Exp Brain Res (2014) 232:1403–1414
DOI 10.1007/s00221-014-3832-1

RESEARCH ARTICLE

Sleep and the processing of emotions

Gaétane Deliens · Médhi Gilson · Philippe Peigneux

Received: 18 October 2013 / Accepted: 5 January 2014 / Published online: 22 January 2014

Abstract How emotions interact with cognitive processes has been a topic of growing interest in the last decades, as well as studies investigating the role of sleep in cognition. We review here evidence showing that sleep and emotions entertain privileged relationships. The literature indicates that exposure to stressful and emotional experiences can induce changes in the post-exposure sleep architecture, whereas emotional disturbances are likely to develop following sleep alterations. In addition, post-training sleep appears particularly beneficial for the consolidation of intrinsically emotional memories, suggesting that emotions modulate the off-line brain activity patterns subtending memory consolidation processes. Conversely, sleep contributes unbinding core memories from their affective blanket and removing the latter, eventually participating to habituation processes and reducing aversive reactions to stressful stimuli. Taken together, these data suggest that sleep plays an important role in the regulation and processing of emotions, which highlight its crucial influence on human's abilities to manage and respond to emotional information.

Keywords Sleep · Emotion · Emotion regulation · Memory · Dreams

G. Deliens (✉) · M. Gilson · P. Peigneux (✉)
Neuropsychology and Functional Neuroimaging Research Unit (UR2NF), Center for Research in Cognition and Neurosciences (CRCN) and UNI, ULB Neurosciences Institute, Université Libre de Bruxelles (ULB), Avenue F.D. Roosevelt 50, 1050 Brussels, Belgium
e-mail: Gaetane.Deliens@ulb.ac.be

P. Peigneux
e-mail: Philippe.Peigneux@ulb.ac.be

Introduction

Despite many studies conducted over the last century, the functions of sleep still remain shrouded in mystery. Available evidence suggests that sleep plays a non-exclusive role in neuronal detoxification, conservation of brain energy, tissue restoration, immunity and neuronal plasticity (for a review see Siegel 2005). Lately, the interaction between the physiology of sleep and the processing of emotions has become a focus of growing interest for the scientific community. In this review, we will broach this topic, looking at how presleep emotional experiences impact on sleep architecture, and conversely how sleep quantity and quality on prior nights impact on our current mood state and emotional reactivity. We will also discuss how sleep modulates the processing of presleep emotional experiences and their reprocessing during re-exposure.

How negative emotions modulate sleep architecture

Modifications in the sleep architecture have been reported after the presentation of aversive or stressful movies presented immediately before sleeping, as compared to neutral movies or a baseline night (Talamini et al. 2013; Baekeland et al. 1968; Cluydts and Visser 1980). However, alterations in the sleep hypnogram are discrepant across studies. In the Baekeland et al. (1968) study, viewing a distressing film in the evening significantly increased both the number of REM sleep awakenings and REM sleep density, whereas Talamini et al. (2013) found a dampening of the expected REM sleep increase during the second half of the night. Besides REM sleep attenuation, Talamini et al. (2013) observed a higher proportion of slow wave sleep (SWS) in the stressing movie condition. At variance, similar distressing conditions were associated with less sleep stage shifts,

a higher proportion of stage 2 sleep, a higher overall fragmentation of sleep by awakenings and a decreased proportion of sleep in the first 3 h after sleep onset (Cluydts and Visser 1980). Finally, others did not disclose any disrupting effects of the presentation of a stressful movie on sleep architecture, but an alteration in the manifest content of the initial dreams (Lauer et al. 1987). Also, promoting negative mood using a failure feedback strategy in different cognitive tasks just before sleeping resulted in lower sleep efficiency, increased sleep latency, higher percentage of intra-sleep wake periods, more awakenings from REM sleep, a reduced total sleep time and a reduced proportion of REM sleep and SWS (Vandekerckhove et al. 2011).

Similarly, daily life stressors seem to exert an impact on sleep architecture, but results are also discrepant. Indeed, non-depressed divorcing spouses were found to exhibit decreased delta sleep, increased amount of REM sleep and shorter REM sleep latency (Cartwright and Wood 1991). Likewise, increased REM intensity was observed after the death of a close relative (Reynolds et al. 1993). However, Åkerstedt et al. (2007) found lower sleep efficiency, higher percentage of wake time and increased latency to SWS in periods of high stress, but no alteration in REM sleep parameters in sleep recordings following the days of low, high and intermediate stress. Anticipation and apprehension of stressful events may also alter sleep architecture. For instance, people apprehending a difficult working day to come exhibited a reduced percentage of SWS and increased amounts of stage 2 sleep (Kecklund and Åkerstedt 2004), while increased sleep fragmentation was found in patients waiting for an impending surgical operation (Edéll-Gustaffson 2002). Closer to the life of scientists, a stress state triggered by announcing a speech to be given the next morning dampened the normal increase in REM density over sleep cycles (Germain et al. 2003).

