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REVIEW SLEEP AND SLEEP DEPRIVATION

TINJAUAN TIDUR DAN KURANG TIDUR

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ABSTRAK

Tidur adalah kondisi mental dan fisik yang tidak banyak bergerak. Tidur diperlukan untuk kesejahteraan emosional, fisik, dan kognitif dan menyumbang sekitar sepertiga dari kehidupan seseorang. Tidur terjadi dalam siklus di mana tubuh bergantian antara dua mode: tidur REM dan tidur non-REM. Pengamatan perilaku, pemantauan fisiologis, atau campuran keduanya dapat digunakan untuk penilaian tidur dan bangun. Rekomendasi klinis saat ini untuk menilai PSG menggambarkan tiga fase secara progresif. Mayoritas hormon dalam plasma menunjukkan siklus 24 jam yang kuat, menyoroti pentingnya jam sirkadian dan dampak terkait tidur pada pelepasan hormon dan/atau metabolisme. Kurang tidur yang cukup untuk durasi atau kualitas dapat mempengaruhi kemampuan seseorang untuk berfungsi secara normal dan menjaga kesehatan. Orang dewasa yang khas membutuhkan tujuh jam atau lebih tidur setiap malam agar sehat. Review ini akan menginformasikan keadaan ilmu pengetahuan saat ini tentang kurang tidur dan kurang tidur

Kata kunci: Tidur, kurang tidur, REM,

ABSTRACT

Sleep is a sedentary mental and physical condition. Sleep is necessary for emotional, physical, and cognitive well-being and accounts for approximately one-third of a person's life. Sleep happens in cycles in which the body alternates between two modes: REM sleep and non-REM sleep. Behavioural observation, physiological monitoring, or a mix of the two can be used for the assessment of sleep and wake. Current clinical recommendations for scoring PSGs describe three phases of progressively. The majority of hormones in plasma show strong 24-hour cycles, highlighting the significance of the circadian clock and sleep-related impacts on hormone release and/or metabolism. Lack of sufficient sleep for duration or quality might affect one's ability to function normally and maintain good health. The typical adult requires seven or more hours of sleep each night to be healthy. This report will inform the current state of the science of sleep and sleep deprivation.

Keywords: *Sleep, sleep deprivation, REM*

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INTRODUCTION

Sleep is a sedentary mental and physical condition. It is distinguished by altered awareness, significantly suppressed sensory activity, decreased muscular activity, and decreased interactions with the environment. It differs from wakefulness in that it has a reduced ability to respond to stimuli, but it is more reactive than comas or disorders of consciousness, with sleep revealing diverse, active brain patterns (Nguyen *et al.,* 2019).

Sleep is necessary for emotional, physical, and cognitive well-being and accounts for approximately one-third of a person's life (Troynikov *et al.,* 2018). Sleep allows the human body to recoup from activities, enabling optimal following performance. Sleep deprivation are linked to cognitive impairment, emotional disorders, and hormone imbalances. Sleep is an important component of general health and well-being.

Sleep happens in cycles in which the body alternates between two modes: REM sleep and non-REM sleep. Although REM stands for "rapid eye movement," this type of sleep includes numerous additional characteristics, including virtual bodily paralysis. Dreams are a series of pictures, ideas, emotions, and feelings that occur unconsciously in the mind during different phases of sleep. Most of the body's systems are in an anabolic condition during sleep, assisting in the restoration of the immunological, neurological, skeletal, and muscular systems (Krueger *et al.,* 2016).

These are critical processes that sustain mood, memory, and cognitive function, as well as play an important part in the endocrine and immune systems' operation . Every night, the internal circadian clock encourages sleep. Sleep's various functions and processes are the topic of extensive ongoing research. Sleep is a characteristic that has likely been preserved throughout animal evolution for hundreds of millions of years.

Sleep is an important component of general health and well-being. This review will look at the recent research and discoveries on the significance of sleep, sleep deprivation and their impacts. We will also go over the influence of lifestyle and environmental

variables on sleep, as well as the necessity of resolving these concerns in order to encourage healthier sleep. We will also go over research and discoveries on the significance sleep deprivation and their impacts. We will also go over the influence of lifestyle and environmental variables on sleep, as well as the necessity of resolving these concerns in order to encourage healthier sleep.

