DOI: https://doi.org/10.54393/pjhs.v7i6.3890

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 7, Issue 06 (June 2026)

Systematic Review

OPEN ACCESS

Sleep Deprivation and Its Effects on Cognitive and Neurophysiological Functions: A Systematic Review

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ARTICLE INFO

Keywords:
Sleep Deprivation, Cognition, Electroencephalography, Event-Related Potentials, Functional MRI, Vigilant Attention

How to Cite:
Mehwish, R., Riaz, A., Ajmal, A., Hameed, S., Baig, I. M., & Samad, A. (2026). Sleep Deprivation and Its Effects on Cognitive and Neurophysiological Functions: A Systematic Review: Sleep Deprivation and Its Effects on Cognitive and Neurophysiological Functions. Pakistan Journal of Health Sciences, 7(6), 172-181. https://doi.org/10.54393/pjhs.v7i6.3890

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Received Date: 26th January, 2026
1st Revision Received: 5th March, 2026
Acceptance Date: 16th March, 2026
Published Date: 30th June, 2026

ABSTRACT

Sleep deprivation is common among students, healthcare workers, and shift-based occupations and is associated with impaired cognition and altered brain function. Modern electrophysiological and neuroimaging methods (EEG/ERP and functional neuroimaging) help explain neurophysiological mechanisms underlying sleep-loss-related cognitive impairment. **Objectives:** To systematically evaluate the effects of sleep deprivation on cognitive outcomes and neurophysiological measures in human participants. **Methods:** This systematic review followed PRISMA 2020 guidelines. PubMed, Scopus, and the Cochrane Library were searched for studies published from January 2021 to December 2025. Eligible studies were original quantitative human research reporting at least one cognitive outcome (e.g., attention, working memory, executive function, reaction time) and one neurophysiological measure (EEG, ERP, fMRI, fNIRS, or multimodal) using total or partial sleep deprivation. Risk of bias was assessed using RoB 2 and the NIH Quality Assessment Tool. Due to heterogeneity, findings were synthesized narratively. **Results:** Sixteen studies were included. Sleep deprivation consistently impaired vigilant attention and reaction time (increased PVT lapses and slower responses). Executive control and cognitive flexibility were frequently reduced, whereas working memory effects were task- and load-dependent. Neurophysiological changes included altered EEG activity, modulation of ERP components (P300, N2, CNV), and disrupted connectivity in frontoparietal and default mode networks. **Conclusions:** Sleep deprivation produces reproducible attention and executive deficits, accompanied by parallel neurophysiological alterations, underscoring the critical role of sleep in performance and safety.

INTRODUCTION

Sleep plays a crucial role in optimal cognitive functioning and stable neurophysiological processes. Partial sleep restriction and acute total sleep deprivation are increasingly common in modern educational and professional environments, particularly among students, healthcare workers, and individuals engaged in shift-based occupations [1]. Even short-term sleep deprivation can negatively affect several cognitive domains, including attention, reaction time, working memory, and executive control, which in turn may influence learning ability, clinical decision-making, and safety-sensitive performance [2, 3]. The cognitive effects of sleep deprivation are not uniform across domains. Vigilant attention and sustained performance, often measured using the Psychomotor Vigilance Test (PVT) appear to be particularly vulnerable to sleep loss and are typically characterized by slower reaction times and increased attentional lapses [4]. Executive functions such as inhibitory control, proactive and reactive control, and cognitive flexibility may also deteriorate, especially during prolonged sleep deprivation

