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Sleep Deprivation, Memory Consolidation, and Cognitive Function: A Neuroscience Analysis

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Abstract. Although the negative effects of sleep deprivation on learning, memory and executive function are well-known, an increasing number of studies have put forward that these impairments are mainly due to a disturbance in sleep-dependent memory consolidation rather than fatigue alone. This paper investigates the connection between sleep and memory and cognition, examines how the brain processes information during sleep to promote learning, and enhances high-order thinking skills. Currently, it has been shown that memories formed in a waking state are organised during sleep through cooperation among the hippocampus, neocortex and prefrontal cortex. Meanwhile, there is temporal coupling between the cortical slow oscillations, thalamocortical sleep spindles and hippocampal sharp-wave ripples which collectively promote hippocampal replay and cortical plasticity and drive a redistribution of newly acquired information through long-term cortical networks. Synaptic homeostasis which keeps the brain efficient and prevents the loss of learning capability also mentioned in this review. All the above mechanisms are disrupted when sleep is lost, there is an impact on the communication between the hippocampus and cortex and memory and executive function are reduced. Finally, behaviour and neuroscience-based interventions to enhance sleep-dependent memory consolidation and thus improve cognitive functioning are discussed. Overall, the available evidence suggests that sleep primarily affects cognition via modulation of memory processes, and better understanding of the physiology of sleep can help improve cognitive function and contribute to new therapeutic approaches.

Keywords: Sleep deprivation, Memory consolidation, Cognition, Neural plasticity

1. Introduction

Sleep is a vital biological function with numerous physiological roles and is believed to play a role in different cognitive functions such as memory consolidation and restoration of the nervous system [1]. But, with growing academic and work demands and the ubiquitous use of electronic devices, sleep deprivation has been increasing across the globe. There are two types of sleep deprivation: chronic sleep restriction (repeatedly restricting sleep for several consecutive nights) and acute sleep deprivation (keeping someone up for a long period of time). Both are associated with impairments in attention and working memory, learning and decision making [2] although the underlying causes of these are different. With the high prevalence of inadequate sleep in the adolescent and young adult

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population, almost three-quarters (72.7%) of high school students reported report sleeping less than eight hours a night on weekdays [3], sleep-related findings on the neural mechanisms of cognitive decline could also facilitate new strategies and approaches to improve cognitive functioning.

Recent findings indicate that sleep is an active process and not only a rest state, and it can help to organise and fix newly learned memories. The active systems consolidation hypothesis posits that the process of memory consolidation involves multiple restatements of memories, which occur during sleep, during which information that was recently learned is slowly incorporated into long-term memory networks. Other scientists have suggested that sleep enhances memory by clearing away the excess neurons that have been firing in the waking brain, and thus by resynchronizing the disturbed neural networks [4]. Sleep deprivation thus could disrupt these consolidation processes and affect memory and cognition. There is consensus that sleep is a key factor in determining cognitive outcome, but how exactly is still a topic of debate.

Meanwhile, sleep deprivation research has shown that due to sleep deprivation, there is a cognitive impairment that is not a simple result of fatigue. Thus, various models have been proposed to explain how sleep loss affects cognition, such as the attentional lapse model, which focuses on the possibility that sleep loss may lead to lapses in attention, and the higher-order brain regions model, which posits that regions of the brain involved in higher-order functions may be more sensitive to sleep loss. These findings indicate that sleep loss is not just about being drowsy and is related to a complicated interconnection between memory and cognition. Knowing these mechanisms is, therefore, important to create effective interventions to minimize the harmful effects of sleep deprivation.

This paper will first look at the link between sleep deprivation and memory consolidation, and then the link between memory deficits and the more general cognitive deficits. It will then examine the neural mechanisms that connect sleep, memory and cognition, and make suggestions on how the harmful effects of sleep deprivation might be prevented.