Various changes in sleep architecture after or in apprehension of a distressing event may be modulated by the type of psychosocial stressors under study (i.e., daily life stress events, experimental stress, traumatic stress), as well as stress duration and severity (for a review see Kim and Dimsdale 2007), but may also depend on the individual coping strategies adopted to face stressful situations. In patients with cancer, adopting an avoidance coping strategy such as denial and suppression was associated with increased sleep latency and nocturnal and early morning awakening, as compared to an approach-oriented coping strategy encompassing problem-solving to reduce treatment side effects, information seeking, expression of emotions and social support seeking (Hoyt et al. 2009). Another study compared the impact of experiential versus analytical emotion regulation strategies on sleep architecture (Vandekerckhove et al. 2012). The analytical approach consisted in evaluating causes, meanings and consequences of the emotional experience, whereas the experiential approach consisted in focusing on emotional experience and expressing emotions. Participants were administered a set of intelligence tests, after what they received a failure feedback stating that their performance was way below the norms. Before sleeping, they were instructed to apply the analytical or experiential emotion regulation strategy. Participants asked to use an experiential approach reported less negative feelings (e.g., irritability, distress and hostility) and exhibited longer total sleep duration and higher sleep efficiency (i.e., mainly reduced sleep fragmentation, fewer awakenings and time spent awake) than those adopting an analytical approach. The authors surmised that an experiential approach is more effective to recover from negative emotional events, with effects extending into sleep physiology. These effects seem pervasive across the development since positive or prosocial coping in 5-year-old kindergarten children was associated with an increased number of NREM S2 sleep spindles, whereas it was decreased in children using an avoidance coping strategy (Mikoteit et al. 2012). Besides state-related effects associated with exposure to ongoing or pending stressful events, trait-like sensitivity to stress was also linked with changes in the sleep architecture. Indeed, greater daytime sleepiness and fatigue, poorer subjective sleep quality and shorter sleep duration were observed in individuals having a natural trend for high stress levels as compared to others usually exhibiting low stress levels (Kashani et al. 2012). It is of course difficult in this case to disentangle the effects on sleep architecture that are attributable to a repeated exposure to stressful events (or experienced as such) from those associated with an intrinsic sensitivity to stress.

Is sleep an emotional regulator?

If emotional distress seems to exert an impact on sleep architecture on the subsequent nights, the reverse is also true. Many studies showed that sleep loss lowers emotional intelligence (Killgore et al. 2008), impairs emotional regulation by increasing irritability (Kahn-Greene et al. 2006) and emotional lability (Horne 1985) and worsens mood in children (Berger et al. 2012; Vriend et al. 2013) as well as in adults (Dinges et al. 1997; Lingenfelser et al. 1994; Rose et al. 2008). Also, increased self-reported feelings of depression, frustration, anger and anxiety are frequently observed over a prolonged sleep deprivation (Caldwell et al. 2004; Durmer and Dinges 2005; James and Gregg 2004; Pilcher and Huffcutt 1996; Scott et al. 2006; Kahn-Greene et al. 2007; Tempesta et al. 2010), which might eventually represent a more deleterious impact on daily activities than sleep deprivation-related

cognitive and behavioral alterations (Vandekerckhove and Cluydts 2010). Delivering theta (5 Hz) anodal transcranial direct current stimulation during REM sleep periods in the second half of the night increased gamma EEG during late REM sleep and was associated with a negative mood state in the morning (Marshall et al. 2011). Furthermore, disruptive daytime events (e.g., when being disrupted during a scheduled activity) are perceived more negatively by medical residents in a state of lack of sleep and reduce the positive effect of goal-enhancing experiences (e.g., an opportunity to perform a medically challenging task; Zohar et al. 2005). The perception of stress is also exacerbated by sleep deprivation (Minkel et al. 2012) or in subjects self-reporting poor sleep quality (Prather et al. 2013). This biased perception of stress seems to depend on both the intensity of the stressor and of the vigilance state. Indeed, administration of a cognitive test inducing low stress levels induced greater subjective stress, anxiety and anger in sleep-deprived participants as compared to rested controls (Minkel et al. 2012). By contrast, increased negative mood and stress levels were observed in a high-stress cognitive testing condition in both normal sleep and sleep-deprived conditions. The authors suggested that the psychological threshold for the perception of stress from cognitive demands is lowered after sleep deprivation, whereas negative affects in response to high-stress performance demands are not increased. This lack of effect of sleep in the latter case might be due to a ceiling effect in stress responses.

Like sleep deprivation, poor sleep quality is associated with decreased ability in cognitive reappraisal (Baglioni et al. 2010; Bower et al. 2010; Mauss et al. 2013), i.e., the ability to cognitively reframe an emotional event in order to lessen its impact (Ochsner and Gross 2005). Mauss et al. (2013) concluded that a lack of sleep, but also impaired sleep quality, might alter cognitive reappraisal ability by impairing underlying higher cognitive functions such as cognitive control processes (Goel et al. 2009; Tucker et al. 2010). Using fMRI, exacerbated emotional reactivity for aversive stimuli (or in other words, decreased cognitive reappraisal ability) after sleep deprivation was associated with an amplified amygdala response. Connectivity between the amygdala and the medial prefrontal cortex (mPFC) was also decreased in response to increasingly aversive pictures (Yoo et al. 2007). Decreased functional connectivity with the mPFC, a region known to exert inhibitory control on amygdala (Davidson 2002), suggests that the deregulation of emotion observed after a sleep deprivation (Yoo et al. 2007), or even after a short-term restricted sleep period (Motomura et al. 2013), is related to a degraded top-down inhibitory control. Increased autonomic reactivity observed under the conditions of sleep deprivation, as reflected by larger pupillary responses to negative pictures, also supports this disinhibition hypothesis (Franzen et al. 2009).

Responses to stimuli with positive valence are less consistent across studies, only partially validating the disinhibition theory. Indeed, Gujar et al. (2011a) found increased activity throughout mesolimbic reward brain networks in response to pleasant stimuli after sleep deprivation, associated with a higher connectivity in early primary visual processing regions and extended limbic areas, and a significant loss of functional connectivity in medial frontal and orbitofrontal regions. However, happy facial expressions did not significantly alter amygdala activity after a 5-day sleep restriction (5 h of sleep per night; Motomura et al. 2013). Moreover, whereas one night of sleep deprivation led to an overestimation of positive emotional stimuli, correlated with activity in mesolimbic regions (Gujar et al. 2011a), Zohar et al. (2005) found that goal-enhancing experiences were less positively perceived by medical residents in lack of sleep. Discrepancies between these studies may be due to different amounts of sleep deprivation (Motomura et al. 2013). Indeed, sleep debt might alter the processing of negative emotional stimuli, but not positive ones, whereas total sleep deprivation would exacerbate emotional reactivity for aversive stimuli but also, by its antidepressant effect (Gillin et al. 2001), amplifies reactivity throughout limbic regions in response to positive emotional stimuli.

