



Electroencephalographic Markers of Working Memory Alterations Associated with Sleep Deprivation: A Systematic Review

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Sleep deprivation (SD) is a condition in which an individual does not get enough sleep. It can also be used to identify the effects of SD on working memory. Working memory is the brain's ability to temporarily hold and manipulate information for short periods to perform tasks that require problem-solving, attention, focus, and multistep instructions. EEG studies have been conducted to detect abnormalities in sleep patterns following sleep deprivation. Electroencephalography (EEG) is an essential indicator of sleep deprivation. There is a direct link between working-memory performance and sleep quantity and quality, with EEG studies suggesting that SD leads to poor working memory in individuals performing attention and decision-making tasks. In this systematic review, we identified nine studies that examined EEG results from working memory tasks. In these studies, SD ranged from 24 to 40 hours and collectively investigated the influence of sleep loss on working memory. We found that Event-Related Potential (ERP), a time-locked waveform measured in response to stimuli, is influenced by SD in both the P3 and N2 components. A decrease in amplitude was observed in the P3 wave, and an increase in latency was observed when the N2 sleep stage was measured after SD. These results suggest that SD negatively affects working memory, leading to prolonged reaction times and impairments

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INTRODUCTION

What is Electroencephalography (EEG)?

Electroencephalography (EEG) is a non-invasive medical test that measures electrical brain activity generated by cortical neurons via scalp electrodes, following the International 10-20 system. EEG recordings consist of spontaneous electrical activity, including postsynaptic potentials generated by large pyramidal cells of the cerebral cortex, with rhythmic EEG waves arising primarily from the thalamus [1].

Scalp-recorded signals are attenuated and smeared by layers of tissues found in the scalp, skull, and cerebrospinal fluid. Recordings are made by measuring the voltage difference between two recording locations over time, yielding a sum of inhibitory and excitatory postsynaptic potentials [1].

What is Sleep Deprivation?

Sleep deprivation (SD) occurs when an individual fails to get the proper amount and quality of sleep. A lack of appropriate, healthy sleep can adversely affect alertness, cognitive performance, and overall health, and can be classified as either chronic or acute [2]. SD can also be divided into partial and total SD; partial SD consists of a singular night of inadequate or reduced sleep, whilst total SD refers to no sleep at all for a minimum of one night during a regular sleep/wake cycle [2]. Working longer hours, shift work, spending more time watching television or scrolling through the internet, and stress are the most common drivers behind SD [3]. According to the Centers for Disease Control and Prevention (CDC), more than one in three adults in the United States and nearly eight out of ten teens don't get enough sleep, in addition to a quarter of adults experiencing a chronic sleep disorder such as sleep apnea or insomnia [4].

What is working memory?

Three types of memory are commonly studied: short-term, long-term, and working memory. In this paper, we will be focusing on working memory, also known as short-term memory. To clarify, short-term memory refers to the information that is processed by an individual in a short period of time, usually up to 30 seconds, that is understood as “temporary storage” with limited capacity and is essential for focusing on everyday current activities, such as reading a sentence or following directions to an unfamiliar place [5]. Working memory is the term used to refer to memory as it relates to the execution of planned behavior, such as solving a math problem, following a recipe, or preparing talking points for an argument [6]. An important point to remember is that working memory includes short-term memory and other processing mechanisms that use it, as working memory influences the ability to control attention and information processing [6]. Because short-term and working memory are used interchangeably, we will use working memory when discussing findings that include both.

EEG and sleep

EEG is commonly used to assess sleep patterns in individuals. Through EEG, researchers can observe changes in brain activity, as distinct frequency and wavelength patterns characterize different sleep stages. There are four main EEG waves: alpha, beta, delta, and theta; alpha and beta waves are associated with wakefulness, whilst delta and theta are associated with sleep. Sleep can be divided into non-rapid eye movement (nREM) and rapid eye movement (REM) sleep. With EEG, we can see that as one falls asleep, the

low-voltage, fast EEG pattern of wakefulness gradually gives way to slower frequencies as NREM sleep goes from stage N1 (decrease in alpha) to stage N2 (spindles, K-complexes) to stage N3 (increasing amplitude and regularity of delta rhythm). Stage N3 is referred to as slow-wave sleep (SWS). SWS is interrupted by periods of rapid eye movement (REM, i.e., active or paradoxical) [7]

