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Original Research Article

Impact of Sleep Loss on Neurocognitive Function and Reaction Speed: An Experimental Analysis

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Abstract:

Background: Sleep deprivation in young adults and professionals is the matter of concern would decrement the attentiveness, working memory, and psychomotor performance. University students specifically have acute and chronic sleep loss due to lifestyle and occupational factors; hence they are susceptible to declines in neurocognitive performance.

Aim: The aim of the current study was to examine the impact of acute and chronic sleep deprivation on speed of reaction and neurocognitive processing, indexed by both behavioral performance and event-related brain potentials P300.

Method: The current study employed seventy healthy adults (20 – 40 years old) who were randomly assigned to either acute sleep deprivation (n = 35) or chronic sleep deprivation (n = 35). EEG recordings, along with a test of reaction time, were administered to participants before and after sleep deprivation under an auditory Oddball paradigm. Data analysis used paired t-tests and repeated measures ANOVA.

Results: Acute sleep deprivation significantly increased reaction time ($p < 0.001$), and increased P300 latency ($p < 0.001$), along with decreased P300 amplitude ($p < 0.001$) indicative of slower neural and behavioral response. Chronic sleep deprivation resulted in smaller changes, not significant, suggesting some adaptation.

Conclusion: Acute sleep deprivation had a large degradation in attentiveness and cognitive efficiency, and chronic sleep deprivation had moderated effects. Adequate sleep remains a vital component for best neurocognitive performance and rapid performance.

Keywords: Sleep deprivation, reaction time, neurocognitive function, P300, EEG, attention, cognitive performance.

Introduction

Sleep is a core biological function that is important for optimal physical, emotional, and cognitive health. Nevertheless, chronic sleep deprivation has become a growing feature among the general population, particularly in university students, whose academic culture implicitly fosters reduced sleep for the sake of productivity and social activity. University life requires a high level of cognitive and emotional performance from students, and yet most students consistently fail to get adequate rest because of academic pressures, lifestyle choices, and environmental conditions. The lack of sufficient sleep has far-reaching impacts related to fatigue on reaction time, decision-making ability, working memory, and overall neurocognitive efficiency [1].

Sleep hygiene among students is commonly compromised by a combination of late-night technology use, caffeine or stimulant consumption, and intake of alcohol-all of which interfere with attaining adequate sleep duration and quality. The epidemiology of this problem is alarming: one cross-sectional survey estimated that 71% of university students do not attain the recommended eight hours of sleep per night, and approximately 60% can be classified as poor sleepers based on sleep quality measures [2]. In some professions, the prevalence of sleep deprivation is even higher. For instance, architecture students have been found to report an average sleep time of only 5.7 hours, with "all-nighters" occurring about 2.7 times per month, mainly because of academic pressure and deadlines for project submissions [3].

While there is extensive literature on the effects of acute sleep deprivation in general populations, there are relatively few studies devoted to university students, despite their specific susceptibility to poor sleep patterns and resultant implications for performance and health [4, 5],. Much of this literature also tends to focus on aspects of disease states or clinical outcomes, limiting a broader comprehension of how sleep deprivation impacts overall wellbeing-encompassing physical, emotional, and cognitive domains [6]. This level of understanding is important, considering that about 52% of students engage in sports or physical activity at least once per week, where reaction time and executive function play critical roles. Students themselves perceive sleep problems as a major barrier to success, rating sleep disturbances second only to stress in terms of negative impact on academic performance [7].

The effects of sleep deprivation on physical performance have been extensively studied. Evidence suggests that brief, high-intensity exercise performance may not significantly decline following acute sleep deprivation, although endurance-based activities tend to suffer due to reductions in motivation and perceived exertion 8. This distinction highlights the complex interaction between physiological endurance and the cognitive drive to sustain performance under conditions of fatigue. However, the neurocognitive consequences of sleep loss-that is, those which affect attention, reaction speed, and decision-making-remain far more serious concerns for populations engaged in academic and mental tasks rather than purely physical ones.