DOI: https://doi.org/10.54393/pjhs.v7i6.3890

or in situations requiring strong top-down cognitive regulation [5]. In contrast, some studies have reported relative preservation of certain working-memory outcomes under specific sleep restriction paradigms, suggesting that vulnerability to sleep loss may depend on task characteristics and cognitive load rather than representing a uniform impairment across all cognitive domains [6, 7]. A growing body of neurophysiological research has demonstrated that sleep deprivation is associated with measurable alterations in brain activity. Electroencephalography (EEG) studies have identified changes in resting-state aperiodic activity and functional network organization, which may reflect disrupted arousal regulation and reduced cortical efficiency [8]. Event-related potential (ERP) research has also shown modulation of attention- and control-related components, including P300, N2, and contingent negative variation (CNV), indicating that sleep deprivation may impair information-processing capacity and preparatory cognitive control mechanisms [9]. Similarly, functional neuroimaging studies have reported altered stability and connectivity within large-scale brain networks, including reduced engagement of frontoparietal control systems and changes in default mode and thalamic network activity, which are associated with behavioral impairments and sleepiness-related declines in performance [10].

Despite the extensive literature on sleep deprivation, recent years have witnessed rapid advances in multimodal neurophysiological techniques, including EEG/ERP, functional magnetic resonance imaging (fMRI), and functional near-infrared spectroscopy (fNIRS), as well as network-based analytical approaches. These developments have provided new insights into attentional lapses as complex neurovascular and physiological processes. However, considerable variability in sleep deprivation paradigms, cognitive assessment tasks, and outcome reporting continues to pose challenges for synthesizing findings and translating them into clear educational and clinical implications. Therefore, this systematic review was conducted to evaluate contemporary human evidence published between 2021 and 2025 on the effects of sleep deprivation on cognitive outcomes and neurophysiological measures. By integrating findings from behavioral assessments and brain-based modalities, this study aims to identify the cognitive domains most consistently affected by sleep loss, characterize reproducible neurophysiological signatures, and highlight implications for performance, safety, and future research.

METHODS

The systematic review was conducted to assess the impact of sleep deprivation on cognitive and neurophysiological functions in human participants. It was reported and structured in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. A predefined review protocol was followed throughout the review process, including literature searching, study selection, data extraction, and synthesis. An electronic literature search was conducted in three major databases: PubMed, Scopus, and the Cochrane Library. Although the search was conducted in three major biomedical databases (PubMed, Scopus, and Cochrane Library), manual screening of reference lists was also performed to identify additional relevant studies. The search included studies published between January 2021 and December 2025, and the final search was performed in December 2025. The search strategy combined controlled vocabulary terms and free-text keywords related to sleep deprivation, cognitive performance, and neurophysiological outcomes. The following Boolean strategy was applied with database-specific adaptations: ("sleep deprivation" OR "total sleep deprivation" OR "partial sleep restriction" OR "sleep loss") AND ("cognitive function" OR "attention" OR "working memory" OR "executive function" OR "reaction time") AND ("EEG" OR "ERP" OR "fMRI" OR "functional connectivity" OR "neurophysiology" OR "brain network"). In addition, reference lists of eligible studies were manually screened to identify any further relevant articles. Inclusion criteria comprised original quantitative human studies published between 2021 and 2025, employing experimental, within-subject, or longitudinal designs that investigated acute total sleep deprivation and/or partial sleep restriction (including time-in-bed restriction). Eligible studies were required to report at least one cognitive outcome (e.g., attention/vigilance, working memory, executive function/cognitive control, processing speed, reaction time, or accuracy/errors) and at least one neurophysiological outcome (EEG, ERP, fMRI, fNIRS, or multimodal measures). Cognitive outcomes were categorized into standardized domains, including vigilant attention, working memory, executive function/cognitive control, processing speed, reaction time, and accuracy, based on established neuropsychological classifications to ensure consistency across studies. Exclusion criteria included review articles, meta-analyses, case reports, conference abstracts, studies focused solely on clinical sleep disorders without an experimental sleep-loss paradigm, and non-human (animal or in vitro) studies. All records retrieved from databases were exported to reference management software, and duplicates were removed before screening. Titles and abstracts were screened to identify potentially eligible studies, followed by full-text assessment using the prespecified inclusion and exclusion criteria. Any disagreements during screening or eligibility assessment were resolved through discussion

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and consensus. The study selection process, including the number of records identified, screened, excluded, and included, is summarized in the PRISMA 2020 flow diagram. The study shows the identification of 473 records from electronic databases, removal of duplicates and ineligible records, screening of titles and abstracts, full-text eligibility assessment, and final inclusion of 16 studies in the qualitative synthesis (Figure 1).

