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Sleep is For the Brain

Contemporary Computational Approaches in the Study of Sleep and Memory and a Novel "Temporal Scaffolding" Hypothesis

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Introduction

The facilitatory effects of sleep on memory consolidation have been well established during the past two decades in both human and animal studies. Sleep has been shown to contribute to a variety of cognitive processes, and two particular sleep stages that alternate throughout the course of a night, slow wave sleep (SWS) and rapid eye movement (REM) sleep, were frequently implicated in distinct cognitive abilities (Rasch & Born, 2013). SWS, a stage most abundant in the early parts of the night and characterized by slow oscillations in EEG activity, has been shown to contribute to the enhancement of declarative memories, ranging from improved recall of paired associates to explicit insight into hidden rules governing recently learned material (e.g., Marshall, Helgadóttir, Mölle, & Born, 2006; Plihal & Born, 1997; Wagner, Gais, Haider, Verleger, & Born, 2004). REM, a sleep stage more pronounced in the latter parts of the night and often referred to as "paradoxical sleep" due to its EEG activity resembling wake time (and also being the period when most dreams appear), has been linked to enhancement of procedural memories (e.g., motor learning; Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994; Maquet et al., Maquet, Laureys, Peigneux, Fuchs, Petiau, Phillips and Meulemans, 2000; Plihal & Born, 1997), as well as to creative thinking in tasks involving linguistic materials (Cai, Mednick, Harrison, Kanady, & Mednick, 2009; Walker, Liston, Hobson, & Stickgold, 2002). Moreover, the typical cycling between SWS and REM along the course of a night was demonstrated to have a functional role in procedural learning as well (Stickgold, James, & Hobson, 2000).

While the experimental data on the effects of sleep on cognition have been rapidly accumulating, questions regarding the biological mechanisms underlying these effects from a computational perspective, as well as the differential role of specific sleep stages, are yet to receive definitive answers. Nevertheless, two theoretical approaches, sometimes seen as competitive, stand out as the prevalent contemporary hypotheses in the field. One, the "memory reactivation" approach (sometimes also referred to as the "active system consolidation theory"; Rasch & Born, 2013), highlights the role of reactivation of recently acquired experiences during offline periods in memory consolidation. The other, the "sleep homeostasis hypothesis" (Tononi & Cirelli, 2006), focuses on the role of renormalization of synaptic connectivity during sleep and its resulting memory benefits. While both approaches are based on physiological findings in human and animal sleep, each offers distinct understanding of how cognitive performance is benefited as a result.

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In the following, I will review the core computational principles of the two approaches, as well as some other, related models, and their respective accounts of sleep-dependent cognitive facilitation. I will then discuss the relations between the models and whether they are best seen as contradictory, or, in fact, complementary. Finally, I will argue that some complex cognitive faculties, such as sleep-dependent facilitation of insight, have not been sufficiently addressed by previous models and will introduce a novel reactivation-based hypothesis that offers an explanation for such effects.

Contemporary Computational Models of Sleep and Cognition

The Memory Reactivation Theory

The memory reactivation theory asserts that memories that are encoded during waking activity go through a secondary process of consolidation during resting periods, including sleep. As part of the consolidation processes, these memories are reactivated, strengthened, reorganized, and integrated into the general knowledge structure. According to the theory, neural networks in the brain face a major computational challenge during learning—*catastrophic interference* (McCloskey & Cohen, 1989). Reactivation of memory during offline periods, in turn, offers a solution to this interference.

In the most seminal of the reactivation models (McClelland, McNaughton, & O'Reilly, 1995), catastrophic interference is exemplified by the distinct challenge the brain is facing when needing to balance between the requirement to learn new things quickly and the need to extract common structure from distinct experiences and store them in an organized way. These two requirements are often in conflict. For example, when parking the car every day before going to work, one needs to remember the location of the car at that particular day and avoid conflating this information with memories of the car's location at previous days. Thus, this "episodic" memory should be encoded in a way that is distinct from similar episodic memories. In computational terms, the representations of the new memory and the previous, somewhat similar, memories should be orthogonal (i.e., without correlations). On the other hand, when one needs to learn where the best places to park the car are, what times the spot right below the office is free, and when is parking on Bleecker Street not allowed, memories from all the relevant parking experiences should be compared and their commonalities extracted. In other words, these memories should be encoded in a "semantic" way that reflects the similar and dissimilar aspects of the experiences they represent rather than being stored in isolation. In computational terms, the representations should be correlated to reflect the inherent structure of the environment. These two contradicting requirements pose a problem regarding how to best encode new memories.

