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Updating Internal Cognitive Models during Sleep

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Review of Lutz et al.

It is well known that a night of good sleep reinforces various cognitive functions, including memory processing (for review, see Lim and Dinges, 2010). Specifically, the consolidation of newly acquired memories is thought to benefit from sleep (Rasch and Born, 2013), based on studies showing improved performance (e.g., memory enhancement or reduced forgetting, depending on the task) after sleep than after a similar time spent awake; Chambers, 2017).

Although the effect of sleep on the consolidation (i.e., the process of transforming newly acquired information into long-term memories) of explicit, hippocampal-dependent, declarative memory seems to be widely supported (for review, see Rasch and Born, 2013), the impact of sleep on more complex functions seems to be less clear. For example, studies testing the effect of sleep on tasks requiring a constant updating of information in working memory, such as rapid serial visual presentation tasks and sequence learning, have provided conflicting results (Fischer et al., 2006; Nemeth et al., 2010; Durrant et al., 2011; Cellini et al., 2015). Nevertheless, accumulating evidence suggests that sleep is an essential period for extracting the overarching rules underlying a set of new information (i.e., the gist) and to integrate them into existing knowledge, leading to an update of existing schemas (Stickgold and Walker, 2013; Lewis et al., 2018).

Grounded on this literature, a recent study published in *The Journal of Neuroscience* (Lutz et al., 2018) investigated the effects of overnight sleep on the formation, consolidation, and abstraction of an internal cognitive model (i.e., a schema representing upcoming stimuli), using a predictive coding approach. Predictive coding theories hypothesize that information processing is organized hierarchically (Rauss and Pourtois, 2013). That is, we would use our learned models to predict upcoming congruent stimuli (top-down processing), whereas we would use the stimuli themselves, when incongruent with our models, to modify our predictions and update our models (bottom-up processing; Rao and Ballard, 1999). In other words, model-congruent stimuli would be processed in a top-down modality, whereas model-incongruent stimuli would be processes in a bottom-up one.

In the study of Lutz et al. (2018), subjects were tested before and after a retention period consisting of either a night of undisturbed sleep or a period of wake consisting of controlled, nonstrenuous, and nonlearning routine activities (between-group design: sleep vs wake). To generate internal models, participants were trained on a 12-item implicit deterministic sequence of visual stimuli. Stimuli were grayscale Gabor gratings sequentially appearing at six peripheral locations on a computer screen, tilted either at an angle of 45° or 135° from a central fixation point. Participants had to indicate the position of each stimulus by pressing the appropriate button on a keyboard. Thirty minutes after the training phase, participants completed the preretention test. In the test phase sequences, stimuli deviating from the original learned sequence were introduced. Specifically, in the test phase, sequences contained standard (congruent with the original sequence), deviant (incongruent from the original sequence), and "follower standard" stimuli (i.e., a standard stimulus that comes right after a deviant one which is supposed to elicit a behavior more likely similar to deviant than standard stimuli). The same sequences were then retested after the retention period, consisting of either sleep or wake. Importantly, to test the effects of sleep on the abstraction of internal models and their use across different temporal contexts, the authors manipulated the response-to-stimulus intervals (i.e., the delay between when a subject made a response and when the next stimulus was presented) between preretention and postretention assessment (e.g., long response-to-stimulus intervals in prephase and short response-to-stimulus intervals in postphase).

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The authors reasoned that if sleep supports the consolidation of an internal implicit sequence model, then subjects tested after a period of sleep should show increased error rates for deviant stimuli, compared with subjects tested after wakefulness, because their predictive model is more consolidated and their sequence learning is stronger. In other words, if the sequence is well consolidated in an internal model, participants should make an error whenever the present sequence deviates from the standard. Moreover, because a consolidated predictive model should allow one to restore behavior as soon as the environment again behaves as expected and produce appropriate motor responses, a more consolidated internal sequence model should also be reflected in reduced error rates for follower standard stimuli Lutz et al. (2018).

Consistent with these hypotheses, in the postretention assessment, the sleep group showed higher error rates for deviant stimuli than the awake group. Moreover, the sleep group showed a greater increase in the prediction strength index (i.e., error rates for deviant stimuli minus error rates for follower-standard stimuli) from preretention to postretention compared with the awake group. Error rates for follower-standard stimuli were comparable to standard stimuli in the postsleep group, whereas in the awake group, error rates for follower-standard items were greater than for standard stimuli. Finally, when response-to-stimulus intervals varied from preretention to postretention assessment, the prediction strength was increased after sleep but not after wake, reflecting the positive effect of sleep on the abstraction of internal models that can be used in different temporal contexts.

This study showed that the sleeping brain is able to update internal models based on information acquired during wakefulness to better predict the external situations during the next waking period. However, how the sleeping brain updates internal models remains unclear. According to Honey et al. (2018), internal models are shaped by a continuous switching between externally and internally biased processing modes. In the externally biased mode, input from the environment shapes the ongoing neural activity in a bottom-up fashion, whereas in the internally biased mode neural activity reflects a top-down process that guides the perception and prediction of the external world. In this framework, the wake–sleep alternation is considered one of the switching situations between internal and external modes. Moreover, even within sleep, Honey et al. (2018) propose another switching mechanism: during non-rapid eye movement sleep (NREM), the sleeping brain processes external-like input via hippocampal replay (Ólafsdóttir et al., 2018), whereas during rapid eye movement sleep (REM), which is characterized by an increased long-range corticocortical effective connectivity (Massimini et al., 2010) and by a high cholinergic tone (Hasselmo and McGaughy, 2004), the brain switches to an internally biased mode, promoting memory integration and model updating. Thus, this within-sleep switching mechanism may promote the consolidation of newly acquired external information during NREM and their integration into preexisting models during REM sleep. These models are then used during wakefulness to interact with the external world and can be updated during the following sleeping period.

