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The effect of acute sleep deprivation on the activity of attention supporting brain areas

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ABSTRACT

More than 30% of adults suffer from sleep deprivation (SD). SD has adverse effects on cognitive functions such as attention. In psychology, attention is defined as the concentration of awareness on some events to exclude other stimuli. It has a very important role in regulating the human behavior. Although several studies have investigated the alteration in activity of different attention supporting brain regions following SD, however, these effects are not still fully addressed. Considering the significance of attention in learning and directing the human behavior and regarding the high prevalence of SD, here we review the consequences of acute SD on activity and connectivity of different regions involved in the attention processing by focusing on neuroimaging studies.

Keywords: Sleep deprivation, Attention, Brain networks, Functional connectivity, Neuroimaging

Abbreviation list

SD:sleep deprivation; NREM: non-rapid eye movement; REM: rapid eye movement; fMRI: functional magnetic resonance imaging; FPN: frontoparietal attention network; DMN: default mode network; ACC: anterior cingulate cortex; EEG: electroencephalography; NBM: nucleus basalis of meynert

Introduction

Sleep is one of the important physiological events playing a crucial role in maintaining the physical and mental health [1]. Sleep is characterized by a remarkable decrease in consciousness and a significant reduction in responding to sensory stimuli, which can be quickly reversed [2]. Sleep is associated with complex alteration in physiology of different systems and affects almost all physical and mental functions [3, 2]. During sleep, the organism body take actions necessary to maintain brain function and body health [3]. Neurogenesis and synaptogenesis occur during different stages of sleep. Sleep also plays an important role in removing the harmful substances accumulated surrounding brain neurons during the wakefulness. In fact, the production and flow of cerebrospinal fluid increases dramatically during sleep and somehow washes and removes toxic substances such as beta amyloid from brain tissue. In addition, sleep plays a key role in regulating emotions and maintaining mental balance. Having sustained and effective cognitive functions including learning, consolidating new memories, attention, language, reasoning, creativity and decision making is dependent on sleep.

The brain neuronal activity undergoes remarkable changes during both non-rapid eye movement (NREM) and rapid eye movement (REM) stages of sleep (Box 1). Thus, sleep is expected to play an important role in brain-related functions [4]. Normally, healthy adults need 7- 9 hours and elderly individuals require 8-9 hours of night sleep [1]. Missing even one hour from normal sleep duration causes sleep deprivation (SD), which is the most common type of sleep complaint [5]. SD implies a reduction in both normal duration or quality of sleep [5]. If sleep deficiency lasts for one or two days, it is considered as acute SD and if it encompasses a long period of time, it is regarded as chronic SD [5]. The severity of SD can vary across individuals [5]. The prevalence of acute SD is more than 30% and the prevalence of chronic SD is estimated to be 6-10% worldwide [5, 6].

Multiple factors can cause or contribute to SD including severe life stress, illness, psychological or physical injuries, as well as environmental parameters such as sound, light, and low or high ambient temperature [7]. People with SD undergo impairment of cognitive functions so that they exhibit reduced performance in analytical and logical reasoning. They also need more time for responding to environmental stimuli and show a weak performance in tasks engaging sustained attention [4]. SD leads to enhancement of sleep pressure and weakens the executive function of the individuals. Then, SD exposes the people at risk when they are doing their everyday activities that require high attention, such as driving [8]. The neurological alterations underlying SD consequences are of interest of many researchers. Various studies have attempted to address the changes in activity or connectivity of the relevant neuronal substrates. By focusing on the neuroimaging studies, here we review how the brain networks underlying sustained attention are affected by acute SD in human.

Box1. Sleep stages

Nocturnal sleep consists of several cycles repeating sequentially, and each cycle consists of a maximum of four stages [65]. In a typical night, a person goes through four to six sleep cycles [65]. Duration of sleep cycles is variable, however each cycle lasts about 90 minutes on average [65].

Characteristics of sleep cycles changes over time from sleep onset [66]. The first sleep cycle is often the shortest ranging from 70 to 100 minutes, while subsequent cycles last 90 to 120 minutes [65, 67]. Additionally, sleep cycles can vary from person to person and from night to night based on a range of factors such as age, recent sleep patterns and taking a medication [65].

In general, sleep consists of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep stages [65, 66]. NREM sleep includes 3 phases comprising N1, N2 and N3, and REM sleep has only one stage [65]. These stages are determined based on the analysis of brain activity during sleep that reveals distinct EEG patterns and characterize each period [66]. During stage 1 of NREM sleep, consciousness is reduced but not completely lost [65]. This stage is actually considered as the stage of transition from waking to real sleep [65]. It is easy to awake a person in this phase [66]. In the recording of EEG signals, the power of alpha waves (8-12 Hz) decreases while the power of theta (4 to 8 Hz) enhances [65].