GENERATION AND MAINTENANCE SLEEP AND WAKE CYCLE

Mechanism that occurred during generation and maintenance sleep and wake cycle is worked by subcortical structure and route network that is extensive (figure 1). Neurochemical that associates with ascending system arousal includes: norepinephrine from Locus Coeruleus (LC), serotonin from midline raphe nuclei, histamine from tuberomammillary nucleus, dopamine from ventral periaqueductal grey matter, acetylcholine from pedunculopontine tegmentum, and the laterodorsal tegmentum of pons and orexin from perifornical area. All of these arousal systems may be required for normal behavioural functioning (Carley and Farabi 2016).

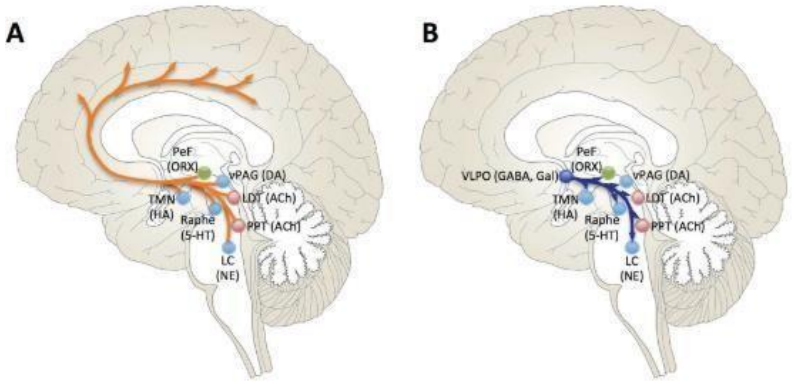

Figure 1. Brain networks regulating sleep and wakefulness. Panel A depicts key elements of the ascending arousal systems, with diffuse excitatory projections to the cortex. Panel B shows pathways arising from the hypothalamus that inacti-vate the ascending arousal system during sleep. ACh, acetylcholine; DA, dopamine; GABA, gamma amino-butyric acid; Gal, galanin; HA, histamine; LDT, laterodor-sal tegmentum; NE, norepinephrine; ORX, orexin; PeF, perifornical region; PPT, pedunculopontine tegmentum; TMN, tuberomammillary nucleus; vPAG, ventral periaqueductal gray matter; 5-HT, 5-hydroxytryptamine (Carley and Farabi 2016)

In order to initiate and maintain sleep, ascending arousal systems must be suppressed, which is performed by inhibitory neurons in the ventrolateral preoptic region (VLPO). The molecular trigger that activates VLPO and initiates sleep onset is not entirely

understood, however the preponderance of data relates to extracellular adenosine build-up. During alertness, adenosine accumulates in the basal forebrain and decreases with continued sleep. VLPO neurons express adenosine receptors, and adenosine stimulates them in vivo (Carley and Farabi 2016)

Other molecules also play crucial signalling roles in the regulation of sleep start and maintenance. The VLPO is projected to by the monoaminergic arousal centres, which may be used to block its activity. Additionally, the suprachiasmatic nucleus provides significant circadian regulation of VLPO (central circadian clock) (Carley and Farabi 2016). Rapid Eye Movement (REM) sleep, which is linked to dreaming, and Non-Rapid Eye Movement (NREM) sleep are the two main categories of sleep. Monoaminergic neuron and a certain subtype of cholinergic neuron in the brainstem appear to be the controlling factors in REM and NREM. Cholinergic neurons in the REM on switch show reciprocal inhibitory connections to serotonergic (raphe) and noradrenergic (LC) neurons. Contrary to adrenergic and serotonergic neurons, REM-on cholinergic neurons have peak activity when REM is induced (Carley and Farabi 2016)

MEASUREMENT AND QUANTIFICATION OF SLEEP AND WAKE STATES

Behavioral observation, physiological monitoring, or a mix of the two can be used for assessment. Adults who are sleeping often exhibit loss of awareness, relative immobility, and a recumbent position with closed eyelids. Reduced tonus of the major skeletal muscles occurs during NREM and proceeds to total or almost complete atonia with the transition to REM. Respiratory pump muscles engage in relative activity sparring as you sleep. All sense responses (visual, olfactory, auditory, somatosensory, and even nociceptive) are diminished but not entirely eliminated during sleep (Carley and Farabi 2016).

Polysomnography is the gold standard method for measuring waking and sleep states in the lab (PSG). Multiple non-invasive sensors are connected to a subject during a PSG. These sensors include skin electrodes that may record the electroencephalogram (EEG), electrocardiogram, eye movement, submental tone, and leg motions. Oral and nasal airflow sensors, thoracic and abdominal strain gauges, and finger probes to detect oxygen saturation can all be used to monitor breathing as you sleep (Carley and Farabi 2016).