Cognitive performance is especially susceptible to sleep loss. Previous studies have shown that sleep deprivation is associated with lower academic achievement, including a lower grade point average for freshmen in college [9]. Deprivation of sleep negatively impacts attention, executive control, and working memory. Working memory capacity and filtering efficiency have been found to be reduced in individuals after acute sleep loss, indicating a negative impact of disturbed sleep on the brain's information processing and manipulation capabilities [10]. Results of the Stroop test-a task commonly used for cognitive performance and speed of processing-present impairments after sleep loss. Its deterioration has largely been considered due to the significant slowing down in reaction times rather than impairment in processing accuracy. These findings indicate that higher-order cognitive reasoning may remain intact for extended periods; however, the speed and efficiency with which individuals can answer or react to a given stimulus are drastically affected.

Taken together, these findings indicate that sleep loss impairs a wide range of human functions, including psychomotor speed, sustained attention, emotional regulation, and motivation. Yet how these functional impairments develop in university students, in particular, following a single night of complete sleep deprivation or an "all-nighter," is not well documented. The lifestyle habits and adaptive behaviors of this population could offer differential neurocognitive responses compared to other populations. Besides, considering the rising dependence on fast information processing, multitasking, and prolonged cognitive effort in today's academic environment, understanding the effects of acute sleep deprivation on reaction speed and executive functioning bears both academic and public health importance.

The present study addresses this gap through the experimental investigation of the effects of acute sleep deprivation, namely one night of lost sleep, on neurocognitive functioning and reaction speed among university students. Specifically, the study investigates the impact of an all-nighter on motor response, executive control, working memory, and cardiopulmonary function. In each of these domains, this research provides a thorough assessment of the degree to which sleep deprivation compromises student performance in various physiological and cognitive respects. The expected outcome will add to the body of evidence with regard to sleep and cognition and will emphasize, from a practical perspective, the importance of sufficient sleep for academic success, mental alertness, and general well-being.

Materials and Methods

Study Design: This study used a comparative experimental design to assess the effects of acute and chronic sleep loss on neurocognitive function and reaction speed in healthy adults. Neurocognitive performance and reaction speed were measured using both behavioral and electrophysiological parameters, namely, EEG-based event-related potentials, before and after sleep deprivation.

Study Area: The research was carried out in Department of Physiology, KMC Medical College and Hospital, Maharajganj, Uttar Pradesh, India.

Study Duration: The study was carried out over a period of 12 months. which included participant recruitment, screening, data collection, and analysis.

Sample Population: The study population consisted of healthy adult volunteers aged 20-40 years, recruited from the hospital staff, postgraduate students, and the local community. All participants were screened to ensure the absence of physical, psychiatric, or neurological disorders.

Sample Size

A total of 70 participants were enrolled, divided into two groups:

- **Acute Sleep Deprivation (ASD) Group:** 35 individuals with regular sleep schedules and no history of night shift work.

- **Chronic Sleep Deprivation (CSD) Group:** 35 individuals have engaged in regular night shift work for at least the past 24 months.

The sample size was determined using power analysis (power = 0.8, $\alpha = 0.05$) based on expected medium effect sizes for P300 latency differences from previous studies.

Inclusion Criteria

Participants who met the following criteria were included:

1. Age between 20 and 40 years.
2. Regular sleep schedule (7–8 hours/night) for the ASD group.
3. Documented night shift work ≥ 24 months for the CSD group.
4. No use of medications affecting sleep or cognition.
5. Normal hearing and vision (corrected or uncorrected).
6. Willingness to provide written informed consent.

Exclusion Criteria

Participants were excluded if they had:

1. History of psychiatric, neurological, or sleep disorders.
2. History of substance abuse or alcohol dependence.
3. Current use of psychoactive or stimulant medications (e.g., benzodiazepines, antidepressants, modafinil).
4. History of head injury or epilepsy.
5. Hearing impairment or difficulty distinguishing auditory tones.

Data Collection: Data collection involved both neurophysiological and behavioral assessments. Neurophysiological data were recorded using a 64-channel EEG system placed according to the international 10–20 electrode placement system in a soundproof, electromagnetically shielded room. Participants sat comfortably in front of the computer screen and were exposed to auditory stimuli designed using E-prime 3.0 software, based on the classical auditory Oddball paradigm. The stimuli consisted of a standard tone (1.0 kHz) presented 80% of the time and a target tone (1.5 kHz) presented 20% of the time. Participants were instructed to press a button as quickly as possible after hearing the target tone. Reaction time and accuracy were recorded automatically by the software. Both groups underwent baseline and post-sleep-deprivation EEG and reaction time measurements to assess changes in neurocognitive performance.