Second phase of NREM sleep is actually the first stage of real sleep as the consciousness is completely lost at the onset of this phase. During this stage, body temperature falls, muscles relax, breathing slows, heart rate decreases and eye movement stops [65]. Stage 2 of NREM sleep last 10 to 25 minutes in the first sleep cycle, but it become longer in later cycles. In this phase. delta (0.5 to 4 Hz) and theta (4 to 8 Hz) waves are dominant pattern of the recorded signal [65]. Furthermore, two distinct patterns of waves are manifested which referred as sleep spindles and k-complex waves [66] (Fig 1). Sleep spindles (10–15 Hz) are transient oscillatory cortical activity arises from local thalamocortical events that periodically alters neuronal activity in grey matter of neocortex. K-complex are short negative high-voltage signal with more than 100 µV in amplitude and are followed by a slower positive deflection around 350 to 550 ms (Fig 1). They often precede a bursts of sleep spindles. sleep spindles and k-complex are considered as hallmarks of stage 2 of NREM sleep.

The third stage of sleep is known as deep sleep because it is more difficult to awake a person from this phase [65]. Muscle tone, heart and breathing rate continue to decrease further [65] and delta waves constitute the dominant pattern in EEG [65]. Even though brain activity is reduced, there is evidence that deep sleep supports thinking, creativity and memory [65]. The most time of deep sleep is spent in the first half of the night. In the first sleep cycles, stage N3 typically lasts 20 to 40 minutes [65]. As sleep continues, these stage becomes shorter, and instead, REM sleep becomes longer [65].

After the 3rd stage of NREM sleep, REM sleep appears [65]. During the REM sleep, brain activity increases and approaches to what observed during wakefu.lness [65, 68]. In this stage, muscle tone is temporarily lost in almost the entire body except for the respiratory muscles, pupil sphincter and the muscles that are responsible for eye movement [65]. The eyes show apparent quick movements so that they are easy to be recognized even with closed eyelids [65, 68]. This is the reason that this stage is referred as REM [65]. It is believed that REM sleep is essential for maintaining cognitive functions like some forms of memory such as emotional memory, learning and creativity [65]. REM sleep is characterized by vivid dreams which is explained by the significant increase in brain cortical activity [68]. As already implied, the duration of REM sleep increase with sleep cycles advancement, especially in the second half of the night [68]. While the first REM sleep may last only a few minutes, the later ones can last up to an hour [65]. On average, the REM stage accounts for about 25% of sleep duration in adults [65].

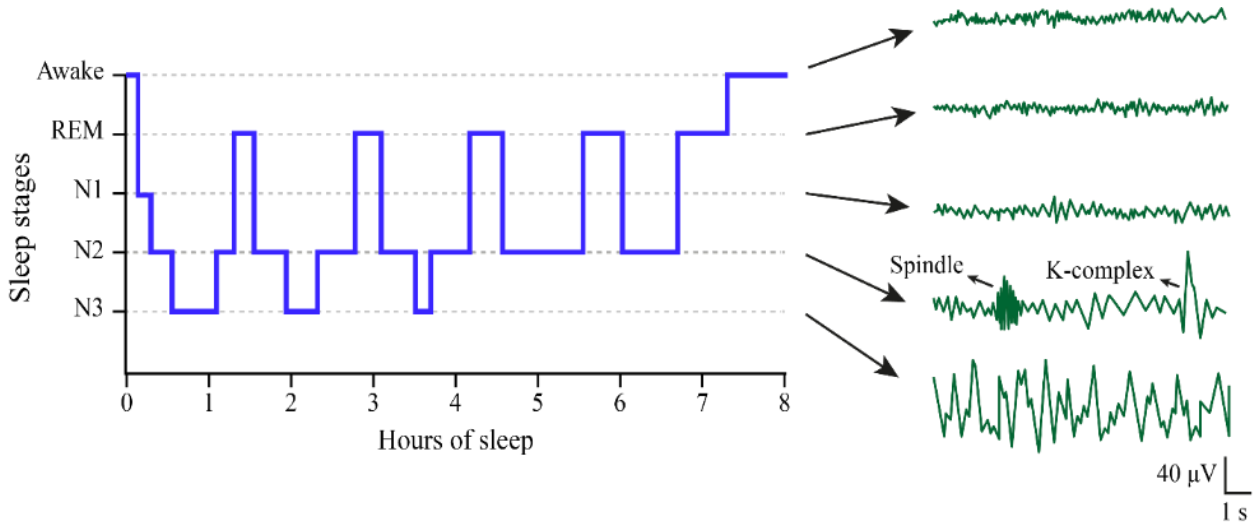

Fig 1. Sleep pattern in a healthy person during 8 hours of night sleep. An example of a hypnogram is shown on the left and a schematic diagram of the EEG pattern in different stages of sleep is shown on the right

Effects of acute SD on attention

Attention is the ability to actively process specific information in the environment while excluding other details (Box 2). It is one of the basic cognitive processes which is considered as the basis of the learning processes and plays a very important role in decision-making, motor execution and behavior [9]. Being conscious and alert is the primary condition for having normal attention [9].

