The Influence of Sleep on Emotional Memory Consolidation Processes

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Abstract
An emerging trend in the literature has accumulated evidence in support for sleep’s role in the processing of episodic emotional memories. This review presents varying perspectives and models regarding information processing and affective functioning as it relates to sleep, emotions, and memory. Adaptive and maladaptive functions as it directly relates to sleep and emotions are also discussed herein. Collectively, the findings attempt to build on the literature and offer some clarity into the interaction of sleep, emotions, and memory.

Keywords: Sleep, Emotions, Memory Consolidation, PTSD

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1. Introduction

The link between emotions and sleep has intrigued the minds of many scholars and sages over the years. However, it is only in recent years that the mechanisms of emotional information processing have been steadily unraveled. Emotions are thought to play a significant role in the safety and survival of the species (Darwin, 1872/1998; LeDoux, 1996). From the perspective of evolutionary theory, reactivation during the offline periods of sleep may have evolved to maintain remembrance of emotionally salient information (e.g., a predator) and/or situations (e.g., escaping or avoiding a predator) active—thereby keeping memories consciously accessible for future use (Fishbein et al., 2010). In the present context, emotional memories characterized by an arousal response elicited during exposure of an event, situation and/or stimulus (Cahill et al., 1994) are enhanced by sleep (see Walker, 2009). The emotional (versus neutral) memory enhancing effect has been shown to benefit most over periods of undisturbed sleep, specifically occurring during rapid eye movement (REM) sleep (van der Helm & Walker, 2010).

Apart from the above-mentioned studies, adverse effects associated with sleep deprivation including impairments in mood, attention, concentration, reaction time, vigilance as well as dysregulation of social and emotional functioning have been reported (Durmer & Dinges, 2005). Conversely, favorable effects on mood and behavior have been shown in clinical populations such as in depressed (Vogel et al., 1980) and anxiety patients (Benca, 2008)—a finding that sheds light into the complex relationship that exists between “sleep, emotional brain function, and clinical mood disorders” (Walker & van der Helm, 2009, p. 731). Supplementing these findings, research on the physiology of emotional arousal and episodic emotional memories has placed considerable focus on the structures within the Medial Temporal Lobe (MTL), particularly the hippocampal formation, as well as the amygdala for the formation of newly created declarative memories (McGaugh, 2004).

Building on the literature, the present review focuses on four areas of research regarding the effects of sleep on emotional memory consolidation: 1) Effects of Sleep Deprivation and The Influence of Emotional Arousal on Cognition and Affect, 2) Sleep’s Role in the Enhancement and Impedance of Episodic Emotional Memories, 3) Neurophysiology of REM Sleep and Modulatory Effects of the Amygdala and Hippocampal Structures in Emotional Memory Information Processing, and 4) Clinical Implications of REM Sleep Deprivation and A Proposed Role for REM Sleep in Cognition and Affective Disorders.

2. Effects of Sleep Deprivation and The Influence of Emotional Arousal on Cognition and Affect

Anyone who has experienced sleep loss can attest to its negative effects on cognition and affect, among other functions. Research has shown that a single night of sleep deprivation can cause impairments in working memory (as measured by retrieval of an eight-digit sequence task), adversely affecting more women than men, in a group of 24 young adults (Rångtell and others, 2018). Sleep loss, even for a part of the night can increase inflammation in the body, significantly more in females than males, as shown in a group of 14 healthy adults (Irwin and others, 2008). More spe-
Specifically, they found that activation of nuclear factor (NF)-κB, a transcription factor linked to inflammatory signaling, was significantly greater in the morning after sleep loss as compared to after baseline or recovery sleep (Irwin et al., 2008). Acute sleep deprivation has also been shown to impair innovative thinking and flexible decision making (Harrison & Horne, 1999). In their study, 10 healthy participants showed a significant deficit in performance on a marketing decision-making game after 32-36 hours of sleep loss (Harrison & Horne, 1999).

These data are complemented by research showing sleep deprivation poses a serious concern for optimal cognitive functioning (see Frenda & Fenn, 2016, for a review). A wide range of cognitive impairments associated with sleep loss may include: deficits in executive functioning and attention (Durmer & Dinges, 2005), increased response times in tasks requiring sustained attention (Alhola & Polo-Kantola, 2007), compromised performance in both speed and accuracy (Smith, McEvoy, & Gevins, 2002), difficulty acquiring and retaining newly learned information (Wimmer et al., 1992), problems initiating and maintaining goal-directed behavior (Dahl, 1996), and increased impulsivity and reduced inhibitory control (Harrison & Horne, 1998), among other deleterious effects. Collectively, these studies underscore the value of sleep to maintaining overall cognitive health.