Procedure: On the day of testing, participants came to the laboratory between 7:00 and 8:00 a.m. after a full night's sleep, verified by a sleep diary and actigraphy data. Baseline EEG and behavioral data were recorded under well-rested conditions. Participants assigned to the acute sleep deprivation group were instructed to stay awake for a continuous 24-hour period under constant supervision within the laboratory setting. Participants were allowed light activities during this time, such as reading or watching movies, but were restricted from engaging in vigorous exercise, closing their eyes for an extended period, or consuming any caffeine, alcohol, or other stimulating substances. Two researchers took turns to monitor compliance throughout the night. Following the 24-hour deprivation period, a second set of EEG and reaction time data was collected. Participants in the chronic sleep deprivation group underwent identical testing procedures following a usual night shift in order to replicate their habitual pattern of sleep loss.

Data Processing and Analysis: All EEG processing and analysis was performed with MATLAB R2023b using the EEGLAB toolbox (v2021_0). Data were band-pass filtered (0.1-30 Hz) for noise reduction, resampled to 250 Hz, and ICA applied for ocular and muscular artifacts removal. The continuous EEG was segmented into epochs extending from 200 ms before to 800 ms after stimulus onset; baseline correction was applied to the pre-stimulus interval. Trials with more than ±100 μV were rejected as artifacts. The P300 component, reflecting cognitive processing and attention allocation, was identified within a post-stimulus window of 300-600 ms, mainly at the Cz electrode and its closest neighbors. Individual mean amplitudes and latencies were obtained for further analysis. RTs were filtered in order to include only correct responses to target tones. All data were anonymized and stored securely for further statistical processing.

Statistical Analysis: All statistical analyses were performed using SPSS version 25.0 and GraphPad Prism 9.0. Quantitative variables were expressed as mean ± standard deviation (SD). Pre- vs. post-deprivation comparisons within each group were analyzed by paired-samples t-tests. The RM-ANOVA, with sleep condition (normal vs. deprived) and sleep loss type (acute vs. chronic) as within-subject factors, was used to evaluate the effects of sleep condition and sleep loss type on reaction time and P300 measures. Interaction effects were also evaluated. The partial eta squared (ηp^2) was reported as a measure of effect size. Bonferroni corrections were applied for multiple comparisons. The correlation between the latency of P300 and the reaction time was analyzed using Pearson's correlation coefficient. A p-value < 0.05 was considered statistically significant for all analyses."

Result

The demographic and night shift characteristics of the 70 participants in the acute and chronic sleep

deprivation groups (n = 35 for each) are shown in Table 1 below. The mean age was comparable between groups, at 28.97 ± 4.21 versus 30.46 ± 4.03 years (p = 0.142). The gender distribution was relatively equal (21 males/14 females in the acute group, and 17 males/18 females in the chronic group). As would be expected, participants in the acute sleep deprivation group had no prior night shift exposure, while participants in the chronic sleep deprivation group had a median night shift duration of 50 months (IQR 32–85) and worked a median of 6-night shifts per month (IQR 5–7).

Table 1: Demographic and night-shift characteristics of study participants (N = 70).

Parameter	Acute Sleep Deprivation Group (n = 35)	Chronic Sleep Deprivation Group (n = 35)	p-value
Age (years, mean ± SD)	28.97 ± 4.21	30.46 ± 4.03	0.142
Gender (Male/Female)	21 / 14	17 / 18	—
Night shift duration (months, Median [IQR])	None	50.0 (32.0–85.0)	—
Number of night shifts per month (Median [IQR])	None	6.0 (5.0–7.0)	—

Table 2 indicates that acute sleep loss produced a significant change in reaction time from 361.95 ± 48.51 ms to 435.03 ± 110.86 ms ($t(34) = 4.50$, $p < 0.001$, $\eta p^2 = 0.110$) which is indicative of slower responses following sleep loss; however, chronic sleep deprivation only resulted in an increase in reaction time that was not significant from 392.16 ± 72.38 ms to 409.40 ± 66.92 ms ($t(34) = 1.48$, $p = 0.148$, $\eta p^2 = 0.028$). The change between the chronic sleep deprivation and acute sleep deprivation groups ($\Delta = 55.84$ ms) was statistically significant ($F(1,68) = 8.43$, $p = 0.005$, $\eta p^2 = 0.110$), showing that acute sleep deprivation produced a greater decline in psychomotor performance compared to chronic sleep deprivation.