SD, which is associated with weak consciousness and low vigilance, impairs attention. A number of studies have indicated that attention deficits persist even when awareness is restored with stimulants such as caffeine following SD, suggesting that SD has a complex

effect on attention impairment rather than simply reducing the vigilance [10–12].

Despite the inter-individual variations observed in SD-mediated attention impairment, generally, performance of sleep-deprived persons in attentional tasks decreases in a dose-dependent manner with increasing duration of wakefulness and therefore with rising the sleep pressure [13–15]. During these tasks, the attention is completely disrupted for short periods of time known as microsleep [16, 13, 14]. In other words, attention becomes very unstable and irregular [16] and causes the performance of people to vary in attention tasks [17]. Therefore, the severity of attention impairment can be predicted with regard to the cumulative duration of wakefulness in both acute and chronic SD [14].

Box2. What is attention?

Attention is one of the topics widely discussed in psychology and cognitive neuroscience, and is considered as one of the main research fields in education, psychology and neuroscience [69]. William James in the 19th century defined attention as follows: "the taking possession by the mind in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. It implies withdrawal from some things in order to deal effectively with others" [69].

According to John Ratey (2001), attention is more than only paying attention to incoming stimuli [69]. "It involves a number of processes including filtering out perceptions, balancing multiple perceptions and attaching emotional significance to these perceptions" [69]. Attention is believed to be the basis of learning, acts as the leader of our brain and determines how neural resources are engaged [69].

In general, there are two types of attention including passive or involuntary attention and active or voluntary attention [69]. In passive attention, a strong and sudden stimulus attracts the attention [69].

Voluntary attention is exactly what its name implies and is divided into 5 different types including focused, sustained, alternating, divided and selective attention [69].

1) Focused attention: It is a type of attention that includes a basic reaction to various environmental stimuli such as auditory, visual or tactile. For instance turning the head after hearing a sound [70].
2) Sustained attention: It is considered as the ability to focus on a specific stimulus or information for a continuous period of time without being distracted. For example, playing a computer game needs sustained attention [71, 72].
3) Alternating attention: It is the ability of mental flexibility to shift attention from one activity to another such as cooking the food [73].
4) Divided attention: It is the ability of an individual to carry out more than one activity or task at the same time, like talking on the phone while surfing the web [74].
5) Selective attention: It implies to focusing on one activity, external stimulus or a certain information among several activities, stimuli or information [75]. for example, listening to a friend at a crowded party [75, 72, 76].

Posner and Rafal (1987) proposed a model that can be used for analyzing the circadian changes of attention [77]. This model was later elaborated further by Cohen (1993) [77]. According to this model, attention can be defined with four components including phasic alertness, tonic alertness, selective attention and vigilance [77]. Alertness is an organism capability to respond to the environmental stimuli [77]. Tonic alertness is the basic capacity of an organism for responding to stimuli during the day and phasic alertness is defined as the ability to respond to a stimulus after a warning signal [77]. Selective attention is the capacity to respond to a specific stimulus and ignore distractors [77]. Vigilance is the capacity of an organism to focus on a task and preserve this activity over time [77]

Total SD for 24 hours reduces components of attention including tonic alertness, selective attention, and sustained attention, while the effects of total SD on phasic alertness are observed only after periods of SD longer than 24 hours (periods of 48 hours or more) [78]

Effect on lateral frontoparietal attention network

Functional magnetic resonance imaging (fMRI) studies have revealed that SD reduces the neuronal activity in lateral frontoparietal attention network (FPN) related regions including dorsolateral prefrontal cortex and intraparietal sulcus in attention tasks [18–25]. In addition, SD decreases activity in the extrastriate visual cortex and also diminishes functional connectivity of this area with both dorsolateral prefrontal cortex and intraparietal sulcus during the visual attention task [18–20]. The behavioral consequences of these alteration in attention tasks are observed as losing the attention to a particular stimulus in the visual field or as a disruption in the top-down allocation of attentional resources, such as looking at the target location where the visual

stimulus is expected to emerge [26, 18, 27]. In addition to focusing attention at a particular moment (selective attention), SD also diminishes the capacity of attention maintenance over time (sustained attention; box 2) [16].

Effect on thalamus

The coordinate activity of cortical neurons is regulated to a large extent by thalamus. It is a diencephalon brain region that has extensive connections with the different areas of cerebral cortex and possesses both sensory and motor functions. In addition, it also plays an important role in cognitive processes including attention, consciousness and sleep-wake transitions [26, 27].

The thalamocortical functional connectivity significantly reduces following SD, which leads to a decreased vigilance level [28]. The thalamus has been suggested to be considered as an interacting hub in the SD-affected network [17]. It is noteworthy that the pattern of alteration in thalamus activity is not uniform among different individuals following SD [17]. While some studies have observed an increased activity in this area [29, 30, 25, 21], others have reported intermittent periods of decreased activity subsequent to SD [19, 21]. Considering the role of thalamus in arousal and regarding the attention instability following SD, reporting such contradictory results are not surprising [17].