The hypothesis that internal model updating mainly (but not exclusively) occurs during REM sleep has been also suggested by theoretical work (Llewellyn, 2015). In this work, Llewellyn (2015) also proposed a distinction between predictive and prospective coding. Predictive coding refers to the anticipation of upcoming input during wake based on a specific ongoing event, whereas prospective coding creates off-line probabilistic patterns (i.e., schema of plausible sequences of events created during sleep or resting periods) based on past events. According to Llewellyn (2015), during REM the sleeping brain scans memories to find meaningful regularities between events and uses these regularities to create associations between the events. These associations generate prospective codes that can be used during wakefulness as a predictive as well as a perceptual/attentional codes to prepare and adapt our behaviors to constantly changing external events.

These theoretical models concerning how sleep influences the updating of internal models fit well with the findings by Lutz et al. (2018). However, Lutz et al. (2018) monitored sleep between testing sessions via actigraphy, which cannot provide information about sleep architecture (e.g., the time spent in NREM and REM) or on the neural dynamics during sleep. Future studies, building on the work of Lutz et al. (2018) should test the specific roles of REM sleep and neural activity during sleep in influencing prospective/predictive coding.

In summary, the study by Lutz et al. (2018) provides evidence for a role of sleep in reinforcing the consolidation and abstraction of an internal sequence model. Which specific sleep stages facilitate these processes is yet to be clarified. Traditionally, REM sleep has been recognized as a key physiological state supporting the consolidation of implicit memories (Whitehurst et al., 2016). However, it is likely that both NREM and REM sleep interact to consolidate the regularities present in the environment and to use this information to update internal cognitive models. This idea prompts several questions. What is the impact of sleep disruption on the updating of internal models? In particular, what are the consequences for clinical populations characterized by abnormal REM sleep, such as narcolepsy or depression (Pillai et al., 2011)? Moreover, given that the quantity of NREM and REM sleep varies across the human life span (Ohayon et al., 2004), what are the consequences of age-dependent sleep changes on the updating of internal models? Further studies, combining behavioral, neurophysiological, and computational approaches, should address these questions to shed light on how our brains create and modify internal cognitive models.

References

Cellini N, Goodbourn PT, McDevitt EA, Martini P, Holcombe AO, Mednick SC (2015) Sleep after practice reduces the attentional blink. Atten Percept Psychophys 77:1945–1954.

Chambers AM (2017) The role of sleep in cognitive processing: focusing on memory consolidation. Wiley Interdiscip Rev Cogn Sci 8:e1433.

Durrant SJ, Taylor C, Cairney S, Lewis PA (2011) Sleep-dependent consolidation of statistical learning. Neuropsychologia 49:1322–1331.

Fischer S, Drosopoulos S, Tsen J, Born J (2006) Implicit learning–explicit knowing: a role for sleep in memory system interaction. J Cogn Neurosci 18:311–319.

Hasselmo ME, McGaughy J (2004) High acetylcholine levels set circuit dynamics for attention and encoding and low acetylcholine levels set dynamics for consolidation. Prog Brain Res 145:207–231.

Honey CJ, Newman EL, Schapiro AC (2018) Switching between internal and external modes: a multiscale learning principle. Netw Neurosci 1:339–356.

Lewis PA, Knoblich G, Poe G (2018) How memory replay in sleep boosts creative problem-solving. Trends Cogn Sci 22:491–503.

Lim J, Dinges DF (2010) A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. Psychol Bull 136:375–389.

Llewellyn S (2015) Dream to predict? REM dreaming as prospective coding. Front Psychol 6:1961.

Lutz ND, Wolf I, Hübner S, Born J, Rauss K

(2018) Sleep strengthens predictive sequence coding. J Neurosci 38:8989–9000.

Massimini M, Ferrarelli F, Murphy M, Huber R, Riedner B, Casarotto S, Tononi G (2010) Cortical reactivity and effective connectivity during REM sleep in humans. Cogn Neurosci 1:176–183.

Nemeth D, Janacsek K, Londe Z, Ullman MT, Howard DV, Howard JH Jr (2010) Sleep has no critical role in implicit motor sequence learning in young and old adults. Exp Brain Res 201:351–358.

Ohayon MM, Carskadon MA, Guilleminault C, Vitiello MV (2004) Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep 27:1255–1273.

Ólafsdóttir HF, Bush D, Barry C (2018) The role of hippocampal replay in memory and planning. Curr Biol 28:R37–R50.

Pillai V, Kalmbach DA, Ciesla JA (2011) A meta-analysis of electroencephalographic sleep in depression: evidence for genetic biomarkers. Biol Psychiatry 70:912–919.

Rao RP, Ballard DH (1999) Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. Nat Neurosci 2:79–87.

Rasch B, Born J (2013) About sleep's role in memory. Physiol Rev 93:681–766.

Rauss K, Pourtois G (2013) What is bottom-up and top-down in predictive coding? Front Psychol 4:276.

Stickgold R, Walker MP (2013) Sleep-dependent memory triage: evolving generalization through selective processing. Nat Neurosci 16:139–145.

Whitehurst LN, Cellini N, McDevitt EA, Duggan KA, Mednick SC (2016) Autonomic activity during sleep predicts memory consolidation in humans. Proc Natl Acad Sci U S A 113: 7272–7277.