Nonetheless, recent studies cast doubt on the disinhibition theory by showing a dampening of emotional reactions for negative and positive stimuli after sleep deprivation, rather than an exacerbation of affective reactivity. Among these studies, Minkel et al. (2010) reported less facial expressiveness, especially in response to positive film clips, in sleep-deprived participants than in rested participants. In line with these results, Schwarz et al. (2013) observed a deceleration of volitional facial reactions to emotional clips but no alteration in affective inhibitory control after one night of restricted sleep. In this study, participants were asked to respond with either compatible or incompatible facial muscles to emotional clips. Following the disinhibition hypothesis, slower facial reactions should mainly be observed in response to incompatible stimuli due to an additional effort to prevent the compatible reaction caused by a degraded top-down inhibition in partially sleep-deprived participants. Since slowed facial reactions were observed for both compatible and incompatible stimuli, the authors concluded to a general slowing down due to a dampening effect on the behavioral response, rather than a disinhibition of the top-down emotional control after one night of restricted sleep.

The sleep stage that is affected by sleep deprivation also determines emotional and cerebral reactivity to emotional stimuli. Rosales-Lagarde et al. (2012) scanned participants

twice during a visual emotional reactivity task, before (baseline condition) and then 24 h after one night of either selective REM sleep deprivation or NREM sleep interruptions. In agreement with the disinhibition theory, emotional reactivity was increased as compared to baseline, but only in the REM sleep-deprived group. Also, functional MRI disclosed increased activity in regions involved in emotional processing in the REM sleep-deprived group during the second test period (relative to the baseline), whereas cerebral activity was similar before and after an experimental night with NREM sleep interruptions. These results suggest that REM sleep may contribute to reset emotional brain reactivity to cope with next day's disruptive events. However, evidence suggests that REM sleep is not the sole stage involved in emotional regulation. For instance, 5-year-old children exhibiting a higher rate of NREM S2 spindles were found more likely to exhibit current and future positive emotional and behavioral functioning (Mikoteit et al. 2013).

Finally, others reported that it is not only affective reactivity in response to emotional stimuli that is affected by sleep debt, but also accuracy in facial emotional expression judgment. Indeed, the ability to recognize moderately angry and happy facial expressions was significantly reduced in women after a total sleep deprivation, and a trend was observed in men (van der Helm et al. 2010). These deficits in recognition of salient affective social cues disappeared following one night of recovery sleep. Similarly, Pallesen et al. (2004) reported a reduction in speed and accuracy for the recognition of facial emotions after total sleep deprivation for one night. Moreover, sleep-deprived participants reported feeling themselves less able to understand their own emotions and these of others (Killgore et al. 2008).

In all of the aforementioned studies, the link between sleep and emotional regulation was investigated using emotional task-relevant targets. Fewer studies have investigated the sensitivity to emotional distractors after sleep deprivation. Chuah et al. (2010) administered a delayed-response working memory task in which the to-be learned neutral faces were followed by one out of three types of distractors: negative emotional, neutral and scrambled patterns. Participants were then asked to indicate whether the probe face was presented earlier. Despite similar behavioral working memory patterns between sleep and sleep-deprived participants, analysis of fMRI data showed that higher distraction by emotional pictures after 24 h of sleep deprivation was associated with increased amygdala activation and reduced functional connectivity between the amygdala and prefrontal regions.

Altogether, these studies indicate that alterations in sleep patterns may impact the functional neurophysiological mechanisms underlying the regulation of our emotions.

Emotion modulates sleep-dependent memory consolidation processes and their neurophysiological substrates

Besides a potential impact of sleep on the perception of emotional daytime events and emotional reactivity in response to these events, as discussed above, post-training sleep is also known to play a beneficial role for memory consolidation (for a review see, e.g., Peigneux and Smith 2010). Among the different memory subsystems, several studies support the assumption that emotional declarative memory also benefits from sleep (Holland and Lewis 2007). For instance, the retrieval of emotional items, as compared to neutral ones, is enhanced when the retention interval contains a sleep episode (Hu et al. 2006; Payne et al. 2008). A beneficial effect on the consolidation of emotional memory can still be found 4 years later (Wagner et al. 2006). Conversely, sleep deprivation on the post-learning night impairs recognition performance for positive and neutral pictures when a recognition test is administered 72 h (Sterpenich et al. 2007) or 1 week (Atienza and Cantero 2008) after the encoding session. However, aversive pictures might be less sensitive to sleep deprivation (Atienza and Cantero 2008) or preferentially consolidated during the recovery nights (Sterpenich et al. 2007). Indeed, neuroimaging data disclosed greater hippocampus and mPFC activity during correct recognition of emotional (vs. neutral) pictures in rested as compared to sleep-deprived participants (Sterpenich et al. 2007). Moreover, activity in the amygdalo–cortical network was higher in sleep-deprived participants during correct recognition of negative pictures. It was surmised that activation of this alternative network would allow sleep-deprived subjects to reach similar performance levels than subjects allowed to sleep (Sterpenich et al. 2007). In a follow-up study, Sterpenich et al. (2009) retested their participants 6 months after the initial encoding session. Although behavioral performance was similar between the two groups, distinct patterns of neuronal activity were evidenced. Sleep-deprived subjects exhibited reduced activity in the ventromedial prefrontal cortex (vMPFC), the amygdala and the occipital cortex, suggesting that sleep impacts on neuronal networks underlying the retrieval of emotional memories even 6 months after encoding.

The sleep stage(s) specifically underlying the consolidation of emotional memories remains a matter of debate. Several studies suggest a key role for REM sleep in the processing of emotional memories. For instance, the recall of emotional but not neutral texts was enhanced after a 3-h period of late sleep (vs. early sleep), i.e., the period richer in REM sleep (Wagner et al. 2001, 2005). Similarly, a better recall performance was observed for emotional memories after a nap than a wake interval. This beneficial effect

correlated with the percentage of REM sleep in the nap as well as with right-dominant prefrontal theta power during REM sleep (Nishida et al. 2009). Also, recall performance for threatening items (e.g., a snake's picture) was both altered after REM sleep deprivation (Grieser et al. 1972) and enhanced after a late sleep episode rich in REM sleep (Wagner et al. 2001, 2005).

