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The Impact of Sleep on Cognitive Function

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Abstract

Sleep is essential for cognitive performance and long-term neurological health, yet the effects of sleep duration, quality, and deprivation on cognition remain characterized incompletely. This literature review synthesizes peer-reviewed research examining associations between sleep and attention, memory, executive function, processing speed, decision-making, and related neural mechanisms. Evidence across healthy and clinical populations indicates that insufficient sleep, poor sleep quality, and irregular sleep patterns are consistently associated with impaired cognitive performance. Proposed mechanisms include disrupted synaptic plasticity and memory consolidation, altered functional connectivity, impaired glymphatic clearance, neuroinflammation, and changes in brain structure and neurotransmission. Findings also suggest that both sleep duration and sleep consistency contribute to cognitive outcomes. Collectively, the evidence supports sleep as a modifiable factor in cognitive and neural health. Future research should prioritize longitudinal and interventional designs using objective sleep measures and standardized cognitive assessments.

Keywords: memory consolidation; executive processes; attention; circadian rhythms; neuroplasticity; glymphatic clearance; brain health

1. Introduction

Sleep is a fundamental physiological process which is vital for maintaining proper physical health, emotional regulation, and optimal cognitive performance. In modern society, changes in lifestyle factors including extended work hours, academic demands, and increased screen exposure have contributed to an increase in sleep restriction and disruption to circadian patterns across diverse populations. Understanding how sleep influences brain function has become a critical focus within cognitive neuroscience, neuropsychology, and public health.

The human brain relies on complex and dynamic neural network interactions which are highly sensitive to changes in sleep duration, quality, and longevity. Research demonstrates that adequate sleep supports a wide spectrum of cognitive development and domains from sustained attention, awareness, and processing speed to higher executive functions including working memory, cognitive flexibility, risk assessment, and decision making. Furthermore, sleep plays a critical role in memory consolidation, facilitating the transition of newly acquired information into long term neural pathways through certain stage-specific psychological processes such as slow-wave sleep and rapid eye movement sleep. Conversely, sleep disruption in the form of acute total sleep deprivation, chronic partial sleep restriction, or poor sleep quality severely impairs these systems which increases reaction times, increases error rates and alters the overall functional connectivity between key regions of the brain.

Despite a large amount of literature documenting these impairments, several major questions remain regarding the precise mechanisms and generalizability of current finding across varying populations. Many existing studies rely on acute laboratory sleep deprivation models which may not reflect the real-world implications and occurrence. Additionally reliance on self-report measurements introduces potential recall bias and obscures the contribution of sleep specific research factors.

This literature review synthesizes peer-reviewed findings from neuroscience and psychology to examine the multifaceted relationship between sleep parameters and cognitive outcomes in both healthy and clinical cohorts. Specifically this review evaluates sleep deprivation and cognitive function, sleep duration on cognitive performance, sleep quality and cognitive outcomes, neural mechanisms, and methodological limitations and research gaps. By integrating evidence across various different methodologies and assessment, this review aims to provide a comprehensive synthesized framework on the essential role of sleep in preserving human cognitive function and brain health.

2. Methods

This study used a narrative literature review design to examine the relationship between sleep and cognitive function. The research team selected 20 peer-reviewed articles relevant to the topic and organized them in a shared Google Sheet. The literature was divided among the major sections of the review, including sleep deprivation and cognitive function, sleep duration and cognitive performance, sleep quality and cognitive outcomes, and neural mechanisms. Each researcher was assigned three articles to independently review, extract information from, and analyze. To standardize data extraction across the research team, the Google Sheet included the following categories for each article: article title, article link, rationale for source selection, study

design, sample size, study population, sleep variable measured, cognitive outcomes measured, main findings, and reported limitations. Sleep-related variables included measures such as sleep duration, sleep quality, sleep deprivation, and sleep variability, while cognitive outcomes included domains such as attention, memory, processing speed, executive function, and decision-making. After data extraction, findings were compared and synthesized within their respective thematic sections. Researchers evaluated similarities and differences across studies, including patterns in cognitive outcomes, proposed neurological mechanisms, study populations, research designs, and methodological limitations. The extracted evidence was then integrated into a qualitative synthesis to identify the primary relationships between sleep characteristics and cognitive function, areas of consistency or disagreement within the literature, and remaining research gaps.