The common view in the field holds that the brain solves this dilemma by applying a complementary learning systems approach (McClelland et al., McClelland, McNaughton and O'Reilly, 1995). One system, residing in the medial temporal lobe (MTL) and particularly the hippocampus, is in charge of storing new experiences through sparse coding (i.e., encoding representations based on the activity of a relatively small number of neurons). Thus, different neurons become responsible for different memories, and interference between them is minimized. As a result, memories of where I parked my car today and where I parked it yesterday remain distinct, despite their inherent similarity. Conversely, the other system, residing in the neocortex (especially the prefrontal cortex), is in charge of storing memories in a way that reflects any inherent structure within the environment that yielded them. In that system, for example, the memory representations of the two times I received a parking ticket in Bleecker Street would be correlated to reflect that at both times, it was a Tuesday afternoon. This structure allows the extraction of generalities and rule learning

(i.e., "never park the car on Bleecker Street on a Tuesday afternoon").

The problem with catastrophic interference becomes apparent when considering what each system requires to encode its characteristic representations. Encoding distinct episodic memories requires "one-shot learning": strong imprinting of the experience after a single exposure. This, in turn, requires relatively large synaptic changes within the network. In contrast, encoding a new experience such that it is integrated into a previously stored semantic structure requires slow learning, in which the new experience is gradually presented to the semantic network interleaved with presentations of previously learned experiences. Only such slow learning allows the network to extract the structure governing these experiences. If, instead, the new experience is imprinted into the semantic network through strong modifications of synaptic connections, or, alternatively, presented repeatedly without interleaving it with former experiences, it will run over previously stored memories, rendering them inaccessible. In other words, a catastrophic interference will emerge.

According to the complementary learning systems approach, the solution to this computational problem is to separate learning into two stages. In the first, the episodic system within the MTL quickly encodes new experiences during active wake. In the second, those new experiences, already encoded in the MTL, are incrementally presented to the neocortical semantic system, interleaved with previously encoded experiences. This slow process does not depend on exposure to new information in the environment and as such can occur during offline periods. According to the reactivation theory, such a semantization process is what happens during rest and sleep (Stickgold, 2009). Specifically, the theory identifies SWS as especially prominent in this transfer of information, given the vast biological evidence of a hippocampal–cortical dialogue during that phase (Buzsaki, 1989; Hasselmo, 1999).

Other models highlight that catastrophic interference could also occur when the statistics of the environment changes (Norman, Newman, & Perotte, 2005; Káli & Dayan, 2004). For example, imagine that for a period of a few months, the hours and days during which parking on Bleecker Street is prohibited change from Tuesday afternoons to Wednesday mornings. While the brain slowly picks up these new statistics, the old ones, which are no longer reinforced, will fade away. When the original environment returns into play (e.g., parking rules in Bleecker Street return to normal), the originally formed memories will no longer be accessible. The solution to this kind of catastrophic interference, however, remains similar: reactivation of the old memories in parallel to the new ones during offline periods, thus preventing them from decaying in face of the environmental change. This reactivation, according to the models, could either occur as part of a hippocampus–cortical dialogue during SWS, or take place during REM within the neocortex itself (Norman et al., Norman, Newman and Perotte, 2005).

Different components of the memory reactivation theory are supported by evidence. Most strongly supported is the linkage between reactivation in the hippocampus during SWS and neocortical learning. First, SWS has been associated with improved performance in a range of declarative memory tasks in humans (e.g., Peigneux et al., 2004; Plihal & Born, 1997; Yordanova et al., 2008). Second, and more crucially, single-cell recordings from the rodent hippocampus during sleep have established that sequential experiences from previous waking periods are replayed in a compressed timescale (about 10–20 times faster than the original wake experience time (Rasch & Born, 2013) during the first hour of SWS, supplying basic biological evidence for the reactivation theory (e.g., Wilson & McNaughton, 1994). Third, this compressed memory replay takes place during specifically defined events called "sharp-wave ripples": 100-ms-long increases in the oscillation frequency of local field potentials in the hippocampus. Sharp-wave ripples, in turn, have been shown to have temporal correlations with short bursts

