PNAS SPECIAL FEATURE: INTRODUCTION

The functions of sleep: A cognitive neuroscience perspective

Katharine C. Simon[a], Lynn Nadel[b], and Jessica D. Payne[c,1]

This Special Feature explores the various purposes served by sleep, describing current attempts to understand how the many functions of sleep are instantiated in neural circuits and cognitive structures. Our feature reflects current experts' opinions about, and insights into, the dynamic processes of sleep. In the last few decades, technological advances have supported the updated view that sleep plays an active role in both cognition and health. However, these roles are far from understood. This collection of articles evaluates the dynamic nature of sleep, how it evolves across the lifespan, becomes a competitive arena for memory systems through the influence of the autonomic system, supports the consolidation and integration of new memories, and how lucid dreams might originate. This set of papers highlights new approaches and insights that will lay the groundwork to eventually understand the full range of functions supported by sleep.

sleep | memory | function | cognitive neuroscience

Humans spend roughly one-third of their lives sleeping, and other animals sleep even more (1, 2). Despite how much time is spent in this offline state, why we sleep remains a mystery. There are various candidate answers related to the immune system, hormonal systems, thermoregulatory systems, and basic metabolic processes, as sleep is essential for all of these bodily functions. Although the entire body benefits from sleep (3), the most immediate, detrimental, and unavoidable consequences of sleep loss impact the brain and the various cognitive functions it supports (2, 4). This insight has led some researchers to conclude that "sleep is of the brain, by the brain, and for the brain" (5). This PNAS Special Feature addresses the topic of sleep from a cognitive neuroscience perspective, one that should be of broad interest given the necessity of sleep in our lives.

One link between sleep and the brain concerns the processes by which newly acquired information is stored. The notion that sleep benefits memory dates back to Ebbinghaus (6), who pioneered the experimental study of memory by demonstrating the effect of time on forgetting. He observed that forgetting occurs rapidly in the first hours after learning but progresses more slowly over the days that follow. Ebbinghaus (6) observed that forgetting seemed to slow or perhaps stop altogether between 8.8 hours postlearning and a day later. While only 2.1% of information was forgotten across this 15.2-hour period, three times that amount was forgotten over the next 24 hours. He noted that much of this period was occupied by sleep, but 40 years passed before Jenkins and Dallenbach (7) experimentally confirmed that memory retention following sleep was superior to retention following an equivalent interval of wake. Jenkins and Dallenbach (7) explained this finding by suggesting that sleep passively protects newly forming memories from the impact of interfering information. That sleep passively shelters memories against interference seems clear, but as the papers in this Special Feature demonstrate, sleep does much more for memory. It also appears to actively strengthen and shape memories as they undergo consolidation—a time-dependent process that helps to stabilize memories in brain circuits. Since the seminal research by Ebbinghaus (6) and then, Jenkins and Dallenbach (7), dozens of studies have reported that sleep benefits consolidation of memories about our daily experiences, termed episodic memory, known to be dependent on the hippocampus (e.g., ref. 7)—especially when compared with a period of wake (refs. 7–9 have reviews). The putative mechanisms by which sleep influences memory consolidation are actively being explored (e.g., ref. 11), and the articles in this collection offer new insights into the essential nature of the sleep–memory connection.

Sleep across the Life Span

One pressing question about the sleep–memory link concerns how it manifests over one's lifetime. Spencer and Riggins (10) examined this link at the younger end of the age spectrum. They review evidence that naps in early childhood are essential for memory consolidation, presenting a fascinating new hypothesis connecting the psychological, physiological, and neurobiological changes that accompany "nap transitions" in early childhood. Transitioning from multiple bouts of sleep each day (i.e., naps) to a single bout of overnight sleep is universal in human development, but why and exactly when this transition occurs remain unknown. Spencer and Riggins (10) argue that as the hippocampal-dependent episodic memory network matures, more efficient memory storage becomes possible. This, in turn, reduces the buildup of the pressure to sleep in the brain, known as sleep homeostasis (12), eventually enabling a young child to abandon naps in favor of consolidated overnight sleep. This hypothesis has interesting implications for how sleep supports cognitive development during the early years of life.