Current clinical recommendations for scoring PSGs describe three phases of progressively deepening NREM, stage N1-N3, in addition to waking and REM sleep. Based on distinctive rhythms and events seen in PSG waveforms, these phases are identified and rated (Carley and Farabi 2016).

An EEG signal with low amplitude and mixed frequencies indicates alert alertness. Alpha waves, which are viewed as a rhythm with peaks in the 8 to 13 Hz range, are connected to drowsy wakefulness. Slow rolling eye movements that may last into light slumber are also linked to drowsiness. The lack of alpha rhythm and the existence of theta waves, which have a typical frequency of 4–7 Hz, are characteristics of NREM's lightest stage. Spindles (a burst-like train of waves in the 11 to 16 Hz range with a total duration >= 0,5 seconds) and K complexes (well-defined biphasic waves with a duration >= 0,5 seconds and often peaking over the frontal cortex) are two features that distinguish stage N2 from other stages. Large (>=75 uV), slow (0,5–3 Hz), and N3 waves are connected to deep NREM (Carley and Farabi 2016). Skeletal muscle activity typically displays increasing amplitude reduction with the transition from alertness to N1, N2, and N3 sleep. Lowest skeletal muscle tone and either sharp theta waves (sawtooth waves) or wake-like EEG patterns are linked with REM (Carley and Farabi 2016)

An overnight PSG recording is separated into 30 second epochs for scoring reasons, with a staff score given to each epoch. The temporal organization of the sleep process is shown by graphically representing this series of Sage scores as a hypnogram. In a typical night, the sleep cycle begins with the start of sleep and is followed by a quick drop into deep stage N3 sleep within the first hour. After thereafter, NREM and REM sleep alternated in cycles every 60 to 90 minutes for the remainder of the night. Most N3 sleeps typically take place in the first half of the night, whereas most REM sleep happens in the second (Carley and Farabi 2016).

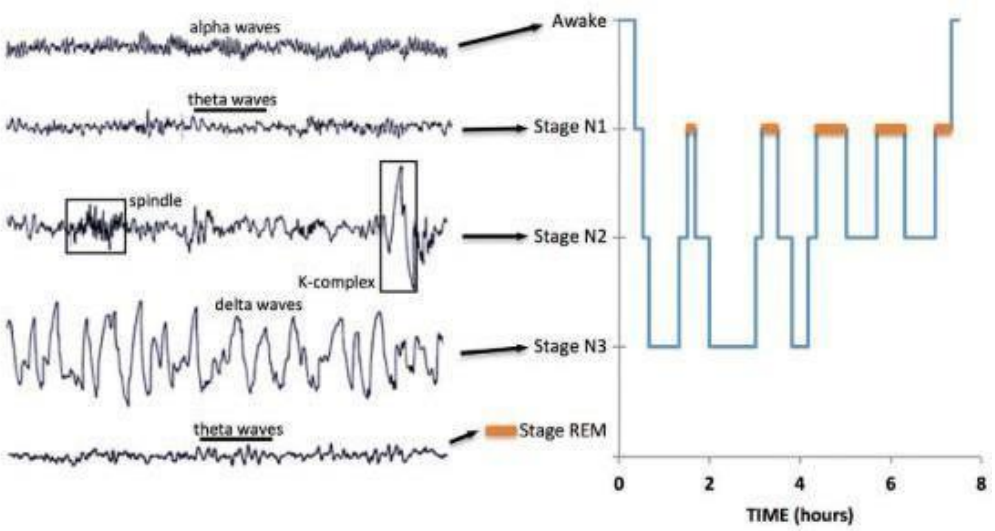

Figure 2. EEG features of sleep/wake stages (left) and typical temporal organization of healthy nocturnal sleep in an adult (right) (Carley and Farabi 2016).

EEG power spectrum analysis has grown to be a very popular sleep research method because it allows researchers to distinguish between EEG rhythms linked to various levels of awareness and NREM sleep based on distinctive frequencies (Carley and Farabi 2016). Self-assessment reports can also be used to subjectively evaluate sleep and sleep quality. There are several different web-based, paper-based, and mobile phone applications that may be used to adapt laboratory or home testing for clinical and research purposes. The two instruments that have been utilized a lot are: Functional Outcomes of Sleep Questionnaire and the Pittsburgh Sleep Quality Index (Carley and Farabi 2016).

The most straightforward of them is actigraphy, in which the person wears a device (usually the size and shape of a wristwatch) on non-dominant wrist. In-home PSG resting has been created that do not contain EEG monitoring. This gadget has numerous accelerometers and can continually record movement for up to several weeks. Then, to distinguish between awake and sleeping phases, these movement profile data were employed (Carley and Farabi 2016).