EEG and memory performance in individuals with SD

Studies have shown that EEG scans that are performed during regular sleep and SD studies showcase a noticeable difference in function in the medial prefrontal cortex (mPFC) for individuals with partial SD; the mPFC is critical for developing judgments, decision-making, emotional responses, and attention [2]. As previously mentioned, SD disrupts memory consolidation, particularly affecting the hippocampus through long-term potentiation (LTP) [2]. The hippocampus and the neocortex are the primary regions in which memory consolidation occurs. When performing cognitive tasks, a key EEG signal is the P3 (P300) wave, an event-related potential (ERP) observed in the parietal area and associated with attention and decision-making [8]. In an EEG, the P3 wave is typically observed within a 250–500 ms time window after stimulus presentation; its maximum positive deflection is at the parietal (P3, Pz, P4) electrodes [8].

In this review paper, we aim to discuss different methods in which working memory is influenced by SD as analyzed through cognitive tasks and EEG studies. The use of EEG in memory and SD studies continues to provide steppingstones in understanding the influence of healthy sleep on cognition and overall health. This paper will provide insight into the methods and findings from various studies that showcase the role of EEG and demonstrate visual and empirical data on the effects of SD on memory performance.

METHODOLOGY

Search Strategy

Searches were conducted using three research platforms: PubMed, Google Scholar, and the University of Texas at Dallas Library, for studies on the use of electroencephalography to assess changes in memory after SD. Keywords for our search included: EEG, electroencephalography, sleep, sleep pattern, SD, long-term memory, short-term memory, working memory, hippocampus, and cognitive performance.

All studies obtained from the three research platforms were filtered using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). A total of 198 studies were received during the literature search. These were then retrieved and uploaded into Rayyan, a review software, for further analysis. Rayyan detected duplicates, and 50 papers were excluded. Included studies had to involve human participants, include details on the original research, and be relevant to working memory, SD, and EEG

applications. Papers that were excluded included any paper that fell under these criteria: non-EEG related, non-memory related, or non-sleep/SD related. An additional screening was conducted to eliminate animal studies, studies with incorrect study designs, non-adult populations, and review articles. Data and findings in this review are taken from the nine remaining papers.