3. Results

Sleep Deprivation and Cognitive Function

Sleep deprivation occurs when an individual does not obtain sufficient sleep for optimal physical and cognitive functioning. It can be acute, resulting from one or a few nights of inadequate sleep, or chronic, caused by repeated sleep restriction over time. Modern lifestyles, extended working hours, and shift-based occupations have increased its prevalence. Researchers study sleep deprivation because it is associated with impairments in several cognitive domains, including attention, memory, learning, executive function, and decision-making[1]. Furthermore, deep sleep (N3) has been identified as a protective factor against cognitive decline, whereas higher insomnia severity (AIS score) and frequent nighttime awakenings are associated with an increased risk of severe cognitive impairment.

Attention and vigilance are among the earliest cognitive functions affected by sleep deprivation. Sleep loss disrupts communication between brain networks and alters activity in the prefrontal cortex, thalamus, amygdala, and hippocampus, reducing the ability to sustain attention and remain alert. Studies consistently report poorer performance on attention-based tasks, characterized by increased lapses in attention, slower responses, and reduced vigilance. Prolonged sleep deprivation also impairs working memory, including the central executive responsible for coordinating cognitive processes, further affecting concentration and higher-order cognitive functions.

Reaction time and processing speed are widely used indicators of cognitive performance and decline significantly following sleep deprivation. Individuals experiencing insufficient sleep exhibit slower reaction times, delayed information processing, and greater variability in task performance. Chronic sleep deprivation may also impair the brain's glymphatic clearance system, reducing the removal of metabolic waste products such as amyloid-beta, thereby increasing the risk of neurodegenerative changes. Although some brain regions temporarily compensate for sleep loss, these mechanisms are limited, leading to progressive deterioration in cognitive performance and increased errors during complex tasks.

Many past studies have concluded that sleep plays a critical role in memory consolidation, and, as a result, sleep deprivation (SD) can hinder these processes. Particularly, Mohammad A Khan and Hamdan Al-Jahdali discuss in their review that sleep deprivation was seen to disrupt memory consolidation inside the hippocampus. The NMDA receptor structure, which is essential for the consolidation phase of memory and moving it from unstable to permanent memory, was found to be modified after sleep deprivation. The hindrance in memory consolidation and, therefore, memory retention, combined with impact of sleep deprivation on attention can also hinder learning. Specifically, SD was found to decrease the activation of the PPA and fronto-parietal cortex in response to task-related activity, causing attention impairments[2]. Similarly, Joseph M Dzierzewski and colleagues highlighted that chronic and long-term insomnia has associations with cognitive decline and dementia, potentially linking to decreased memory retention and consolidation.

Moreover, their article suggests that during slow wave NREM sleep, synapses throughout the brain are reduced to counteract the net strengthening of network synapses during wakefulness, including learning, where the absorption of surrounding information necessitates solidifying networks within the brain. This processes during wakefulness requires increased cellular processes for energy and learning consolidation, while spontaneous activity (neural activity not driven by a task) can renormalise the synaptic strength to restore cellular homeostasis during sleep, increasing synaptic plasticity the next day, improving memory acquisition and consolidation as energy is saved when counteracting the network effects of synaptic excitation and neural activity during the wakeful period, enhancing learning and memory retention[3]. Additionally, Adam J. Krause and his colleagues have shown that the offline consolidation of hippocampus-dependent memories are benefited by sleep after learning. SD was found to reduce the ability of the brain to induce hippocampal long-term potentiation (LTP), leading to the decaying of memory after neuroplasticity. They also suggested that sleep deprivation leads to the extracellular build-up of the metabolic adenosine, which was found to disrupt intracellular cAMP signalling, decreasing hippocampal NMDA receptor signalling essential for stable LTP. They suggested that sleep is crucial for this process because adenosine is cleared from the brain during sleep, which increases plasticity and memory consolidation after learning[4].