Level 3 cardiorespiratory monitoring devices based on a variety of technologies are increasingly used for clinical applications. These devices typically track heart rate variability, airflow and effort during breathing, and oxygen saturation. These devices are frequently used for clinically significant sleep apnoea syndrome screening (Carley and Farabi 2016).

ENDOCRINE MANIFESTATION OF SLEEP AND WAKE STATES

The majority of hormones in plasma show strong 24-hour cycles, highlighting the significance of the circadian clock and sleep-related impacts on hormone release and/or metabolism. Adrenocorticotropic hormone, cortisol, and melatonin are a few hormones that are not significantly affected by sleep versus waking (Carley and Farabi 2016).

Thyroid Stimulating Hormone (TSH) and prolactin are two that are substantially regulated by sleep, whereas growth hormone is affected by specific sleep stages (GH). Prolactin levels are typically low during the day and high at night while people are sleeping. A significant dependent rhythm is also seen in GH, with secretion being especially linked to stage N3. Similar to GH, cortisol has a strong 24-hour pattern, however unlike GH, this pattern appears to be predominantly influenced by circadian factors rather than sleep specifically. In contrast to FH, cortisol rises near the conclusion of sleep or in the early morning hours and reaches its lowest point early in a nocturnal sleep phase. TSH readings are low throughout the day, rising during the evening, and reaching their highest just before falling asleep. TSH secretion appears to be inhibited by sleep and the N3 stage (Carley and Farabi 2016).

GLYMPHATIC SYSTEM – NEW APPROACH BRAIN WASTE CIRCULATION

In 2013, the perivascular system, also known as the lymphatic clearance route, was identified as the vertebrate CNS's system for clearing waste. Maiken Nedergaard, a Danish neurologist, gave this structure its name in honour of how dependent it is on glial cells and how similar its functions are to those of the peripheral lymphatic system (Hablitz and Nedergaard 2021). This concept proposes that CSF enters the perivascular region surrounding cerebral arteries, combines with interstitial fluid and parenchymal solutes, and then leaves the region through the venous perivascular regions (Benveniste *et al.*, 2019).

CSF reaches the brain parenchyma by a para-arterial channel, and extracellular solutes and interstitial fluid are cleared from the interstitial compartments of the brain and spinal cord. Vascular pulsation, which is regulated throughout sleep by the expansion and contraction of brain extracellular space, is the main source of the solute exchange between the CSF and ISF. Aquaporin 4 (AQP4) water channels in astrocytes facilitate convective

bulk flow of ISF, removing waste products, extracellular fluid, and soluble proteins (Hablitz and Nedergaard 2021).

CSF enters the brain parenchyma through a para-arterial route after interstitial fluid and extracellular solutes have been removed from the interstitial compartments of the brain and spinal cord by a clearance process. While in sleep, the expansion and contraction of brain extracellular space controls arterial pulsation, which is principally in charge of the solute exchange between the CSF and ISF (Naganawa and Taoka 2022).

Initially, it was thought that the lymphatic system's flow was the only method to fully address the long-standing conundrum of how the CNS's brain tissue could function without a lymphatic drainage channel for extracellular proteins, surplus fluid, and metabolic wastes. But two following papers from the University of Virginia School of Medicine's Louveau *et al.,* and the University of Helsinki's Aspelund *et al.,* reported separately (Aspelund *et al.,* 2015; Louveau *et al.,* 2016).

The meninges, which are layers of tissue that wrap the brain and contain blood arteries and cerebrospinal fluid, were investigated by the researchers. The researchers discovered vessel-like patterns while looking for meninges-related structures (Aspelund *et al.,* 2015). These veins carried lymphatic system markers. They discovered that the capillaries transported fluid and immune cells from the CSF fluid down veins in the sinuses and into close-by deep cervical lymph nodes by injecting dye into anesthetized animals and following its course. The researchers hypothesize that once the cerebrospinal fluid is drained from the brain through the glymphatic system, these veins may act as a second phase in the process (Benveniste *et al.,* 2019).

The identification of an exit route for immune cells from the central nervous system prompts the issue of whether obstruction of this passageway may contribute to neurological conditions including multiple sclerosis, meningitis, and Alzheimer's disease that are linked to immune system malfunction.

SLEEP DEPRIVATION (SD)

Lack of sufficient sleep for duration or quality might affect one's ability to function normally and maintain good health. It may be acute (lacks all sleep for a brief period of time, often lasting 24 to 48 hours) or chronic (individual routinely sleeps less than an optimal amount for ideal functioning). Chronic SD is frequently mistaken for sleeplessness

(Sateia 2014). The distinction between chronic SD and insomnia is in the capacity to fall asleep, even if both conditions share decreased sleep quantity and/or quality as well as function impairment. When given the chance, sleep deprived people can fall asleep quickly (Doghramji 2021).