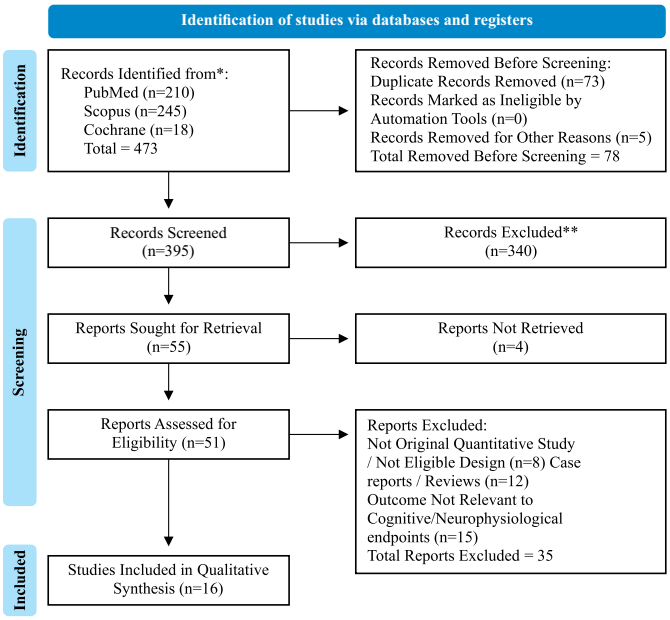

Figure 1: Study Selection Process for the Systematic Review of Sleep Deprivation and Cognitive and Neurophysiological Outcomes

Data were extracted using a standardized form that captured study design, sample characteristics, sleep deprivation protocol, cognitive domains assessed, neurophysiological modality, and key findings. Variability in sleep deprivation paradigms (e.g., total sleep deprivation, partial sleep restriction, and time-in-bed restriction protocols) was considered during narrative synthesis by examining the consistency and direction of reported outcomes across studies. Methodological quality was assessed using the Cochrane Risk of Bias 2 (RoB-2) tool for randomized and within-subject experimental studies and the NIH Quality Assessment Tool for non-randomized/observational designs. Given substantial clinical and methodological heterogeneity across the included studies (including variation in sleep-loss paradigms, cognitive tasks, neurophysiological techniques, and outcome reporting), a meta-analysis was not performed. Instead, results were synthesized using a structured narrative approach, emphasizing consistency in direction and patterns of effects across cognitive domains and neurophysiological measures. Because individual participant data were not available across studies, associations between neurophysiological alterations and behavioral impairments were interpreted descriptively based on study-level findings. A formal sensitivity analysis was not performed because the review employed qualitative narrative synthesis; however, methodological quality assessment using validated risk-of-bias tools was incorporated when interpreting the strength of evidence.

RESULTS

A total of 16 studies published between 2021 and 2025 were included in the final qualitative synthesis. Most studies employed experimental or within-subject designs and were conducted in healthy adult or adolescent populations. Sleep deprivation protocols included acute total sleep deprivation (24–36 hours), partial sleep restriction, or time-in-bed manipulation. Across studies, cognitive domains such as vigilant attention, executive function, working memory, reaction time, and processing speed were evaluated alongside neurophysiological measures including EEG, ERP, fMRI, and fNIRS. The results summarize the characteristics of the included studies examining the cognitive and neurophysiological effects of sleep deprivation. Most studies used experimental or within-subject designs, allowing within-participant comparison of sleep-deprived and rested states. The majority of studies implemented acute total sleep deprivation protocols ranging from overnight deprivation to 36 hours of wakefulness, although some studies also investigated partial sleep restriction or time-in-bed manipulation. Cognitive outcomes frequently assessed included vigilant attention, inhibitory control, working memory, and cognitive flexibility, while neurophysiological measures primarily involved EEG, ERP, resting-state fMRI, or fNIRS-based hemodynamic responses. Across studies, sleep deprivation was consistently associated with impaired attention and executive performance, as well as measurable alterations in neural activity and brain network organization (Table 1).