Table 2: Comparison of reaction time (ms) before and after sleep deprivation in acute and chronic groups.

Group	Baseline (BS)	Post Sleep Deprivation (SD)	Δ Change (SD − BS)	RM-ANOVA / t-test Results
Acute group (n = 35)	361.95 ± 48.51	435.03 ± 110.86	+73.08 ± 96.09	$t(34) = 4.50$, $p < 0.001$, $\eta p^2 = 0.110$
Chronic group (n = 35)	392.16 ± 72.38	409.40 ± 66.92	+17.24 ± 58.32	$t(34) = 1.48$, $p = 0.148$, $\eta p^2 = 0.028$
Between-group comparison (Δ)	—	—	55.84	$F(1,68) = 8.43$, $p = 0.005$, $\eta p^2 = 0.110$

Note: BS – Baseline; SD – Sleep Deprivation; Δ – Difference between SD and BS. A significant increase in reaction time was observed in the acute group.

Table 3 demonstrates that P300 latency significantly increased following sleep deprivation in both groups, indicating delayed cognitive processing. In the acute sleep deprivation group, latency rose from 356.08 ± 28.01 ms to 389.87 ± 41.69 ms ($t(34) = 6.07$, $p < 0.001$, $\eta p^2 = 0.181$), whereas in the chronic group, it increased from 342.98 ± 36.12 ms to 359.59 ± 25.71 ms ($t(34) = 2.77$, $p = 0.009$, $\eta p^2 = 0.128$). The between-group comparison revealed a significant difference in change ($\Delta = 17.17$ ms; $F(1,68) = 5.64$, $p = 0.020$, $\eta p^2 = 0.077$), suggesting that acute sleep deprivation had a more pronounced effect on cognitive latency compared to chronic deprivation.

Table 3: Comparison of P300 latency (ms) before and after sleep deprivation.

Group	Baseline (BS)	Post Sleep Deprivation (SD)	Δ Change (SD − BS)	RM-ANOVA / t-test Results
Acute group (n = 35)	356.08 ± 28.01	389.87 ± 41.69	+33.79 ± 32.94	$t(34) = 6.07$, $p < 0.001$, $\eta p^2 = 0.181$
Chronic group (n = 35)	342.98 ± 36.12	359.59 ± 25.71	+16.62 ± 28.40	$t(34) = 2.77$, $p = 0.009$, $\eta p^2 = 0.128$
Between-group comparison (Δ)	—	—	17.17	$F(1,68) = 5.64$, $p = 0.020$, $\eta p^2 = 0.077$

Note: Significant prolongation of P300 latency was observed after sleep deprivation in both groups, with a greater effect in the acute group.

Table 4 shows that P300 amplitude significantly decreased after acute sleep deprivation, indicating reduced cognitive efficiency. In the acute group,

amplitude dropped from 4.76 ± 0.91 μV to 3.84 ± 1.02 μV ($t(34) = -5.78$, $p < 0.001$, $\eta p^2 = 0.061$). In contrast, the chronic group showed a slight, non-significant increase from 3.40 ± 1.07 μV to 3.58 ± 0.94 μV ($t(34) = 1.35$, $p = 0.186$, $\eta p^2 = 0.045$). The between-group comparison of changes (Δ = 1.11 μV; $F(1,68) = 4.08$, $p = 0.048$, $\eta p^2 = 0.061$) revealed a significant difference, suggesting that acute sleep deprivation caused a more marked reduction in P300 amplitude than chronic deprivation.

Table 4: Comparison of P300 amplitude (μV) before and after sleep deprivation.

Group	Baseline (BS)	Post Sleep Deprivation (SD)	Δ Change (SD − BS)	RM-ANOVA / t-test Results
Acute group (n = 35)	4.76 ± 0.91	3.84 ± 1.02	−0.93 ± 0.86	$t(34) = -5.78$, $p < 0.001$, $\eta p^2 = 0.061$
Chronic group (n = 35)	3.40 ± 1.07	3.58 ± 0.94	+0.18 ± 0.92	$t(34) = 1.35$, $p = 0.186$, $\eta p^2 = 0.045$
Between-group comparison (Δ)	—	—	1.11	$F(1,68) = 4.08$, $p = 0.048$, $\eta p^2 = 0.061$

Note: P300 amplitude decreased significantly in the acute group but showed a mild nonsignificant increase in the chronic group.