Executive functions include processes like planning, problem-solving, decision-making, judgement, and reasoning, and there is strong evidence from many studies showing the impact of sleep deprivation on these critical functions. The study conducted by Mohammad A Khan and Hamdan Al-Jahdali also concluded that individuals with SD lose the functional connectivity between the amygdala and the mPFC (medial prefrontal cortex), which is known for its involvement in executive functions by exhibiting inhibitory projections to the amygdala. It was found that, with a lack of sleep, an amygdala hyperlimbic reaction increases, causing reactions to stimuli with negative emotional connotations, especially as the brain misses a corrective reset reactivity because of the dysfunctional integrity of the mPFC-amygdala circuit, causing inappropriate decision making, behavioral responses, and social judgements, causing a higher level of difficulty when deciding on a suitable course of action, leading to impaired executive performance[2]. Taking a different approach on SD and lack of sleep, Dzierzewski and colleagues concluded that participants with insomnia performed significantly worse on executive functioning tasks compared to healthy control groups, especially as a result of early morning awakening associated with worse cognitive performance within executive functions. This paper also highlighted that sleep disordered breathing showcased significant negative effects on attention and executive function, suggesting PAP therapy as a promising approach, though with inconsistent cognitive benefits[3]. Another paper by Anna Hyndych, Rima El-Abassi, and Edward C Mader Jr found that executive functions like decision making were significantly impaired as a result of the prefrontal cortex's heightened sensitivity to insufficient sleep, as a result of increased amygdala reactivity, as seen in previous studies. This was seen to contribute to emotional dysregulation, impulsiveness, and risk-taking behaviors, which was concluded to have impacts on emotional instability and motor performance decline. The study found that key physiological processes like homeostatic regulation, molecular biosynthesis, and neural circuit development are more effectively performed during sleep, while also being essential for daytime cognition and behavior, as well as executive function and decision-making. The disruption of these processes as a result of sleep deprivation was, as a result, found to impair judgement and cognitive and behavioral processes. Sleep deprivation was also found to cause a decline in cognitive flexibility, referring to the capacity to adjust thinking and behavior according to changes in goals and environmental stimuli, causing sleep deprivation to lead to individuals struggling to modify their cognitive strategies, causing a tendency towards rigid, inflexible decision making and thinking[5].

Acute sleep deprivation is defined by a single period of extended wakefulness, while chronic sleep deprivation may result from continuous sleep loss and obtaining insufficient sleep repeatedly, over time. A study conducted by Zhongkai Ren and company found that individuals who work regular night shifts are at a higher risk of chronic cognitive impairment due to chronic SD, while also indicating that these individuals who experience long-term circadian rhythm reversal cannot escape the cumulative effects of cognitive impairments even if they start getting adequate sleep. The study utilised an objective P300 auditory component, which refers to the event-related potential brain wave that peaks around 300 milliseconds after a specific stimulus, which is audio in this case (auditory oddball paradigm), reflecting attention and decision-making components. The study found a significant increase in P300 latency and reaction time among the participants following a 24 hour SD period, with a significant decrease in P300 amplitude. An unexpected