Table 1: Characteristics of Included Studies (2021–2025) on Sleep Deprivation and Cognitive + Neurophysiological Outcomes

References	Design/Sample	Sleep Deprivation Exposure	Cognitive Outcomes Assessed	Neurophysiological Outcomes	Key Result (Direction)
[11]	Experimental; healthy adults	Total sleep deprivation and partial sleep restriction	Inhibitory control + vigilant attention	(Neurocognitive task-based outcomes)	Vigilant attention impairment partly explained inhibitory-control deficits after sleep loss.
[12]	Experimental; healthy participants	Acute SD	Vigilant attention	Baseline brain activity changes	SD altered baseline brain activity and worsened vigilant-attention performance.

DOI: https://doi.org/10.54393/pjhs.v7i6.3890

[13]	Within-subject; 24 healthy women	24 h SD	Digit symbol, digit span, TMT, complex figure	Resting-state fMRI (functional stability metrics)	SD changed the stability of functional architecture and was related to poorer neurocognitive scores.
[14]	Observational/ experimental; adults	Sleep deprivation (SD) exposure	Vigilance / sleepiness-related performance	Resting-state networks (DMN/FPN/thalamus activity patterns)	SD is associated with ↑ DMN/ thalamus activity and ↓ FPN activity, correlating with sleepiness.
[15]	Experimental; adults	Total SD	Working memory under different cognitive loads	ERP	Cognitive load moderated SD effects on working memory; ERP markers shifted with SD.
[16]	Multisite; healthy young adults	Total SD with recovery	Global cognitive state (brain-age prediction)	Neuroimaging-based brain-age metrics	SD increased "brain age" prediction reversibly after recovery.
[17]	Experimental (fNIRS); adults	Acute SD	Executive/cognitive task performance	fNIRS hemodynamic response in PFC	SD altered prefrontal hemodynamic responses, consistent with reduced executive efficiency.
[18]	Experimental; adults	Sleep deprivation	Situational cognitive impairment	"Brain vital signs" (ERP-based monitoring approach)	Brain vital-sign monitoring detected SD-related cognitive impairment in a situational paradigm.
[19]	Experimental; n=13	Extended wakefulness / SD protocol	Psychomotor Vigilance Test (PVT)	EEG functional networks (graph metrics)	EEG network properties at baseline related to vulnerability/ resilience on PVT under SD.
[20]	Experimental	Total SD	Self-reported vigilance / alertness-linked outcomes	Resting EEG (aperiodic parameters; alpha bandwidth)	SD increased aperiodic offsets (notably occipital) and related to reduced vigilance.
[10]	Dose–response, semi-longitudinal; adolescents/young adults	Time-in-bed restriction (7 vs 8.5 vs 10 h for 4 nights)	Attention control, cognitive flexibility, PVT, working memory	(Behavioral emphasis)	Restriction to 7 h impaired attention control & flexibility; PVT worsened dose-dependently; WM relatively spared.
[21]	Experimental; adolescents/young adults	24 h SD	Cognitive flexibility tasks	Neuroelectrophysiology (ERP/EEG measures)	SD impaired cognitive flexibility with measurable neuroelectrophysiological alterations.
[22]	Experimental: young vs older groups	Partial SD	Emotional/cognitive performance indices	fMRI/network dynamics (energy landscape)	Partial SD altered brain-state switching/dynamics; age-related differences reported.
[23]	Within-subject; 60 healthy males	36 h acute SD	PVT, sleepiness (ESS), mood (POMS)	Resting-state fMRI (visual-network FC)	SD disrupted FC in visual-related circuits; changes correlated with slower PVT and worse sleepiness/ mood.
[24]	Within-subject; n=33	36 h SD	AX-CPT (proactive vs reactive control), RT/errors	ERP (P3, CNV, N2)	SD impaired both proactive/ reactive control; ERP indices showed altered control dynamics under SD.
[25]	Within-subject; n=26	Overnight total SD	Attentional failures during PVT	Fast fMRI–EEG + pupil + CSF flow	Attentional lapses after SD aligned with coupled brain–body dynamics, including neurovascular and CSF-flow pulsations.