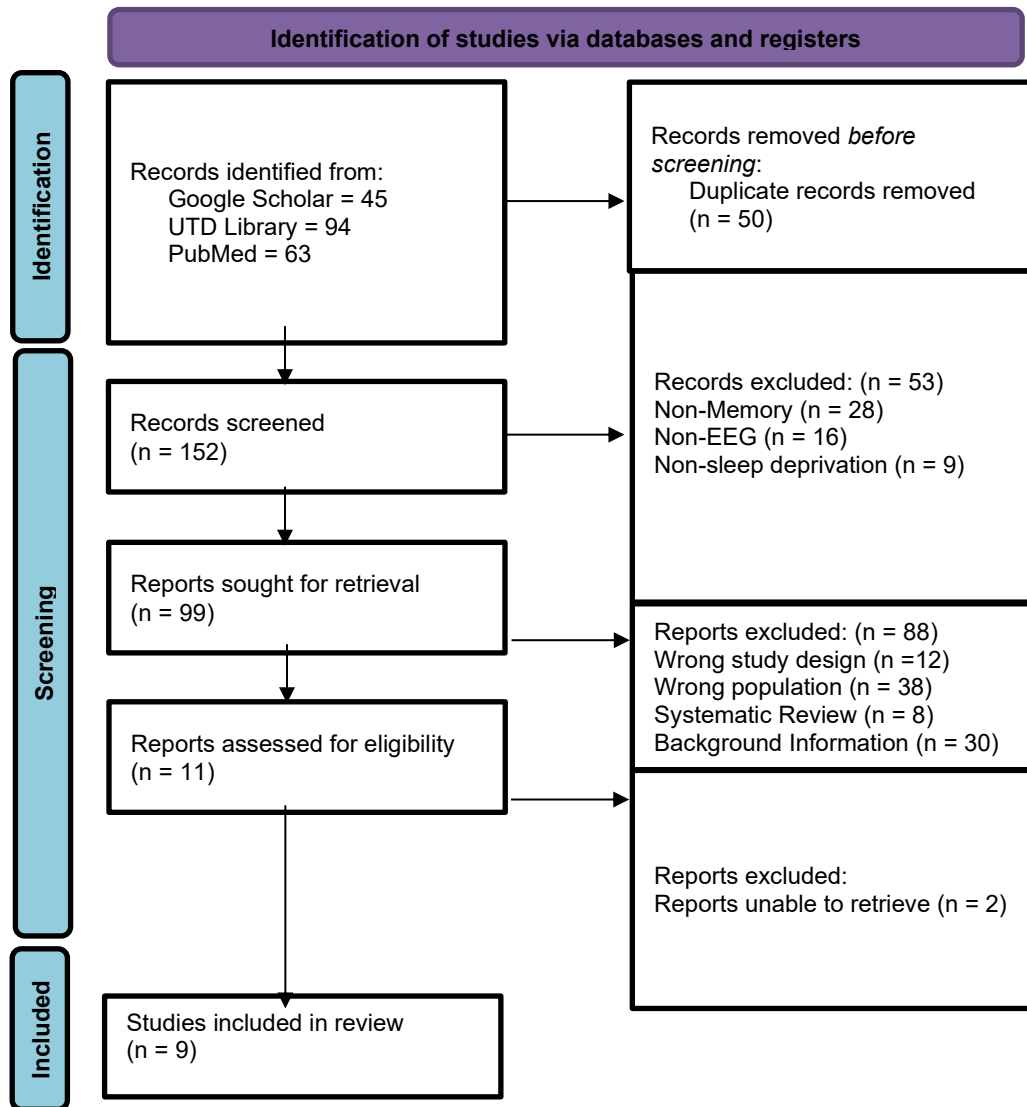


Figure 1: PRISMA Chart.

Study Selection

Electroencephalography (EEG)

Electroencephalography is a noninvasive technique used to record and monitor the brain's bioelectrical activity. Using the International 10-20 system, electrode placement points are measured throughout the

scalp from the bridge of the nose to the back of the head, and from ear to ear. Once points are measured, gold-cup electrodes are placed on the scalp with a conductive gel. A baseline recording is conducted by following standard EEG parameters, including a low-cut filter (1 Hz), high-cut filter (70 Hz), notch filter (60 Hz), and sensitivity (7 μ V). Recordings of postsynaptic potential generated by pyramidal cells and rhythmic cortical activity will arise from the thalamus. This technique has high temporal resolution but is limited to only recording from large synchronous neurons [9]. EEG frequency signals are delta 0–4 Hz, theta 4–8 Hz, alpha 8–12 Hz, or beta 12–30 Hz. Both delta and theta are classified as slow waves. Delta waves are seen during deep sleep, whereas theta waves are seen during drowsiness. Alpha waves are seen in the occipital lobe when the eyes are closed. Beta waves are seen in the frontal lobe when eyes are open, and the patient is awake and alert.

Event Related Potentials (ERP)

Event-Related Potentials (ERPs) are measured brain responses derived from EEG data. It is essentially a focused section of EEG data that usually starts at the onset of stimuli, particularly ones that differ from the event pattern. To obtain ERPs, the individual is presented with a stimulus, and the EEG records brain activity over a specific time period that begins at the onset of the stimulus. This process is done repeatedly. Once stimulus presentations conclude, the continuous EEG data are divided into small segments for each trial. Unwanted signals are filtered out, and the individual time segments with relevant information are averaged to identify specific waveforms that represent the ERP. Different peaks in ERPs can help identify sensory and cognitive processing.

Memory and N-Back Tasks

While multiple types of memories were assessed using EEG and ERP, working memory was the primary focus in most articles, with some focusing on specific aspects of working memory, such as spatial and pronunciation. Working memory was typically assessed using N-back tasks. An N-back task is a type of task in which an individual must match sequential stimuli shown “N” trials ago, where “N” indicates the number. For example, in a two-back task, an individual would be shown several cards, one at a time. They would have to decide whether the current card they are shown matches the one from the two prior sequences. In this scenario, the “N” is two. N-back tasks are often used as a measurement of cognitive function, as the higher the number, the more cognitive function is required. Most of the studies we reviewed used two-back tasks to assess cognitive function.

Sleep Deprivation

Sleep deprivation is a method to facilitate and obtain a sleep EEG tracing, since it can be challenging to obtain spontaneous sleep with an EEG. It is recommended that 24 hours of SD will get the best efficacy. Many factors may affect EEG tracing done with SD. Among these are the duration of SD, the recording duration, the sleep state, and patient characteristics such as age, sex, brain injury, and other neurological conditions. The state of sleep can affect the observed brain waves, and abnormal brain waves, such as

generalized discharges, are generally increased during rapid eye movement (REM) sleep and are not affected during slow-wave sleep. The sleep-wake stage of the sleep cycle can impact the effects of SD. An EEG recording should be done before, during, and after the patient's sleep. There is no specific outline on SD as an activating method when compared to regular sleep [10].