Denis et al. (13) extend this life span analysis to include individuals from young adulthood through middle age (18 to 59 years) using a large sample of participants spanning

Author affiliations: [a]Department of Psychology, University of California, Irvine, CA 92697; [b]Psychology and Cognitive Science, University of Arizona, Tucson, AZ 85721; and [c]Department of Psychology, University of Notre Dame, Notre Dame, IN 46556

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[1]To whom correspondence may be addressed. Email: jpayne7@nd.edu.

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various backgrounds. Denis et al. (13) confirmed the importance of the sleep–memory connection across the life span, demonstrating that sleep selectively benefits memory for negative emotional information at the expense of memory for neutral information (14). This "emotional memory trade-off effect" was observed in both young and middle-aged adults, with sleep's benefit selective to negative (but not positive) emotional memories. This work begins to address a gap in our knowledge about sleep and cognition in middle-aged adults.

Sleep within the Brain and How It Is Influenced by the Body

Guthrie et al. (15) provide a bold new insight into human sleep—replicating animal research—concerning the various stages of sleep. Basically, sleep throughout the night can be subdivided into epochs of "rapid eye movement" (REM) sleep and "nonrapid eye movement" (NREM) sleep, defined as the terms suggest by the presence or absence of eye movements, respectively. These epochs alternate over the typical night's sleep, with NREM sleep being most prominent early in the night and REM sleep being most prominent later on. Guthrie et al. (15) confirm that two regions critical for memory—the hippocampus and cortex—can simultaneously be in different stages of sleep. In evaluating the sleep data from eight patients with both intracranial and scalp electrodes, the authors found that overall, the cortex and hippocampus spent more time in divergent than congruent states of sleep. Interestingly, different patterns emerge in the divergent, contrasting stages of sleep. The cortex appears to spend more time in wake and REM compared with the hippocampus, yet the amount of time spent in various phases of NREM appeared equivalent across the two brain regions. The findings of Guthrie et al. (15) have important functional implications for sleep-dependent cognition mechanisms during congruent and divergent states and are sure to invigorate the field of sleep research.

Chen, Zhang et al. (16) provide novel insights into the overlooked influence of the autonomic nervous system (ANS) on sleep-dependent memory mechanisms. The ANS shows clear physiologic shifts across wake and sleep stages and has recently been implicated in sleep-dependent cognition (17). In their review of the literature, these authors provide robust evidence for two distinct ANS–central nervous system networks, in which electrophysiologic features and changes in heart rate beats, known as heart rate variability, are interconnected and facilitate either the consolidation of episodic memory or performance gains in working memory. Their slow oscillation switch model highlights the competitive trade-off between these two functional networks for the limited resources available during NREM sleep and how either episodic memory or working memory domains could gain or lose performance benefits. Given the ANS life span–associated changes, their model provides new directions and testable hypotheses for sleep-dependent cognition during early and later life.

Mechanisms Underlying the Sleep–Memory Connection

Sleep, as we have seen, contributes to the storage and consolidation of memories, and developmental changes in memory storage needs may, in turn, have an impact on sleep. The "active systems consolidation" hypothesis (18) offers an integrative account of the role of sleep in memory, arguing that memory representations are repeatedly reactivated and reorganized across large-scale neuronal networks during sleep. The hippocampus is thought to orchestrate this process, which stabilizes some memories and transforms others. According to this view, the neocortex serves to integrate related and overlapping memory traces, yielding abstract representations that can be flexibly and efficiently used for the purposes of generalization and adaptive future forecasting (11, 19). This view of sleep dovetails with modern views of memory, which hold that medial temporal lobe (MTL) regions, including the hippocampus, are at least as important for predicting the future as they are for recollecting the past (20, 21); these regions comprise part of the default mode network (DMN), a set of brain regions active when individuals are not engaged in specific experimenter-defined tasks (22) but rather, in mental activities, such as reminiscing, future thinking, and generally constructing scenarios that help make sense of the world—all of which involve memory (23).

There remains some debate about how long the hippocampus is needed to support a fully consolidated episodic memory (24, 25), with the active systems consolidation hypothesis favoring the view that a literal "transfer" of memory from the hippocampus to the neocortex occurs during consolidation (e.g., refs. 11 and 18). According to this view, episodic memories lose their dependence on the hippocampus over time and with the intervention of sleep.