2. Sleep and memory

2.1. Sleep deprivation

Sleep deprivation is the reduction in sleep requirement to achieve a normal physiological state and/or cognitive functioning. In general, it is divided into two types, acute sleep deprivation (the subject is not allowed to sleep for a considerable amount of time), and chronic sleep restriction (the subject sleeps less than the minimal amount for a series of nights) [2]. While acute sleep deprivation is the more common form of sleep deprivation used in laboratory studies, such sleep deprivation is more like that of today's society, particularly among adolescents and young adults [3]. Acute sleep loss leads to impairments in encoding of new information and consolidation of newly formed memories, while chronic sleep loss leads to impairments in learning, working memory and long-term memory performance due to multiple nights of poor sleep [2, 3].

While sleep deprivation is known to cause fatigue, it has other effects as well. Adequate research has shown that sleep deprivation negatively affects attention, working memory, learning and executive functioning [2]. Furthermore, sleep deprivation has been associated with impaired cognitive flexibility and decision making, both acute and chronic, indicating that sleep is crucial for normal cognitive functioning. The results suggest that sleep deprivation not only reduces the amount of information that can be recalled, but also the cognitive functions which require intact memory formation and retrieval, which forms the basis for the understanding of the role of sleep in memory consolidation.

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2.2. Relationship between sleep and memory

The three stages of memory formation are regarded as an important aspect in determining whether the information learnt will be stored and subsequently retrieved – these are encoding, consolidation and retrieval. The encoding, the consolidation and the retrieval process are three mechanisms that are involved in the storage of information [5]. While each of these processes influence memory, sleep seems to play a special role in memory consolidation.

This is supported by a growing body of evidence that sleep is not a passive process for consolidating memories learned during wakefulness, but sleep may be an active process that strengthens and recognises neural representations learned during wakefulness [6]. Different kinds of memory are associated to different sleep stages. Declarative memory (facts, events) appears to be related to slow-wave sleep (SWS) and procedural and emotional memory to rapid eye movement (REM) sleep [1, 5].

There are other pieces of evidence that confirm the importance of sleep in memory consolidation. Yoo et al. randomly allocated healthy adults to either a normal sleep condition, or around 35 hours of total sleep deprivation prior to memory encoding [7]. The sleep-deprived group had significantly poorer recognition memory (approx. 19% lower than sleep group) and reduced activation of the hippocampus during encoding, suggesting that the sleep deprivation affected the brain's ability to form new episodic memory.

In line with these insights, studies have revealed that memory is generally better following a similar amount of sleep and a similar amount of awakeness, suggesting that sleep plays an active role in consolidating newly learned information [5, 6]. However, sleep deprivation has been found to affect the consolidation of newly acquired information and encoding of new experiences, thus decreasing the ability of the hippocampus to support new learning [1, 5].

2.3. Neural mechanisms underlying sleep-dependent memory consolidation

There are many proposed models of the role of sleep in memory consolidation, but one of the most influential is the active systems consolidation hypothesis that proposes that memory items encoded while awake may be replayed many times during sleep and eventually transferred from an ephemeral (hippocampal) store to a permanent (cortical) store [6]. This process is thought to rely on coordinated patterns of neural activity in the part of sleep in which rapid eye movement (REM) is not occurring. Slow oscillations, sleep spindles, and hippocampal sharp-wave ripples can impart a temporal structure which allows communication between widely distributed brain areas and reactivation and consolidation of newly acquired memories [8]. Slow oscillations originating in the neocortex are believed to be involved in timing sleep spindles which synchronize with sharp-wave ripples in the hippocampus. Sharp-wave ripples play a role in the replay of recently encoded memory traces in hippocampus, and spindles play a role in synaptic plasticity of cortical networks, and they happen at the appropriate times. The coupling over time facilitates transfer of multiple memory related information from the hippocampus to the neocortex, and results in the consolidation of long-term memory representations in the neocortex over time [6, 8]. These oscillating rhythms are coordinated to repeatedly reactivate newly acquired memories and to link them more with long-term memory networks in the cortex, which aids in retaining the memory [6, 8].