The typical adult requires seven or more hours of sleep each night to be healthy. Weight gain, high blood pressure, diabetes, heart disease, stroke, and depression have all been connected to SD. High levels of anxiety, impatience, unpredictable behaviour, subpar cognitive functioning, and psychotic episodes are further side effects of SD (Jin *et al.*, 2022).

There are some causes of SD:

1. Insomnia

   The signs of insomnia include: strong mood swings or irritability, excessive daytime drowsiness, annoyance or anxiety about sleep, difficulty paying attention, controlling one's emotions, lack of energy or motivation, poor school or job performance, and tension headaches or stomach-aches.

   Primary insomnia is a form of sleep problem that cannot be attributed to environmental, psychological, or medical factors. There are three basic types of primary insomnia: idiopathic (usually starts in infancy and lasts the remainder of a person's life, suggesting neurochemical abnormality), psychophysiological (anxiety-induced), and sleep state misperception (people inaccurately perceive not get enough sleep). Other medical, neurological, psychological, and psychiatric problems might cause secondary insomnia (Riemann *et al.*, 2020).

2. Sleep apnoea

   Obstructive sleep apnoea is frequently brought on by the upper airway collapsing as you sleep and decreasing airflow to the lungs. Awakening gasping or choking, having trouble falling asleep, waking up with morning headaches, being confused or agitated in the morning, and restlessness are all signs of sleep apnoea. One in ten Americans are affected by this illness. The first line of treatment for sleep apnoea is positive airway pressure therapy utilizing CPAP, APAP, or BPAP (Olaithe *et al.*, 2018).

The inability of the Central Nervous System (CNS) to instruct the body to breathe during sleep is the cause of central sleep apnoea. Some drugs, including opioids, can either exacerbate or induce central sleep apnoea (Olaithe *et al.*, 2018).

3. Mental illness

   In bipolar patients, the onset of mania is frequently preceded by episodes of sleeplessness, and SD has been proven to cause mania in roughly 30% of individuals. Manic individuals typically have a persistently decreased desire for sleep, and SD may constitute a last common route in the origin of mania (Riemann *et al.*, 2020).

   In a normal psychiatric practice, 50–80% of patients suffer from chronic sleep issues. Patients with schizophrenia, bipolar disorder, anxiety, and depression are more likely to experience sleep issues than healthy individuals (Riemann *et al.*, 2020).

4. School

   According large multicentre study by Becker, S. P. *et al.*, in US, college- and university-aged students slept for an average of fewer than 6 hours each night. As they get used to the pressures and social events of college life, SD is typical among first-year college students. The night before a test, students tend to sleep less than normal, and exam performance was positively connected with sleep length, according to Estevan *et al.*, Adolescents' sleep, health, and academic performance have consistently benefited from studies on schools with later start hours (Becker *et al.*, 2018). A 1997 study from the University of Minnesota contrasted students who arrived for class at 7:15 a.m. with those who arrived at 8:40 a.m. They discovered that students who started later had better marks and slept more over the week (Adolescence and Graham 2000).

   It is well known that circadian rhythm, and by extension, sleep habits, alter significantly during the adolescent era. According to EEG research, stage 4 deep sleep is reduced by 50%, and the peak amplitude of delta waves during NREM is reduced by 75% (Tarokh and Carskadon 2009).

5. Caffeine

   Large caffeine intake might have a detrimental impact on one's sleep pattern. Caffeine use might improve short-term function, but excessive usage can aggravate current sleeplessness or cause its symptoms (Reichert *et al.*, 2022).

6. Hospital stays

Compared to sleeping at home, general ward patients in the Netherlands' largest study had shorter total sleep times (83 minutes less), more night-time awakenings, and earlier awakenings. Seventy percent of these patients reported being awakened by outside factors like hospital staff, other patients' noise, medical equipment, pain, and bathroom visits (Wesselius *et al.,* 2018). SD is more severe in ICU patients because there is no naturally occurring night-time peak in melatonin production (Shilo *et al.,* 1999).