Table 5 demonstrates that changes in electrophysiological parameters showed weak, non-significant correlations with changes in reaction time. The correlation between Δ P300 latency and Δ reaction time was positive but not significant ($r = 0.247$, $p = 0.058$), indicating a trend where increased latency was modestly associated with slower reaction time. Conversely, Δ P300 amplitude showed a weak negative, non-significant correlation with Δ reaction time ($r = -0.221$, $p = 0.081$), suggesting that a reduction in amplitude was slightly related to prolonged reaction time, though not statistically meaningful.

Table 5: Correlation between changes (Δ) in electrophysiological parameters and reaction time (N = 70).

Variable Pair	Pearson's r	p-value	Interpretation
Δ P300 latency vs Δ Reaction time	0.247	0.058	Weak positive trend (not significant)
Δ P300 amplitude vs Δ Reaction time	−0.221	0.081	Weak negative trend (not significant)

Discussion

The present study demonstrated that acute sleep deprivation produced significant impairments in both behavioral and electrophysiological measures of neurocognitive performance, whereas chronic sleep deprivation due to long-term night-shift exposure led to milder, nonsignificant changes. The mean reaction time in the acute sleep deprivation group increased significantly from 361.95 ± 48.51 ms to 435.03 ± 110.86 ms, indicating pronounced psychomotor slowing. In contrast, the chronic group exhibited only a modest increase from 392.16 ± 72.38 ms to 409.40 ± 66.92 ms, suggesting partial adaptation to sleep restriction. These findings are consistent with previous research demonstrating that acute total sleep deprivation (TSD) severely impairs vigilance, attention, and reaction performance (Dawson & Reid, 1997; Aidman et al., 2018). Dawson and Reid (1997) [11] showed that 24 h of sleep loss produced cognitive deficits equivalent to a blood alcohol concentration of 0.10%, aligning with the notable decline in reaction speed observed in the current study. Similarly, Aidman et al. (2018) [12] reported significant decrements in executive functioning and decision-making following acute TSD, reinforcing that short-term sleep loss has immediate and measurable cognitive consequences."

The significant increase in P300 latency following acute sleep deprivation—from 356.08 ± 28.01 ms to 389.87 ± 41.69 ms—further supports the notion that sleep loss delays cortical information processing. The smaller increase in latency observed in the chronic group (342.98 ± 36.12 ms to 359.59 ± 25.71 ms) suggests neural adaptation over prolonged exposure. Comparable findings were reported by Lee et al. (2003) [13], who observed prolonged P300 latencies and reduced amplitudes after 24 h of total sleep deprivation, indicative of impaired cognitive processing speed and attention allocation. Likewise, Choshen et al. (2021) [14] found compensatory activation during conflict monitoring after TSD, suggesting that the brain may temporarily recruit additional resources to maintain performance despite fatigue. These results mirror the trend seen in the chronic group, where the absence of a significant amplitude decrease might reflect compensatory neural adjustments.

The decline in P300 amplitude in the acute sleep deprivation group (from 4.76 ± 0.91 μV to 3.84 ± 1.02 μV) underscores the reduction in cortical responsiveness and attentional resource allocation. Similar reductions have been reported by Ray et al. (2012) and Zhang et al. (2019) [15,16], , who found that acute sleep deprivation decreased P300 amplitude during working memory and attention tasks.

Ray et al. (2012) [15] suggested that modafinil administration could restore P300 amplitude, emphasizing its sensitivity as a neural marker of alertness. In contrast, the chronic group in the present study exhibited a slight, nonsignificant increase in amplitude, which may indicate long-term neural adaptation or a shift in attentional strategy. Kusztor et al. (2019) [17] reported that sleep deprivation differentially affects cognitive control subcomponents, with prolonged exposure leading to selective resistance in certain executive functions—possibly explaining the chronic group's relative stability in amplitude.

