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Reviewing the effect of Acute Sleep Deprivation on Functional Connectivity of Reward Network and Adverse Emotional Experience

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ABSTRACT

While the precise functions of sleep are not fully understood, it is an important part of daily life and involves a series of events that follow a consistent night-time cycle and enable the human body to function at its best. More than 30% of adults suffer from sleep deprivation (SD). SD can lead to negative effects on cognitive function, including reward and emotional processing. Here, we review the consequences of acute SD on reward network and emotional processing, and the activity and connectivity of different brain regions involved in reward network and emotional processing, with a focus on neuroimaging studies.

Keywords:

sleep deprivation, reward network, emotional processing, brain networks, functional connectivity, neuroimaging.

Introduction

Sleep is a vital physiological process that plays an essential role in maintaining both physical and mental health [1]. It is marked by a significant reduction in consciousness and responsiveness to sensory stimuli, both of which can be rapidly reversible [2]. Sleep affects almost every bodily and mental function and causes intricate alterations in the physiology of numerous systems [3]. The body performs the necessary processes to support brain function and general health while you sleep [3]. At various stages of sleep, important processes like neurogenesis and synaptogenesis take place [4]. Sleep is also essential for removing toxins that build up around brain cells while we are awake [4]. Cerebrospinal fluid production and circulation rise significantly while you sleep, aiding in the removal of toxins like beta-amyloid from brain tissues [4]. Sleep is also essential for preserving mental balance and controlling emotions [4]. Getting enough sleep is essential for all cognitive processes, including learning, memory consolidation, attention, language, reasoning, creativity, and decision-making [4].

Brain activity undergoes significant changes during both non-rapid eye movement and rapid eye movement sleep stages [5]. Sleep is therefore anticipated to be essential for brain function [6]. Older adults usually need 8 to 9 h of sleep per night, while healthy adults need 7 to 9 h [1]. The most prevalent sleep-related complaint, sleep deprivation (SD), can result from losing even one hour of sleep [7]. A reduction in the typical length or quality of sleep is a hallmark of SD [7]. If SD lasts for one or two days, it is classified as acute SD, while prolonged deprivation is referred to as chronic SD [8]. The severity of SD can differ among individuals. Acute SD affects more than 30% of the population, and chronic SD is estimated to affect 6-10% globally [8].

People who suffer from SD may suffer severe consequences [9]. In addition to impairing cognitive brain functions like working memory, attention, and decision-making, SD also causes emotional experience to deteriorate [10]. According to studies, SD makes people feel depressed and makes their nerves more sensitive to unpleasant emotional stimuli [11–13]. For instance, increasing waking hours causes agitation, hostility, and anxiety. Furthermore, less control over one's emotions may result in more destructive behavior [10]. Emotional instability can cause major human failure and impair cognitive function, both of which can lead to workplace accidents. Emotional decision-making can be affected by SD. Since rewards and punishments can modify emotions, the emotional state after SD can affect decisions pertaining to gain and loss. Emotional instability following SD and reward network communication, however, has not received much attention in research.

Additionally, the reward network's alterations and the connection between it and unpleasant emotional experiences are still poorly understood. So here, we review neuroimaging studies investigating how SD causes reduced functional connectivity in the reward network, which is positively associated with negative emotional experiences in humans.

Effect of acute SD on reward and incentive processing

A network of interrelated brain areas, including the striatum, prefrontal cortex (PFC), hippocampus, amygdala, midbrain ventral tegmental area and nucleus accumbens make up the brain reward system [14]. The striatum receives dopaminergic innervation from the ventral tegmental area [14]. This innervation is linked to and controlled by PFC regions, specifically the inferior orbitofrontal cortex and medial PFC, which direct learning and motivated behavior [14]. It has been repeatedly shown that this system is sensitive to SD, which causes changes in motivated behaviors like impulsivity, risk-taking [14].

By altering dopamine receptor sensitivity and availability in basal ganglia regions, SD impairs dopaminergic function in rats [15, 16]. In a mixed monetary gamble task, humans who have been sleep-deprived for one night exhibit increased ventral striatum activity when they anticipate and receive monetary rewards [17, 12]. Following SD, there is also a significant increase in activity in affect-related frontal cortex regions, such as the insula and medial PFC, that are linked to valuation and viscerosensory functions [12, 17].

These results collectively imply that the state of acute SD appears to hypersensitize subcortical reward-related brain regions as well as associated cortical regions coding salience and valuation [14]. However, there appears to be no difference between rested and deprived conditions in the extent to which functional magnetic resonance imaging (fMRI) signal amplitude tracks reward magnitude in these subcortical and cortical regions [18] . Instead, SD causes a widespread rise in reward sensitivity that undermines the accuracy of reward discrimination, making it harder for the brain to correctly code small increases in reward value from low to high [14].