It has been argued that the neuroanatomical, neurophysiological and neurochemical features of REM sleep make it the ideal site to process emotional information (van der Helm and Walker 2009). Several animal studies also suggested a crucial role of REM sleep in emotional memory consolidation (see, e.g., Graves et al. 2003; Hennevin et al. 1995). In humans, from a neuroanatomical standpoint, the amygdala and hippocampus are two regions well known to play a pivotal role in the consolidation of emotional memories (Cahill et al. 1996; Phelps and LeDoux 2005; for a review see McGaugh 2004). These regions are also part of the most active brain regions during REM sleep (Hennevin et al. 1995; Maquet et al. 1996). During the encoding phase, the emotional features of the stimulus trigger the hormonal and brain processes involved in the regulation of memory consolidation, releasing stress hormones (cortisol, norepinephrine) that in turn increase the activation of the amygdala. Activation in the amygdala promotes memory consolidation by modulating striatal, prefrontal and hippocampal activity (for a review, see McGaugh 2004), the latter region playing a pivotal role for the consolidation of declarative memories (for a review see Moscovitch and Nadel 1998). Hippocampus modulation by amygdala activity, through direct and indirect projections (see Petrovich et al. 2001, for a review), has been observed during the encoding (Kilpatrick and Cahill 2003), retrieval (McGaugh 2004) and consolidation (Strange and Dolan 2004) stages. From a neurophysiological standpoint, theta waves featured in REM sleep are thought to be involved in the strengthening of the distributed features of specific events across related but different anatomical networks (Buzsaki 2002; Jones and Wilson 2005), and particularly in the strengthening of emotional items (Nishida et al. 2009; Popa et al. 2010; for a review see Pare et al. 2002). On the neurochemical side, REM sleep is featured by increased acetylcholine levels (Marrosu et al. 1995), a neurotransmitter involved in the modulation of emotional memory consolidation (Introini-Collison et al. 1996; McGaugh 2004). Similarly, increased cortisol levels are observed during late night sleep episodes rich in REM sleep, cortisol being a stress hormone that modulates emotional memory via the amygdala–hippocampus interactions (Payne and Nadel 2004). Considering the wide body of evidence supporting the hypothesis that sleep promotes memory consolidation processes and the specific neuroanatomical, neurophysiological and neurotransmitter features of REM sleep, it is tempting to surmise that the consolidation of emotional memories would also benefit from REM sleep.

However, other studies have evidenced a role of NREM sleep rather than REM sleep in the consolidation of emotional memories. In a study, participants learned a set of emotional pictures and then received zolpidem (ZOL), sodium oxybate (SO) or a placebo before a daytime nap in a within-subject design (Kaestner et al. 2013). ZOL is known to increase sleep spindle density (Brunner et al. 1991; Feinberg et al. 2000), hippocampal sharp wave ripples (Koniaris et al. 2011) and REM sleep latency and/or to reduce the amount of REM sleep (Brunner et al. 1991; Feinberg et al. 2000). Conversely, SO is known to reduce sleep spindle density and increase the percentage of SWS (Walsh et al. 2010). Post-nap recognition performance was enhanced both for negative and for high-arousal stimuli when ZOL was administrated before the nap compared with placebo, whereas memory for negative and high-arousal stimuli was unchanged after a nap with SO. Taken together, these results indicate that pharmacologically increased sleep spindle density eventually led to a memory bias toward negative and highly arousing stimuli, suggesting that sleep spindles may also facilitate emotional memory consolidation.

Is sleep stripping associated emotions from experience?

The studies reported in the prior section suggest that emotional events are preferentially consolidated over sleep. However, it is less clear whether the emotional tone (in other words, the context) associated with this event is similarly consolidated and benefits the retrieval of the stored memory trace. The sleep to forget and sleep to remember (SFSR) hypothesis (van der Helm and Walker 2009) surmises that REM sleep actually supports an unbinding process between the target memory and its emotional tagging. According to these authors, emotional experiences associated with hippocampal activity are strengthened in terms of a core memory, whereas the emotional charge coating these experiences, associated with the activity of the limbic network, is progressively weakened over sleep. Supporting their hypothesis, amygdala activity during exposure to emotional pictures is reduced when participants are re-exposed to the same pictures after a night of sleep, but not after a similar interval spent awake (van der Helm et al. 2011). Moreover, the same study found that participants' arousal ratings for emotional pictures decreased at the second exposition after sleep, but increased in the wake condition. Also, there was a sharper decline of amygdala activity during picture re-exposition in participants exhibiting reduced gamma EEG activity during REM sleep, reduced gamma EEG being indicative of low adrenergic activity (Maloney et al. 1997). At the neurochemical level,

increased concentrations of aminergic neurotransmitters and amygdala activation during wakefulness promote the encoding of emotional memories and their affective context. During subsequent REM sleep, the dramatic rise of cholinergic activity associated with theta oscillations participates in the reactivation of the learned information, eventually strengthening emotional memories (Jones and Wilson 2005). By contrast, the concurrent decrease in aminergic activity may simultaneously lead to a depotentiation of the emotional tone initially associated with the learned stimulus. Based on these elements, the SFSR model (van der Helm and Walker 2009) surmises that the specific neurochemical imbalance dominating REM sleep participates in the strengthening of recent memories while removing their learning-related affective blanket.