Effect on default mode network

Study on the sleep deprived human brain also indicated that the default mode network (DMN) is also sensitive to SD [31]. The DMN comprises a network of interacting brain regions that are more active during awake resting state when the mind is not focused on anything in outside. By contrary, the DMN activity consistently reduces when an individual focuses on something in external or internal worlds. This network is also called medial frontoparietal network and includes the medial prefrontal cortex, posterior cingulate cortex and angular gyrus [32]. Several studies have suggested that instability in the functional connectivity within DMN also participate in the attention impairment following SD [22, 25, 33]. In addition, it has been observed that the reduction in the activity of this network does not occur following SD when the person needs to concentrate in an attention task [17].

Normally, there is a pivotal inhibitory relationship between FPN and DMN activity [17, 34]. In the resting state, when the person does not concentrate on anything in the outside, DMN activity is high and FPN activity is low [34]. The opposite change happens during a sustained attention [34, 17]. This reciprocal inhibitory association is required for establishment of the sustained and effective attention. The thalamus, which sends projections to both networks, participates in setting up the inhibitory interplay between these two networks [35]. When a person has adequate night sleep, thalamus receives continuous inputs from the ascending arousal pathways and causes establishment of effective reciprocal inhibition of the FPN and DMN [35, 36]. Following acute SD, the activity of ascending arousal inputs to the thalamus becomes erratic and irregular, and then, inhibitory pivotal relationship between DMN and FPN activity alter into an unstable state [36, 17]. This causes the increased activity in FPN does not reach the normal level in an attention task. Moreover, the activity of this network also shows intermittent reductions due to the general increase in DMN activity following the SD. As a consequence, performance of attention and working memory weakens and becomes even unstable [17]. This disturbance is worsened with a decrease and improves with an increase in thalamic activity [17].

Effect on salience network

The salience network, which is also regarded as one of the major brain networks, is also vulnerable to SD [37, 17]. It is more active than other networks in many complex cognitive functions such as attention, and hence, it is also called attention network [37]. This network comprise the anterior insula, dorsoanterior cingulate cortex, inferior parietal cortex, right parietal temporal junction and lateral prefrontal cortex [37].

It has been shown that the activity of frontoinsular areas of this network are impaired in attention tasks following acute SD [38–40]. In fact, the insula itself is considered as a core hub in the attention network and also plays an important role in decision making, sensory processing and salience detection [41–43, 35]. Neuroimaging studies has reported that activity, gray matter volume and blood flow in the insula increase following SD [44]. This increment may reflect upregulated bottom-up modulation of cortical attention networks that maintain attention [45]. In addition, SD results in a decreased functional connectivity between the insula and several regions of executive control network including some areas of temporal lobe (such as the inferior temporal gyrus, middle temporal gyrus and fusiform gyrus), the superior parietal

lobule, certain regions in prefrontal cortex (comprising orbital gyrus, dorsolateral superior frontal gyrus, middle frontal gyrus and medial prefrontal cortex) and anterior cingulate cortex (ACC) [45]. All of these areas are involved in attentional processes, and to some extent, the decrease in the functional connectivity of the insula with them may account for the attention impairment following SD [45].

As already pointed out, the reciprocal inhibitory connectivity between DMN and FPN becomes irregular and unstable following SD [17]. Although the cause of such disturbance is not known yet, however, considering that salience network controls the switch between DMN and FPN activity, the abnormal activity of salience network may explain this instable connectivity following SD.

ACC is the frontal part of the cingulate cortex and is involved in some higher-level functions, such as attention allocation [46]. Neuroimaging studies have reported a decrease in ACC activity during attention tasks after an acute SD [47]. An electroencephalography (EEG) study has demonstrated that SD disrupts the functional connectivity between ACC and posterior cingulate cortex [46]. However, the functional connectivity of the ACC with some cortical and subcortical areas including the frontoparietal, putamen and anterior insula has been observed to increase following one night of SD [48].

Effect on nucleus basalis of meynert

Another brain area involved in attention is the nucleus basalis of meynert (NBM), which is located in the substantia innominate of the basal forebrain. This area contains mostly cholinergic neurons projecting to various parts of the brain [49]. The NBM is involved in multiple functions, especially supporting wakefulness and maintaining visuospatial attention [47]. Activity of cholinergic neurons in the NBM has been shown to support supramodal attention by modulating activity in the FPN [50].

High concentrations of adenosine in extracellular space, which occur during prolonged wakefulness, has been shown to suppress the activity of basal forebrain cholinergic neurons [51]. It indicates that NBM activity is sensitive to SD due to impaired sleep homeostasis [47]. In a recent study, Jing Qi et al. (2021) has shown that 36 hours of total SD reduces functional connectivity of the left NBM with superior parietal lobule while it enhances the functional connectivity of the right NBM with thalamus and also with the middle and anterior parts of the cingulate cortex [47]. Reduction of activity in cholinergic projections to superior parietal lobule is associated with impaired performance of verbal episodic memory and visuospatial attention in Parkinson's disease [52]. Therefore, the disruption of the connectivity between NBM and superior parietal lobule may also participate in memory and attention disturbances after total SD [47]. Enhanced functional connectivity of the NBM with thalamus confirms the recruitment of arousal networks by brain to maintain vigilance and cognitive abilities following SD [47]. Furthermore, increased functional connectivity between NBM and ACC reflects a synergistic effect in regulating the attention in sleep-deprived states [47].