7. Internet

Teenage sleep problems and problematic internet use are negatively correlated. It was discovered that it can cause symptoms of insomnia and impact both the quantity and quality of sleep. The exposure to strong light just before bedtime may be a role in sleep disruption. Due to the suppression of melatonin, exposure to the bright lights of electronics might induce a shift to later hours (Kokka *et al.,* 2021). Melatonin is a hormone that synchronizes the sleep-wake cycle and controls circadian rhythm. By doing this, it facilitates the transition to sleep and encourages regular sleep. In addition to problematic users, moderate users were also more likely to exhibit longer sleep latency, which resulted in less sleep, according to a research by Canan *et al.,* (Canan *et al.,* 2013).

EFFECT AND CONSEQUENCES OF SLEEP DEPRIVATION

A. Brain

According to a study using neuroimaging, 35 Total SD in healthy controls has a detrimental effect on the brain's capacity to put an emotional event into correct perspective and to respond to the event in a regulated, appropriate manner. The detrimental effects of SD on cognitive function and attentiveness point to a decline in brain activity. The thalamus and prefrontal cortex are where they mostly occur (Yoo *et al.,* 2007).

Measurements of glucose (absolute regional CMRglu), cognitive function, alertness, mood, and subjective sensations all progressed with SD. NREM is essential for turning off neurotransmitters, enabling their receptors to rest and restore sensitivity, and allowing monoamines (norepinephrine, serotonin, and histamine) to remain active at naturally occurring levels, according to a 2002 University of California animal

research. This enhances emotional management and boosts learning capacity (Thomas *et al.,* 2003).

That study also discovered that REM deprivation resembled selective serotonin reuptake inhibitors, which may help treat clinical depression (SSRISs). This is due to the suppression of the naturally occurring reduction in monoamines during REM, which produces an increase in the concentration of neurotransmitters in the brain that are deficient in people with clinical depression. Enzymes may be able to repair free radical-induced brain cell damage during non-REM sleep. The enzymes themselves are damaged by high metabolic activity when awake, inhibiting effective repair. In this study, the first signs of sleep deprivation-related brain injury in rats were discovered (Siegel 2003).

B. Attention and working memory

The most major effect of SD may be attention and working memory problems, which can result in mistakes like forgetting an ingredient when cooking or a phrase while taking notes. Choice response time tests are one type of tool used to assess working memory. Attentional lapses can also occur in more serious situations when the results could mean the difference between life and death, such as industrial accidents and vehicle accidents caused by distracted driving (SD) (Alhola and Polo-Kantola 2007).

Researchers frequently use the Psychomotor Vigilance Task (PVT), which asks participants to press a button in response to a light at random intervals, to gauge the severity of attention problems. Failure to click the button in response to a stimulus is noted as a mistake, which can be attributed to the microsleeps that result from lack of sleep (Innes *et al.,* 2013).

C. Mood

One may become angry after staying up all night or working an unexpected night shift. One's mood will frequently revert to baseline or normal once they get caught up on their sleep. One study found that after one to two full nights of sleep, participants had greater tiredness, exhaustion, disorientation, stress, and overall mood disruption before returning to baseline (Dinges *et al.,* 1997).

There is a reciprocal association between depression and sleep. Depression can induce insomnia, hypersomnia, or obstructive sleep apnea, and depression can also cause these sleep disorders (Dinges *et al.,* 1997).

D. Driving ability

The effects of SD are noticeable on the road; according to the American Academy of Sleep Medicine (AASM), driver weariness is a factor in one in every five catastrophic motor vehicle injuries, with 80.000 drivers falling asleep at the wheel each day and 250.000 accidents annually. To combat fatigue, the AASM advises stopping off the road and having a 15–20 minute sleep (Brown 2022)

SD may have some of the same dangerous effects as drinking, according to a study from Australia and New Zealand that was published in the British Medical Journal (BMJ) in 2000. Driving after being awake for 17 to 19 hours resulted in lower performance than driving with a blood alcohol level of 0.05%, which is the legal limit for drunk driving in the majority of western European nations (Brown 2022)

E. Sleep transition

The capacity to maintain sleep when already asleep or the willingness to transition from awake to sleep are both examples of sleep propensity (SP). This tendency, which may be detected by polysomnography (PSG) as a decrease in sleep latency, is increased by lack of sleep (the time needed to fall asleep). Shorter transitions from light phases of non-REM sleep to deeper slow-wave oscillations can also be assessed as a sign of a predisposition to sleep, which is indicative of sleepiness (Goel *et al.,* 2009).

After a night without sleep, healthy individuals typically experience a few minutes of latency reduction, as well as a halving of the time between the start of sleep and the initiation of slow-wave sleep. The multiple sleep latency test is typically used to evaluate sleep latency (MSLT). The maintenance of wakefulness test (MWT), in contrast, makes use of sleep latency as a measurement of participants' ability to remain awake (when instructed to do so) as opposed to nodding off (Goel *et al.,* 2009).