Affective functioning is another notable area considerably impacted by sleep loss. Pilcher and Huffcutt (1996) conducted a meta-analytic study and found sleep deprivation affected mood far more intensely than either cognitive or motor functioning. As reported by Dinges and colleagues (1997) sleep restricted to 4-5 hours per night during a week contributed to increased symptoms of tension-anxiety, fatigue, confusion, and overall mood disturbance. Along similar lines, research has shown sleep deprivation contributes to depressive symptoms in non-clinical participants (Cutler & Cohen, 1979). Anxiety symptoms and a range of psychopathology associated with sleep deprivation have also been reported (Kahn-Greene and others, 2007; Harvey et al., 2011). Current research has shown sleep deprivation appears to selectively increase negative over positive emotions (Franzen, Siegle, & Buysse, 2008; Paterson and colleagues, 2011). Taken together, these studies suggest that sleep loss is more associated with negative (anger, depression, fear and fatigue) than positive (happiness, excitement and/or activation) affective states.

As reported in the literature, a bidirectional model (Kahn, Sheppes, & Sadeh, 2013) has been proposed as a framework for understanding the relationship between sleep, emotions, and affective functioning. According to this model, bidirectional links between sleep and emotions are influenced and maintained by input and output systems that include: emotional brain networks, REM sleep related mechanism, emotional information processing, energy regulation and coping style, as well as physiological-emotional reactivity/arousal (Kahn et al., 2013). The dynamic interaction of these “bidirectional links” allow for the emotional experience to emerge across cognitive, behavioral, and physiological domains. As described by Fairholme and Manber (2015), “…the act of emotional regulation may be aimed at amplifying, attenuating, maintaining or even preventing emotional responding and can occur in the
context of motivated behavior intended to promote or prevent desired or undesired future states, respectively” (p. 46). Thus, the model by Kahn et al. (2013) offers one perspective on how cognitive and physiological emotional information processing occurs.

Similar to the bidirectional model (Kahn et al., 2013), the modal model of emotion as proposed by Gross (2014) interprets the intimate relationship between sleep and affect in the context of how an emotional situation and/or stimulus is experienced, and “highlights the process by which emotions are generated and unfold over time” (Fairholme & Manber, 2015, p. 56). In their comprehensive overview, Fairholme and Manber (2015) described Gross’s model as such:

According to this model, the emotion-generative process begins with the situation, broadly construed to capture the internal experience or construction of the situation, which could be based on external or internal cues. Attentional resources are then allocated to particular aspects of the situation and appraisals are assigned to help interpret and make meaning of the current situation. The appraisal results in an emotional response that could be experienced and/or expressed across multiple modes (cognition, behavior/action tendencies, and physiological sensations). The emotional response, which may or may not be subjectively and/or objectively observable, then feeds back into and modifies the current situation, and the process continues (p. 56-57).

At each stage of this process sleep interacts with emotions in a manner that serve adaptive or non-adaptive functions. For example, sleep loss may diminish energy and activity levels thus influencing the type of decisions and/or activities an individual plans to take (Gross, 2014). As caused by sleep loss “activity reduction (particularly reduced social contact) and failure to fulfill roles or obligations can each contribute to negative emotions” (Fairholme & Manber, 2015, p. 57). As such, sleep loss in this scenario can put an individual at risk for experiencing negative affect. Similarly, non-adaptive or maladaptive outcomes as a result of sleep loss or sleep restriction may also impact attention, appraisal and response processes.

Overall, these behavioral studies and proposed model (Kahn et al., 2013) support the idea that sleep plays a critical role in cognitive health and its interaction with other systems perhaps through “bidirectional links” may contribute to outcomes that can either serve adaptive or maladaptive functions. From this perspective, sleep and its interactions with emotions can influence the decisions any one individual can take as well as impact the affective state he or she is experiencing.