Studies having investigated a possibly sleep-dependent evolution of the affective tag of memories had divergent outcomes. In one study (Baran et al. 2012), participants had to rate the valence and arousal of emotionally positive or negative pictures. After a 12-h interval filled with either nocturnal sleep or daytime wakefulness, participants were administered a recognition task and had to rate again the valence and arousal of each picture. Recognition performance was better after sleep than after wake, suggesting a beneficial role of sleep in the long-term consolidation of emotional memories. Contrary to the predictions of the SFSR model, however, valence and arousal ratings for emotional pictures presented before and after sleep were similar, whereas the pictures were rated more positive than after the first exposition after a wake interval, suggesting that sleep consolidates memories while preserving their emotional blanket. Similarly, Wagner et al. (2002) did not find decreased arousal or valence ratings for emotional pictures after versus before late, REM-rich sleep. However, it can be argued that rating valence and arousal for stimuli before sleep (or wakefulness) may have biased ratings at the second presentation of the same stimuli. To address this criticism, Groch et al. (2013) asked participants to rate valence and arousal for the learned pictures during the recognition test only, after an episode of early (SWS-dominant) or late (REM-dominant) sleep. Late but not early sleep enhanced recognition of emotional pictures as compared to neutral ones, again suggesting that REM sleep supports the consolidation of emotional memories. However, emotional reactivity, valence and arousal ratings did not differ between early and late sleep conditions, which might contradict the SFSR hypothesis of a "cathartic" role of REM sleep while being in agreement with the results of the Baran et al. (2012) study. Alternatively, one could argue that REM sleep episodes during early sleep have modulated emotional reactivity, valence and arousal additional parameters to the same extent than during late sleep. Neuroimaging studies also disclosed increased amygdala activity during the recognition of aversive pictures after a night of sleep (Payne and Kensinger 2011; Sterpenich et al. 2009). In another study, negative pictures were better recognized than neutral ones after late than early sleep, better performance being associated with an increased late positive ERP over the frontal cortex 300–500 ms after stimulus onset (Groch et al. 2013), an electrophysiological effect generally interpreted as an index of memory accuracy (Yu and Rugg 2010). On the other hand, valence and arousal were rated similarly after early or late sleep. Taken together, these results are in favor of the hypothesis that REM sleep facilitates memory consolidation of emotional events while preserving their emotional blanket. In another setting, emotional reactivity in an emotional face recognition task was tested after a 90-min nap or a wake interval (Gujar et al. 2011b). In the wake condition, participants rated angry and fearful faces as reflecting more intensely fear and anger than after the first exposure to the same stimuli. This trend was reversed in the nap condition but only if the nap contained REM sleep. Indeed, participants then presented a dampened emotional reactivity for fearful and angry faces, but also greater affective reactivity for happy expressions. Hence, this study suggests that REM sleep would initiate a depotentiation of negative reactivity in response to fearful and angry faces, in agreement with the van der Helm and Walker (2009) finding of a sleep-dependent reduced amygdala activity for familiar negative pictures during a second exposition to a set of emotional pictures.

Finally, three studies gave support to the hypothesis of a sleep-dependent unbinding process but contrarily to the SFSR model suggested that this decoupling takes place during SWS rather than REM sleep. In one study, extended REM sleep deprivation eventually led to a greater adaptation to previously seen negative pictures (as measured by arousal ratings) than limited REM sleep deprivation (Lara Carrasco et al. 2009). It was also found that habituation to previously seen negative pictures (i.e., the reduction in behavioral and physiological responses during re-exposure to the pictures comparing to the initial exposure) was increased after a nap, more than after a similar wake interval (Pace-Schott et al. 2011). Furthermore, participants getting REM sleep during their nap exhibited a lower arousal habituation (as measured by the skin conductance response), and SWS occurrence was positively correlated with habituation of the frowning response (as recorded using EMG on the corrugator supercilii). Still, subjective ratings on pictures did not differ between the wake and nap groups, suggesting a lack of sleep-dependent subjective habituation. Altogether, the results of this study suggest that sleep would play a role in emotional regulation by promoting habituation at the level of somatic responses but not at the subjective level, which may explain why studies based on self-reported emotional reactivity have failed to

disclose sleep-dependent unbinding effects (Baran et al. 2012; Groch et al. 2013; Gujar et al. 2011b). Finally, a third study reported a higher SWS proportion, the flattening of the normal increase in REM density and a correlation between increased SWS and emotional attenuation over sleep after viewing a stressful film (Talamini et al. 2013). Furthermore, emotional attenuation correlated with the baseline amount of SWS during early sleep, suggesting a trait-like emotional attenuation phenomenon over sleep in subjects presenting a higher proportion of SWS in their sleep.

The studies discussed up to now have investigated memory consolidation processes for an intrinsically emotional material. Therefore, they probed the emotional content of the learned information without distinguishing the effects associated with the affective state (or background) surrounding the items from the effects associated with the core to-be-learned material itself (Lewis and Critchley 2003). However, sleep may selectively enhance specific components of memories. In the Payne et al. (2008) study, participants were shown a set of pictures depicting neutral or negative objects on a neutral background and then slept or stayed awake before being administered separate recognition tests on the studied objects and backgrounds. Results indicated a strengthening of negative objects at the expense of their background after sleep, whereas recognition was similarly impaired for objects and backgrounds in the wake condition. In another study, neutral or negative context pictures were associated with a neutral foreground object. At delayed recall, participants decided whether a foreground object presented alone was previously coupled with a negative or neutral encoding context, or not remembered (Lewis et al. 2011). Results disclosed a sleep-dependent enhancement of context memory recognition, to the same extent for emotional and neutral backgrounds. The authors surmised that the reported emotion-specific enhancement in memory consolidation after sleep (Hu et al. 2006; Nishida et al. 2009; Payne and Kensinger 2011; Payne et al. 2008; Sterpenich et al. 2007, 2009; Wagner et al. 2005, 2006) may actually be restricted to the memory of the emotional picture in itself, or of embedded details, but does not extend to the associative link between an emotional context and a neutral stimulus.