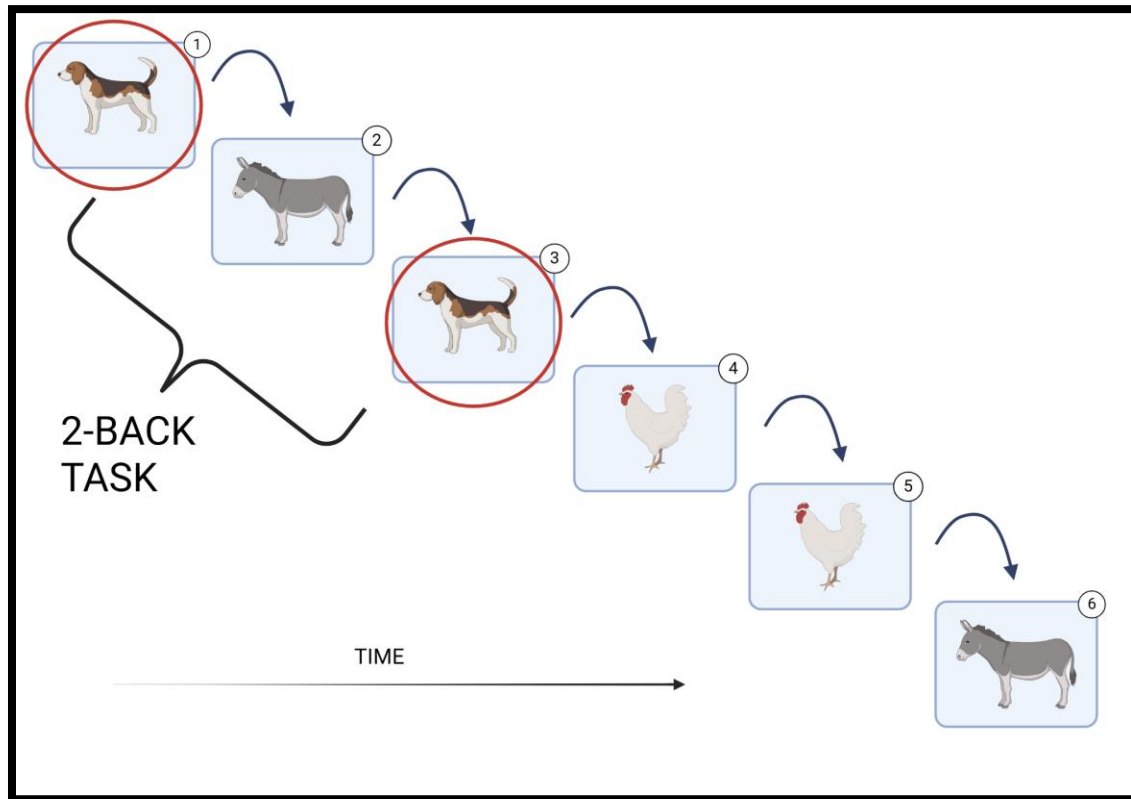


Figure 2: Two-Back Tasks.

RESULTS

Throughout the nine studies, a total of 203 patients participated, with 155 males and 48 females. Except for one study that included adult patients up to age 50, the studies were conducted on young adults, aged 18 to 30. All the studies used EEG to help measure the before-and-after effects of SD. Moreover, eight of the nine studies focused on working memory, and six of those eight used the N-back task to assess it. The remaining study, which did not focus on working memory, examined episodic memory via face-name recognition. All studies tested SD at levels ranging from 24 to 40 hours. Overall, the studies found that SD increased reaction time and decreased the P3 amplitude [11, 12, 13, 14]. Additionally, two studies that focused more on the frequency of EEG waveforms found an increase in theta responses [15, 16]. Key findings from the studies

not only show that SD negatively affects working memory, but also that EEGs can help identify its effects on working memory tasks.

