evident: while sleep supports immunological defenses, infection and inflammation also alter sleep architecture, often increasing NREM sleep as part of the body's restorative response [38]. One of the primary ways sleep influences immunities is through its effects on cytokine regulation. Slow-wave sleep is associated with enhanced production of pro-inflammatory cytokines such as interleukin-1β and tumor necrosis factor-α, which not only play a role in sleep regulation itself but also contribute to the activation of immune cells during infection [39]. Sleep loss, in contrast, suppresses cytokine release and reduces natural killer (NK) cell activity, impairing the body's first line of defense against viral and bacterial pathogens [40]. Sleep also enhances adaptive immunity by promoting the interaction between antigen-presenting cells and T lymphocytes. During NREM sleep, the low cortisol and high growth hormone milieu creates an optimal hormonal environment for antigen presentation and the proliferation of helper T cells. Experimental studies have shown that vaccination responses are significantly stronger in individuals who obtain sufficient sleep compared to those who are sleep-deprived [41]. This effect reflects sleep's role in the formation of long-lasting immunological memory. REM sleep contributes to immune regulation as well, particularly in the modulation of stress responses. While NREM stages promote the initiation of immune activation, REM appears to counterbalance excessive inflammatory activity, helping to prevent overactivation that could lead to tissue damage. The alternation between NREM and REM stages therefore creates a balanced immunological profile across the night [41]. The immunological effects of sleep disruption are clinically important. It has been found that chronic sleep restriction is related to increased vulnerability to upper respiratory tract infections, delayed healing of wounds, and augmented systemic inflammation. The causes of long-term sleep deficiency are also associated with the onset of chronic inflammatory diseases, such as cardiovascular disease, diabetes, and neurodegenerative disorders [42]. Contrastingly, the ability to combat infections is partially recovered after a recovery sleep after deprivation, thus making sufficient rest restorative [43]. Overall, sleep is a part of the defense mechanism in the body. In the control of cytokine synthesis, the maintenance of adaptive response, and the regulation of inflammatory processes, sleep enhances the short-term immune response to infection and the long-term defense against disease.

3.4. Neural Detoxification and Brain Homeostasis

In addition to memory, hormonal maintenance, and immune surveillance, sleep also offers a special biological opportunity to eliminate the waste products of metabolism in the brain. The glymphatic system is the mediator of this process and is activated in a very active way during sleep, especially in slow-wave phases. Both animal and human research indicate that interstitial spaces become expanded in deep sleep, which allows increased cerebral flow of cerebrospinal fluid, which in turn removes neurotoxic proteins like amyloid-beta and tau [3]. This is a vital biological function. In Alzheimer's disease, the buildup of amyloid-beta has been closely linked to poor or fragmented slow-wave sleep, with such disturbances repeatedly associated with greater amyloid levels. Changes in sleep patterns also become more rapid, strengthening the connection between disrupted sleep architecture and neurodegenerative disorders [20]. Supporting this, brain imaging studies in older adults demonstrate that reduced slow-wave sleep predicts faster memory decline and cognitive impairment. These findings highlight how sleep-related waste clearance is directly tied to brain health [12]. REM sleep also contributes indirectly to brain homeostasis. While slow-wave sleep promotes direct clearance of metabolites, REM facilitates neural plasticity and adaptive network reorganization, processes essential for maintaining efficient cognitive functioning. The alternation between NREM and REM therefore ensures both physical detoxification and cognitive optimization across the sleep period [21]. Overall, sleep should be regarded not only as a state of rest but also as an active period of cerebral maintenance. By enabling glymphatic clearance and neural reorganization, sleep preserves long-term brain integrity and reduces the risk of neurodegenerative disease, making it an indispensable factor in sustaining cognitive resilience.