Along the same lines, our group investigated sleep-dependent memory consolidation processes for emotionally neutral items learned in a specific mood state (Deliens et al. 2013a, b). To do so, we used a contextual interference paradigm aimed at testing mood-state-dependent memory (MDM) effects (Bartlett and Santrock 1979; Kenealy 1997), i.e., decreased memory performance for a neutral item learned in a specific mood state but recalled in another mood state, as compared to learning and recall in the same mood state. In a first study (Deliens et al. 2013a), participants learned a list of word pairs after a mood (sad vs. happy) induction procedure and then slept at home or stayed awake during the post-learning night. After two recovery nights, they were induced in the same mood as learning and had to recall 50 % of the learned word pairs, and then were induced after a break in the opposite mood and had to recall the remaining 50 % of learned word pairs. Results disclosed an MDM effect in the post-training wake but not in the sleep condition, supporting the SFSR hypothesis (van der Helm and Walker 2009) of a demodulation between memories and their "affective blanket" during post-learning sleep, eventually protecting memories against emotional retroactive interference. As discussed above, the SFSR model posits that the decoupling between the core information and its emotional context takes place during REM sleep. Against this hypothesis, however, we found preserved MDM effects after both early (SWS-dominant) and late (REM-dominant) sleep episodes (Deliens et al. 2013b). Surmising that the unbinding process may require several NREM–REM cycles to achieve, we subsequently tested for MDM effects after 12 h either spent awake during daytime or including one night of sleep (Deliens and Peigneux 2013). Results again disclosed preserved MDM effects in both the sleep and wake conditions, suggesting that the unbinding process initiated during the first post-learning night might need several nights to achieve as suggested by MDM suppression after 3 days when sleep was allowed on the first post-learning night (Deliens et al. 2013a). Whether REM sleep plays a role in the process still remains to be demonstrated. Notwithstanding, an emotional unbinding process spanning over several nights of sleep might explain why other studies failed to observe an overnight decrease in self-reported emotional reactivity when tested after one night of sleep only (e.g., Baran et al. 2012; Wagner et al. 2002; Groch et al. 2013), actually reflecting an unfinished emotional unbinding process.

Finally, it was suggested that sleep might also favor the retention and generalization of extinction learning after exposure therapy (Pace-Schott et al. 2012). In a fear conditioning paradigm, two-colored lights were coupled with mild electric shocks. Then, the fear response was extinguished to one of the lights only by repeatedly presenting the light without the previously associated shocks. After a wake or sleep episode (12 h delay), fear conditioning was assessed measuring skin conductance responses. Participants in the sleep condition exhibited decreased fear responses for both the extinguished and non-extinguished stimuli, whereas dampened fear responses were found for the extinguished stimulus only in the wake condition, suggesting a generalization of fear extinction over sleep. Additionally, subjective and physiological responses to phobic stimuli were better retained and generalized when the extinction was conducted in the evening, closer to the usual

bedtime, than in the morning (Pace-Schott et al. 2012). Additionally, it was shown that unreinforced re-exposure to a fear-conditioned context during SWS enhances fear extinction (Hauner et al. 2013). In this study, two face pictures were coupled with mild electric shocks in the presence of two distinct background odorants. During the subsequent night, only one odorant context was re-presented during SWS, aimed at reactivating the paired face picture in the absence of electric shocks. At post-sleep retrieval, stimulus-specific fear extinction was associated with decreased hippocampal activity and a reorganization of amygdala patterns. Hence, these results suggest that sleep allows a selective extinction of targeted memories by a mere re-presentation of the odorant context without the associated feared stimulus itself.

Sleep and dreaming

Evidence accumulated so far suggests that NREM and REM sleep are appropriate environments to process and/or reprocess emotional information. Emotion processing during sleep may also be reflected in the production of dreams. More than a century ago, Freud (1990) proposed that emotions are processed during dreams, whose latent contents would express unconscious desires (Freud 1990). In the neuroscience domain, dreaming is currently defined as a mental activity occurring during sleep (Schredl and Wittmann 2005) that can be recollected at wake. Dreaming can therefore be construed as a hallucinogenic experience subtended by the specific neurobiological patterns featuring sleep states. Looking at the patent content of dreams, only a limited percentage of dreams appear to feature emotions, whereas half of them contain perceptual elements and about two-third are linked to reasoning aspects. While the precise implication of sleep stages in the content of dream is still controversial, the sleep mentation likely reflects the underlying physiological brain state. Indeed, dreams associated with emotional contents are more often recollected after REM than after NREM sleep, whereas reasoning aspects are more preponderant after NREM than after REM sleep, perceptual features being equally distributed (Strauch and Meier 1996). Besides a REM sleep-related dominance for emotion in dreams (see also, e.g., Smith et al. 2004; McNamara et al. 2005), NREM dreams have been shown to feature more emotional content at the end than at the beginning of the night (Wamsley et al. 2007). Higher emotional expression during late sleep may stem from a combination of decreased homeostatic sleep pressure and high circadian propensity to sleep (Schmidt et al. 2007) and/or from the fact that light NREM (S2) alternating with consolidated REM sleep episodes characterizes late sleep. Considering the emotional tonality of dreams, negative emotions such as aggressive behaviors have been found more prevalent during REM sleep dreams, suggesting dissimilarities between sleep states in the emotional valence of dream contents (McNamara et al. 2005).

At the neurobiological level, REM sleep is associated with decreased activity in the inferior parietal, dorsolateral prefrontal and orbitofrontal cortex, the posterior cingulate gyrus and the precuneus (Maquet et al. 1996; Schwartz and Maquet 2002). By contrast, there is increased activity in brain regions that may contribute to the experience of dreaming during REM sleep, including the hippocampus, the limbic and paralimbic structures, the mPFC, the basal forebrain, the pedunculopontine tegmental nuclei and anterior cingulate cortex (Maquet et al. 1996; Nofzinger et al. 1997). In this respect, specific amygdala activity might subtend the experience of intense emotions such as fear and anxiety (Smith et al. 2004), whereas cortical deactivations (Schwartz and Maquet 2002; Schwartz 2004) and the temporal dissociation of EEG activity between executive and perceptual regions (Corsi-Cabrera et al. 2003) would account for disorientation and illogical thinking phenomena often found in dreams. While REM sleep is commonly considered as the equivalent of dreaming, recent findings suggest that the cholinergic brain stem mechanisms that control REM sleep interact with a second, dopaminergic, forebrain mechanism, which is the final path leading to the dreaming experience per se (for a review see Solms 2000).