finding was that the chronic SD group showed a less significant cognitive impairment after the sleep deprivation experiment compared to the acute SD group which showcased a notable decline in cognitive function, especially in reaction time. Ultimately, it was concluded that, despite the chronic group showing a higher tolerance to sleep deprivation, they experienced chronic cognitive impairments which accumulated as a result of frequent night shifts, while the acute group demonstrated more severe cognitive deficits after the sleep deprivation period[6]. The paper written by Anna Hyndych and company further highlighted that individuals accumulate sleep debt through consistent, mild sleep deprivation, which can subtly but significantly impair memory and executive function over time. The study found that functional neuroimaging studies show the reduced activation of the DLPFC region following sleep loss in acute SD, which is often related to the temporary storage and manipulation of information, significantly disrupting executive function as a result. Significant findings in chronic sleep deprivation, on the other hand, are related to the contribution of consistent sleep loss to the dysregulation of the cognitive and emotional systems, with neuroimaging studies clarifying how sleep-related disturbances influence large-scale brain networks involved in executive function and emotional regulation. It was found that individuals with primary insomnia particularly exhibited altered connectivity between the striatum, default mode network, and sensorimotor networks, hindering the reorganisation of resting-state activity. The study concluded that both acute and chronic SD significantly impair cognitive and motor function, though chronic SD may present more severe deficits in the long-term, decreasing productivity and increasing the risk of accidents and errors in the work place[5]. Another study done by Adam J Krause and company focused on chemical signalling and sleep pressure-related molecules, particularly on adenosine, which was found to accumulate with increasing time spent awake, as the byproduct of cellular energy (ATP) breakdown[4]. This activates adenosine receptors in order to suppress wakefulness and promote sleep pressure, but drops in concentration during restorative sleep. However, it was found that sleep deprivation may overdrive this process and cause adenosine to build up to abnormally high levels in regions including the cerebral cortex, significantly increasing sleep pressure. In order to cope with this, adenosine receptors will be upregulated, increasing the density by up to 15% in areas like the orbitofrontal cortex, which dampens overall neuronal excitability and directly triggers brain fog, slow reaction time, and impaired attention, as a result of continuous, chronic sleep loss[7]. Moreover, it was found that certain actions after SD may be compensatory and adaptive, particularly as the human brain can compensate for SD to a certain degree through behavioral strategies including talking or standing up when attempting to stay awake when sleep deprived, despite the cumulative deficits that are involved in chronic sleep deprivation, compared to the severe yet short-term impacts of SD in acute groups[4].

Sleep Duration and Cognitive Performance

A paper written by Xin You Tai and colleagues on the impact of sleep duration on executive function and brain structure concluded that 7 hours of sleep per day was the optimal sleep duration for maximum cognitive performance. Associated with the highest cognitive function, 7 hours of sleep was found to be optimal as a result of cognitive performance decreasing by every hour below or above this sleep duration, this quadratic relationship remaining consistent in older individuals as well. One key finding in this study was that individuals that had between 6-8 hours of sleep were found to have significantly greater grey matter volume in 46 of 136 different brain regions, within a distributed anatomical pattern in the frontal, temporal, parietal, and cerebellar regions. An increased grey matter volume corresponds to high cognitive performance, as well as acting as a buffer against age-related brain shrinkage, acting as a defense mechanism against dementia and Alzheimer's. Within the cohort, individuals who reported longer sleep durations were found to have a smaller cerebellum, while other brain regions also showed lower volume only with longer sleep durations. Furthermore, on top of the impacts observed in the structural grey matter integrity, a smaller volume of white matter hyperintensities was also observed in individuals who reported between 6-8 hours of sleep. With white matter being considered as a brain marker that is closely related to cardiovascular burden, this can be seen as a further implication of sleep duration as an important factor within the cardiovascular effects which can impact brain health. The paper also states that a low sleep duration of six hours or less was associated with a higher risk of developing dementia in later life compared to the optimal 7 hours, though a longer sleep duration of eight hours or more was not significantly associated with this risk. The study found a clear decline in executive function with increasing age with stable sleep durations across ages, with a median of 7 hours, while the negative effects of very short and long sleep durations were more apparent in younger individuals. Moreover, the paper concluded that shorter sleep durations from 6-3 hours corresponded with

increasingly worse performance, and a similar negative effect was present from 8-12 hours of sleep. Additionally, it was found that sleep duration of 6-8 hours remained a significant and only positive predictor of executive function, after controlling for vascular comorbidities and genetic risk (including the APOE ε4 genotype). The APOE ε4 allele status was found to have an effect on cognition and the risk of developing sporadic dementia, and to score this risk factor, a point was given to each ε4 allele an individual carried in their genotype[8].

