

# EFFECT OF SLEEP DEPRIVATION ON THE PSYCHOLOGY OF BEHAVIOR, BRAIN ACTIVITY, AND COGNITION

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## ABSTRACT

Sleep deprivation (SD) is a widely spread disorder and its impact on psychological well-being, brain performance, and cognitive abilities is too significant. This is a review of the existing literature on the behavioral, neurobiological, and molecular impacts of sleep deprivation. SD interferes with mood control, attention and executive function, and prevents memory consolidation. These cognitive deficits are attributed to abnormal activity in major brain areas including prefrontal cortex and hippocampus as seen under neuroimaging. Multiple neuromodulators are increased by SD at the cellular level, including adenosine, and produces oxidative stress, and impairs gene expression associated with immune function and cell repair. Such changes of the molecules are raised to the excessive inflammation and impaired DNA repair, as well as an increased susceptibility to neurodegenerative and psychiatric conditions. The awareness of the multi-level effect of sleep deprivation explains the importance of sleep deprivation as a crucial, social health concern, and the necessity to implement interventions that help to prevent sleep integrity deterioration and sustain cognitive and psychological well-being

**Keywords:** Sleep deprivation, Sleep fragmentation, reduced sleep, Cognitive Impairment, Memory Consolidation, Oxidative Stress, Neuroinflammation, Hippocampus

## INTRODUCTION

Sleep is a complicated physiological condition, which is marked by less awareness, decreased sensory attentiveness, and reduced movement (Baranwal *et al.*, 2023, Coenen, 2024). It is a cyclic alternation between rapid eye movements (REM) and non-REM sleep, both of which have a specific contribution to neural repair and homeostasis (He, 2025, Le Bon, 2021). During sleep, anabolic processes mediate the repair of the immune system, nervous system, skeletal system, and muscular system, as well as

aid in mood control, memory retention, and thinking (Kaczmarek *et al.*, 2025).

Three interactive processes determine the timing and architecture of sleep:

1. The circadian clock (Process C)- an inherent biological clock, that is synchronized mainly by the exposure to light which controls the sleep-wake cycles through the hormones of melatonin and cortisol (Luo *et al.*, 2024).
2. Sleep-wake homeostasis (Process S)- control that adjusts the sleep requirement based on the

prior wakefulness, facilitated by such factors as adenosine buildup and glycogen depletion(Gao *et al.*, 2024).

3. Volitional control, which is a manifestation of behavioral effects and personal sleep patterns. Epidemiological research suggests that the habitual sleep time of six and seven hours a night is linked with lower cardiovascular risk and higher life span(Guarana *et al.*, 2021).

The idea of sleep deprivation (SD) as a decrease in the amount or quality of sleep below the physiological norms can be provided as sleep restriction, sleep fragmentation, or complete sleep loss(Stroemel-Scheder and Lautenbacher, 2021). Sleep restriction is a limitation of sleep duration and sleep fragmentation is periodic waking up, which interferes with the continuity of sleep(Xin *et al.*, 2022). Total sleep deprivation is characterized by prolonged duration of wakingness and lack of sleep.

The effects of SD are multi-scaled in terms of biological aspects: on the macroscopic scale, it changes the brain activity and behavior; on the mesoscopic scale, it influences hormonal reactions and attention; and on the microscopic scales, it affects neuromodulators like adenosine and causes molecular and genetic alterations.

#### **Sleep deprivation affects Behavior, memory, mood, decision-making, and brain activity**

The role of sleep is very important in the functioning of the central nervous system especially in terms of mood, cognition, and neural restoration(Desai *et al.*, 2024). Sleep deprivation (SD) when acute and chronic disrupts these processes, causing major brain structure and functionality impairment(Gilley, 2023).