3. Sleep’s Role in the Enhancement and Impedance of Episodic Emotional Memories

An abundance of research supports the idea that memory for emotionally arousing information is better remembered than neutral (or non-emotional) information (Christianson, 1992; McGaugh, 2003; De Jesús, 2012). This finding has been reported across a number of experimental paradigms and/or conditions such as those using: pictures (Bradley and others, 1992), film clips
(Cahill et al., 1996), scenes (Payne et al., 2008), faces (Gupta & Srinivasan, 2009), words (Kensinger & Corkin, 2003), naturally occurring sounds (Bradley & Lang, 2000), and narrated slide shows (Heuer & Reisberg, 1990; Cahill et al., 1994). Episodic emotional memories are enhanced by two dimensions: 1) valence (ranging from unpleasant [negative] to pleasant [positive], with neutral considered an intermediate value; Lang, Bradley, & Cuthbert, 1992) and, 2) arousal (ranging from low [calm] to high [excitement]; Greenwald, Cook, & Lang, 1989). In fact, both valence and arousal are known to correlate remarkably well with each other and facilitate memory enhancement (Kensinger, 2004).

Using functional magnetic resonance imaging (fMRI), Kensinger and Corkin (2004) identified separate pathways for the distinct processing of valence and arousal: the former dependent on a prefrontal cortex-hippocampal network, while the latter, is dependent on an amygdalar-hippocampal network. With respect to stress, Payne and colleagues (2006; 2007) found pre-training stress affected memories differently—emotional memories were better retained whereas neutral memories were impaired. Payne et al. (2007) state, “hormone release might encourage encoding and consolidation of only those memory elements that were themselves emotionally arousing and salient” (p. 864). Elaborating on this process Payne and Kensinger (2018) note:

…the sleeping brain’s ability to selectively enhance emotional memory consolidation depends on stress and arousal levels at the time of learning, with stress responses during learning setting in motion a cascade of neurochemical events that lead to downstream selective consolidation of emotional aspects of memories (p. 36).

A working model proposed by Payne and Kensinger (2018) places significant importance of arousal at the time of learning to emotional memory processing. As described in the model by Payne and Kensinger (2018): “During an emotional experience, stress-related and arousal-related neuromodulators are released. Their presence helps set molecular tags that mark key features of an emotional experience” (p. 38). The neural activity from this experience is propagated across neural networks thus facilitating information from short-term into long-term consolidation (Payne & Kensinger, 2018). Similarly, Walker (2005) proposed a framework highlighting two different processes of memory formation: 1) stabilization, and 2) enhancement. Refining ideas of traditional consolidation models (McGaugh, 2000), Walker’s framework of memory formation postulates the following:

In this alternative model, the process again starts with an acquisition stage requiring a period of exposure to the task or experience. Following or during acquisition, another time-dependent (but not sleep-dependent) mechanism occurs, involving a process of consolidation-based stabilization. As a result, the memory representation is now resistant to interference, while behavioral performance (learning) is maintained, but not improved. However, only during periods of sleep can the additional process of consolidation-based enhancement, a brain-state dependent process, take place, regardless of whether this is immediately after acquisition (i), or several
hours later (ii and iii). As a consequence, behavioral performance indicates additional learning over and above that achieved during acquisition (Walker, 2005, p. 55).

The enhancement of memory and its differential processing by distinct sleep stages in animal (Fishbein & Gutwein, 1977; Smith, 1985) and human studies (Alger and colleagues, 2015) has accumulated in significant number over the years from a wide variety of sources (Walker & Stickgold, 2006). A common finding reported in the literature suggests early sleep (first half) consisting of non-REM (NREM) sleep (N1-N3) is more associated with the processing of declarative memories, specifically slow-wave sleep (SWS or N3) (Plihal & Born, 1997), whereas REM sleep and N2 is more associated with procedural memories (Born, Rasch, & Gais, 2006). This evidence, referred to as the “dual process hypothesis” (Peigneux and others, 2001) is contradicted by earlier research showing there is no difference between NREM and REM with respect to task performance (Cipolli & Salzarulo, 1975; Cipolli & Salzarulo, 1979). A similar model, as proposed by Giuditta and colleagues (Ambrosini & Giuditta, 2001; Giuditta, 2014) in their “sequential hypothesis,” place SWS and REM in complementary roles for memory processing. In this model, as Ficca and Salzarulo (2004) state, “SWS is associated with both adaptive and non-adaptive memories, whereas adaptive memories are strengthened during REM sleep” (p. 227). This point may help explain discrepancies in sleep-stage specific memory processing, but also underscore the adaptive role emotions play in survival and information-processing (Al-Shawaf and others, 2015).