3.5. Emotional and Psychological Health

Sleep is a major factor affecting emotions and mental wellbeing. Sleep instability or sleep disorders are closely linked with mood instability, greater stress reactivity, and increased psychiatric disorder risk. These processes are complementary in both NREM and REM sleep. The processing of emotional experiences has been associated with REM sleep, especially. Functionally, it has been shown that there is increased activity in the amygdala and hippocampus during REM, suggesting that this stage of sleep allows emotional memories to be integrated and, simultaneously, decreases over-reactivity to traumatic events [22]. This may be why sleep loss is often associated with increasing levels of anxiety, irritability and difficulty in regulating negative emotions. In addition, memory traces are recombined during the REM phase, which leads to adaptive coping and resiliency through a reduction in emotional response intensity with repeated exposures [31]. The NREM sleep also has an indirect influence on emotional balance. Slow-wave sleep is associated with decreased sympathetic activity and increased parasympathetic dominance, which provides a physiologic state that is conducive to stress recovery. Specifically, NREM can stabilize declarative memory and strengthen cognitive control networks, and thus may buffer emotional experiences and decrease an individual's vulnerability to stress-related disorders [12]. Age-related reductions in both slow-wave and REM sleep have been implicated in the decline of emotional regulation seen in older adults. Diminished continuity of these stages has been correlated with increased prevalence of mood disorders, including depression and anxiety, in later life [20]. Conversely, interventions that restore or extend sleep duration improve emotional stability, highlighting sleep's therapeutic potential for mental health [21]. In summary, sleep functions as a natural regulator of psychological well-being. Through its combined influence on emotional memory processing, stress recovery, and mood stabilization, it plays a central role in maintaining mental health across the lifespan. Functions of sleep and their physiological roles are given in **Table 2**.

Table 2 Functions of Sleep and Their Physiological Roles

Function	Primary Sleep Stage(s) Involved	Key Physiological Mechanisms	Outcomes / Benefits
Memory consolidation and learning	NREM (slow-wave sleep) and REM	Hippocampal–neocortical transfer during SWS; limbic activation during REM	Stabilization of declarative memory, consolidation of procedural skills, emotional memory integration, creativity
Hormonal regulation and metabolic balance	NREM (especially SWS) and circadian-timed transitions	Growth hormone release in SWS; cortisol nadir in early night; melatonin secretion in darkness; leptin/ghrelin modulation	Tissue repair, stress regulation, circadian alignment, appetite control, insulin sensitivity
Immune function and repair	NREM (SWS) and REM (balancing role)	Cytokine secretion (IL-1β, TNF-α); enhanced antigen presentation; optimized T cell activation	Improved vaccine response, resistance to infection, reduced systemic inflammation

Neural detoxification and brain homeostasis	NREM (SWS) with REM complement	Glymphatic clearance of amyloid-beta and tau; REM-linked neural reorganization	Brain waste removal, prevention of neurodegeneration, maintenance of cognitive integrity
Emotional and psychological health	REM (emotional processing) and NREM (stress recovery)	Amygdala–hippocampal reactivation during REM; parasympathetic dominance during NREM	Emotional regulation, mood stabilization, resilience to stress and trauma

4. Sleep Deprivation Effects

4.1. Cognitive and Neurobehavioral Impairments

Adequate sleep is essential for sustaining optimal cognitive performance, and sleep deprivation produces measurable deficits in attention, memory, and executive functioning. Experimental studies consistently show that both total and partial sleep loss impair neurobehavioral performance in a dose-dependent manner, with effects comparable to those of alcohol intoxication [31]. Reduced alertness and slower reaction times are among the earliest consequences, increasing the likelihood of accidents in occupational and transportation settings. For instance, prolonged wakefulness exceeding 17 hours has been associated with decrements in psychomotor vigilance comparable to blood alcohol levels above legal driving limits [44]. Working memory and higher-order decision-making processes are also particularly vulnerable to sleep restriction. Functional neuroimaging has demonstrated reduced prefrontal cortex activation during tasks requiring sustained attention and executive control in sleep-deprived individuals [12]. This diminished cortical activity contributes to lapses in reasoning, poor judgment, and reduced problem-solving capacity. The hippocampus, which is central to forming declarative memories, functions poorly under sleep-deprived conditions, resulting in weaker retention of newly acquired information [45]. Emotional regulation also suffers. Studies show that people who lack sleep respond to negative experiences with heightened amygdala activity and reduced connectivity to the prefrontal cortex. This imbalance makes them more irritable, anxious, emotionally unstable, and less able to cope with stress [21]. These neurological changes help explain why long-term sleep loss is strongly tied to mood disorders such as depression and generalized anxiety. The behavioral and cognitive effects also extend beyond the individual. Large-scale studies have linked sleep deprivation to higher rates of workplace errors, industrial mishaps, and road traffic accidents. Because many people underestimate how much chronic sleep loss affects them, they may not notice their declining performance, allowing cognitive deficits to accumulate over time [46]. In sum, insufficient sleep undermines core processes of thinking and behavior by weakening attention, memory consolidation, and emotional balance. These impairments not only lower personal performance and safety but also create widespread health and economic burdens on society.