Vanasse et al. (26) tested this idea using the publicly available Natural Scenes Dataset to examine memory recognition in eight human subjects on a weekly basis over the course of a year. This study deployed high-resolution (7-tesla) functional MRI to take an unprecedented look at memory consolidation as it unfolds over time. The authors examined whether memory recognition continues to engage the hippocampus and other MTL structures over the long term or whether the memory comes instead to rely entirely on the neocortex in the course of sleep-assisted consolidation. They found that recognition memory was associated with increased MTL activity at both early and late time points, with the surviving memory traces becoming more robust in and around the hippocampus in the weeks after encoding and persisting for more than 200 days. This finding is inconsistent with aspects of the active systems consolidation framework, favoring the idea that the hippocampus remains involved in those memories that retain detailed episodic information, as suggested by multiple trace theory (27).

The systems consolidation account for the role of sleep in memory consolidation is not the only viable notion of how sleep affects the brain. According to the synaptic homeostasis hypothesis, there is a net, and unsustainable, increase in synaptic strength in brain circuits that accumulates during exposure to the events of the day (12). Sleep promotes a general downscaling of synaptic weights as an antidote. Synaptic downscaling, in this view, avoids saturation of synaptic connections and keeps the high energy costs of synaptic activity under control. Because sleep-dependent downscaling is thought to be selective and to afford relative protection to synapses recently engaged in new learning, sleep-dependent

downscaling could also promote memory consolidation by increasing signal to noise in key brain regions. To test this hypothesis, Vanasse et al. (26) examined whether the relative enhancement of some memory traces is linked to the concomitant forgetting of others. They found that maximal forgetting of learned material (images) overall was positively correlated with stronger brain activation generated by surviving memory traces, which they argue might reflect a reduction of noise in the system. Given that only postsleep recognition memory displayed this noise removal effect, the authors suggest that sleep may have renormalized synaptic weights, which in turn, produced the increased activation of surviving memory traces. While an increased memory signal alone would support the active systems model of consolidation discussed above, the observed correlation with forgetting supports the synaptic homeostasis hypothesis, whereby synaptic downscaling results in the selective preservation of some (previously active and/or especially relevant) memories but not others.

This idea may be related to the selective memory effects observed by Denis et al. (13) noted above, namely that the emotional elements of one's experience are selectively retained postsleep, even while neutral elements deteriorate. Of course, such emotional selectivity in memory could reflect active consolidation of important memories only, which underscores the idea that active systems and synaptic downscaling accounts may complement each other.

Using a rodent model, Pedrosa et al. (28) also focus on hippocampal–neocortical interactions during sleep-based memory consolidation. In the rodent literature, numerous studies have demonstrated an association between "replay" of memories in the hippocampus and several cortical areas, including the prefrontal, visual, retrosplenial, and entorhinal cortices (e.g., 29, 30, and 31). Exactly how hippocampal activity relates to this broader, cortex-wide activity is not well understood. Pedrosa et al. (28) used voltage imaging, electrocorticography, and laminarly resolved hippocampal potentials in mice to provide a wide-scale picture of spontaneous cortical activity during sleep and to examine how this activity organizes itself into functional networks. Their data-driven procedure revealed spontaneous neocortical activation signals spanning various spatial scales, which were organized in a small number of functional networks (retrosplenial cortex and medial cortical bank of the cortex, somatosensory cortex, and lateral cortex). The authors then analyzed the hippocampal CA1 layer–resolved local field potential correlates of spontaneous waves involving these three cortical networks. They found that a particular form of brain oscillatory activity, "slow gamma" (20 to 50 hertz), was strongly correlated with the retrosplenial network in particular. This, they claim, argues for a role for slow gamma in memory processing, such that spontaneous activity in the cortex acts as a "cue" for such processing, indicating that interactions between the neocortex and hippocampus are bidirectional. Importantly, the retrosplenial network most involved in this spontaneous dialogue strongly overlaps with the DMN, pointing to a potentially dynamic interchange that may help us understand the involvement of the DMN in memory (22).

Aleman-Zapata et al. (32) explored the connection between hippocampal ripples and high-frequency oscillations in the cortex during sleep-dependent consolidation after one-trial spatial learning in rats. With this hippocampal ripple–dependent task, they report cortical oscillations of two high frequencies, with each high frequency involving a distinct neural network—a prefrontal–parietal network for faster oscillations and a hippocampal–parietal network for slower oscillations. Disrupting hippocampal ripples reduced learning and diminished parietal high-frequency oscillations, suggesting that when learning is interrupted, there is less of a need for information in cortex to be consolidated.