The study presents the direction of cognitive outcomes across studies following sleep deprivation. The most consistently reported deficit was impaired vigilant attention, commonly measured using the Psychomotor Vigilance Test (PVT), which demonstrated increased lapses and slower reaction times in multiple studies. Executive function and cognitive control, including inhibitory control and cognitive flexibility, were also frequently affected. In contrast, working memory findings were less consistent, with several studies showing task-dependent or load-dependent impairment while others reported minimal change. Overall, sleep deprivation was associated with slower processing speed, prolonged reaction time, and increased error rates, reflecting reduced cognitive efficiency and performance stability (Table 2).

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Table 2: Cognitive Outcomes Affected by Sleep Deprivation (Study-Wise Direction of Effect)

References	Attention/Vigilance	Working Memory	Executive Function / Cognitive Control	Processing Speed	Reaction Time	Accuracy/Errors
[11]	↓ (PVT)	↓ (Go/ No-Go inhibition)	—	—	↓ (PVT + Go RT slower)	↓ (↑ omission errors Go/No-Go)
[12]	↓ (PVT)	—	—	—	↓ (PVT RT)	↓ (PVT lapses/poorer performance)
[13]	↓	↓	↓	↓	↓	↓
[14]	↓ (sleepiness-linked performance reported)	—	—	—	—	—
[15]	—	↓ (N-back; load-dependent)	—	—	—	↓ (worse WM performance)
[16]	—	—	—	—	—	—
[17]	↓	↓	↓ (executive/ interference resolution)	—	↓	↓
[18]	↓ (attention indexed via P300 "brain vital signs")	—	↓ (processing-related ERP indices)	—	—	—
[19]	↓ (PVT lapses)	—	—	—	↓ (PVT RT)	↓ (PVT lapses/poorer PVT)
[20]	↓ (self-reported vigilance/alertness)	—	—	—	—	—
[21]	—	—	↓ (cognitive flexibility)	—	↓	↓
[22]	—	—	—	—	—	— (emotional performance was highlighted rather than classic domains)
[23]	↓ (PVT + sleepiness)	—	—	—	↓ (PVT)	↓ (PVT lapses/poorer performance)
[24]	—	—	↓ (proactive + reactive control)	—	↓	↓ (↑ errors; slower responses)
[25]	↓ (attentional failures during PVT)	—	—	—	↓	↓

↓ worsened/declined, ↔ no clear change/preserved, — not assessed/not reported. SD = sleep deprivation; PVT = psychomotor vigilance test; RT = reaction time; EF = executive function

The results summarize the neurophysiological alterations observed following sleep deprivation across included studies. EEG-based studies commonly reported changes in aperiodic activity, spectral dynamics, and functional network organization, suggesting disrupted cortical efficiency and arousal regulation. ERP studies demonstrated alterations in attention- and control-related components such as P300, N2, and CNV, indicating slowed stimulus evaluation and reduced cognitive control under sleep-deprived conditions. Functional neuroimaging findings showed altered connectivity and stability within large-scale brain networks, particularly the default mode network (DMN), thalamic circuits, and frontoparietal control networks. Multimodal studies integrating EEG, fMRI, pupil dynamics, and cerebrospinal fluid (CSF) flow further demonstrated that attentional lapses during sleep deprivation were associated with coordinated neurovascular and physiological fluctuations (Table 3).