4.2. Metabolic and Endocrine Consequences

Sleep is essential for keeping metabolism in balance, and losing sleep disrupts several hormonal systems. Short and long-term sleep deprivation changes the glucose regulation, appetite, and energy status, which predispose to the risk of obesity, diabetes, and other metabolic syndromes. The impact of sleep restriction on insulin sensitivity is one of the most stable results. Experimental studies have shown that even a single week of curtailed sleep reduces insulin responsiveness, leading to impaired glucose tolerance [37]. These metabolic changes mirror the early stages of type 2 diabetes and occur independently of caloric intake, highlighting the direct physiological impact of insufficient sleep. Prolonged sleep restriction further amplifies these effects, creating a metabolic environment conducive to chronic disease development [32]. Appetite-regulating hormones are also strongly influenced by sleep duration. Short sleep leads to reductions in leptin, the hormone responsible for satiety, and increases in ghrelin, which stimulates hunger [36]. This hormonal imbalance increases appetite, particularly for calorie-dense foods, and provides a mechanistic explanation for the robust epidemiological association between insufficient sleep and obesity risk. Chronic elevation of ghrelin, together with reduced leptin, promotes overeating and weight gain in sleep-deprived populations [17].

Cortisol, the body's primary stress hormone, demonstrates a circadian rhythm that is sensitive to sleep patterns. Normally, cortisol levels are lowest in the early part of the night and rise toward morning. However, sleep restriction and fragmentation elevate nocturnal cortisol concentrations, a change associated with impaired glucose metabolism and heightened cardiovascular risk [34]. Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis through poor sleep thus contributes to both metabolic and stress-related pathologies. Melatonin, secreted during the night, not only regulates circadian alignment but also exerts antioxidant and metabolic effects. Disruption of melatonin secretion by light exposure or irregular sleep schedules contributes to circadian misalignment, which has been associated with increased risk of metabolic syndrome in both clinical and shift-working populations [35]. Together, these findings demonstrate that sleep deprivation alters multiple endocrine systems, creating a cascade of metabolic disturbances.

Reduced insulin sensitivity, dysregulated appetite hormones, elevated cortisol, and altered melatonin secretion collectively explain the strong link between insufficient sleep and long-term metabolic disease burden.

4.3. Cardiovascular and Immune Dysregulation

Sleep deprivation has profound consequences for both cardiovascular regulation and immune competence. Normal sleep, particularly slow-wave stages, is characterized by parasympathetic predominance, lowered heart rate, and reduced blood pressure, changes that collectively provide restorative benefits for the cardiovascular system. In the absence of sufficient sleep, however, sympathetic nervous system activity remains elevated, leading to persistent increases in heart rate and blood pressure [7]. Over time, this imbalance contributes to the development of hypertension, arrhythmias, and heightened risk of ischemic heart disease. REM sleep further illustrates the delicate cardiovascular balance maintained by sleep. This stage is marked by autonomic instability, with irregular heart rhythms and fluctuations in blood pressure. When sleep is fragmented or curtailed, the proportion of REM decreases, yet the instability it normally balances with NREM processes becomes dysregulated. Such alterations have been linked to increased vulnerability to acute cardiac events, particularly in individuals with underlying disease [21]. Immune function is similarly impaired by sleep deprivation. Experimental studies show that sleep restriction suppresses cytokine production and reduces natural killer cell activity, thereby weakening the innate immune defense [40]. The adaptive arm of the immune system is also disrupted, with impaired antigen presentation and weaker antibody responses following vaccination in sleep-deprived individuals [41]. These alterations explain why insufficient sleep is associated with increased susceptibility to infections and slower recovery from illness. Chronic sleep loss amplifies systemic inflammation, as evidenced by elevated circulating pro-inflammatory markers. This sustained low-grade inflammatory state creates a biological environment conducive to atherosclerosis, diabetes, and neurodegenerative disease [43]. Together, cardiovascular stress and immune dysregulation form a central pathway by which sleep deprivation accelerates long-term morbidity. In summary, the loss of restorative cardiovascular patterns during NREM and the disruption of immunological balance highlight how sleep deprivation undermines two systems critical for survival. By maintaining sympathetic overactivation and weakening host defense, insufficient sleep significantly increases vulnerability to both acute and chronic disease.