Emotional (re)processing of negative events during dreams is also at the heart of the threat simulation theory (Revonsuo 2000). This theory suggests that dreaming acts as a defense mechanism, which repeatedly simulates threatening events, leading to the rehearsal of the cognitive mechanisms subtending the efficient threat perception and avoidance. This model fits well with the idea that dreaming is associated with fear conditioning (especially avoidance behaviors) and that fearful elements are often embedded in dreams. Likewise, Levin and Nielsen (2009) proposed that the neurophysiological and neurobiological states characterizing REM sleep favor essential cognitive processes occurring during dreams. These processes would facilitate fear-memory extinction via the activation of memory elements, their subsequent recombination and finally the expression of the associated emotions, allowing a down-regulation of negative and arousing emotions.

Nonetheless, neuroimaging studies have also highlighted markedly increased activation in reward networks during REM sleep including the ventral tegmental area and nucleus accumbens (Solms 2000), potentially reflecting the reprocessing of memories of high emotional or motivational relevance. According to the Reward Activation Model (Perogamvros and Schwartz 2012), the mesolimbic dopaminergic (ML–DA) system—involved in reward and

emotion processing—is also active during sleep, suggesting its participation both in memory consolidation processes and in the generation of dreams. Accordingly, the administration of dopaminergic agents enhances the appearance of vivid dreams (Balon 1996; Pinter et al. 1999), whereas dopaminergic antagonists reduce nightmares and vivid dreams in the post-traumatic stress disorder syndrome (David et al. 2006; Lambert 2006). In support of this model, approach behaviors during dreams have been found three times more frequent than avoidance behaviors (Malcolm-Smith et al. 2012), against the hypothesis that dream mechanisms are specialized in the simulation of the threatening events (Revonsuo 2000) and that dreams are mostly involved in fear conditioning processes.

As a whole, available evidence suggests that specific brain activity and neurobiological patterns subtend emotional regulation and memory processing during sleep and dreaming.

Conclusions

In the present review, we have provided evidence supporting a strong link between sleep and the processing of emotions in various ways. Stressful and emotional experiences in general seem to induce changes in the sleep architecture under both experimental and ecological conditions. Individual features such as coping strategies and personality traits might also modulate the way sleep is impacted by emotional experiences. Conversely, sleep alterations might trigger emotional disturbances possibly favoring the emergence of psychiatric pathologies such as anxiety and depression disorders. Additionally, sleep appears beneficial for the consolidation of emotional memories, above consolidation levels for neutral memories, suggesting that emotions modulate the off-line cerebral activity patterns subtending memory consolidation processes. However, these sleep-dependent consolidation processes would not apply to the whole emotional experience. Indeed, available data suggest that sleep contributes to unbind the core, to-be-learned memories from their emotional background context. Hence, by strengthening core memories and weakening their affective tone, sleep would eventually contribute to habituation processes and to reduce aversive reactions to stressful stimuli, participating in the regulation of emotions. Future studies should investigate further the interactions between sleep and emotion and especially understand causal and bidirectional links between sleep alterations and emotional disturbances.

Acknowledgments M.G. and G.D. were FRS-FNRS Research Fellow at the time of writing this manuscript.

References

Åkerstedt T, Kecklund G, Axelsson J (2007) Impaired sleep after bedtime stress and worries. Biol Psychol 76(3):170–173

Atienza M, Cantero JL (2008) Modulatory effects of emotion and sleep on recollection and familiarity. J Sleep Res 17(3):285–294

Baekeland F, Koulack D, Lasky R (1968) Effects of a stressful presleep experience on electroencephalograph recorded sleep. Psychophysiology 4(4):436–443

Baglioni C, Spiegelhalder K, Lombardo C, Riemann D (2010) Sleep and emotions: a focus on insomnia. Sleep Med Rev 14(4):227–238

Balon R (1996) Bupropion and nightmares. Am J Psychiatry 153(4):579–580

Baran B, Pace-Schott EF, Ericson C, Spencer RMC (2012) Processing of emotional reactivity and emotional memory over sleep. J Neurosci 32(3):1035–1042

Bartlett JC, Santrock JW (1979) Affect-dependent episodic memory in young children. Child Dev 50(2):513–518

Berger RH, Miller AL, Seifer R, Cares SR, Lebourgeois MK (2012) Acute sleep restriction effects on emotion responses in 30- to 36-month-old children. J Sleep Res 21(3):235–246

Bower B, Bylsma LM, Morris BH, Rottenberg J (2010) Poor reported sleep quality predicts low positive affect in daily life among healthy and mood-disordered persons. J Sleep Res 19(2):323–332

Brunner DP, Dijk DJ, Münch M, Borbély AA (1991) Effect of zolpidem on sleep and sleep EEG spectra in healthy young men. Psychopharmacology 104(1):1–5

Buzsaki G (2002) Theta oscillations in the hippocampus. Neuron 33(3):325–340

Cahill L, Haier RJ, Fallon J, Alkire MT, Tang C, Keator D, Wu J, McGaugh JL (1996) Amygdala activity at encoding correlated with long-term, free recall of emotional information. Proc Natl Acad Sci 93(15):8016–8021

Caldwell JA, Caldwell JL, Brown DL, Smith JK (2004) The effects of 37 hours of continuous wakefulness on the physiological arousal, cognitive performance, self-reported mood, and simulator flight performance of F-117A pilots. Mil Psychol 16(3):163–181

Cartwright RD, Wood E (1991) Adjustment disorders of sleep: the sleep effects of a major stressful event and its resolution. Psychiatry Res 39(3):199–209

Chuah LYM, Dolcos F, Chen AK, Zheng H, Parimal S, Chee MWL (2010) Sleep deprivation and interference by emotional distracters. Sleep 33(10):1305