Table 3: Neurophysiological Outcomes Affected by Sleep Deprivation (Study-Wise Direction of Effect) (2021–2025)

References	EEG Spectral / Aperiodic Activity	ERP (P300 / N2 / CNV / N400)	fMRI Connectivity / Stability	Neurovascular / Pupil / CSF Dynamics	Other Neurophysiology
[11]	—	—	—	—	—
[12]	—	—	—	—	Baseline brain activity altered
[13]	—	—	Altered functional stabilit (shift vs baseline)	—	—
[14]	—	—	↑ DMN / thalamus activity; ↓ FPN activity	—	—
[15]	—	ERP altered with SD (load-dependent change)	—	—	—
[16]	—	—	Neuroimaging "brain-age" ↑ after SD; reversible after recovery	—	—
[17]	—	—	—	—	fNIRS PFC hemodynamic response altered (task-related change)

DOI: https://doi.org/10.54393/pjhs.v7i6.3890

[18]	—	ERP "brain vital signs" altered (P300/N400 indices changed)	—	—	—
[19]	EEG functional networks altered (graph metrics changed)	—	—	—	—
[20]	↑ aperiodic offset; alpha bandwidth altered	—	—	—	—
[10]	—	—	—	—	— (primarily behavioral dose–response)
[21]	EEG/ERP altered (neuroelectro-physiological changes)	ERP altered (reported)	—	—	—
[22]	—	—	Brain-state dynamics altered (energy landscape /switching)	—	—
[23]	—	—	Visual-network FC disrupted (direction: altered connectivity vs baseline)	—	—
[24]	—	P3, N2, CNV altered (control-related ERP changes)	—	—	—
[25]	EEG coupled with lapses (change reported)	—	Fast fMRI dynamics linked to lapses (change reported)	Neurovascular + pupil + CSF-flow dynamics coupled to attentional failures	—

↑ increased, ↓ decreased, ↔ no clear change, — not assessed/not reported. EEG = electroencephalography; ERP = event-related potentials; fMRI = functional MRI; FC = functional connectivity; fNIRS = functional near-infrared spectroscopy; DMN = default mode network; FPN = frontoparietal network; CNV = contingent negative variation; P3/P300 = P300 component

The findings present the risk-of-bias assessment for the included studies. Overall methodological quality was generally high, with most studies rated as having low risk of bias across major domains, including outcome measurement and handling of missing data. Some studies were classified as having "some concerns", primarily due to limitations in participant blinding, small sample sizes, or incomplete control of potential confounding factors. Importantly, no study was classified as having a high overall risk of bias, suggesting a moderate-to-high level of confidence in the reliability of the synthesized evidence. The quality appraisal supports the robustness of the overall findings linking sleep deprivation with measurable cognitive and neurophysiological alterations(Table 40.

Table 4: Risk of Bias Assessment of Included Studies(2021–2025)