Another paper by Samantha A. Keil and colleagues on longitudinal sleep patterns and cognitive impairments in older adults also supports the findings from the paper by Xin You Tai and co. This paper highlighted the idea that a shorter sleep duration and increased sleep variability was significantly associated with increased risk of cognitive impairments, also concluding that higher instability in sleep duration over long periods could influence higher amounts of cognitive decline in older adults. This paper not only supported the idea of 7 hours of sleep being the optimum sleep duration for maximum cognitive performance, but it also discussed the impact of sleep duration on the development of dementia in further detail. For instance, the paper summarised that observational clinical studies done in the past highlighted the idea that amyloid ß deposition, related to progressive memory impairments, began at least 15 years before the onset of the clinical dementia diagnosis. The paper also states that many studies in the past indicated that 90% of patients experienced disrupted sleep before the emergence and onset of cardinal disease symptoms. It was concluded that short sleep duration is associated with increased risks of cognitive impairments in healthy aging adults, while also concluding that AD biomarker studies had found that poor sleep quality and short sleep duration may be linked to a greater AD-related pathological burden in the brain, which directly links sleep duration and chronic sleep disruption to dementia diagnosis[9]. AD-related pathological burden refers to the total accumulation of Alzheimer's disease brain changes, specifically related to amyloid ß plaques and neurofibrillary tau tangles, hindering normal communication between neurons and potentially cause the neurons to fail as a result of the build up of protein fragments and misfolding of tau proteins, leading to long term, progressive memory loss[10]. In order to test the impact of sleep duration on cognitive decline, the Cox proportional hazards regression technique was used, observing a higher rate of cognitive impairment. Overall, being a short sleeper with a median sleep duration of under 7 hours was seen to cause a hazard ratio of 3.67 for cognitive impairment, with higher sleep variability across ages being associated with a cognitive impairment hazard ratio of 3.06[9].

Next, a paper written by Molly E Zimmerman and colleagues, related to the effects of insufficient sleep and adequate sleep on cognitive function in healthy adults, demonstrated that stable sleep for at least 7 hours per night improves the working memory and response inhibition in healthy adults. Just like the previous two papers discussed, this study emphasised the idea that consistent short sleep durations were associated with poor attention. It was hypothesised that manipulating sleep duration would affect cognitive functions like processing speed and the working memory, and it was later found that this hypothesis was partially supported as it was found that adequate sleep intervention would improve the working memory. It was also demonstrated that adequate sleep intervention would improve performance on tasks of response inhibition and attention beyond a practice effect. The working memory was investigated as a result of its importance towards a wide range of everyday activities which involve multitasking and effective function to effectively achieve complex goals, while response inhibition was tested as a result of its influence on executive functions which underlie activities requiring self-control, performance monitoring, as well as resisting the urge for impulsive responses to stimuli. The paper highlights the conflicts between the findings of different studies, especially by stating that despite some prior studies demonstrating that sleep loss coils lead to working memory impairments, other studies did not, referring to a chronic sleep restriction study in adolescents and young adults that found no impact on the accuracy of working memory performance, despite reaction time declines being observed. It is also stated in this paper that a speed/accuracy trade-off was observed within participants that went 34.5 hours of wakefulness and therefore sleep duration restriction, where, though their responses to a test of response inhibition compared to baseline testing was not less accurate, they were seen to have responded more slowly to the tests, while their performance improved with recovery sleep, with decreased response inhibition being reported in individuals with insomnia and sleep restriction. Moreover, it was summarised that in another study, it was found that experimentally manipulated insufficient sleep showed that sleep had detrimental effects on cognitive function across sustained attention, memory, and executive function. Additionally, the paper concluded that participants experiencing 21 days of chronic sleep restriction of 5-5.6 hours of sleep followed by 9 nights of recovery sleep demonstrated persistent cognitive and vigilance deficits despite the recovery sleep, while 6 weeks of chronic sleep restriction with 5

hours on weekdays and 8 hours of weekends showcased poorer performance on spatial orientation and vigilance as well, on top of not improving with two nights of weekend recovery sleep [11].