F. Sleep wake cycle

Seven to nine hours of sleep every night are necessary for those ages 18 to 64. Due to a disruption of the sleep-wake cycle and an increased tendency to sleep, research on sleep deprivation demonstrates its effects on emotional, cognitive, and motor functions.

The significance of the hypothalamus and several brain systems in regulating circadian rhythms and homeostasis was revealed in a number of research, which helped us better understand sleep deprivation. The two-process model of sleep regulation can be referred to in order to characterize the temporal progression of the sleep-wake cycle (Borbély *et al.*, 2016).

In this model, the timing and amount of sleep are determined by an interaction between a homeostatic process (Process S) and a circadian process (Process C). Process C is the oscillator that controls these levels, whereas Process S symbolizes the need for sleep, which rises during alertness and falls during sleep until a set threshold level. Even at the maximum circadian urge for awake, waking functions will deteriorate when one is sleep deprived due to the build-up of homeostatic pressure (Borbély *et al.*, 2016).

G. Microsleeps

A person with a high level of SD is prone to the phenomena of microsleeps, which are tiny periods of sleep. When someone is striving to stay awake while feeling drowsy, this phenomenon normally lasts for a few seconds. This behaviour may occur when performing boring tasks like driving, reading, or sitting in front of a computer. That individual is unaware of the condition, which is comparable to blackouts (Ayyagari *et al.*, 2021)

In laboratory study setting, rats kept awake for extended periods of time have been shown to experience an even lighter kind of sleep. Specific localized brain areas entered brief (80 ms) but frequent (40/min) NREM-like states, a phenomenon known as local sleep. The rats looked to remain awake during on and off times when neurons switched down, despite their dismal test results (Vyazovskiy *et al.*, 2011).

H. Cardiovascular morbidity

Reduced sleep duration is linked to a variety of harmful cardiovascular effects. Sleep deprivation has been identified by the American Heart Association as a risk factor for negative cardiometabolic profiles and outcomes. Along with other well-known variables including blood pressure, cholesterol, food, glucose, weight, smoking, and physical exercise, the organization suggests good sleep practices for optimal heart health. According to the Centers for Disease Control and Prevention, persons who sleep fewer than 7 hours a day are more prone than those who get an appropriate

amount of sleep to suffer from chronic illnesses such heart attacks, coronary heart disease, and stroke (Jin *et al.,* 2022)

In research that tracked more than 160,000 healthy, non-obese people, those who self-reported having less than 6 hours of sleep each day were at a higher risk of acquiring a number of cardiometabolic risk factors. They had symptoms of metabolic syndrome, including increased central adiposity, higher fasting glucose, hypertension, low high-density lipoprotein, and hypertriglyceridemia. In this study, the effects of sleep duration were not affected by the presence or absence of insomnia symptoms (Deng *et al.,* 2017).

Over a 7-year follow-up period, the United Kingdom Biobank examined over 500,000 persons without cardiovascular illness, finding that those who slept fewer than 6 hours each night had a 20% higher chance of suffering a myocardial infarction (MI). Surprisingly, getting more than 9 hours of sleep each night put them at danger (Daghlas *et al.,* 2019).

I. Immunosuppression

Disruption of the immune system is one of the many negative effects that sleep deprivation may have on one's health. Although it is unclear why, experts think that sleep is crucial for giving the immune system the energy it needs to function and for allowing inflammation to happen when we are sleeping. Additionally, sleep strengthens memories in the brain exactly as it does for the immune system or adaptive immunity (Irwin 2019).

Less than 6 hours of sleep a night increases the risk of infection and increases the likelihood of getting the flu or a cold. Patients in critical care units may take longer to recuperate if they get insufficient sleep (ICU) (Pisani *et al.,* 2015).

J. Weight gain

A number of hormones that are important for weight growth might become unbalanced as a result of sleep deprivation. Lack of sleep causes levels of the hunger hormone ghrelin to rise while leptin levels fall, increasing a person's sense of hunger and appetite for high-calorie foods. Loss of sleep is linked to higher cortisol levels and reduced growth hormone, both of which are linked to obesity. People who don't get enough sleep may also experience daytime drowsiness and exhaustion and engage in

less physical activity. Additionally, obesity might result in poor sleep quality. Obstructive sleep apnoea, gastroesophageal reflux disease (GERD), depression, asthma, and osteoarthritis are among conditions that can make it difficult to get a decent night's rest for those who are overweight or obese (Taheri *et al.*, 2004).