References	Study Design	Randomization /Allocation	Blinding of Participants and Assessors	Outcome Measurement	Missing Data /Attrition	Confounding Control	Overall Risk of Bias
[11]	Experimental	Low	Some concerns	Low	Low	Low	Low risk
[12]	Experimental	Low	Some concerns	Low	Low	Low	Low risk
[13]	Within-Subject	Low	Some concerns	Low	Low	Low	Low risk
[14]	Observational/Experimental	Some concerns	High	Low	Low	Some concerns	Some concerns
[15]	Experimental	Low	Some concerns	Low	Low	Low	Low risk
[16]	Multisite Experimental	Low	Some concerns	Low	Low	Low	Low risk
[17]	Experimental (fNIRS)	Low	Some concerns	Low	Low	Low	Low risk
[18]	Experimental	Low	Some concerns	Low	Low	Low	Low risk
[19]	Experimental (Small N)	Low	Some concerns	Low	Some concerns	Low	Some concerns
[20]	Experimental	Low	Some concerns	Low	Low	Low	Low risk
[10]	Semi-Longitudinal	Some concerns	High	Low	Low	Low	Some concerns
[21]	Experimental	Low	Some concerns	Low	Low	Low	Low risk
[22]	Experimental	Low	Some concerns	Low	Low	Some concerns	Some concerns
[23]	Within-Subject	Low	Some concerns	Low	Low	Low	Low risk
[24]	Within-Subject	Low	Some concerns	Low	Low	Low	Low risk
[25]	Multimodal Experimental	Low	Some concerns	Low	Low	Low	Low risk

Risk of bias was assessed using the Cochrane Risk of Bias 2 tool for randomized and within-subject experimental studies and the NIH Quality Assessment Tool for non-randomized and observational designs. Each study was rated as having low risk of bias, some concerns, or high risk of bias based on randomization procedures, blinding, outcome measurement, handling of missing data, and control of confounding

DOI: https://doi.org/10.54393/pjhs.v7i6.3890

DISCUSSION

This systematic review synthesizes recent human evidence published between 2021 and 2025, examining the cognitive and neurophysiological consequences of sleep deprivation. Overall, the findings demonstrate that sleep deprivation produces consistent impairments in several cognitive domains, particularly vigilant attention and reaction time, accompanied by measurable neurophysiological alterations. Across the included studies, deficits in vigilant attention commonly assessed using the Psychomotor Vigilance Test (PVT) were the most consistently reported behavioral outcomes. These impairments typically manifested as increased lapses, slower responses, and reduced performance accuracy during prolonged wakefulness or under cognitively demanding conditions [10, 19]. Similarly, several studies reported worsening of vigilant attention and reaction time, reflected by increased PVT lapses, slower responses, and reduced task accuracy when sleep deprivation was prolonged, or tasks were cognitively demanding [10, 11]. Similarly, several studies reported worsening of vigilant attention and reaction time, reflected by increased PVT lapses, slower responses, and reduced task accuracy when sleep deprivation was prolonged, or tasks were cognitively demanding [15, 18]. Supporting this, a recent ERP and reaction-time study demonstrated a significant delay in P300 latency accompanied by slower reaction times following sleep deprivation, highlighting the sensitivity of P300 dynamics to acute sleep loss and their association with behavioral performance [26]. Similarly, EEG studies have shown that sleep deprivation alters spectral activity and functional network organization, which may reflect decreased cortical efficiency and disruption of arousal regulation [20]. These electrophysiological findings provide plausible mechanistic explanations linking sleep loss with deficits in vigilance, cognitive flexibility, and executive control. A network-level explanation for impaired cognition following sleep deprivation is further supported by neuroimaging evidence. Several studies included in this review reported alterations in resting-state functional stability and connectivity, particularly involving decreased activity or connectivity in cognitive control systems such as the frontoparietal network and increased activity within internally directed networks such as the default mode network (DMN) and thalamic circuits [13, 14]. This pattern of reorganization is consistent with reduced top-down cognitive regulation and increased intrusion of internally directed processing during prolonged wakefulness. Such changes may contribute to attentional lapses and impaired response control. Additionally, more region-specific studies have demonstrated that acute sleep deprivation disrupts thalamocortical connectivity, which is strongly associated with declines in attentional performance [23, 27]. Dynamic connectivity analyses further suggest that cognitive impairment following sleep loss is not static but fluctuates over time, with increased transitions between unstable brain states that correspond with variations in PVT performance during extended wakefulness [28]. These findings suggest that sleep deprivation progressively destabilizes neural systems responsible for maintaining alertness and cognitive performance. The vulnerability to sleep deprivation may also vary according to age and exposure patterns. Evidence from adolescent and young adult populations suggests that sleep restriction produces dose-dependent impairments in attention control and cognitive flexibility while sometimes sparing certain working-memory tasks under low cognitive load. However, more prolonged deprivation, such as 24-hour sleep loss in adolescents, has been associated with neuroelectro-physiological alterations accompanied by reduced cognitive flexibility [21]. Comparisons between younger and older adults also indicate that partial sleep deprivation may influence brain-state dynamics differently across age groups, suggesting possible age-related differences in neural compensation or vulnerability to sleep loss [29, 30]. Together, these findings indicate that developmental stage and the duration or type of sleep deprivation (acute total deprivation versus repeated restriction) influence the magnitude and profile of cognitive impairment. Beyond cognitive performance, emerging evidence suggests that sleep deprivation may also affect emotional processing and decision-making. Experimental research has demonstrated that sleep deprivation reduces the ability to engage in cognitive reappraisal, a key strategy involved in emotional regulation, which may negatively influence real-life decision-making under conditions of sleep debt [31]. Other studies have shown that sleep deprivation can increase reward sensitivity during sequential decision-making tasks, potentially altering decision strategies even when overall accuracy remains relatively preserved [32, 33]. These findings extend the impact of sleep deprivation beyond traditional cognitive domains and highlight its potential implications for decision-making in professional environments such as healthcare, education, and other safety-sensitive occupations [34, 35]. The certainty of the evidence synthesized in this review is generally favorable, as most studies were rated as having a low risk of bias.