Authors and Year	# of patients	Age	Gender	Amount of SD	Type of memory assessed		Modality/methods used	Key findings
Chai et al. 2020	54	21-50	28 M, 26 F	24 hrs.	Episodic memory		EEG, face-name pairs	The TSD group showed decreased episodic memory performance and impaired hippocampal connectivity, which were restored after 2 nights of recovery sleep.
Chen et al. 2023	22	18-24	M	36 hrs.	Pronunciation memory	working	ERP, 2-back task	After TSD, reaction time decreased, but accuracy was reduced significantly. The N2 amplitude was larger after TSD. A negative correlation was found between the P3 amplitude and reaction time.
Meisel et al. 2017	8	Young adult, mean age: 23	M	40 hrs.	Long-range correlations (decision-making and working memory)	temporal (decision-making and working memory)	EEG	LRTCs decline as SD progresses.
Peng et al. 2019	16	21-28	M	36 hrs.	Spatial working memory		EEG, two-back tasks	P3 amplitude decreased for as long as the prolonged latency of N2 components. After TSD, P3 amp. Decreased more in the right hemisphere than the left, where spatial working memory presides.
Posada-Quintero et al. 2019	10	25-35	7 M, 3 F	24 hrs.	Working memory		EEG, EAT	An increase in alpha and theta waves indicated impairment on memory tasks, and gamma waves were indicative of reactivity. Cognitive impairment was seen after 18 hours of SD.
Vasquez et al. 2023	10	25-35	7 M, 3 F	25 hrs.	Working Memory		EEG, EDA, ECG, N-back, PVT, Error awareness task (EAT), ship search	Increase in the EEG after SD, increase in delta for working memory tasks, and increase in alpha for tasks requiring attention.
Yin et al. 2023	22	18-25	M	36 hrs.	Working memory		EEG, one and two-back tasks	Reduction in amplitude of P3 in 2-back tasks when compared to 1-back. After SD, the correct number per unit decreased.
Yirikogullari et al. 2025	30	18-30	14 M, 16 F	24 hrs.	Working memory		2-back tasks, EEG	TSD group prolonged reaction times and decreased frontocentral ERP delta and theta responses.
Zhang et al. 2019	31	23-27	M	36 hrs.	Working memory		EEG, 2-back task	TSD participants showed impairments in working memory and longer reaction times, as well as decreased P3 and N2 amplitudes. This was partially restored after 8 hours of sleep.

Table 1: All nine studies that matched our search criteria are summarized.

Author and Year	Title
Chai et al. 2020	Two nights of recovery sleep restore hippocampal connectivity but not episodic memory after total SD
Chen et al. 2023	Total SD triggers a compensatory Mechanism During the Conflict Monitoring Process: Evidence from Event-Related Potentials.
Meisel et al. 2017	Decline of long-range temporal correlations in the human brain during sustained wakefulness
Peng et al. 2019	Total SD impairs the lateralization of spatial working memory in young men.
Posada- Quintero et al. 2019	Brain Activity Correlates with Cognitive Performance Deterioration During SD
Vasquez et al. 2023	Mutual Information between EDA and EEG in Multiple Cognitive Tasks and SD Conditions
Yin et al. 2023	Cognitive Load Moderates the effects of total SD.
Yirikogullari et al. 2025	Frontocentral delta and theta oscillatory responses are sensitive to SD during a working memory task.
Zhang et al. 2019	Decreased Information Replacement of Working Memory After SD: Evidence from an Event-Related Potential Study

Table 2: All nine studies listed with their respective article titles.

DISCUSSION

Based on the findings of the various research studies that were discussed in this paper, we can interpret that SD influences overall memory performance as seen through EEG studies. When combining SD and activation of short-term or working-memory, subjects were seen to have decreased performance when compared to those who did not undergo SD. Our bodies follow an “internal clock”, which creates the circadian rhythm that controls the body’s natural 24-hour cycle of physical, mental, and behavioral changes. This helps us determine when our body needs sleep and when it’s ready to maintain wakefulness. Circadian cycles can be synchronized to external time signals, such as lack of sunlight [17], but can still persist in the absence of such signals, such as interference of melatonin production caused by excessive screen use, which