of cortical oscillations termed "sleep spindles" (Ji & Wilson, 2007; Siapas & Wilson, 1998), substantiating the idea of a memory-related hippocampal–cortical dialogue during SWS. Finally, memory replay, sharp-wave ripples, and sleep spindles have been directly and indirectly associated with performance on memory tasks in rodents and humans (Axmacher, Elger, & Fell, 2008; Clemens et al., 2007; Gais, Mölle, Helms, & Born, 2002; Girardeau, Benchenane, Wiener, Buzsáki, & Zugaro, 2009; Marshall et al., 2006; Pfeiffer & Foster, 2013).

To a lesser degree, there is also evidence for a role of replay in REM sleep. First, imaging data in humans have shown that cortical regions that are activated during learning of a visuomotor task are reactivated during REM (Maquet et al., 2000). This effect corresponds to a number of studies associating REM sleep with the facilitation of procedural memories in humans (Plihal & Born, 1997) and animals (Pearlman & Becker, 1974). Second, there is some evidence from single-cell recordings of sequential replay in the rodent hippocampus during REM (Louie & Wilson, 2001).

The reactivation theory, however, is not without its limitations when attempting to account for sleep-related cognitive benefits. First, although it suggests that reactivation can serve as a mechanism for detecting structure within learned stimuli, it has predominantly been demonstrated in relation to simple stabilization of memory. Specific simulations of behavioral results showing facilitation of processes such as rule learning, insight, and creativity are scarce (but see Kumaran & McClelland, 2012). Moreover, structure governing newly learned stimuli is often detected during active wake as well, possibly within the MTL itself (Gluck & Myers, 1993). It is therefore not perfectly clear what sleep adds to these processes. Finally, the reactivation theory does not give a definitive answer as to the differential roles of REM and SWS. First, why is there more than one sleep stage to begin with? And second, how do the unique physiological properties of each stage relate to their function role in memory?

The Synaptic Homeostasis Hypothesis

The synaptic homeostasis hypothesis (Tononi & Cirelli, 2006), rather than addressing sleep-related facilitation of cognitive processes directly, aims at answering an even more ambitious question: why do we sleep at all? The answer, according to this theory, is that "sleep is the price we pay for plasticity" (Tononi & Cirelli, 2014). It asserts that learning during wake results in a net increase of synaptic strength in the brain. This increase is problematic: First, since synaptic connectivity cannot increase ad infinitum, it limits the ability to learn new things once reaching values close to ceiling. Second, it is energetically costly, and possibly toxic, for neurons to constantly secrete high amounts of synaptic neurotransmitters. Third, when networks continually operate within a regime of strong synaptic connectivity, the neurons tend to synchronize their activity, reducing the selectivity of their responses to inputs (Olcese, Esser, & Tononi, 2010). According to the theory, the solution to all of these challenges is to regain synaptic homeostasis by decreasing the net strength of synaptic connections during offline periods when active learning of the environment is minimized. This "renormalization" (sometimes also referred to as "downscaling," "down-regulation," or "down-selection") happens during, and is driven by, SWS, and has a selective nature: Rather than being reduced indiscriminately, the reduction is a function of the synaptic strength before sleep. In some versions of the model, synapses decrease proportionally to their original strength and are eliminated if they fall below a certain threshold (Hill, Tononi, & Ghilardi, 2008). In other versions, stronger synapses are protected from decrease compared to weaker ones (Nere, Hashmi, Cirelli, & Tononi, 2013). Regardless of the particular mechanism of reduction, all models lead to a similar outcome: An increase in the signal to noise ratio of stored memories. Thus, while intended to solve a biological stability issue, this process can have significant computational consequences as well, which, in turn,

have the potential to facilitate a variety of cognitive processes. Nere and colleagues (2013) have demonstrated this point by showing how learning in a neural network model leads to the creation of strong, desired connections between neurons, representing true associations between stimuli, alongside weaker connections that reflect spurious associations (resulting from noise or errors during encoding). These weak connections can lead to below-par performance when testing the quality of learning. For example, in sequence learning, where each learned segment is associated with the next item in the chain of events (e.g., learning a sequence of turns when driving from home to work), spurious associations may lead to eliciting the wrong segment in the sequence during task performance; in paired-associates learning, the wrong association may be recalled; and in gist learning, where only the commonalities among several learned examples should be remembered rather than each example on its own (e.g., recalling that on Tuesday afternoons parking in Bleecker Street is not allowed, rather than recalling the fact that it was raining on a particular day I got a ticket), the unnecessary details of each example may overtake the commonalities, leading to a failure to recognize the gist. In all of these examples, preserving the strong synaptic connections while degrading—or eliminating—the weak ones, as is assumed to occur during SWS, can improve performance.

