REM, DREAMS AND EMOTIONAL BRAIN HOMEOSTASIS

By Matthew P. Walker

Relative to cognition, surprisingly less research attention has been given to the interaction between sleep and affective brain function, despite the prominent co-occurrence of abnormal sleep in almost all clinical mood disorders. Nevertheless, recent work has begun to describe a clarifying role for sleep in emotion regulation, vignettes of which I review below, followed by a proposed REM sleep hypothesis of emotional brain homeostasis.

Emotional Reactivity

Numerous studies have described an increase in subjective affective disturbance following sleep loss, intensifying negative mood reports while reducing positive mood ratings associated with daytime activities (Walker and van der Helm, 2009). Objectively, it has been demonstrated that one night of sleep deprivation amplifies amygdala reactivity in response to negative emotional stimuli (Yoo et al., 2007), further associated with the loss of top-down medial prefrontal connectivity, yet increased coupling with autonomic activating brainstem regions. Larger pupillary response (indexing greater autonomic reactivity) to negative picture stimuli following a similar duration of sleep deprivation have also recently been reported (Franzen et al., 2009). These and other such studies support a role for sleep in recalibrating the appropriate connectivity of limbic circuits, resetting affective brain reactivity for next-day emotional challenges.

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Affective Memory (Re)processing

Several reports have described the selective memory consolidation of negative (unpleasant) emotional stimuli across periods containing sleep, relative to equivalent daytime periods awake, as well as late-night versus early-night sleep (rich in REM) (Walker and van der Helm, 2009). Most recently, Nishida et al. (2009) reported that REM not only facilitates the consolidation of negative memories, but that specific theta brain oscillations during REM correlate with these improvements. REM sleep may therefore offer a unique neurophysiological substrate for the (re)processing of emotional experiences.

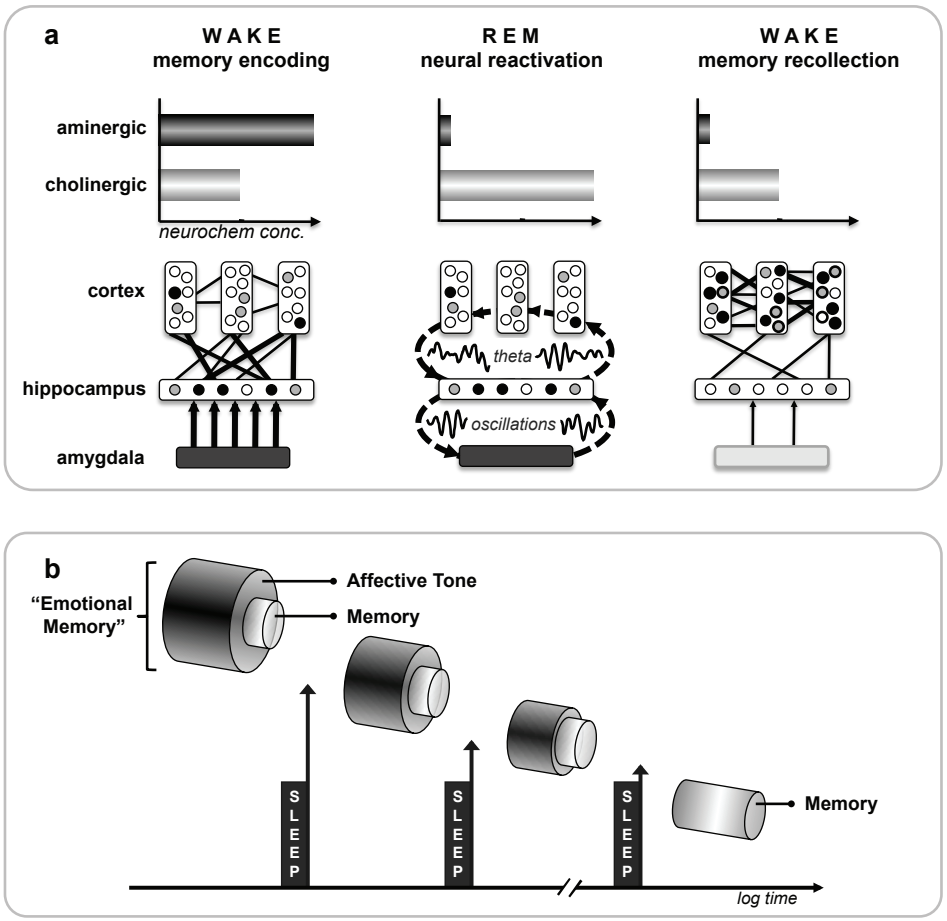

Figure 1. The sleep to forget and sleep to remember (SFSR) model of emotional memory processing: a) Neural dynamics of waking formation of an episodic emotional memory, followed by the subsequent REM sleep reprocessing, resulting in, on the one hand, a depotentiation of the affective tone initially associated with the event(s) at encoding, while on the other, a progressive neocortical consolidation of the experience in the context of prior semantic knowledge (the conscious expression of which may contribute to dreaming). Cross-connectivity between structures is represented by number and thickness of lines. Circles within cortical and hippocampal structures represent information nodes; depth of shade reflects extent of connectivity. Shading and arrows from amygdala represent magnitude of co-activation and influence on the hippocampus. b) Conceptual outcome. Through multiple iterations of this mechanism across the night, and/or across multiple nights, the long-term consequence of such REM sleep reprocessing allows for the strengthening and retention of salient information previously tagged as emotional at the time of learning, yet the divorcing of the autonomic charge acquired during learning.

A REM Sleep Hypothesis

While abundant evidence suggests that emotional experiences persist in our autobiographies over time, an equally remarkable but less noted change is a reduction in the affective "charge/tone" associated with their recall. The reason emotional experiences persist more robustly than neutral memories is due to autonomic neurochemical reactions elicited at the time of the experience, creating what we term an "emotional-memory" (Figure 1). However, the later recall of these memories tends not to be associated with anywhere near the same magnitude of autonomic (re)activation – suggesting that, over time, the affective "blanket" previously enveloped around the memory during learning has been stripped away, whereas the information contained within that experience (the memory) remains. This proposed REM hypothesis predicts that such decoupling takes place within the unique biological theater of REM (and potentially dreaming), such that we sleep to forget the emotional tone, yet sleep to remember the tagged memory of that episode (SFSR model; Figure 1). Specifically, increased activity within limbic networks during REM sleep dreaming offers the ability for reactivation of previously acquired affective experiences. Secondly, dominant REM-theta oscillations within subcortical and cortical nodes offer large-scale synchronous network cooperation, allowing the integration and, as a consequence, greater understanding of recently experienced emotional events in the context of pre-existing neocortical memories. Thirdly, these REM dreaming processes critically take place within a brain devoid of aminergic stress neurochemistry. As a consequence, REM dreaming achieves a balanced neural facilitation of the informational core of emotional experiences (the memory), yet depotentiates the autonomic charge originally acquired during learning (the emotion) – a form of overnight therapy.

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