The part of the brain attacked by SD most is the prefrontal cortex (PFC) which is the area of executive functions, including attention, judgment, and emotional regulation(Friedman and Robbins, 2022). It has been demonstrated by functional imaging studies that the PFC activity has decreased after loss of sleep causing impaired attention, slower response time, and worsened cognitive flexibility(Pesoli *et al.*, 2022). In addition to that, the hippocampus, which is vital

in memory consolidation, is also compromised. Animal experiments support that SD worsens the learning of information that depends on the hippocampus, which is an illustration of the functions that sleep can play in maintaining neural plasticity(Weerasinghe-Mudiyanselage and Moon, 2024).

SD is behaviorally expressed in the form of irritability, emotional instability, and being stress sensitive. In some severe instances, it can result in hallucinations. A study that examined the effect of 32-hour shifts on physicians found significant changes in mood and cognitive performance which highlights the fact that sleep deprivation has serious repercussions in real life(Jaradat *et al.*, 2022). These effects might not necessarily be because of sleep deprivation but rather the physiological stress that is brought about by sleep deprivation. The high cortisol concentrations seen when SD is present indicate that it is a chronic cause of stress thus it is an allostatic load that has a negative effect on the nervous system as well as the immune system(Knezevic *et al.*, 2023, Noushad *et al.*, 2021).

The epidemiological data also makes the situation worse. Although chronic SD is pathological, it has been found that moderate sleep deprivation in habitual long sleepers can be potentially mortality-reducing, perhaps by a similar mechanism as caloric restriction(Grandner and Drummond, 2007). On the other hand, spending too much time in bed can make sleep worse in terms of fragmentation and daytime drowsiness causing a poor sleep and dysfunctional cycle(Aman).

#### **Cellular and Molecular Effects of Sleep Deprivation**

Sleep deprivation (SD) at the cellular level triggers neurochemical and molecular changes which affect the homeostasis of the brain(Krause *et al.*, 2017). Adenosine (AD) is a purinergic messenger involved in neuronal excitability and energy metabolism, which is one of the most important neuromodulators that participate in sleep regulation(Huang *et al.*, 2024). AD concentration in the brain during extended wakefulness and is a homeostatic factor of sleep

facilitating sleep through inhibiting neurotransmitters of arousal like glutamate and acetylcholine(Reichert *et al.*, 2016).

The elevation of extracellular adenosine, especially in basal forebrain have been reported to increase after prolonged waking, and fall in the course of recovery sleep. The role of adenosine in controlling sleep has also been proven by experimental studies which show that adenosine or its agonists induce sleep whereas its antagonists such as caffeine prevent sleep(Reichert *et al.*, 2022).

In addition to neurotransmission, SD has an impact on intracellular signaling, oxidative stress signaling and gene expression. Prolonged wakefulness disturbs mitochondrial activity and increases reactive oxygen species (ROS) and causes neuronal stress and disrupted synaptic activity. These molecular impairments are especially more acute in areas of the brain requiring a large amount of energy like the cortex and hippocampus (the place where learning and behavior occur), which play a role in the cognitive and behavioral impairments experienced during SD(Davinelli *et al.*, 2024).

Altogether, these discoveries contribute to highlighting that sleep is not only a behavioral condition but an essential cellular process, which is needed to restore metabolic balance, control activity of synaptic activities, and maintain the health of neurons.

### **Molecular and Genetic Impact of Sleep Deprivation**

Sleep deprivation (SD) interferes with the major genetic and molecular pathways that play a significant role in cell repair, immunity, and metabolic homeostasis. On the gene expression level, SD changes the transcriptional activity of numerous systems, which leads to a higher susceptibility to diseases(Lyons *et al.*, 2023).

Oxidative stress, loss of antioxidant capacity and damage of DNA repair pathways have been linked to chronic SD. The study by Trivedi *et al.* indicated that the levels of glutathione, ATP, and cysteine were significantly reduced after SD-metabolites with great importance in redox regulation and the DNA methylation

process(Trivedi *et al.*, 2017). This intrusion is also associated with the increase in oxidative DNA damage, as the increase in urinary 8-hydroxydeoxyguanosine among the shift workers. Moreover, genotoxic stress caused by chronic sleep loss was supported by the fact that Cheung *et al.* discovered that the process of repairing DNA was impaired in sleep-deprived people(Cheung *et al.*, 2019).