The basic principle suggested by the synaptic homeostasis hypothesis, namely, a net increase in synaptic strength after wake and a decrease after sleep, is supported by a variety of biological findings from different animal species. In *Drosophila*, synapse size and number, as well as the levels of proteins related to synaptic transmission, increase and decrease with wake and sleep, respectively. Miniature excitatory postsynaptic currents in the rodent frontal cortex, indicative of synaptic efficacy, increase after wake and reduce after sleep (Liu, Faraguna, Cirelli, Tononi, & Gao, 2010). A similar pattern was demonstrated with synaptic receptors in the rat frontal cortex, as well as with the slope of the frontal cortex response to electric stimulation (a known indicator of excitability), the latter also being correlated to the degree of slow wave activity during sleep (Vyazovskiy, Cirelli, Pfister-Genskow, Faraguna, & Tononi, 2008). Similar results were obtained with the slope of response to transcranial magnetic stimulation (TMS) in humans.

Some indirect evidence also supports the relations between sleep-dependent synaptic renormalization and improvement in cognitive tasks. Experimental and computational findings suggest that learning during wake leads to local increases in slow wave activity in the same brain regions responsible for that learning, as well as to the expression of plasticity-related genes. This slow wave activity then decreases following SWS in proportion to the pre-sleep increase (Huber, Ghilardi, Massimini, & Tononi, 2004; Huber, Tononi, & Cirelli, 2007; Olcese et al., 2010). It therefore stands to reason that SWS-dependent overnight gains in learning are at least partly the result of the synaptic decrease taking place during SWS (Tononi & Cirelli, 2014).

Nevertheless, the synaptic homeostasis hypothesis, too, is limited in its ability to explain cognitive benefits following sleep. First, unlike the reactivation theory, it does not initiate from a fundamental computational principle (such as the need to avoid catastrophic interference) but, rather, from a biomechanical one. Therefore, any cognitive facilitation predicted by the theory is, to some degree, incidental. Second, unlike the evidence for a net decrease in overall synaptic strength following sleep, evidence that synapses decay as a function of their original strength is scarce (Frank, 2012). Third, and quite similar to the reactivation theory, the function of REM sleep and its effects on cognition remains largely uncharted territory (but see Tononi & Cirelli, 2014, for some initial suggestions).

Common Principles in Current and Past Models

Both the memory reactivation and the synaptic homeostasis models share similarities

with earlier semi-computational models of sleep and cognition. Crick and Mitchison (1983) suggested that the function of sleep—and specifically, REM sleep—is to allow spurious, parasitic memories, which do not reflect any actual experiences but are unavoidably created during the encoding of real memories, to be removed through "reverse learning." This resembles the synaptic homeostasis hypothesis, in which a general synaptic down-regulation mechanism yields memory benefits by improving the signal to noise ratio in the network. Conversely, in the sequential hypothesis of sleep (Giuditta et al., 1995), SWS and REM are suggested to act successively to, first, dispose of irrelevant, nonadaptive memories (during SWS) and then reorganize and integrate the remaining memories within the general knowledge structure (during REM). This theory shares properties with both the memory reactivation and synaptic homeostasis theories, with each mechanism assigned to a different sleep stage in a functional order (see also Walker & Stickgold, 2010).

Contrasting the memory reactivation and synaptic homeostasis models directly, apparent contradictions seem to arise. One model suggests that novel neuronal associations are formed during sleep, particularly SWS, whereas the other highlights the elimination of such associations during SWS; one advocates a systems approach to sleep in which slow waves orchestrate different brain regions to act in concert and allow the transfer of memories from one region to another, whereas the other emphasizes the ability of local circuitry to engage in slow wave activity and thus reduce its synaptic overload independently of other neural circuits in the brain; and while the evidence for memory replay in the hippocampus is at the core of the memory reactivation model, it is viewed by the synaptic homeostasis approach as a rather trivial indication of the fact that some synaptic connections were formed more recently than others, rendering the involved neurons more prone to repeating the same activation during subsequent sleep (Olcese et al., 2010).