The issue of spontaneous hippocampal–neocortical dialogue is further explored in a computational model by Singh et al. (33), who raise the question of how these brain systems are able to interact and accomplish useful learning and representational sculpting during periods with virtually no environmental input. Their proof-of-concept neural network model shows that when new information is acquired, the hippocampus can facilitate stabilization and integration during sleep by replaying the newly learned neural representations, providing the opportunity for the cortex to integrate and dissect common features of the material being learned. Their model is able to account for the fact that sleep is particularly supportive of new learning about aspects of experience that share features in categorically meaningful ways. Their model also provides insight into the dual roles of consecutive, alternating NREM and REM epochs, wherein NREM facilitates the stabilization of the newly learned representations and REM sleep reduces potential interference between old and new neocortical representations that share overlapping features. Their contribution provides insight into how consolidation mechanisms are initiated and maintained without external influence. However, as already noted, Guthrie et al. (15) demonstrated that sleep stages in the neocortex and hippocampus can diverge, so it will be important to see if and how future versions of this model could account for divergent sleep stages influencing hippocampal–cortical communication during consolidation.

Although most prior studies have linked the mechanisms of sleep and memory processing indirectly, more recent work has used specific facets of sleep to experimentally and directly alter the fate of memories. One means of doing so involves the use of a procedure called targeted memory reactivation (TMR). A seminal study in humans examined the effect of presenting olfactory cues during sleep that had been part of memory associations learned during the day. In this study, these olfactory cues, which did not wake the participants, improved memory performance (34, 35). A growing body of literature has established that presenting olfactory or auditory reminder cues during sleep, especially during slow-wave sleep, can meaningfully boost memory consolidation (36). Other effects of TMR have also been demonstrated, including studies that have tried to weaken, instead of strengthen, certain memory traces (37, 38).

Ngo and Staresina (39) take the novel step of pairing TMR with experimental augmentation of slow-wave sleep, another method of experimentally enhancing memory consolidation (40). By combining the two approaches, these authors show that delivering TMR cues during the depolarizing up states of slow oscillations both triggered strong reactivation of memory representations and led to enhanced memory consolidation as measured by improvement in performance. Ngo and Staresina (39) speculate that the relatively late increase in

reinstatement they observe in the scalp electroencephalography (EEG; at about one second) might reflect a slower hippocampal contribution to memory reactivation. However, they note that intracranial EEG recordings would be necessary to test this conjecture.

Creery et al. (41) do just that by testing five patients with depth electrodes implanted in or near the hippocampus to determine the feasibility of surgery to relieve their epilepsy. While patients slept in the hospital, EEG responses to sounds (half of which had previously been associated with spatial memories) were recorded. These sounds elicited oscillatory intracranial EEG activity increases in the theta, sigma, and gamma EEG bands, with gamma responses in particular predicting the degree of improvement in memory after sleep. Similar to the results presented by Pedrosa et al. (28), this study provides additional evidence that gamma oscillations might be especially important for sleep-based memory-processing effects.

Sleep and Dreaming

One of the most fascinating aspects of sleep is the occurrence of dreams. Simor et al. (42) provide a novel multicomponent neurocognitive framework for the onset and maintenance of what is known as lucid dreaming. They extend and build upon prior theories of dreaming suggesting that lucid dreaming is induced when top-down models of the self clash with bottom-up interoceptive pathways, generating prediction errors. They argue that when prediction error is high between current dream content and interoceptive input signaling, the body is in a state of muscle atonia, and lucid dreaming can arise. In nonlucid dreamers, the same mismatch and resultant prediction error might result in sensory input being incorporated into the dream or in an arousal from sleep. Further, top-down attentional control governs prediction error, attenuating the contrast between dream content (i.e., flying) and the true state of the body and motor system, determining whether arousal, dream inclusion, or a lucid dream state results.

Conclusion

This Special Feature provides a current, although necessarily selective, snapshot of the broad and exciting landscape of the field of sleep research and a sense of where the field is moving. The papers included here highlight the dynamic nature of sleep, tying in the role of the autonomic system and shedding light on the mechanisms influencing the sleep–memory connection and how it changes across the life span. They also address aspects of one of the most fascinating aspects of sleep—the nature of dreaming and in particular, the phenomenon known as lucid dreaming. While a full understanding of the range of functions of sleep remains elusive, this set of papers highlights aspects of sleep and sleep research that will eventually provide the detailed mechanistic picture such understanding requires.

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