4.4. Long-Term Health Risks and Psychiatric Outcomes

The consequences of chronic sleep deprivation extend beyond immediate cognitive, metabolic, and cardiovascular impairments, contributing significantly to long-term health risks and psychiatric disorders. A growing body of evidence indicates that insufficient sleep is a major factor in the development of neurodegenerative diseases. Disruption of slow-wave sleep reduces glymphatic clearance of amyloid-beta, thereby accelerating its accumulation in the brain and predisposing individuals to Alzheimer's disease [3]. Similar mechanisms involving impaired removal of neurotoxic proteins have been implicated in other neurodegenerative processes, including Parkinson's disease [20]. Psychiatric outcomes are also tightly linked to sleep disturbances. Chronic restriction or fragmentation of sleep has been consistently associated with depression, anxiety, and emotional instability. Heightened amygdala reactivity and reduced prefrontal cortical control, both observed in sleep-deprived individuals, provide neurobiological explanations for the increased prevalence of mood disorders in those with insufficient sleep [22]. These mechanisms also account for the common clinical finding that insomnia is both a risk factor for, and a symptom of, major depressive disorder [12]. Furthermore, sleep deprivation contributes to cumulative health risks by sustaining systemic inflammation and hormonal dysregulation. People who consistently sleep too little often show higher cortisol levels and ongoing low-grade inflammation, both of which raise the risk of heart disease, metabolic problems, and a weakened immune system [34]. These effects also help explain the strong link between poor sleep, mental health issues, and physical illness. Large population studies further reveal that short, chronic sleep is tied to higher overall mortality, showing that the negative effects of poor sleep extend beyond specific conditions to impact life expectancy itself [46].

Altogether, the evidence suggests that sleep loss is a major risk factor for psychiatric disorders and long-term health decline. Poor-quality sleep leaves the body more vulnerable to disease and shortens lifespan through unstable brain function, poor emotional control, and reduced cellular repair. These wide-ranging consequences of sleep deprivation are summarized in **Figure 2** and **Table 3**.