The two approaches, however, are only contradictory as far as they are taken to extreme. Acknowledging that potentiation and depression of synapses during sleep can occur in parallel may lead to the conclusion that, in fact, they are complementary. As suggested by Lewis and Durrant (2011), both replay and downscaling may take place during SWS. Memory replay could allow the transfer of sparse hippocampal memories to the cortex where their commonalities are identified, whereas downscaling, by reducing the strength of all cortical connections, could lead to the elimination of connections that support the idiosyncratic attributes of each memory. This two-phase process ensures that only the gist of common experiences (or "cognitive schemata" in the words of the authors) remains vivid in the network. Indeed, such cyclic alternations between potentiation and depression were previously suggested as characterizing general sleep-dependent consolidation processes within a pure reactivation model (Norman et al., 2005). In other words, from a computational perspective, there is nothing in the memory reactivation model that forbids depression of synapses, and, as a result, an increase in signal-to-noise ratio similar to the one suggested by the synaptic homeostasis hypothesis. The conclusion is clear: the mechanisms allowing memory consolidation through reactivation encompass those of the synaptic homeostasis theory. The theories may differ on the biological constraints speculated to take place, but they do not adhere to completely unrelated learning mechanisms.

Going Beyond Modeling of Simple Sleep Effects on Memory

One common feature of contemporary computational models of sleep is their focus on basic consolidation processes. The influence of sleep on other, more unique types of learning is not explained in detail. An example of such process is "insight" learning: the sudden realization of hidden patterns within encoded

stimuli, patterns that, once identified, significantly facilitate performance in relevant tasks. Insight learning was shown to improve following sleep compared to an equivalent period of wake. Evidence for this effect comes from experiments using paradigms such as the Number Reduction Task (NRT) and the Serial Reaction Time Task (SRTT).

In the NRT, subjects perform computations on a series of digit pairs in succession (Fig. 18.1, left; see details in Rose, Haider, Weiller, & Büchel, 2002). For each pair, they need to produce a third digit based on some simple rule. Throughout each trial, subjects produce a total of seven digits one after the other by continually employing the rule, with the final digit considered as the ultimate answer for that trial. Unrevealed to the subjects, there is a hidden regularity that determines that final response. If subjects recognize the regularity, they can produce the answer as soon as they compute the second response in the sequence, allowing them to skip the rest of the successive computation.

In the SRTT (Fig. 18.1, right), subjects are exposed to a series of successive cues appearing in one of several possible locations. They are asked to respond to each cue as quickly as possible by pressing a corresponding button. Unknown to the subjects, a hidden regularity governs the order of the cues (e.g., two successive locations probabilistically predict the location of the next; Fischer, Drosopoulos, Tsen, & Born, 2006). Subjects who recognize the regularity can predict where the next cue will appear and reduce their reaction time considerably.

Sleep was found to significantly increase the probability of discovering the hidden regularity in NRT compared to similar time in wake (Wagner et al., 2004). Further, when comparing sleep rich with SWS compared to sleep with little SWS ("split night design"; see Plihal & Born, 1997) effects were found to occur only following sleep rich in SWS and to correlate with markers of sleep spindles (Yordanova et al., 2008; Yordanova, Kolev, Wagner, Born, & Verleger, 2012). These findings, coupled with functional imaging studies showing that performance in the NRT involves activation of the medial temporal lobe, including the hippocampus (Rose et al., 2002) strongly suggest that it is SWS-related processes in the hippocampus that facilitate the sleep-induced discovery of the hidden regularity. Similar findings were found