Emergent studies examining differential processing of emotional versus neutral information have confirmed an emotional memory benefit following periods of undisturbed sleep relative to equal durations of wakefulness across varying retention intervals (30-min: Payne et al., 2008; 90-min: Nishida et al., 2009; 3-hrs: Wagner, Gais, & Born, 2001; Wagner, Fischer, & Born, 2002; 12-hrs: Hu, Stylos-Allan, & Walker, 2006; Payne et al., 2008; Payne & Kensinger, 2011) including conditions of total sleep deprivation (Atienza & Cantero, 2008; Sterpenich et al., 2009). Wagner and colleagues (2007) found an 8-hr retention interval consisting of nocturnal sleep relative to an 8-hr wake control enhanced memory accuracy for faces. Other experimental studies corroborated this finding demonstrating impairment in recognition of faces after one night of sleep deprivation (van der Helm, Gujar, & Walker, 2010; Pallesen and others, 2004), which was reversed after a single night of recovery sleep (van der Helm et al., 2010). These findings make clear emotionally arousing information relative to neutral information is enhanced following periods of undisturbed sleep and conversely impaired by sleep deprivation.

4. Neurophysiology of REM sleep and Modulatory Effects of the Amygdala and Hippocampal Structures in Emotional Memory Information Processing

Since the time of Freud, theories linking sleep and emotions have focused on a diversity of functions including sleep’s cathartic role in counteracting or ridding the mind of negative emotionality (Freud, 1900/1996), and how random neural activity that give rise to the “bizarre” nature of the dream phenomenon occurring during
REM sleep is biologically programmed (Hobson & McCarley, 1977). The emotional tinge ascribed to dreams “may reflect the mental representations of high limbic activations in conjunctions with deactivation of high-order cortical regions” (Germain, Buysse, & Nofzinger, 2008, p. 187). Neuroimaging data has confirmed increased activation and regional cerebral blood flow of limbic forebrain structures including the pontine tegmentum, thalamus, amygdaloid complexes, and neighboring regions during REM sleep (Maquet and colleagues, 1996). This finding would suggest, as Germain and colleagues (2008) point out, “that REM sleep is an endogenous state of heightened activity in emotional arousal brain centers” (p. 187).

Although the mechanisms underlying sleep-dependent emotional memory processing is not fully understood, REM sleep appears to play a significant role in this process given its functional role in dreaming and unique biology (Stickgold, 2005). Wagner and colleagues (2001) found 3-hr retention intervals comprised of post-learning late-night sleep (rich in REM and N2) relative to early-night sleep (predominantly SWS) benefited emotional as compared to neutral memories for texts—a finding that was reported to last for years (Wagner et al., 2006). Rauchs and colleagues (2004) showed performance for a task resembling a Remember/Know paradigm (Tulving, 1983) using factual (i.e., memorizing a word), spatial (i.e., its location), and temporal (i.e., what list it belongs to) components was dependent on REM sleep. Neurophysiology studies corroborate this finding showing support for REM sleep as an active process in the consolidation of emotional memories (see Walker & van der Helm, 2009 for a review). Nishida and colleagues (2009) found a significant relationship between the amount of REM sleep and selective enhancement of emotional as compared to neutral memory in participants obtaining a 90-minute nap relative to no-nap (wake) controls. They also report this offline emotional memory benefit is correlated with spectral activity in the theta-band range, which has been shown to support encoding processes during wakefulness (Buzsáki, 1998). Buzsáki (1998) posits a “hippocampal-neocortical dialogue” whereby memories initially dependent on the hippocampus become progressively less dependent on this structure as memories are reorganized in the neocortex during “transfer” of stored representations via sharp wave bursts occurring in sleep.