Sleep Quality and Cognitive Outcomes

In postmenopausal women, sleep quality metrics rather than duration alone are associated with cognitive performance. Benýšková, Beníčková, and Gimunová found that reduced sleep efficiency and greater fragmentation corresponded to weaker memory and attention, while prolonged wake after sleep onset and extended sleep onset latency tracked with poorer cognition and higher mild cognitive impairment risk, though each of these parameters was examined in only a small number of included studies[12]. The same review reported a U-shaped duration effect, with short sleep below six hours and long sleep above eight hours both linked to attentional difficulty and slower information processing relative to roughly seven hours. Findings were not uniform: three included studies detected no significant sleep-cognition association, all of them questionnaire-based with comparatively small samples.

Synthesized evidence on sleep architecture offers a partial account of why quality may matter beyond total sleep time. Weighall and Kellar, summarizing nineteen systematic reviews and meta-analyses, describe slow wave sleep and spindle activity as playing critical roles in consolidation[14]. One pooled analysis of fifty-three studies found a small-to-moderate spindle-memory association, stronger for procedural than declarative memory, but also detected publication bias and cautioned that effects may be overestimated. Sleep-dependent consolidation declines in older adults, particularly for hippocampus-dependent declarative memory, which the authors suggest is likely linked to age-related loss of slow wave sleep. The picture is not fully consistent, since one included meta-analysis found that slow wave sleep and REM duration did not significantly moderate sleep's episodic memory benefit. Interpretive caution is warranted for the Benýšková review in particular, which relied heavily on self-report, pooled heterogeneous instruments, and states in its methods that data extraction and quality scoring were carried out by a single reviewer[12]. Weighall and Kellar report a different set of constraints: single screening, PubMed as the sole database, an English-language restriction, and no formal critical appraisal, which they concede stops short of a true umbrella review.

Neural Mechanisms

Sleep loss affects cognition through several interconnected biological mechanisms rather than a single pathway. Khan and Al-Jahdali describe the Synaptic Homeostasis Hypothesis, which proposes that slow-wave sleep weakens synapses strengthened during wakefulness, restoring the brain's signal-to-noise ratio and preparing it for new learning[2]. When sleep is restricted, this process is disrupted, reducing neuronal selectivity and impairing memory consolidation. They also note that evidence remains mixed, as some studies report poorer memory encoding after sleep deprivation, whereas others show improved encoding following brief sleep with enhanced slow oscillations. Expanding on memory processing, Weighall and Kellar explain the Complementary Learning Systems theory, in which the hippocampus rapidly stores new experiences while sleep gradually transfers them to stable neocortical networks. Sleep spindles and slow-wave activity support this transfer, although age-related reductions in slow-wave sleep appear to weaken this consolidation process[14].

Several studies focus on the structural and physiological changes associated with poor sleep. Tai, Chen, Manohar, and Husain found that individuals sleeping 6–8 hours had greater grey matter volume in regions including the hippocampus, orbitofrontal cortex, and thalami, together with fewer white matter abnormalities[8]. They suggest that insufficient sleep reduces slow-wave sleep density and may impair amyloid-β clearance, contributing to cortical thinning. Supporting this mechanism, Keil, Iliff, and colleagues identify impaired glymphatic clearance as a likely link between chronic sleep disruption and cognitive decline, citing evidence that sleep promotes the

removal of amyloid-β, tau, and α-synuclein from the brain[9]. Xu, Qin, Wen, Zhang, and Zhou further propose that the astrocyte circadian clock helps coordinate toxin clearance, synaptic regulation, neurotransmitter balance, and inflammation while also enhancing cerebrospinal fluid clearance during high-quality sleep[15]. Neural network dysfunction provides another explanation for cognitive impairment. Krause, Ben Simon, Mander, and colleagues report that sleep deprivation reduces prefrontal and parietal activity, destabilizes the default mode network, weakens hippocampal memory encoding, increases impulsive reward processing through altered dopamine signaling, and amplifies amygdala responses while reducing prefrontal regulation of emotion[4]. Similarly, Ren, Mao, Zhang, and Wang found that total sleep deprivation significantly prolonged P300 latency and reaction time, reflecting slower cortical processing and reduced brainstem responsiveness[6]. Although chronic night-shift workers showed smaller performance declines, the authors suggest this may represent physiological adaptation rather than protection from long-term neural damage.