research has shown can be a contributor to SD [18]. Various side effects have been studied, notably a decline in performance in learning and memory. A study found that sleep restriction impairs both memory encoding and memory consolidation, which are crucial steps that occur during sleep, beginning during slow-wave sleep (SWS) and strengthening during rapid-eye-movement (REM) sleep [19]. Apart from memory formation and consolidation, sleep influences the quality of memories by detecting statistical regularities, integrating into existing working networks, and generalizing new information with existing knowledge [19]. Lack of sleep or interrupted sleep disrupts memory encoding and consolidation processes, regardless of whether an individual is facing partial or total SD [20, 21]. Without proper memory consolidation, the process of turning short-term memories into long-term memories can be negatively altered. Throughout the night, we begin sleep by entering N1 sleep, which is characterized by theta waves; this would be considered the lightest sleep stage. N2 sleep consists of sleep spindles and k-complexes. Sleep spindles are brief, powerful bursts of neuronal firing, whilst k-complexes are long delta waves. We spend most of our sleep in N2, a sleep stage essential for sleep consolidation and maintenance [22]. Based on the findings of this review, SD increases N2 latency, meaning that an individual takes longer to reach this stage of sleep. Because of increased latency, a link between decreased memory consolidation and poor cognitive performance, as reflected in increased reaction time and reduced accuracy, has been reported [11, 14].

There are various ways to assess working memory. To test working memory, researchers can administer an N-back task, in which participants are presented with a continuous stream of stimuli (e.g., letters, pictures) and must indicate when the current stimulus matches the one given at earlier positions [23]. Cognitive tests for memory are often paired with EEG studies, in which researchers can observe changes in electrical and blood-flow activity in regions related to memory formation. Additionally, frequencies and wavelengths differ between tasks and activity levels. Low-frequency oscillatory responses are characterized by delta waves (0 to 4 Hz) and theta waves (4 to 8 Hz). During wakefulness, we can observe beta waves, which have the highest frequency at 13-30 Hz and are present during focused, alert states. Alpha waves, which possess the lowest amplitude, are seen during quiet/relaxed wakefulness [22]. One study found that sleep-deprived individuals exhibited reduced low-frequency activity in frontal and central regions, where alpha and beta waves are commonly observed [13]; these oscillatory findings were associated with poor performance on memory tasks [13]. Another study found an increase in theta on EEG after SD, an increase in delta for working memory tasks, and an increase in alpha for tasks requiring attention [16]

With EEG, clinicians can diagnose epilepsy, observe changes in blood flow, such as ischemia, detect tumors, conduct research studies, and use the test in sleep studies. Sleep studies can aid in detecting abnormal sleep patterns and disorders such as insomnia and night terrors, narcolepsy, obstructive sleep apnea, and epilepsy. Studies on SD and its adverse effects on human health and performance have been conducted using EEG across various research populations. When it comes to memory studies and EEG, mainly when influenced by behavioral and physiological changes caused by SD, there is consistently a change seen in both P3 and N2. P3 (P300) and N2 (N200) have both been linked to cognitive processes and attention. Both come from Event-Related Potential (ERP), which averages EEG signals and records time-locked data [24].

N2 is usually observed following stimuli by 180 to 325 ms and exhibits a negative amplitude wave [24]. It is generally observed in the frontal and central areas during cognitive processing. On the other hand, P3 is seen following stimuli by 300 to 400 ms and has a positive amplitude. P3 likely reflects working memory, recognition, and memory-updating processes [24]. This wave is found mainly in the parietal area during tasks. Both waves show significant and valid changes during memory tasks, with decreases in P3 and N2 most common. After SD, both waves decrease in amplitude, with N2 becoming less negative and latency increasing [14]. This likely reflects a longer reaction time and impairment in working memory [14]. One study found that an SD group showed decreased episodic memory performance and impaired hippocampal connectivity, which were restored after 2 nights of recovery sleep [25]. Sleep deprivation affects the brain's state in both sleep and wakefulness, as observed in a study that found a decline in long-range temporal correlations in the human brain during sustained wakefulness [26]. We can conclude that the effects of TSD impair recognition memory and retrieval processes, as previously observed through fluctuations in ERPs when attention and decision-making are tested in individuals [27, 28]. Using EEG in conjunction with the studies reviewed in this paper, we can conclude that the P2-N3 complex is essential for proper cortical dynamics, memory consolidation, and effective signal communication during cognitive tasks in everyday life. Some limitations identified across the literature include small sample sizes, gender differences, participant-controlled sleep deprivation, the applicability of functions to real-life performance, and limited spatial resolution of EEG [8, 13, 14, 15, 26]. Being able to detect and monitor fluctuations in the N2-P3 complex properly can aid in understanding the neural mechanisms underlying working memory, as studies have observed that the P3 component is linked to the continuous updating of information in working memory. At the same time, the N2 is associated with cognitive control and inhibitory processes. Impairments in the N2-P3 complex caused by SD can alter the efficacy of working memory performance despite restorative sleep following periods of TSD [14].