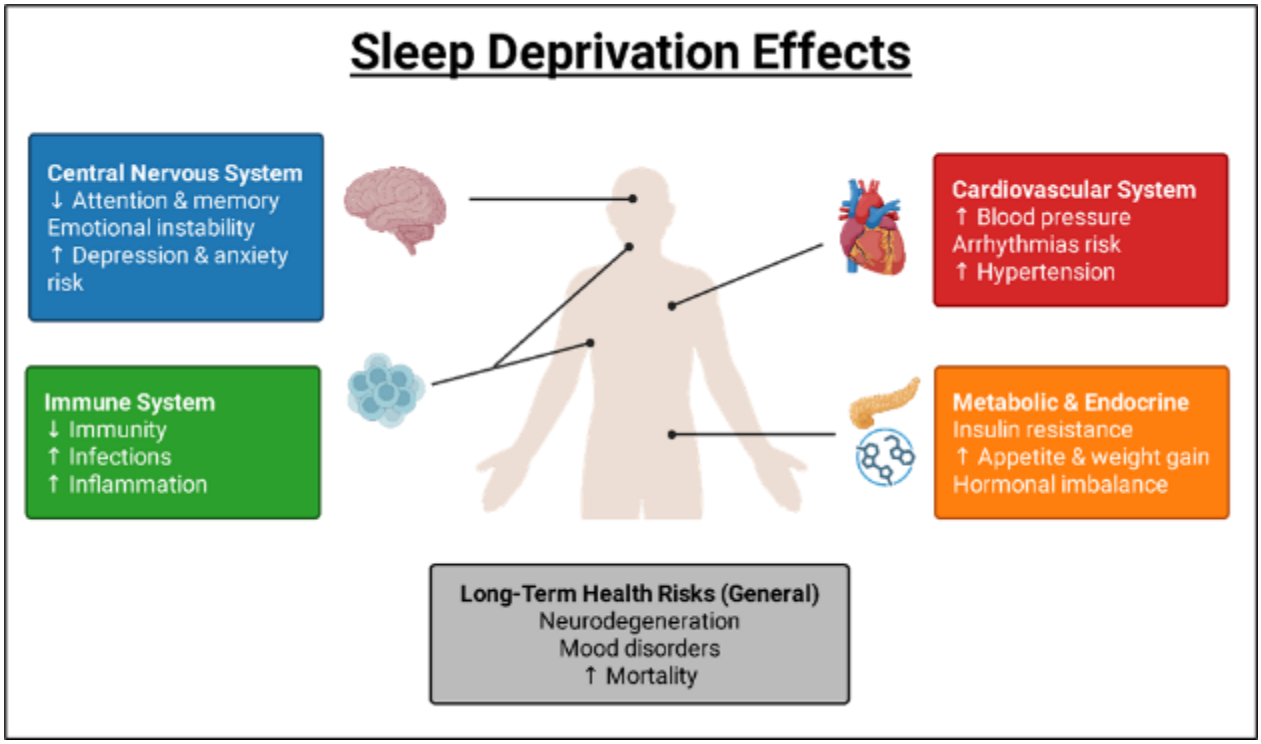


Figure 2 Physiological effects of sleep deprivation on major body systems

Table 3 Effects of Sleep Deprivation on Human Health

Domain	Key Changes	Consequences
Cognitive & Neurobehavioral	Reduced vigilance, impaired working memory, decreased prefrontal activation, heightened amygdala activity	Slower reaction times, poor decision-making, emotional instability, higher accident risk
Metabolic & Endocrine	Reduced insulin sensitivity, altered leptin ↓ and ghrelin ↑, elevated nocturnal cortisol, disrupted melatonin	Weight gain, obesity, impaired glucose tolerance, increased diabetes risk, circadian misalignment
Cardiovascular & Immune	Persistent sympathetic activation, irregular HR & BP, reduced NK cell activity, weaker vaccine response, systemic inflammation	Hypertension, arrhythmias, impaired immunity, higher infection risk, chronic inflammation
Long-Term Risks & Psychiatric Outcomes	Reduced glymphatic clearance, elevated cortisol, chronic inflammation, impaired amygdala-prefrontal regulation	Alzheimer's and neurodegenerative risk, depression, anxiety, all-cause mortality

5. Conclusion

Sleep is one of the four foundations of good health and is closely tied to the regulation of nearly every system in the body. Its rhythm alternating between the deep restorative stages of NREM sleep and the active phases of REM sleep forms the basis of internal balance. NREM sleep supports cell renewal, strengthens the immune system, and consolidates declarative memory, while REM sleep helps regulate emotions, promotes brain flexibility, and enhances creative problem-solving. Together, these processes create harmony between body and mind. When sleep is disrupted or denied, the consequences are serious: thinking slows, reaction times worsen, and decision-making suffers, raising the risk of mistakes and accidents. Over time, poor sleep also contributes to obesity, diabetes, reduced insulin sensitivity, cardiovascular problems, and appetite and hormone imbalances. The immune system weakens, leaving the body more prone to infections and long-term inflammation. With aging, these disruptions amplify vulnerability to psychiatric illness and accelerate neurodegenerative processes, ultimately reducing both quality of life and life expectancy.

Because the long-term effects of poor sleep are well known, it is essential to encourage healthy sleeping habits. Staying healthy across the lifespan depends on getting enough sleep, keeping regular sleep schedules, and aligning rest with the body's natural circadian rhythm. Sleep quality improves through simple practices such as maintaining consistent bedtimes, limiting artificial light at night, creating comfortable sleep environments, and managing stress. On a wider scale, public health initiatives that highlight the value of sleep alongside diet and physical activity are urgently needed as chronic sleep deprivation becomes increasingly common in modern society. Sleep is far from wasted time; it is an active and essential process that safeguards health. A good night's sleep enhances memory, emotional balance, metabolism, and immune strength. For this reason, policymakers and healthcare systems should regard sleep as a pillar of preventive medicine. Protecting and promoting sleep benefits not only individuals but also builds healthier, more resilient communities.

Compliance with ethical standards

Disclosure of conflict of interest

The author has no relevant financial or non-financial interests to disclose.

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