Other evidence highlights additional interacting mechanisms. Dzierzewski, Perez, Ravyts, and Dautovich propose that neuroinflammation, disrupted synaptic plasticity, vascular and metabolic changes, altered neurotransmission, and psychosocial factors collectively determine cognitive outcomes[3]. Sun, Qu, Zhang, and colleagues suggest that reduced cerebral oxygen supply and impaired cerebrovascular reactivity may further limit cognitive flexibility[16]. while Xia, He, Sun, and Wang show that irregular sleep schedules independently predict poorer cognition through circadian disruption, hormonal imbalance, and structural brain changes[17]. Together, these findings indicate that sleep supports cognition through multiple overlapping pathways, making adequate and consistent sleep essential for maintaining healthy brain function.

4. Discussion

Overall, the literature reviewed demonstrates a consistent relationship between sleep and cognitive functioning, with inadequate sleep duration, poor sleep quality, and sleep deprivation associated with impairments in attention, memory, processing speed, executive function, and decision-making. These findings support the broader understanding that sleep is not just a period of rest, but an active biological process required for maintaining efficient neural processing and cognitive performance. Across the reviewed studies, attention and vigilance appeared to be among the earliest functions affected by sleep loss, while prolonged or repeated sleep disruption was associated with broader impairments in working memory, executive functioning, emotional regulation, and learning.

These findings are consistent with prior research suggesting that sleep plays a central role in memory consolidation and neural plasticity. Previous studies have demonstrated that sleep deprivation can interfere with hippocampal long-term potentiation, NMDA receptor signaling, and the transfer and stabilization of newly acquired memories. Sleep loss has also been associated with reduced connectivity between the medial prefrontal cortex and amygdala, potentially explaining the increases in impulsivity, emotional reactivity, and impaired decision-making observed following insufficient sleep. The current review therefore reinforces existing evidence while showing that sleep-related cognitive effects extend across several interconnected neural systems rather than being limited to a single cognitive domain.

Sleep duration also emerged as an important predictor of cognitive performance. Evidence across multiple studies suggests that approximately seven hours of sleep is associated with relatively optimal cognitive functioning, while both shorter and substantially longer sleep durations may be associated with poorer outcomes. Individuals obtaining approximately six to eight hours of sleep were also reported to have greater gray matter volume across several brain regions and fewer white matter abnormalities, while shorter sleep duration and greater long-term sleep variability were associated with increased risk of cognitive impairment. Importantly, these findings suggest that sleep consistency may be nearly as important as total sleep duration. The many cognitive risks even after recovery sleep in some studies further indicates that repeated sleep restriction may produce cumulative effects that cannot always be corrected by a small number of nights of additional sleep.

Sleep quality appears to provide additional information beyond sleep duration alone. Reduced sleep efficiency, increased nighttime awakenings, sleep fragmentation, and prolonged sleep onset were associated with weaker attention and memory performance in several populations. These findings are particularly important because they suggest that simply measuring the number of hours an individual spends sleeping may underestimate the cognitive consequences of disrupted sleep. Sleep architecture, including slow-wave sleep and spindle activity, may contribute to these effects through its role in memory consolidation and neural restoration.

The importance of these findings extends beyond short-term academic or occupational performance. The mechanisms identified across the reviewed research suggest that chronic sleep disruption may contribute to long-term changes in brain health. Sleep loss has been associated with impaired glymphatic clearance, changes in gray and white matter structure, disrupted synaptic regulation, neuroinflammation, altered neurotransmission, reduced prefrontal control, and impaired functional connectivity. Because sleep promotes the clearance of metabolic waste products such as amyloid-β and tau, prolonged sleep disruption may also contribute to processes associated with cognitive aging and neurodegenerative disease. These findings emphasize the potential importance of sleep as a modifiable behavioral factor that could influence both immediate cognitive performance and long-term neurological health

A major strength of the current review is its broad synthesis of evidence across multiple dimensions of sleep, including deprivation, duration, quality, sleep architecture, and underlying neural mechanisms. It also incorporates findings from healthy and clinical populations and considers both behavioral cognitive outcomes and physiological mechanisms. Examining these areas together provides a more complete understanding of how sleep may influence cognition than focusing on sleep duration alone. However, across the literature reviewed here, the direction of the sleep cognition relationship is consistent, but the evidence base cannot yet specify that relationship precisely. Four limitations are consistently recurring throughout the papers.