Long-term, full sleep deprivation in rats increased food intake and energy expenditure, which resulted in weight loss and eventual death. In civilizations where high-calorie food is readily available, this study's hypothesis is that mild chronic sleep debt caused by habitual short sleep is linked to increased hunger and energy expenditure, with the equation favouring food intake rather than expenditure (Taheri *et al.*, 2004).

K. Type 2 diabetes

It has been hypothesized that type 2 diabetes is more likely to develop in those who have short-term sleep limitations because they metabolize glucose more slowly than people who get the full 8 hours of sleep. In people with diabetes and prediabetes, poor sleep is associated with high blood sugar levels, although the cause of this association is unclear. Lack of sleep may have an impact on insulin, cortisol, and oxidative stress, which in turn alter blood sugar levels. Lack of sleep can cause ghrelin levels to rise and leptin levels to fall (Pahceco and Singh 2020).

When people don't get enough sleep, they are more prone to seek food to make up for their lack of energy. As a result of this practice, they run the danger of becoming obese and developing diabetes. A study from Gottlieb *et al.*, include more than 1400 individuals found that people who regularly slept for little hours had a higher risk of type 2 diabetes. The direction of cause and effect between insufficient sleep and diabetes is not clear, though, because this study was purely correlational. The authors cite a previous study that shown that experimental rather than regular sleep deprivation led to reduced glucose tolerance (IGT) (Gottlieb *et al.*, 2005).

Assessment

The following are indications and symptoms of sleep deprivation: exhaustion, drowsiness, difficulty thinking clearly, and sleepy driving. In order to assess sleep length and quality as well as the root of sleep deprivation, it is important to ask many questions. When discussing the quality of sleep, it is important to rule out conditions like obstructive

sleep apnoea and restless leg syndrome as well as sleep patterns (average bedtime or rising time on weekdays and weekends), shift work, and frequency of naps (Hanson and Huecker 2022).

Detailed information regarding sleep habits may be found in sleep diaries. They are affordable, widely accessible, and simple to use. The diaries can be as straightforward as a 24-hour log to record when a person goes to bed or they can be elaborate and contain other pertinent information. If there is any doubt about the patient's adherence, sleep surveys like the Sleep Timing Questionnaire (STQ) might be used in place of sleep diaries (Hanson and Huecker 2022).

If there are any doubts about the reliability of self-reported sleep diaries or questionnaires, actigraphy is a helpful, unbiased wrist-worn instrument. In order to determine total sleep time, sleep onset delay, the amount of waking time following the start of sleep, and sleep efficiency, actigraphy records movements and applies computerized algorithms. To measure exposure to light, certain gadgets contain light sensors (Smith *et al.*, 2018).

Management

Organizations including the Centres for Disease Control and Prevention, the National Institutes of Health, the National Institute on Aging, and the American Academy of Family Physicians have recommended several actions to support healthy sleep. Sleep hygiene is the secret to better sleeping patterns. This includes establishing a regular bedtime, keeping a sleep environment that encourages sleep, exercising every day, abstaining from alcohol, caffeine, large meals in the evening, calming down and avoiding physical or electronic activity close to bedtime, and getting out of bed if you can't sleep (Wilkinson 2008; Redinger *et al.*, 2022).

After an excluding physical diagnosis, Cognitive Behavioural Therapy for Insomnia is frequently advised as the first line of treatment for long-term involuntary SD. Cognitive therapy, sensory control, sleep restriction, sleep hygiene, and relaxation are the five components of CBT for insomnia. Together, these elements have demonstrated efficacy in adults, with clinically significant effect sizes. It is frequently chosen over long-term pharmacological treatment (Redinger *et al.*, 2022)

There are several methods to lessen the effects of SD and improve attentiveness. When experiencing acute SD, caffeine is frequently utilized for brief periods of time to promote alertness. The AASM also suggests preventive sleep prior to deprivation, naps, various stimulants, and combinations of these (Wilkinson 2008).

CONCLUSION

Overall health and wellbeing depend on getting enough sleep. It makes up around one-third of a person's life and is essential for their emotional, physical, and cognitive health. Cognitive decline, emotional problems, and hormonal abnormalities are all associated with sleep deprivation. The study also emphasizes how crucial it is to comprehend the factors that contribute to sleep problems, how to treat them, and how well different interventions, may increase the quantity and quality of sleep. The review also included the relationship between environmental and lifestyle variables and sleep, as well as the significance of resolving these problems to encourage better sleep. The review also emphasizes how sleep is a dynamic process that takes place in cycles and has a number of roles and processes that are the subject of continuing research.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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