Effect on hypothalamus

Another brain area that has been shown to be sensitive to SD is hypothalamus [35]. It is a heterogeneous subcortical region located below the thalamus and has numerous nuclei. It has extensive connections with the brainstem, thalamus, limbic areas, basal ganglia and cerebral cortex, and plays a crucial role in regulating sleep and wakefulness, temperature, glucose metabolism, body fluid balance and ingestion [53]. In addition, it also participates in regulating attention because some important nuclei of the arousal system are located within the hypothalamus [54].

SD is also associated with alterations in hypothalamus activity that can be followed as an impairment in the function of hypothalamus-pituitary-adrenal axis [55]. It has been observed that cortisol release from adrenal gland in response to stressful factors decreases flowing SD [55]. Fifel et al. (2018) have reported alteration in neural activity of different rat hypothalamus subregions including paraventricular nucleus, lateral hypothalamus, mammillary bodies and arcuate nucleus after a SD [56]. Schmidt et al. (2009) found that with increased homeostatic sleep pressure, the hypothalamic suprachiasmatic

area activity during a sustained attention task decreases [57]. This nucleus plays a central role in controlling the body's circadian rhythms and regulating sleep and wakefulness [58]. A decreased functional connectivity between hypothalamus and middle frontal gyrus following acute SD has also been reported [35]. As discussed before, the prefrontal cortex is involved in a wide range of cognitive processes including top-down attention control [59]. The middle frontal gyrus is a key hub that mediates the interaction between dorsal and ventral attention networks and contributes to the integration of neural circuits associated with attention [60]. Therefore, it has been suggested that the hypothalamus acts as a key controller in the regulation of attention networks, such that the disturbance of hypothalamic circuits activity can lead to attention deficit [35].

In the recent study conducted by Jing Qi et al. (2021), 36 hours of total SD caused a change in the functional connectivity of the hypothalamus with the brain regions involved in attention control as enhancement of the connectivity with thalamus, ACC, insula and right amygdala and a decreased connectivity with middle frontal gyrus [35]. The role of the thalamus, insula and ACC in controlling the attention has already implied. Amygdala also participate in attention modulation, as it has a strong connection with the sensory networks and indirect connections with the attention and arousal network [61–64].

The increase in the functional connectivity of the hypothalamus with the mentioned areas again demonstrate that the brain organizes more attentional resources to meet the requirements of cognitive performance under increased sleep pressure [35]. However, redistribution of attentional resources is not sufficient to counteract the negative effects of SD [35].

Conclusion

According to what reviewed above, acute SD causes a complex set of changes in the activity and connectivity of different brain areas which participate in processing or modulating the attention. While the functional connectivity between some of these areas reduces, the activity and functional connectivity between others enhances. The brain seems to recruit various resources to maintain vigilance and attention following an SD. However, these compensatory solutions are not sufficient and then, the sustained attention impairs and becomes even unstable. Of course, it should be noted that attention does not decrease to the same extent in all people following SD, and inter-individual differences are usually observed. It demonstrates that the activity of brain attention networks may be affected differently in each individual following SD. Another noteworthy point is that most of SD consequences on SD reviewed above are related to fMRI studies. Despite the high spatial resolution, this method has a low time resolution for examining the alteration in activity or connectivity of different brain areas, while momentary fluctuations in the activity or connectivity of these regions in a small timescale are significant. Therefore, repeating the experiments using high temporal resolution supplementary methods, such as EEG, are required.

The last point is that our current knowledge about the effect of chronic SD on human brain is still largely unknown, because most studies have focused on effects of acute SD. Also, there is no comprehensive understanding of how human brain networks may recover from acute and chronic SD and more studies should be carried out to address this issue.

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Conflict of interest

The authors declare that they have no conflicts of interest.

References

[1] Mason GM, Lokhandwala S, Riggins T, Spencer RMC. Sleep and human cognitive development. Sleep Med Rev 2021; 57:101472.

[2] Cordi MJ, Rasch B. How robust are sleep-mediated memory benefits? Curr Opin Neurobiol 2021; 67:1–7.

[3] Ferrara M, Gennaro L de. How much sleep do we need? Sleep Med Rev 2001; 5(2):155–79.