The first is measurement. A large proportion of this work relies on self-reported sleep rather than objective assessment, in some cases a single questionnaire item repeated only three to five times across a twenty year window, which leaves findings exposed to recall bias. Several survey studies compound this by measuring cognition through self-assessment scales rather than performance-based testing, capturing perceived rather than demonstrated ability. Even actigraphy, which is objective, tracks movement without resolving sleep architecture, so questions about the specific contribution of slow wave sleep, spindles, and REM remain out of reach. Reviews inherit these weaknesses, and in at least two cases data extraction and quality appraisal were carried out by a single reviewer rather than in duplicate[12],[14].

The second is design. Much of the evidence is cross-sectional and observational, supporting association but not causation. The three level meta-analysis of sleep variability pooled 103 effect sizes from 15,828 participants and found a statistically robust association ($r = -0.12$), yet cannot rule out reverse or bidirectional pathways[17]. Experimental work is comparatively scarce, with only 6 of 17 studies in one scoping review being randomized controlled trials, and several deprivation studies lacking adequate control groups.

The third is ecological validity. The most consistent findings come from total sleep deprivation protocols of 24 to 38 hours, but chronic partial restriction is what most people actually experience, and results there are markedly more mixed. Single-night designs speak only to acute effects and cannot address cumulative deficits[18]. Recovery is a particular blind spot, since little of this literature establishes how long restored sleep takes to reverse impairment or which sleep stages do the restorative work. The fourth is standardization and generalizability. There is no agreed threshold for what counts as sleep restriction, and cognitive batteries differ across studies, which blocks precise dose-response curves and finer mechanistic conclusions[19]. Sample sizes range from 7 to roughly 66, populations vary widely, and sex-specific and ethnic differences are flagged as understudied. Mechanistic questions also remain open, including the unresolved relationship between objective markers and subjective performance[6].

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Future work should therefore prioritize longitudinal and interventional designs, objective sleep measurement paired with performance-based cognitive testing, protocols modelling chronic partial restriction, standardized definitions and outcome measures, and deliberate recruitment of currently underrepresented populations.

5. Conclusion

This review indicates that sleep is a fundamental determinant of cognitive performance and neural health. Across the literature, insufficient sleep duration, poor sleep quality, and sleep disruption were associated with deficits in attention, memory, executive function, processing speed, and decision-making, alongside alterations in neural mechanisms involved in synaptic plasticity, functional connectivity, and metabolic clearance. Collectively, these findings support the importance of maintaining adequate and consistent sleep as a modifiable factor in preserving cognitive function and potentially reducing long-term neurological risk. Future research should prioritize longitudinal and interventional designs, objective sleep assessment, standardized cognitive measures, and more diverse study populations to clarify causal mechanisms and establish more precise sleep-related recommendations.

Abbreviations

AIS: Athens Insomnia Scale

NMDA: N-methyl-D-aspartate

PPA: Parahippocampal Place Area

NREM: Non-Rapid Eye Movement

cAMP: Cyclic Adenosine Monophosphate

PAP: Positive Airway Pressure

DLPFC: Dorsolateral Prefrontal Cortex

ATP: Adenosine Triphosphate

APOE ε4: Apolipoprotein E ε4 allele

AD: Alzheimer’s Disease

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Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethics Statement

Ethics approval was not required.

Data Availability Statement

No new datasets were generated or analyzed for this study. All information synthesized in this review was obtained from publicly available peer-reviewed articles cited within the manuscript.

AI Use Statement

Some authors used ChatGPT solely for language editing and proofreading. All AI-generated content was reviewed, edited, and verified by the authors, who take full responsibility for the accuracy and integrity of the manuscript.

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