With regard to episodic emotional memories, a key structure found to play an important role in encoding and memory enhancement processes is the amygdala (see McGaugh, 2004; Phelps, 2006). The amygdala is believed to influence the formation of episodic emotional memory via its interaction with the hippocampus and prefrontal cortices and surrounding cortical regions (Cahill & McGaugh, 1998). These processes contribute to the memory enhancing effects reported in studies where emotional arousal is elicited (Bradley et al., 1992; Dolcos & Cabeza, 2002), but more importantly, support the necessary brain-state conditions whereby emotional saliency is retained following sleep, specifically REM sleep, as compared to wakefulness (Hu et al., 2006) and NREM sleep (early sleep vs. late sleep comparisons; Wagner et al., 2001; Wagner, Fischer, & Born, 2002). At a molecular level, animal studies have confirmed a contributing role of gene expression including those associated with synaptic plasticity (i.e., zif-268) following
learning, which are orchestrated during REM sleep (Ribeiro and colleagues, 1999). These processes, apparently “up-regulated during REM sleep in response to manipulations and memory tasks targeting the amygdala and hippocampus” (Payne & Kensinger, 2008, p. 36) would suggest as reviewed in the literature by Payne and Kensinger (2008) “that sleep constitutes a privileged window for consolation of emotional memories within larger associative networks” (p. 36). Neuroimaging data from Payne and colleagues (2011) corroborate this finding showing greater engagement and activation of limbic areas including the amygdala, ventromedial prefrontal cortex (vmPFC) and cingulate gyrus in response to retrieval of negative stimuli following periods of nocturnal sleep as compared to a “diffuse memory network” during wakefulness activity. Similarly, these areas coupled with associated memory networks implicated during encoding at learning also appear active during sleep with a rise in cortisol in response to emotional arousal reflecting further elaboration or reactivation of experience-dependent neural activity (as in encoding processes) (Bennion and colleagues, 2015).

The engagement of structures and networks sensitive to perceived emotional saliency as evidenced by the above neuroimaging data in corroboration with behavioral responses to negative versus neutral stimuli following sleep as compared to wakefulness add support for an active role of sleep in information-and-consolidation processes and not “merely a passive role” (Payne & Kensinger, 2018) attributed to reduced interference and/or circadian effects. Moreover, these data show undisturbed sleep, especially nocturnal periods of REM, interact with emotionally charged information to contribute to the “stabilization and enhancement” (Walker, 2005) of memory for emotional information.

5. Clinical Implications of REM Sleep Deprivation and A Proposed Role for REM Sleep in Cognition and Affective Disorders

Presently, there is no cure for post-traumatic stress disorder (PTSD), only symptom management through pharmacotherapy and/or cognitive-behavior therapy (Otto, Smits, & Reese, 2004). As outlined in the Diagnostic and Statistical Manual of Mental Disorders—5th ed. (DSM-5; American Psychiatric Association [APA], 2013) abnormal levels of anxiety in Anxiety Disorders and Trauma- and Stress-related Disorders appear to result from impaired emotion-regulatory mechanisms (Pace-Schott, Germain, & Milad, 2015). Evidence from a number of studies suggests sleep disturbance, a symptom of PTSD as per DSM-5 (APA, 2013), may be involved in the genesis and/or maintenance of PTSD (Mellman and others, 2002). Moreover, as notes Kahn and colleagues (2013), “clinical evidence implies that nearly all psychiatric and neurological mood disorders are associated with alterations in REM sleep” (p. 220). Research has shown REM sleep deprivation (REMD) appears to be sensitive to the impairing effects of emotional versus neutral memory (Wagner et al., 2001). Greenberg and colleagues (1983) report a selective impairment of emotionally meaningful past memory (Wagner et al., 2001). Greenberg and colleagues (1983) report a selective impairment of emotionally meaningful past memory (Wagner et al., 2001). Greenberg and colleagues (1983) report a selective impairment of emotionally meaningful past memory (Wagner et al., 2001).
of negative emotions and leads to a decreased intensity of dream emotions (Lara-Carrasco et al., 2009). Assuming an active role for REM in emotional-memory processing and an opposing role for REMD, researchers have intuitively sought out ways in which sleep deprivation following a highly charged emotional experience such as a trauma can be used as a potential intervention for the prevention of PTSD, or at the very least, therapeutically provide some form of symptom management.