[4] Orzeł-Gryglewska J. Consequences of sleep deprivation. Int J Occup Med Environ Health 2010; 23(1):95–114.
[5] Landolt H-P, Holst SC, Sousek A. Effects of acute and chronic sleep deprivation: European Sleep Research Society; 2014.
[6] Vargas I, Nguyen AM, Muench A, Bastien CH, Ellis JG, Perlis ML. Acute and Chronic Insomnia: What Has Time and/or Hyperarousal Got to Do with It? Brain Sci 2020; 10(2).
[7] Sanches I, Teixeira F, dos Santos JM, Ferreira AJ. Effects of Acute Sleep Deprivation Resulting from Night Shift Work on Young Doctors. Acta Med Port 2015; 28(4):457–62.
[8] Powell NB, Chau JKM. Sleepy driving. Med Clin North Am 2010; 94(3):531–40.
[9] Collet J, Ftouni S, Clough M, Cain SW, Fielding J, Anderson C. Differential Impact of Sleep Deprivation and Circadian Timing on Reflexive Versus Inhibitory Control of Attention. Sci Rep 2020; 10(1):7270.
[10] Killgore WDS. Effects of sleep deprivation on cognition. Prog Brain Res 2010; 185:105–29.
[11] Whitney P, Hinson JM. Measurement of cognition in studies of sleep deprivation. Prog Brain Res 2010; 185:37–48.
[12] Bonnet MH, Balkin TJ, Dinges DF, Roehrs T, Rogers NL, Wesensten NJ. The use of stimulants to modify performance during sleep loss: a review by the sleep deprivation and Stimulant Task Force of the American Academy of Sleep Medicine. Sleep 2005; 28(9):1163–87.
[13] van Dongen HPA, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. Sleep 2003; 26(2):117–26.
[14] Belenky G, Wesensten NJ, Thorne DR, Thomas ML, Sing HC, Redmond DP et al. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. J Sleep Res 2003; 12(1):1–12.
[15] Borbély AA. A two process model of sleep regulation. Hum Neurobiol 1982; 1(3):195–204.
[16] Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. Semin Neurol 2005; 25(1):117–29.
[17] Krause AJ, Simon EB, Mander BA, Greer SM, Saletin JM, Goldstein-Piekarski AN et al. The sleep-deprived human brain. Nat Rev Neurosci 2017; 18(7):404–18.
[18] Chee MWL, Goh CSF, Namburi P, Parimal S, Seidl KN, Kastner S. Effects of sleep deprivation on cortical activation during directed attention in the absence and presence of visual stimuli. Neuroimage 2011; 58(2):595–604.
[19] Chee MWL, Tan JC. Lapsing when sleep deprived: neural activation characteristics of resistant and vulnerable individuals. Neuroimage 2010; 51(2):835–43.
[20] Chee MWL, Tan JC, Parimal S, Zagorodnov V. Sleep deprivation and its effects on object-selective attention. Neuroimage 2010; 49(2):1903–10.
[21] Chee MWL, Tan JC, Zheng H, Parimal S, Weissman DH, Zagorodnov V et al. Lapsing during sleep deprivation is associated with distributed changes in brain activation. J Neurosci 2008; 28(21):5519–28.
[22] Czisch M, Wehrle R, Harsay HA, Wetter TC, Holsboer F, Sämann PG et al. On the Need of Objective Vigilance Monitoring: Effects of Sleep Loss on Target Detection and Task-Negative Activity Using Combined EEG/fMRI. Front Neurol 2012; 3:67.
[23] Drummond SP, Brown GG, Stricker JL, Buxton RB, Wong EC, Gillin JC. Sleep deprivation-induced reduction in cortical functional response to serial subtraction. Neuroreport 1999; 10(18):3745–8.
[24] Thomas M, Sing H, Belenky G, Holcomb H, Mayberg H, Dannals R et al. Neural basis of alertness and cognitive performance impairments during sleepiness. I. Effects of 24 h of sleep deprivation on waking human regional brain activity. J Sleep Res 2000; 9(4):335–52.
[25] Tomasi D, Wang RL, Telang F, Boronikolas V, Jayne MC, Wang G-J et al. Impairment of attentional networks after 1 night of sleep deprivation. Cereb Cortex 2009; 19(1):233–40.
[26] Halassa MM, Chen Z, Wimmer RD, Brunetti PM, Zhao S, Zikopoulos B et al. State-dependent architecture of thalamic reticular subnetworks. Cell 2014; 158(4):808–21.
[27] Steriade M, Llinás RR. The functional states of the thalamus and the associated neuronal interplay. Physiol Rev 1988; 68(3):649–742.
[28] Shao Y, Wang L, Ye E, Jin X, Ni W, Yang Y et al. Decreased thalamocortical functional connectivity after 36 hours of total sleep deprivation: evidence from resting state FMRI. PLoS One 2013; 8(10):e78830.
[29] Portas CM, Rees G, Howseman AM, Josephs O, Turner R, Frith CD. A specific role for the thalamus in mediating the interaction of attention and arousal in humans. J Neurosci 1998; 18(21):8979–89.