CONCLUSION

While an EEG has multiple diagnostic uses, it can be an essential factor in determining and measuring the amount of sleep deprivation. The significant effects of SD are evident on the EEG after 24 hours, as evidenced by decreased P3 amplitude and increased N2 latency. These waveforms are both considered hallmarks for assessing cognitive capability via EEG. There is a direct link between the N2-P3 complex, with decreased P3 amplitude and increased N2 latency, and working memory performance. We believe that understanding and identifying potential fluctuation markers in the N2-P3 complex can provide critical information on the negative consequences of SD and cognitive processes. Future research can examine the adverse effects of SD on uncharted cognitive behaviors, brain region-specific abnormalities influenced by SD, and the particular origins of N2 and P3 neural subcomponents and use advanced technology to detect mechanisms underlying impaired health and cognitive processing.

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REFERENCES

1. Kirschstein, T., & Köhling, R. (2009). What is the Source of the EEG? *Clinical EEG and Neuroscience*, 40(3), 146–149. <https://doi.org/10.1177/155005940904000305>
2. Khan, M. A., & Al-Jahdali, H. (2023). The consequences of sleep deprivation on cognitive performance. *Neurosciences*, 28(2), 91–99. <https://doi.org/10.17712/nsj.2023.2.20220108>
3. Hanson, J. A., & Huecker, M. R. (2023, June 12). *Sleep deprivation*. StatPearls - NCBI Bookshelf. <https://www.ncbi.nlm.nih.gov/books/NBK547676/>
4. *FastStats: Sleep in adults*. (2024, May 15). Sleep. <https://www.cdc.gov/sleep/data-research/facts-stats/adults-sleep-facts-and-stats.html>
5. Cascella, M., & Khalili, Y. A. (2024, June 8). *Short-Term memory impairment*. StatPearls - NCBI Bookshelf. <https://www.ncbi.nlm.nih.gov/books/NBK545136/>
6. Cowan, N. (2008). Chapter 20 What are the differences between long-term, short-term, and working memory? *Progress in Brain Research*, 323–338. [https://doi.org/10.1016/S0079-6123\(07\)00020-9](https://doi.org/10.1016/S0079-6123(07)00020-9)
7. Nayak, C. S., & Anilkumar, A. C. (2023, May 23). *EEG Normal Sleep*. StatPearls - NCBI Bookshelf. <https://www.ncbi.nlm.nih.gov/books/NBK537023/>
8. Yin, Y., Chen, S., Song, T., Zhou, Q., & Shao, Y. (2023). Cognitive Load Moderates the Effects of Total Sleep Deprivation on Working Memory: Evidence from Event-Related Potentials. *Brain Sciences*, 13(6), 898. <https://doi.org/10.3390/brainsci13060898>
9. Cohen, M. X. (2017). Where does EEG come from and what does it mean? *Trends in Neurosciences*, 40(4), 208–218. <https://doi.org/10.1016/j.tins.2017.02.004>
10. Marinig, R., Pauletti, G., Dolso, P., Valente, M., & Bergonzi, P. (2000). Sleep and sleep deprivation as EEG activating methods. *Clinical Neurophysiology*, 111, S47–S53. [https://doi.org/10.1016/S1388-2457\(00\)00401-6](https://doi.org/10.1016/S1388-2457(00)00401-6)
11. Chen, S., Song, T., Peng, Z., Xu, L., Lian, J., An, X., & Shao, Y. (2023). Total sleep deprivation triggers a compensatory mechanism during conflict monitoring process: evidence from Event-Related Potentials. *Archives of Clinical Neuropsychology*, 39(3), 367–377. <https://doi.org/10.1093/arclin/acado83>
12. Peng, Z., Dai, C., Ba, Y., Zhang, L., Shao, Y., & Tian, J. (2020). Effect of sleep deprivation on the working Memory-Related N2-P3 components of the Event-Related Potential waveform. *Frontiers in Neuroscience*, 14, 469. <https://doi.org/10.3389/fnins.2020.00469>