A complementary explanation is called the synaptic homeostasis hypothesis, which posits that in wakefulness, synapses are generally enhanced as a person learns about the world, and that during sleep the synapses that are less important for the person's learning and information processing are selectively downscaled, freeing up resources for learning the following day. Sleep can filter out the

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"noise" or irrelevant information in synapses, while keeping behaviourally important information, thus optimising the ability of neural networks to encode and store memories.

It could therefore be predicted that sleep loss would result in poor retention of new memories, due to disrupted reactivation of newly learned memories, as well as disrupted regulation of the strength of synapses. These disruptions may result in the hippocampus not transferring information to other regions of the brain, resulting in learning difficulties and other cognitive deficits.

3. Memory and cognition

3.1. Role of memory in cognitive function

Memory is the basis of many cognitive processes and the ability to encode, store and recall information gained from experience. The capacity to integrate the new information with the old information stored in long-term memory is important for processes like reasoning, decision-making and problem-solving [9]. Memory is not a stand-alone system but works together with attention and executive functions to facilitate adaptive behaviour and complex cognition [10].

Working memory has been especially suggested to participate in higher order cognitive processes as a temporary storage space where information can be held and manipulated in real time [11, 12]. This is the system that is used for tasks like language comprehension and heavily depends on the prefrontal cortex [11]. Declarative memory, on the other hand, includes episodic and semantic memory, which allows people to consciously remember past events and information and involves the hippocampus and related areas of the cortex. The interplay of these systems enables learning to influence current action and enables the learning of new information [5, 10].

3.2. Cognitive impairments associated with memory deficits

Memory deficits are linked to other cognitive deficits. A decrease in working memory capacity results in reduced capacities for sustained attention and manipulation of information, which translates into lower reasoning and decision-making abilities. Similarly, disturbances of declarative memory impair the embedding of new experiences in old knowledge and, in turn, affect the efficiency with which learning takes place and the flexibility of cognition [10, 11].

In line with these results, Lim and Dinges (2010) performed a meta-analysis of short-term deprivation studies and concluded that sleep restriction had significant effects on a variety of cognitive measures, most notably for attention, working memory and executive functioning. The authors concluded that cognitive tasks that demand a high level of mental engagement are most sensitive to sleep deprivation, meaning that memory related processes are especially sensitive to sleep deficiency [12, 13].

These findings in combination indicate sleep deprivation interferes with memory processes (working memory and declarative memory) and therefore the higher order cognitive functioning (attention, reasoning, executive control, and decision-making). So, memory deficits are an important way that poor sleep leads to more general cognitive dysfunction.

4. Interactions between sleep, memory, and cognition

4.1. From sleep-dependent memory consolidation to cognitive function

Sleep, memory and cognition are not independent but are part of an ongoing neurobiological process where sleep-dependent memory consolidation is a prerequisite for higher order cognitive functions.

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Sleep does not mark independent processes but rather appears to facilitate the consolidation and reorganisation of newly learnt information in a coordinated manner across the hippocampus, which through a gradual process is integrated into neocortical networks [6, 8]. These representations will be more stable and abstracted and can be integrated into pre-existing knowledge structures and be used more flexibly when engaged in later cognitive processing [6]. Thus, the cognitive dysfunction of sleep loss is not only due to a reduction in alertness, but to a breakdown of the neural systems involved in consolidating new information and analysing it into long-term, cortical networks.

Memory traces formed while awake are very labile until they are consolidated during sleep (as stated in Section 2). Coordinated hippocampal replay transfers newly acquired information from transient storage in the hippocampus to distributed networks in the neocortex to consolidate and render memory resistant to interference [14]. This change is not just to save a particular experience. Sleep processes can abstract, generalise and make schemas of new information, incorporating it into previous knowledge stored in the cortex, enabling previous experiences to influence future reasoning and decision making [14].