[30] Chee MWL, Choo WC. Functional imaging of working memory after 24 hr of total sleep deprivation. J Neurosci 2004; 24(19):4560–7.
[31] Havas JA de, Parimal S, Soon CS, Chee MWL. Sleep deprivation reduces default mode network connectivity and anti-correlation during rest and task performance. Neuroimage 2012; 59(2):1745–51.
[32] Buckner RL, Andrews-Hanna JR, Schacter DL. The brain's default network: anatomy, function, and relevance to disease. Ann N Y Acad Sci 2008; 1124:1–38.
[33] Drummond SPA, Bischoff-Grethe A, Dinges DF, Ayalon L, Mednick SC, Meloy MJ. The neural basis of the psychomotor vigilance task. Sleep 2005; 28(9):1059–68.
[34] Cui J, Tkachenko O, Gogel H, Kipman M, Preer LA, Weber M et al. Microstructure of frontoparietal connections predicts individual resistance to sleep deprivation. Neuroimage 2015; 106:123–33.
[35] Qi J, Li B-Z, Zhang Y, Pan B, Gao Y-H, Zhan H et al. Altered Hypothalamic Functional Connectivity Following Total Sleep Deprivation in Young Adult Males. Front Neurosci 2021; 15:688247.
[36] Fu W, Dai C, Chen J, Wang L, Song T, Peng Z et al. Altered insular functional connectivity correlates to impaired vigilant attention after sleep deprivation: A resting-state functional magnetic resonance imaging study. Front Neurosci 2022; 16:889009.
[37] Seeley WW. The Salience Network: A Neural System for Perceiving and Responding to Homeostatic Demands. J. Neurosci. 2019; 39(50):9878–82.
[38] Ma N, Dinges DF, Basner M, Rao H. How acute total sleep loss affects the attending brain: a meta-analysis of neuroimaging studies. Sleep 2015; 38(2):233–40.
[39] Gumenyuk V, Roth T, Korzyukov O, Jefferson C, Bowyer S, Drake CL. Habitual short sleep impacts frontal switch mechanism in attention to novelty. Sleep 2011; 34(12):1659–70.
[40] Gazes Y, Rakitin BC, Steffener J, Habeck C, Lisanby SH, Butterfield B et al. Dual-tasking alleviated sleep deprivation disruption in visuomotor tracking: an fMRI study. Brain Cogn 2012; 78(3):248–56.
[41] Naqvi NH, Bechara A. The insula and drug addiction: an interoceptive view of pleasure, urges, and decision-making. Brain Struct Funct 2010; 214(5-6):435–50.
[42] Cauda F, D'Agata F, Sacco K, Duca S, Geminiani G, Vercelli A. Functional connectivity of the insula in the resting brain. Neuroimage 2011; 55(1):8–23.
[43] Uddin LQ. Salience processing and insular cortical function and dysfunction. Nat Rev Neurosci 2015; 16(1):55–61.
[44] Elvsåshagen T, Mutsaerts HJ, Zak N, Norbom LB, Quraishi SH, Pedersen PØ et al. Cerebral blood flow changes after a day of wake, sleep, and sleep deprivation. Neuroimage 2019; 186:497–509.
[45] Qi J, Li B-Z, Zhang Y, Pan B, Gao Y-H, Zhan H et al. Altered insula-prefrontal functional connectivity correlates to decreased vigilant attention after total sleep deprivation. Sleep Med 2021; 84:187–94.
[46] Piantoni G, Cheung BLP, van Veen BD, Romeijn N, Riedner BA, Tononi G et al. Disrupted directed connectivity along the cingulate cortex determines vigilance after sleep deprivation. Neuroimage 2013; 79:213–22.
[47] Qi J, Li B-Z, Zhang Y, Pan B, Gao Y-H, Zhan H et al. Altered functional connectivity between the nucleus basalis of Meynert and anterior cingulate cortex is associated with declined attentional performance after total sleep deprivation. Behav Brain Res 2021; 409:113321.
[48] Fang Z, Spaeth AM, Ma N, Zhu S, Hu S, Goel N et al. Altered salience network connectivity predicts macronutrient intake after sleep deprivation. Sci Rep 2015; 5:8215.
[49] Zaborszky L, Hoemke L, Mohlberg H, Schleicher A, Amunts K, Zilles K. Stereotaxic probabilistic maps of the magnocellular cell groups in human basal forebrain. Neuroimage 2008; 42(3):1127–41.
[50] Ljubojevic V, Luu P, Gill PR, Beckett L-A, Takehara-Nishiuchi K, Rosa E de. Cholinergic Modulation of Frontoparietal Cortical Network Dynamics Supporting Supramodal Attention. J. Neurosci. 2018; 38(16):3988–4005.
[51] Porkka-Heiskanen T, Alanko L, Kalinchuk A, Stenberg D. Adenosine and sleep. Sleep Med Rev 2002; 6(4):321–32.
[52] Zhang C, Wu C, Zhang H, Dou W, Li W, Sami MU et al. Disrupted Resting-state Functional Connectivity of the Nucleus Basalis of Meynert in Parkinson's Disease with Mild Cognitive Impairment. Neuroscience 2020; 442:228–36.
[53] Arrigoni E, Chee MJS, Fuller PM. To eat or to sleep: That is a lateral hypothalamic question. Neuropharmacology 2019; 154:34–49.
[54] Jones BE. Arousal systems. Front Biosci 2003; 8:s438-51.
[55] Vargas I, Lopez-Duran N. Investigating the effect of acute sleep deprivation on hypothalamic-

pituitary-adrenal-axis response to a psychosocial stressor. Psychoneuroendocrinology 2017; 79:1–8.