These findings are supported by the fact that sleep-deprived people's medial PFC, orbitofrontal cortex, and anterior insula cortex fMRI signals cannot distinguish between trials with monetary reward and punishment values and trials without either [12, 17].

The outcome phase of incentive decision trials also shows this erroneous representation of reward value within frontal regions, indicating a further failure to update accruing reward history and probability [12, 17]. Therefore, after sleep loss, the PFC may not be able to update the changing incentive value of reinforcing stimuli over time due to inaccurate coding of reward and/or punishment valence [12, 17]. This would also lead to less-than-ideal reward-dependent choices and behaviors [14]. According to this profile, people who are sleep-deprived when completing the Iowa Gambling Task take more chances and give more weight to rewards that have been given recently [19, 9]. This pattern, which reflects temporally shorter-sighted reward updating, shows an inability to properly integrate rewards and their increasing value over time after SD [14].

In addition to processing financial rewards, impulsivity is another behavioral trait that is impacted by sleep deprivation [14]. Since impulsivity is a complex concept, there are various ways in which it can show up in behavior [14]. Nonetheless, many incentive-based decision-making processes heavily rely on impulse control [20, 21]. Therefore, after SD, changes in reward decision-making and risk-taking may be influenced by changes in impulsivity [14].

Participants who experienced either partial sleep restriction or one night of complete sleep deprivation demonstrated significantly lower response inhibition and cognitive control, more frequent errors of inhibition, and slower learning of cue–incentive associations in tasks that require either cued motor response execution or withholding, also known as a Go/No-Go task [22–26].

According to research to date, SD interferes with reward-value updating and integration and dramatically raises the propensity for reward sensitivity, risk-taking, and impulsivity [27]. Changes in dopamine signaling, which is linked to prolonged wakefulness-possibly more so than sleep deprivation could be one mechanism explaining these increases in approach and consummatory behavior [14]. According to

research to date, SD interferes with reward-value updating and integration and dramatically raises the propensity for reward sensitivity, risk-taking, and impulsivity [14]. Changes in dopamine signaling, which is linked to prolonged wakefulness-possibly more so than sleep deprivation could be one mechanism explaining these increases in approach and consummatory behavior [14]. This dopaminergic framework is supported by multiple lines of evidence [14].

First, dopamine is linked to arousal; lower sleep propensity is predicted by naturally higher dopamine levels [28]. Second, research on rodents has shown that wake-promoting stimulant medications, like amphetamine, appear to work in part by preventing dopamine metabolism, which raises dopamine transmission and, consequently, arousal [14]. Third, reducing catecholamines, including dopamine, lowers vigilance, induces sleep, and decreases wake propensity [29, 30].

After SD, changes in reward-driven behavior may also be caused by modifications in dopamine receptors. One night of complete SD has been shown to decrease the availability of dopamine D2 and D3 receptors (D2/3Rs) in the dorsal striatum (which includes the caudate and putamen) as well as the ventral striatum, according to studies using Positron emission tomography ligand [15]. fMRI-measured drops in thalamic activity during an attentional task were predicted by Positron emission tomography-measured downregulation of D2/3Rs in the ventral striatum [31]. This finding implies that the levels of various dopamine receptors may serve as a proxy for sleep pressure; in line with this, adenosine builds up with longer periods of waking, activating A2A receptors and promoting D2R internalization [15, 32]. Additionally, D2R and A2A receptor agonists exhibit allosteric interaction, meaning that D2R agonists reduce the D2Rs' affinity for their agonists, such as dopamine [33]. Therefore, there are at least two ways that adenosine accumulation, which is linked to longer periods of waking, could reduce D2/3R activity: by increasing D2/3R internalization and by decreasing dopamine binding to D2Rs [14].

Although the downregulation of D2/3Rs associated with SD may seem to contradict the SD-associated increases in neural and behavioral reward sensitivity discussed above, there is a plausible explanation for this paradox involving D1Rs: D2/3R decreases may result in an imbalance of dopamine receptor availability, which would cause the remaining D1Rs to be disproportionately stimulated by the same amount of presynaptically released dopamine [14]. Outside of SD, this imbalance in rodents leads to increased approach behavior to food rewards [34] and an increased risk of cocaine addiction [35], two behavioral outcomes that have been notably independently linked to SD [36, 37]. Additionally, it appears that D1R action is primarily responsible for the rise in striatal fMRI signal brought on by reward incentives under SD conditions [38]. Therefore, SD-associated reductions in D2/3R availability may enhance approach-driven and reward-driven behavior and fMRI-indexed striatal activity by indirectly increasing dopamine binding to the remaining D1Rs [14]. Mechanistic and therapeutic insights into obesity, addiction, and substance use disorders can also be gained from the neural and behavioral effects of sleep deprivation on incentive processes [14].