Coordinated communication between hippocampus, neocortex, and prefrontal cortex is essential to efficient functioning of this process. The hippocampus is a rapidly encoding region that is necessary for the encoding of new experiences whilst the prefrontal cortex is implicated in executive function, such as attentional control, working memory, planning, and behaviour regulation. Cognitive performance thus relies on these areas communicating one with the other, with each area providing unique, but complementary functions [8]. Hippocampal replay enhances the representation within cortex, which then can be retrieved by prefrontal networks during complex cognitive tasks. Thus, memory consolidation gives the neural bases on which EF functions and enables stored knowledge to be flexibly used to address new situations [6, 14].

Sleep dependent synaptic plasticity additionally helps with this interaction. The synaptic homeostasis hypothesis states that during wake-promoting periods, there is a general increase in synaptic connections as new information is acquired. Sleep then enables the selective renormalisation of synapses [15] and thereby, leads to a neural efficiency with preserved behaviourally relevant connections while redundant activity is weakened. Sleep helps to balance synaptic stabilization and plasticity, so that neural networks can learn without interfering with the retention of older memories. Systems Consolidation and Synaptic Homeostasis are two complementary processes that sleep may serve to optimize long-term memory storage as well as the efficiency of cognitive processing [15].

4.2. Neural network dysfunction following sleep deprivation

The neural effects of sleep deprivation are related to the fact that continuous wakefulness affects oscillatory architecture on which sleep-dependent memory consolidation depends. A decrease in slow-wave activity leads to a decrease in temporal correlation of the cortical slow oscillations, thalamocortical sleep spindles and sharp-wave ripples in the hippocampus, resulting in a decrease in hippocampal replay and redistribution of newly encoded memories to distributed networks in the neocortex [14]. Therefore, newly learnt information is not yet well consolidated and is vulnerable to disruption and not readily accessible for later cognitive functions.

Widespread changes in functional connectivity in hippocampal-prefrontal-cortical networks accompany disruption of systems consolidation. Functional neuroimaging studies have shown that sleep deprivation decreases the activation of the hippocampus under memory encoding and also decreases functional connectivity between the hippocampus and prefrontal cortex [7]. The prefrontal cortex relies on stable representations of the cortex to control the system, and reducing the

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communication between the hippocampus and prefrontal cortex leads to a reduction in available goal-directed decision making. Thus, executive dysfunction is not a sort of prefrontal cortex dysfunction but reflects impaired access to consolidated memory [8].

Sleep deprivation also affects the synaptic plasticity, which in turn decreases the efficiency of processing information in the networks of the cortex. The synaptic homeostasis hypothesis suggests that wakefulness is accompanied by a broad excitatory synaptic potentiation that leads to an increase in metabolic demand and basal firing rates of neurons, as wakefulness proceeds [15]. Synaptic downscaling during normal sleep selectively weakens fewer active synapses while keeping behaviourally relevant connections, which have been repeatedly reactivated, stable, therefore restoring optimal balance between stability and plasticity [16]. A reduction in sleep causes this renormalisation process to not go to completion. Synaptic strength continues to build up, so that the variety of neuronal activity in the cortex is not distinguished from background signalling. This leads to a decrease in the signal to noise ratio and decreases the efficiency with which new information is encoded in these cortical networks, which could explain why learning is impaired after sleep deprivation [15].

Altogether, these results suggest that sleep deprivation's impact on cognition is a product of a continuous neurobiological pathway that connects sleep, memory and cognition. Insufficient sleep leads to impairment in hippocampus replay, disruption of hippo-cortical communication and prevents normal synaptic renormalisation, which compromises the neural architecture that is important for efficient executive processing. Thus, it is important to view cognitive dysfunction because of impaired sleep-dependent memory consolidation and not merely a by-product of fatigue [6, 8, 15].