[56] Fifel K, Meijer JH, Deboer T. Long-term effects of sleep deprivation on neuronal activity in four hypothalamic areas. Neurobiol Dis 2018; 109(Pt A):54–63.

[57] Schmidt C, Collette F, Leclercq Y, Sterpenich V, Vandewalle G, Berthomier P et al. Homeostatic sleep pressure and responses to sustained attention in the suprachiasmatic area. Science 2009; 324(5926):516–9.

[58] Venner A, Todd WD, Fraigne J, Bowrey H, Eban-Rothschild A, Kaur S et al. Newly identified sleep-wake and circadian circuits as potential therapeutic targets. Sleep 2019; 42(5).

[59] Gaggioni G, Ly JQM, Chellappa SL, Coppieters 't Wallant D, Rosanova M, Sarasso S et al. Human fronto-parietal response scattering subserves vigilance at night. Neuroimage 2018; 175:354–64.

[60] Song P, Lin H, Liu C, Jiang Y, Lin Y, Xue Q et al. Transcranial Magnetic Stimulation to the Middle Frontal Gyrus During Attention Modes Induced Dynamic Module Reconfiguration in Brain Networks. Front Neuroinform 2019; 13:22.

[61] Sarter M, Hasselmo ME, Bruno JP, Givens B. Unraveling the attentional functions of cortical cholinergic inputs: interactions between signal-driven and cognitive modulation of signal detection. Brain Res Brain Res Rev 2005; 48(1):98–111.

[62] Abivardi A, Bach DR. Deconstructing white matter connectivity of human amygdala nuclei with thalamus and cortex subdivisions in vivo. Hum Brain Mapp 2017; 38(8):3927–40.

[63] Pruessner L, Barnow S, Holt DV, Joormann J, Schulze K. A cognitive control framework for understanding emotion regulation flexibility. Emotion 2020; 20(1):21–9.

[64] Shao Y, Lei Y, Wang L, Zhai T, Jin X, Ni W et al. Altered resting-state amygdala functional connectivity after 36 hours of total sleep deprivation. PLoS One 2014; 9(11):e112222.

[65] Patel AK, Reddy V, Araujo JF. StatPearls: Physiology, Sleep Stages. Treasure Island (FL); 2022.

[66] Gaudreau H, Carrier J, Montplaisir J. Age-related modifications of NREM sleep EEG: from childhood to middle age. J Sleep Res 2001; 10(3):165–72.

[67] Memar P, Faradji F. A Novel Multi-Class EEG-Based Sleep Stage Classification System. IEEE Trans Neural Syst Rehabil Eng 2018; 26(1):84–95.

[68] Vazquez J, Baghdoyan HA. Basal forebrain acetylcholine release during REM sleep is significantly greater than during waking. Am J Physiol Regul Integr Comp Physiol 2001; 280(2):R598-601.

[69] Ocasio W. Attention to Attention. Organization Science 2011; 22(5):1286–96.

[70] Bennett KB, Flach JM. Graphical displays: implications for divided attention, focused attention, and problem solving. Hum Factors 1992; 34(5):513–33.

[71] Doran SM, van Dongen HP, Dinges DF. Sustained attention performance during sleep deprivation: evidence of state instability. Arch Ital Biol 2001; 139(3):253–67.

[72] Berger A, Posner M. Pathologies of brain attentional networks. Neuroscience & Biobehavioral Reviews 2000; 24(1):3–5.

[73] Jamro D, Zurek G, Lachowicz M, Lenart D, Dulnik M. Alternating Attention and Physical Fitness in Relation to the Level of Combat Training. Healthcare (Basel) 2022; 10(2).

[74] Spelke E, Hirst W, Neisser U. Skills of divided attention. Cognition 1976; 4(3):215–30.

[75] Johnston WA, Dark VJ. Selective Attention. Annu. Rev. Psychol. 1986; 37(1):43–75.

[76] Squire RF, Noudoost B, Schafer RJ, Moore T. Prefrontal contributions to visual selective attention. Annu Rev Neurosci 2013; 36:451–66.

[77] Valdez P, Ramírez C, García A, Talamantes J, Armijo P, Borrani J. Circadian rhythms in components of attention. Biological Rhythm Research 2005; 36(1-2):57–65.

[78] García A, Angel JD, Borrani J, Ramirez C, Valdez P. Sleep deprivation effects on basic cognitive processes: which components of attention, working memory, and executive functions are more susceptible to the lack of sleep? Sleep Sci 2021; 14(2):107–18.