Nevertheless, several limitations should be acknowledged. First, there was considerable heterogeneity in sleep deprivation paradigms, cognitive testing protocols, and neurophysiological methodologies, which limited the feasibility of conducting a quantitative meta-analysis and comparing effect sizes across studies. Second, many studies involved relatively small sample sizes, particularly in EEG-based network analyses, which may increase

DOI: https://doi.org/10.54393/pjhs.v7i6.3890

variability and limit generalizability. Third, sleep deprivation studies often face methodological challenges related to participant blinding, and some designs cannot fully exclude expectancy effects (Table 4). Finally, the predominance of healthy volunteer populations limits the generalizability of findings to clinical populations or individuals exposed to chronic sleep disruption, such as shift workers. Despite these limitations, consistent evidence across behavioral, electrophysiological (EEG/ERP), and neuroimaging (fMRI/fNIRS) modalities supports the conclusion that sleep deprivation disrupts neural systems responsible for maintaining cognitive stability and attentional control. The convergence of findings across multiple methodologies highlights sleep deprivation as a measurable neurocognitive stressor with identifiable neural signatures. Future research should focus on standardized cognitive assessment batteries, harmonized reporting of neurophysiological outcomes, multimodal study designs, and ecologically valid settings such as healthcare shift work environments. Longitudinal studies examining recovery sleep will also be important to determine whether sleep deprivation leads to transient or persistent neurocognitive alterations.

CONCLUSIONS

Evidence from 2021–2025 demonstrates that sleep deprivation produces consistent impairments in vigilant attention, reaction time, and executive control, paralleled by reproducible neurophysiological alterations on EEG/ERP and network-level reorganization on functional neuroimaging. These findings support sleep loss as a measurable neurocognitive stressor with identifiable electrophysiological and connectivity signatures, underscoring the importance of sleep-protection strategies in students, shift workers, and safety-critical professions.

Authors' Contribution

Conceptualization: RM
Methodology: RM, AR, AA, SH, IMB, AS
Formal analysis: AR, AA, SH, AS
Writing and Drafting: RM, AR, SH
Review and Editing: RM, AR, AA, SH, IMB, AS

All authors approved the final manuscript and take responsibility for the integrity of the work

Conflicts of Interest

All the authors declare no conflict of interest.

Source of Funding

The authors received no financial support for the research, authorship and/or publication of this article.

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