5. Interventions

5.1. Sleep hygiene and behavioural interventions

In fact, since cognitive dysfunction after sleep deprivation is driven by impaired memory consolidation during sleep, interventions that maintain adequate sleep architecture could indirectly help to enhance cognitive function by maintaining memory formation mechanisms. The behavioural interventions facilitate the occurrence of slow-wave and REM sleep which in turn facilitates hippocampal replay, systems consolidation and synaptic plasticity, but not directly impacting cognition itself [8, 13].

Sleep hygiene is thus regarded as the first line of behaviour change interventions. Factors such as keeping regular sleep-wake times, limiting electronic devices exposure before bedtime, reducing caffeine intake before retiring to bed and providing a conducive environment for uninterrupted sleep are promoting more stable sleep architecture and hence the provision of more time for slow-wave and REM sleep to occur [13]. Maintenance of continuity of these sleep stages facilitates the oscillatory activity that is necessary for hippocampal replay, systems consolidation, and synaptic plasticity and thus decreases the risk of newly acquired memories being interfered with. Thus, sleep-behavioural interventions that promote better sleep could indirectly conserve long-term memory processes and therefore save learning and executive function [8].

5.2. Sleep recovery strategies

While the best way to avoid the effects of sleep deprivation is to prevent it, there are some neural and cognitive effects that are at least partially reversible. Recovery sleep replenishes processes that

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have been disturbed during extended periods of waking. Experimental studies show that a single or multiple nights of unrestricted sleep can enhance performance on hippocampal-dependent memory tasks and partially recover executive functioning, yet after multiple or long periods of sleep restriction, recovery is sometimes incomplete, suggesting that disruption of sleep-dependent plasticity may build up over time [1].

If there is not sufficient sleep at night, then it might be possible to nap during the day to reinforce the memory. Even brief naps (including NREM) have been shown to improve declarative memory performance and restore the ability to learn through permitting "hippocampal replay" to occur before new information is acquired [16]. These benefits aren't related to the amount of sleep, but its structure. Slow-wave sleep (SWS) nap is associated with greater improvement in hippocampus-dependent memory consistent with the role of systems consolidation in SWS through slow oscillations, sleep spindles and hippocampus sharp-wave ripples [6]. Therefore, daytime naps are beneficial for the brain not just because they reduce sleepiness, but because they offer yet another chance to consolidate memories during sleep.

New developments of sleep neuroscience have also contributed to interventions directly aimed at physiological processes of memory consolidation. Closed-loop auditory stimulation provides acoustic stimulation time-locked to endogenous slow oscillations, increases slow-wave activity and increases the coupling between slow oscillations and sleep spindles [17]. The same approach is also called targeted memory reactivation, and has been shown to selectively enhance, during slow-wave sleep, hippocampal replay and consolidation of specific memories when learning-associated sensory cues are re-presented [18]. These methods give direct experimental evidence that a selective enhancement of memory consolidation can be achieved by a manipulation of sleep oscillations, thus revealing the causal role of sleep physiology in LTM formation.

Together, all these analyses have shown that interventions that are successful go beyond simply getting more sleep. Recovery works best if it restores oscillatory activity and hippocampal-cortical communication, which are essential for sleep-dependent memory consolidation, and synaptic plasticity. As knowledge of these mechanisms grows, there is potential for interventions that directly impact sleep physiology to offer more targeted strategies for maintaining memory and cognition after sleep loss.

6. Conclusion

In conclusion, sleep plays an important role in cognition and the consolidation of memory is an important process in the brain that happens during sleep. The findings presented in this paper indicate that sleep loss leads to a disruption of hippocampal-cortical communication, systems memory consolidation and synaptic plasticity, and thus to a loss of stable memory representations essential for higher-order cognitive processes including learning, reasoning and decision making. These results suggest that sleep deprivation can have cognitive effects that are not just due to fatigue. Progress in the sleep neuroscience field might then help development of better sleep deprivation interventions to maintain memory consolidation and limit cognitive consequences of sleep loss.

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