Recent studies are being performed to examine the use of this therapeutic paradigm (intentional sleep deprivation) that “could potentially prevent development of PTSD” (Sarode et al., 2013). Wagner and colleagues (2006) found that memory for emotional texts were retained following 3-hour periods of post-learning sleep as compared to wake controls, and that this “enhancing effect” was maintained at retrieval after 4 years. Thus, Wagner et al. (2006) have proposed that sleep deprivation “in the immediate aftermath of traumatic events” may serve as “a possible therapeutic measure to prevent a long-term engraving of these events in memory, thereby at least partly countering the development of PTSD” (p. 789). This idea is further supported by research suggesting that PTSD may be linked to “overconsolidated emotional memories” (Wagner et al., 2006; Charney and colleagues, 1993). Mellman and Hipolito (2006) further argue that “in order to relieve distress ensuing from sleep disturbances in the aftermath of trauma and potentially having impact on the development of PTSD, it may be important to target disruption of REM sleep” (p. 614). Curtailing sleep through sleep deprivation following exposure to traumatic content (trauma film) reduced emotional affect and intrusive memories in a sleep-deprived group (22 subjects) as compared to a sleep control group (20 subjects) (Porcheret and colleagues, 2015). This research further strengthens the above-mentioned studies by advancing the idea that “initial sleep may actually worsen outcomes to psychologically traumatic stimuli and paradoxically, sleep deprivation on the first night may be advantageous for the amelioration of analogue trauma-related responses” (Porcheret et al., 2015).

It is no surprise to find that although there’s research supporting the idea that REMD may be adaptive for individuals afflicted with trauma (Mellman & Hipolito, 2006) the infancy of this scope of research has already yielded conflicting results. Using a trauma film paradigm (similar to Porcheret et al., 2015), Kleim and colleagues (2016) found sleep as compared to wake controls experienced fewer and less distressing intrusive trauma memories as confirmed through diary and questionnaire data. This finding underscores the importance of better understanding conditions by which sleep, especially REM sleep, and its restriction by sleep deprivation impacts the consolidation of emotionally charged memories. Of equal importance is clarifying the dual and opposing role of REM sleep’s strengthening of emotional memories, on the one hand, and REMD disruptive effects on emotional memories, on the other hand. Thus, a role for REM sleep strengthening and REMD disrupting effects on emotional memory information processing needs to be further clarified. Offering some clarity into the possible role of REM sleep with respect to information processing and affective functioning, Walker (2009) has proposed the Sleep to Forget and Sleep to Remember (SFSR)
model. As postulated in the model (Walker and van der Helm, 2009):

Waking formation of an episodic emotional memory involves the coordinated encoding of hippocampal-bound information within cortical modules, facilitated by the amygdala, and modulated by high concentrations of aminergic neurochemistry. During subsequent REM sleep, these same neural structures are reactivated, the coordination of which is made possible by synchronous theta oscillations throughout these networks, supporting the ability to reprocess previously learned emotional experiences...As a consequence, emotional memory reprocessing can achieve, on the one hand, a depotentiation of the affective tone initially associated with the event(s) at encoding, while on the other, a simultaneous and progressive neocortical consolidation of the information (p. 742).

The perspectives advanced by behavioral research studies and proposed model (Walker, 2009) such as reviewed above contribute to a growing literature attempting to elucidate the complex relationship between sleep, emotions, and memory. With respect to PTSD, although medication management has proved effective (Yehuda, 2002) concerns over margin of safety, side effects, and adherence has spurred research with less traditional pharmacological agents including propranolol (Pitman et al., 2002) and morphine (Holbrook and colleagues, 2010) to provide amelioration of symptoms. Sleep deprivation or sleep restriction, can therefore, offer one possible avenue for manipulating conditions whereby individuals experiencing trauma-and-other affective disorders are able to achieve adaptive functioning. Exploring this relationship more closely through experimental paradigms as the one proposed above may prove to be useful in impeding the consolidation of negative thoughts or imagery as experienced in these clinical populations.

6. Conclusion

Based on the findings reviewed above, the effects of sleep and emotions on memory are not unitary. Independently of each other, and perhaps most importantly, is the evolutionarily role both sleep (Siegel, 2001) and emotions (Tooby & Cosmides, 1990) have played in the survival of the species. As notes, Fishbein et al. (2010), “evolutionary adaption evolved neural circuits that may have been exploited for different uses, and one such use may be the cognitive processes engaged in memory consolidation that occur during the neurobiological states of sleep” (p. 273). Therefore, it is not hard to imagine from this perspective a mutually adaptive role for sleep-dependent emotional memory processing, which has served to maintain emotional versus neutral information preferentially consolidated for the purpose of future use. Unlike more extensive reviews on this topic (Walker, 2005; Stickgold, 2005) the review herein covered findings reported in the literature from a narrow focus, mostly on the emerging trend related to sleep’s role in the enhancement and impedance of episodic emotional memories.

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