The between-group difference in the magnitude of P300 changes aligns with earlier observations that repeated night-shift exposure leads to partial tolerance. Choshen et al. (2021) [14] demonstrated that physicians exposed to long-term night shifts showed blunted cortisol responses and elevated inflammatory markers, reflecting chronic physiological strain but reduced acute vulnerability to sleep loss. Similarly, Hayashiet al. (2005) [18] found that chronic sleep deprivation disrupts hippocampal reactivation essential for memory consolidation, suggesting enduring but more stable neurocognitive impairment compared to the acute, reversible deficits seen after single-night deprivation. Together, these findings suggest that while acute deprivation exerts an immediate and severe cognitive cost, chronic exposure may foster adaptive mechanisms that reduce the perceptual impact of fatigue at the expense of long-term neural efficiency.

The weak correlation in the present study between the changes in reaction time and P300 parameters ($r = 0.247$ for latency and $r = -0.221$ for amplitude) further supported this hypothesis, in suggesting that behavioral and neural indices may reflect different aspects of cognitive deterioration. Indeed, Delorme et al. (2004) [19] also found that with greater cognitive load, a longer P300 latency was associated with mild cognitive impairment and interpreted this as evidence that the P300 is a more sensitive electrophysiological index of subtle neural inefficiency. Behavioral and electrophysiological findings diverged possibly as a result of compensatory cortical activation, as Franken et al. (2009) [20] found impairments only in the alerting and executive control networks following TSD.

Furthermore, the chronic group's apparent tolerance to acute sleep deprivation may reflect adaptive alterations in circadian regulation and neuroinflammatory pathways. Niu et al. (2022) [21] demonstrated that chronic sleep deprivation modifies circadian clock gene expression, leading to stable but maladaptive neurobiological rhythms. Similarly, Barger et al. (2005) [22] identified IL-12 as a potential neuroprotective factor mitigating neuroinflammation, suggesting that long-term exposure to sleep restriction might trigger compensatory cytokine-mediated neuroadaptation. These findings parallel the current observation that chronic sleep-deprived individuals exhibit blunted physiological responses to further sleep loss, albeit with evidence of baseline cognitive impairment.

Overall, the findings of the current study confirm that acute sleep deprivation results in immediate measurable declines in reaction speed and cognitive processing efficiency, as evidenced by significant increases in reaction time and P300 latency and a decrease in amplitude. Chronic sleep deprivation, while associated with long-term neural strain, results in less pronounced short-term impairments, likely due to partial physiological adaptation and circadian adjustment. This contrast agrees with previous evidence showing that while the human brain can develop some kind of tolerance against repeated sleep restriction, this adaptation comes at the cost of sustained neurocognitive degradation and altered neural signaling (Kusztor et al., 2019; Choshen-Hillel et al., 2021). Collectively, these findings emphasize that acute and chronic sleep loss are separate yet interconnected phenomena-one characterized by immediate cognitive slowing, the other promoting long-term neural compensation that masks but does not eliminate the underlying impairment.

Conclusion

This research shows that loss of sleep has an identifiable and negative impact on certain aspects of neurocognitive performance, primarily speed of reaction (or reaction time) and the electrophysiological markers of cognitive information processing. Acute sleep deprivation increased reaction time, and this prolongation of the reaction time was related to a significant prolongation of the P300 latency, and a significant reduction in P300 amplitude, both scores which reflect impaired processing of attention, and a decreased neural efficiency. In contrast, sleep deprived participants showing chronic sleep deprivation globally had differences that were less pronounced (when compared to acute sleep deprivation), suggesting some level of physiological adaptation or compensatory mechanisms could be having some effect in ameliorating the effects of acute cognitive sleep deprivation. Even though chronic sleep deprivation had lesser pronounced effects on performance speed and amplitude, both participant groups had evidence of neurocognitive strain, with the cognitive event-related potential delayed and thus the delay implied that sustained or recurring sleep restriction is still taxing cortical speed and efficiency. The weak and nonsignificant correlation in the delay of cognitive event-related potentials on reaction speed suggested behavioral and neural response to sleep deprivation may be partly dissociated, and further the variance in performance may be a product of several interacting and/or combined mechanisms. Collectively, it lends further support to the conclusion that acute sleep loss is more immediate and significantly cognitively, and neurologically adversely

affects cognitive alertness and neural efficiencies as compared to chronic sleep loss, and having adequate sleep is important to have optimal neurocognitive performance; especially in cognitive tasks, requiring high speed and highly accurate responses.

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