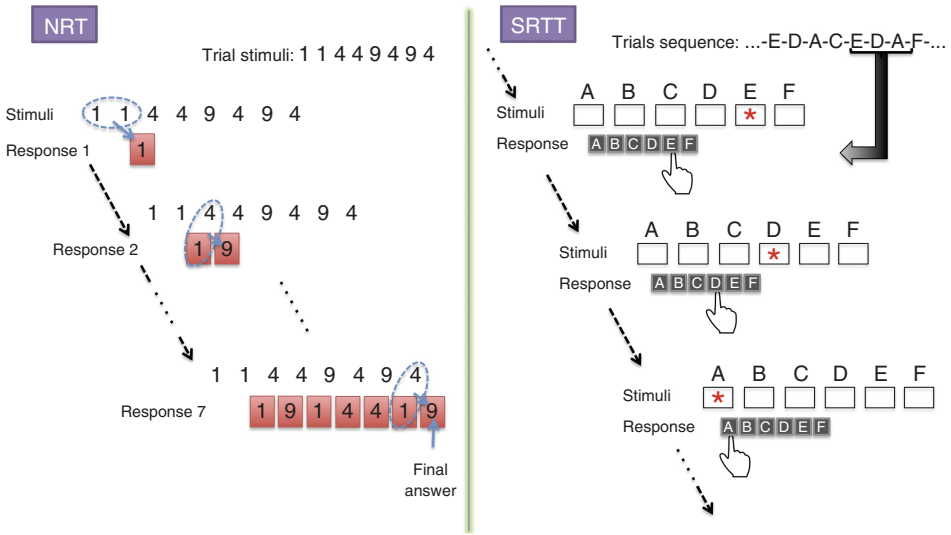

Figure 18.1 Illustrations of the Number Reduction Task (NRT) and Serial Reaction Time Task (SRTT).

using the SRTT paradigm (Fischer et al., 2006; Wilhelm et al., 2013).

Can Current Models Account for Sleep-dependent Insight?

Current models deal with evidence for sleep-related insight in vague terms. The memory reactivation approach explains such results by assuming that representations of the stimuli, encoded as isolated episodic memories, are transferred to the neocortex during sleep to allow the extraction of their internal hidden structure. However, stimuli structure can generally be extracted during wake as well. In the NRT, it was shown that some implicit knowledge of the hidden pattern, reflected by a reduction in response time to the last digit in each sequence, often exists before sleep (Wagner et al., 2004). It is therefore not clear why sleep, in contrast to wake, should provide a unique opportunity to enhance explicit detection of the hidden regularities. Conversely, the synaptic homeostasis theory, due to its complete dependence on synaptic down-regulation as the sleep-dependent memory enhancement mechanism, asserts that detection of the hidden regularities is already achieved during wake, but is blurred by spurious associations that need to be degraded to allow the crucial associations to fully manifest (Tononi & Cirelli, 2014). However, this explanation is no less problematic: first, since the hidden relations remain implicit before sleep, it stands to reason that they are only weakly represented by the synaptic connections following learning. In that case, why should their fate be any different than the spurious connections that are being eliminated through the renormalization process? Second, results in the NRT experiment indicate that the insight achieved following sleep is based on different mechanisms than the ones active during wake. Specifically, the reduction in response times to the predictable digits, achieved before sleep, was not correlated with the probability of having insight into the hidden structure following sleep (Wagner et al., 2004). This finding directly questions the core assertion of the synaptic homeostasis account regarding insight learning.

The Temporal Scaffolding Hypothesis—a Possible Solution?

One novel approach to explain sleep-inspired insight from a memory reactivation perspective can arise from the particular biological characteristics of memory replay—specifically, its time-compressed nature. This approach is based on an important observation: the hidden rule in tasks that yield insight following sleep (such as the NRT and SRTT) is not a general rule *per se*, but, rather, a *temporal* rule. That is, the nature of the hidden regularity embedded in these tasks is of the form "occurrence of event x predicts the occurrence of a future event y," with event y typically arising several seconds later, not necessarily in succession (e.g., the second response in the NRT predicts the seventh response). The significance of this observation becomes clear when considering the common view of how the brain picks up unexpected regularities in the environment. It is believed that such regularities are encoded in the hippocampus using Hebbian mechanisms (Gluck & Myers, 1993), which neurally associate representations of events that happen in close temporal proximity ("Neurons that fire together, wire together"; Shatz, 1992). However, the flipside of such mechanism is that regularities spanning longer periods of time than the typical Hebbian timescales (50–200 ms; August & Levy, 1999) should be difficult to detect, especially given that subjects are not instructed to look for regularities and thus do not deliberately attempt to keep representations active in working memory while attending to the stimuli.