These findings are backed by animal studies. SD caused the increased levels of reactive oxygen species (ROS), mitochondrial dysfunction, and apoptosis in *Drosophila melanogaster*, accompanied by gene expression changes(Yang *et al.*, 2022). The same case was found in rodents, where neuroinflammation and sustained microglial activation were observed even after recovery sleep, suggesting that effects are long-term genomic.

On the protein level, chronic SD in disorders influences serum biomarkers of inflammation, metabolism, and cardiovascular health. Ma *et al.* found that there were four major proteins that were changed in sleep-deprived individuals, namely, KNG1, PFN1, PKM and CLU(Luo *et al.*, 2020). These proteins are implicated in mechanisms like vascular inflammation, cardiac hypertrophy, metabolic regulation as well as cell stress response mechanism. Their dysregulation evidences the proteomic level of SD systemic implications(Tian *et al.*, 2025).

Additionally, SD is connected with immunogenetic alterations, such as high concentrations of pro-inflammatory cytokines (e.g. IL-6, TNF-alpha), the decrease in toll-like receptor 4 in older adults and the autoimmune responses(Garbarino *et al.*, 2021). This immunogenetic disregulation is a factor that leads to the susceptibility to infections and autoimmune diseases.

### **Sleep Tips for Better Rest**

The quality and duration of sleep is key to sustaining mental acuity, emotional stability, and health. The evidence-based measures that can be used to encourage improved sleep hygiene include the following:

- At least have a regular bedtime and wake up time, (go to sleep and get up at the same hour daily, even on a weekend)(2022).
- Take frequent exercise (but not within three hours of bedtime) so as not to overstimulate(2024b).
- Reduce the use of caffeine, alcohol, and nicotine in the pre-sleep hours, since they may affect sleep architecture(Drake *et al.*, 2013).
- Create a pre-sleep ritual that is soothing like a warm bath, reading a book or drinking caffeine-free herbal tea. Avoid stress or tension causing activities.
- Eat full meals at least two to three hours before going to bed so as to avoid discomfort and disturbance in the gastrointestinal system during sleep(Nagalla).
- Make your sleeping environment as dark, cool and as quiet as possible. One may consider white noise machines to cover disruptive sounds(2024a).
- Wearable biometrics such as Fitbit, Apple Watch, Garmin, or Whoop should be used to track sleeping behavior, heart rate and variability, and adjust these variables to make personalized changes. Emerging technologies include the Apollo wearable that offers subtle light vibrations that can potentially impact positively the nervous system to lower stress levels, increase focus, and improve the quality of sleep(Schvovens *et al.*, 2025).
- Use the bedroom only as a place of sleep and sex; do not look at any electronic devices like TV, computer or smartphone in bed because the stronger you make the connection of your bedroom and a sleep-time(AlShareef, 2022). A healthy style of living, allowing one to have regular good sleep, is not always easy particularly when one is under a lot of stress like work deadlines, exams, etc. Nevertheless, you need to give sleep the first priority- always remember that in the matters of learning and memory it is always good to sleep on it.

### Conclusion

Sleep loss can have far reaching effects on behavior, brain functioning and thought in a

process that is elaborate and interdependent. It interferes with mood stability, increases stress, and worsens the key mental abilities, including attention, memory, and executive control, which is psychologically disastrous. Neurobiologically, sleep deprivation causes an activity in the main parts of the brain, especially the prefrontal cortex, and hippocampus, which are critical in the control of the higher order cognition. Cellular and molecular pathways Sleeplessness causes neuromodulator imbalance, oxidative stress, and impairment of gene expression and DNA repair signaling pathways, both of which facilitate inflammation and neuronal susceptibility. Combined, they not only lead to acute impairment of cognitive and emotional functioning but also exposure to the risks of developing long-term neurodegenerative and immune-related conditions. These studies demonstrate that sleep is extremely important in neurological health and that effective policies of public health and clinical intervention strategies are necessary to ensure sleep integrity to protect cognitive and psychological health.

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