13. Yırkoğulları, H., Dalmızrak, E., & Güntekin, B. (2025). Frontocentral Delta and Theta Oscillatory Responses are Sensitive to Sleep Deprivation During a Working Memory Task. *Clinical EEG and Neuroscience*, 56(6), 497–506. <https://doi.org/10.1177/15500594251316914>
14. Zhang, L., Shao, Y., Liu, Z., Li, C., Chen, Y., & Zhou, Q. (2019a). Decreased Information Replacement of working memory after sleep deprivation: Evidence from an Event-Related Potential Study. *Frontiers in Neuroscience*, 13. <https://doi.org/10.3389/fnins.2019.00408>
15. Posada-Quintero, H. F., Reljin, N., Bolkhovsky, J. B., Orjuela-Cañón, A. D., & Chon, K. H. (2019). Brain activity correlates with cognitive performance deterioration during sleep deprivation. *Frontiers in Neuroscience*, 13, 1001. <https://doi.org/10.3389/fnins.2019.01001>
16. Vásquez, D. a. M., Posada-Quintero, H. F., & Pinzón, D. M. R. (2023). Mutual Information between EDA and EEG in Multiple Cognitive Tasks and Sleep Deprivation Conditions. *Behavioral Sciences*, 13(9), 707. <https://doi.org/10.3390/bs13090707>
17. Vitaterna, M. H., Takahashi, J. S., & Turek, F. W. (2001). *Overview of circadian rhythms*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC6707128/>
18. Hatori, M., Gronfier, C., Van Gelder, R. N., Bernstein, P. S., Carreras, J., Panda, S., Marks, F., Sliney, D., Hunt, C. E., Hirota, T., Furukawa, T., & Tsubota, K. (2017). Global rise of potential health hazards caused by blue light-induced circadian disruption in modern aging societies. *Npj Aging and Mechanisms of Disease*, 3(1). <https://doi.org/10.1038/s41514-017-0010-2>
19. Crowley, R., Alderman, E., Javadi, A., & Tamminen, J. (2024). A systematic and meta-analytic review of the impact of sleep restriction on memory formation. *Neuroscience & Biobehavioral Reviews*, 105929. <https://doi.org/10.1016/j.neubiorev.2024.105929>
20. Banks, S., & Dinges, D. F. (2007, August 15). *Behavioral and physiological consequences of sleep restriction*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC1978335/>
21. Pilcher, J. J., & Huffcutt, A. I. (1996). Effects of Sleep Deprivation on Performance: A Meta-Analysis. *SLEEP*, 19(4), 318–326. <https://doi.org/10.1093/sleep/19.4.318>
22. Patel, A. K., Reddy, V., Shumway, K. R., & Araujo, J. F. (2024, January 26). *Physiology, sleep stages*. StatPearls - NCBI Bookshelf. <https://www.ncbi.nlm.nih.gov/books/NBK526132/>
23. Blacker, K. J., Negoita, S., Ewen, J. B., & Courtney, S. M. (2017). N-back versus complex span working memory training. *Journal of Cognitive Enhancement*, 1(4), 434–454. <https://doi.org/10.1007/s41465-017-0044-1>
24. Patel, S. H., & Azzam, P. N. (2005). Characterization of N200 and P300: Selected studies of the Event-Related potential. *International Journal of Medical Sciences*, 2(4), 147–154. <https://doi.org/10.7150/ijms.2.147>
25. Chai, Y., Fang, Z., Yang, F. N., Xu, S., Deng, Y., Raine, A., Wang, J., Yu, M., Basner, M., Goel, N., Kim, J. J., Wolk, D. A., Detre, J. A., Dinges, D. F., & Rao, H. (2020). Two nights of recovery sleep restores hippocampal connectivity but not episodic memory after total sleep deprivation. *Scientific Reports*, 10(1), 8774. <https://doi.org/10.1038/s41598-020-65086-x>
26. Meisel, C., Bailey, K., Achermann, P., & Plenz, D. (2017). Decline of long-range temporal correlations in the human brain during sustained wakefulness. *Scientific reports*, 7(1), 11825. <https://doi.org/10.1038/s41598-017-12140-w>
27. Alberca-Reina, E., Cantero, J., & Atienza, M. (2015). Impact of sleep loss before learning on cortical dynamics during memory retrieval. *NeuroImage*, 123, 51–62. <https://doi.org/10.1016/j.neuroimage.2015.08.033>
28. Mograss, M., Guillem, F., Brazzini-Poisson, V., & Godbout, R. (2009). The effects of total sleep deprivation on recognition memory processes: A study of event-related potential. *Neurobiology of Learning and Memory*, 91(4), 343–352. <https://doi.org/10.1016/j.nlm.2009.01.008>