The solution to this challenge could be the compressed nature of memory replay. Assuming those sequences are encoded in the hippocampus and then replayed during SWS in an accelerated manner, disparate representations may be brought "together," within Hebbian timescales, and become associated. Consequently, temporal regularities

will be picked up by Hebbian mechanisms during sleep much like stationary regularities are detected during wake. Upon awakening, those associations could be used as "scaffolding" to permit the extraction of the hidden rule governing the stimuli and result in huge "insight-like" performance improvements. Fig. 18.2 illustrates this for the NRT task: before sleep, sequential sensory experiences are encoded in the hippocampus. During SWS, these sequences are replayed in a compressed manner, allowing associations between temporally disparate segments in the hippocampus and consequently detection of previously ignored temporal correlations by the prefrontal cortex (e.g., response 2 and 7 are always the same). The following day, those temporal correlations are utilized to predict future events (possibly through wake-replay in the hippocampus; see Pfeiffer & Foster, 2013).

The "temporal scaffolding" hypothesis goes beyond the general framework of the memory reactivation theory in several ways: first, it identifies what type of patterns are more likely to yield insightful learning, namely, patterns that are based on a temporal structure; second, it explains why these patterns would not be easily recognized during wake and why their learning during sleep could manifest itself as sudden insight; and third, it suggests a specific role for the compressed timescale characterizing memory replay, a phenomenon that has so far remained largely unaccounted for (Abel, Havekes, Saletin, & Walker, 2013).

Conclusion

While it is now clear that sleep affects cognitive processing in a variety of ways, the mechanisms supporting these effects are less understood. Contemporary computational models suggest several biologically plausible processes, ranging from sleep-dependent

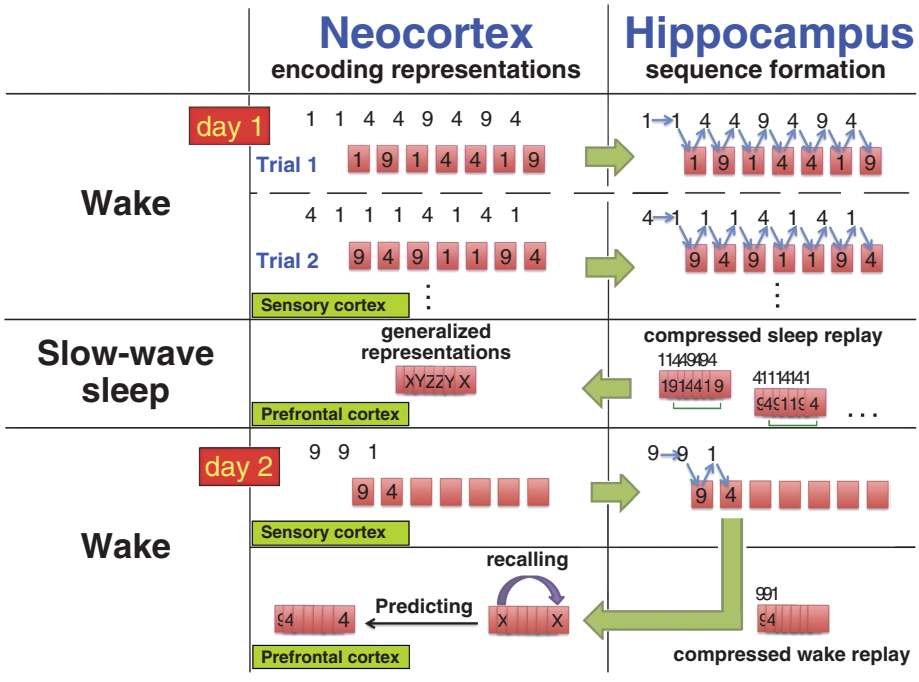

Figure 18.2 Illustration of how the temporal scaffolding mechanism accounts for sleep-dependent insight in the Number Reduction Task.

memory reactivation that allows learning without running into catastrophic interference, to sleep-dependent synaptic renormalization that elevates signal-to-noise ratio when accessing stored memories. However, these models, in their present form, are limited. They are generally centered on SWS rather than the whole sleep cycle, and they mostly focus on basic memory stabilization processes, largely neglecting the mechanisms that allow sleep to facilitate more complex cognitive processes. Future advancements in computational modeling of sleep and cognition will need to address these unaccounted phenomena. One possible step forward, the temporal scaffolding hypothesis, has been presented.

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