Effect of acute SD on emotional processing

In addition to suicidal ideation, suicide attempts, and suicide completion [39–41], sleep deprivation consistently causes changes in negative emotional processing, such as irritability, emotional volatility, anxiety, and aggression [42–45]. These results imply that SD modifies certain process domains, such as basic affective reactivity, as well as more complex ones like emotional expression and discrimination.

The effects of SD on reactivity to simple aversive stimuli were the focus of early neuroimaging studies. For instance, it was demonstrated that a single night of SD increased amygdala reactivity to negative images, including weapons, and poisonous snakes and spiders, by 60% [46]. Notably, rapid, subconscious viewing of fear-expressing faces following sleep restriction has been shown to cause amygdala hyper-reactivity, suggesting that this increased limbic reactivity can happen subconsciously and independently of conscious, deliberate cognition [47].

One possible mechanism underlying such amygdala hypersensitivity involves a loss of regulatory control, resulting in contextually inappropriate amygdala reactivity [48]. Decreases in functional connectivity between top-down

control regions of the medial prefrontal cortex and the amygdala have been reported following SD [46, 47, 49–51].

The brain's anticipation of cued emotional experiences is further altered by SD [14]. One night of SD, for instance, increases anticipatory responses of the peripheral autonomic nervous system and cue-evoked activity in the amygdala, anterior insula, and anterior cingulate cortex in anticipation of upcoming emotional picture slides [52, 53].

An additional phenotype of the sleep-deprived human brain emerges when participants are challenged with the more complex task of disambiguating between varied emotional signals [14]. Areas of the cingulate, insula, and amygdala linked to salience detection in participants who have had sleep can distinguish between stimuli with varying emotional intensities [14]. In contrast, these areas show a saturated and flattened reaction to emotional stimuli in participants who are sleep deprived [14]. As a result, people who are sleep deprived exhibit a generalized excess of emotional sensitivity along with a reduction in emotional discriminatory specificity [48].

Recent work has begun to uncover the neural basis of these impairments in emotional discrimination. Consistent with the failure to accurately code positive, reward incentive signals, one night of SD impairs the discrimination of faces expressing negative emotions by viscerosensory regions of the anterior insula and anterior cingulate cortices, and to a degree, the subcortical amygdala [54]. Similarly, sleep loss results in generalized, nonspecific increases in amygdala activity in response to aversive and neutral emotional pictures [55, 56].

Due to these results, it has been suggested that SD results in a condition of both central and peripheral emotional hypersensitivity, which makes it impossible to grade emotional reactivity correctly [48]. The afferent-efferent communication between the brain and body, which is essential for the "embodied" mapping of and differentiation between various emotions, is thus compromised by SD [48].

The cellular and molecular processes that underlie the emotional brain and body dysfunction brought on by sleep deprivation are less well understood. The locus coeruleus's elevated noradrenergic tone has been the focus of one theory put forth; sleep deprivation inhibits the typical overnight decrease in noradrenergic tone that typically takes place during REM sleep [48, 57, 58]. Thus, elevated and over-generalized responsiveness may result from the ensuing hypernoradrenergic tone in the amygdala and viscerosensory cortical regions, which are part of the noradrenaline-innervated affective salience network [48]. As a result, affective signaling within this network may lose specificity, which could help to explain the SD-associated inaccuracy in emotional discrimination [48]. It is unlikely, though, that noradrenaline is the only neurochemical component responsible for the affective dysregulation brought on by SD [14]. An explanation model that incorporates alterations in other sleep-dependent neurochemicals, like dopamine, might be more accurate [14].

Conclusion

In summary, sleep deprivation has a profound effect on how rewards are processed and how emotions are controlled, which drastically changes cognitive and behavioral outcomes. The brain's reward system is impacted by acute sleep deprivation, which impairs judgment and increases reward sensitivity. Changes in dopamine signaling are linked to this altered reward processing, which may lead to an increase in impulsivity and risk-taking behaviors. Anxiety, irritability, and volatility are also increased when sleep deprivation worsens emotional reactivity and impairs the capacity to distinguish between different emotional stimuli. A loss of top-down control and modifications in the functional connectivity between important brain regions, including the amygdala and prefrontal cortex, are the causes of emotional processing dysregulation. All things considered, these results highlight how crucial getting enough sleep is to preserving emotional and cognitive well-being. It is becoming more and more obvious that treatments targeted at increasing the quantity and quality of sleep may be essential for boosting mental health and lowering the risks of emotional dysregulation and poor decision-making as our knowledge of the neurobiological processes underlying the effects of sleep deprivation advances.

Conflict of interest

The author declares no conflicts of interest.

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