Cluydts R, Visser P (1980) Mood and sleep. II. Effects of aversive pre-sleep stimulation. Wak Sleep 4(3):199

Corsi-Cabrera M, Miró E, del-Rıo-Portilla Y, Pérez-Garci E, Villanueva Y, Guevara MA (2003) Rapid eye movement sleep dreaming is characterized by uncoupled EEG activity between frontal and perceptual cortical regions. Brain Cogn 51(3):337–345

David D, De Faria L, Mellman TA (2006) Adjunctive risperidone treatment and sleep symptoms in combat veterans with chronic PTSD. Depress Anxiety 23(8):489–491

Davidson RJ (2002) Anxiety and affective style: role of prefrontal cortex and amygdala. Biol Psychiatry 51(1):68–80

Deliens G, Peigneux P (2013) One night of sleep is insufficient to achieve sleep-to-forget emotional decontextualization processes. Cogn Emot 1–9. doi:10.1080/02699931.2013.844105

Deliens G, Gilson M, Schmitz R, Peigneux P (2013a) Sleep unbinds memories from their emotional context. Cortex 49(8):2221–2228. doi:10.1016/j.cortex.2012.11.014

Deliens G, Neu D, Peigneux P (2013b) Rapid eye movement sleep does not seem to unbind memories from their emotional context. J Sleep Res 22(6):656–662. doi:10.1111/jsr.12065

Dinges DF, Pack F, Williams K (1997) Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. Sleep 20:267

Durmer JS, Dinges DF (2005) Neurocognitive consequences of sleep deprivation. Semin Neurol 25(1):117–129

Edéll-Gustaffson UM (2002) Insufficient sleep, cognitive anxiety and health transition in men with coronary artery disease: a self-report and polysomnographic study. J Adv Nurs 37(5):414–422

Feinberg I, Maloney T, Campbell IG (2000) Effects of hypnotics on the sleep EEG of healthy young adults: new data and psychopharmacologic implications. J Psychiatr Res 34(6):423–438

Franzen PL, Buysse DJ, Dahl RE, Thompson W, Siegle GJ (2009) Sleep deprivation alters pupillary reactivity to emotional stimuli in healthy young adults. Biol Psychol 80(3):300–305

Freud S (1990) The interpretation of dreams (trans: Crick J). Oxford University Press, London

Germain A, Buysse DJ, Ombao H, Kupfer DJ, Hall M (2003) Psychophysiological reactivity and coping styles influence the effects of acute stress exposure on rapid eye movement sleep. Psychosom Med 65(5):857–864. doi:10.1097/01.psy.0000079376.87711.b0

Gillin JC, Buchsbaum MS, Wu J, Clark C, Bunney W (2001) Sleep deprivation as a model experimental antidepressant treatment: findings from functional brain imaging. Depress Anxiety 14(1):37–49

Goel N, Rao H, Durmer JS, Dinges DF (2009) Neurocognitive consequences of sleep deprivation. Semin Neurol 29(4):320–339. doi:10.1055/s-0029-1237117

Graves LA, Heller EA, Pack AI, Abel T (2003) Sleep deprivation selectively impairs memory consolidation for contextual fear conditioning. Learn Mem 10(3):168–176

Grieser C, Greenberg R, Harrison RH (1972) The adaptive function of sleep: the differential effects of sleep and dreaming on recall. J Abnorm Psychol 80(3):280–286

Groch S, Wilhelm I, Diekelmann S, Born J (2013) The role of REM sleep in the processing of emotional memories: evidence from behavior and event-related potentials. Neurobiol Learn Mem 99:1–9

Gujar N, McDonald S, Nishida M, Walker MP (2011a) A role for REM sleep in recalibrating the sensitivity of the human brain to specific emotions. Cereb Cortex 21(1):115–123

Gujar N, Yoo SS, Hu P, Walker MP (2011b) Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. J Neurosci 31(12):4466–4474

Hauner KK, Howard JD, Zelano C, Gottfried JA (2013) Stimulus-specific enhancement of fear extinction during slow-wave sleep. Nat Neurosci 16(11):1553–1555. doi:10.1038/nn.3527

Hennevin E, Hars B, Maho C, Bloch V (1995) Processing of learned information in paradoxical sleep: relevance for memory. Behav Brain Res 69(1–2):125–135

Holland P, Lewis PA (2007) Emotional memory: selective enhancement by sleep. Curr Biol 17(5):R179–R181

Horne JA (1985) Sleep function, with particular reference to sleep deprivation. Ann Clin Res 17(5):199–208

Hoyt MA, Thomas KS, Epstein DR, Dirksen SR (2009) Coping style and sleep quality in men with cancer. Ann Behav Med 37(1):88–93

Hu P, Stylos-Allan M, Walker MP (2006) Sleep facilitates consolidation of emotional declarative memory. Psychol Sci 17(10):891–898

Introini-Collison IB, Dalmaz C, McGaugh JL (1996) Amygdala β-noradrenergic influences on memory storage involve cholinergic activation. Neurobiol Learn Mem 65(1):57–64

James JE, Gregg ME (2004) Effects of dietary caffeine on mood when rested and sleep restricted. Hum Psychopharmacol Clin Exp 19(5):333–341

Jones MW, Wilson MA (2005) Theta rhythms coordinate hippocampal–prefrontal interactions in a spatial memory task. PLoS Biol 3(12):e402

Kaestner EJ, Wixted JT, Mednick SC (2013) Pharmacologically increasing sleep spindles enhances recognition for negative and high-arousal memories. J Cogn Neurosci 25(10):1597–1610. doi:10.1162/jocn_a_00433

Kahn-Greene ET, Lipizzi EL, Conrad AK, Kamimori GH, Killgore WD (2006) Sleep deprivation adversely affects interpersonal responses to frustration. Pers Individ Differ 41(8):1433–1443

Kahn-Greene ET, Killgore DB, Kamimori GH, Balkin TJ, Killgore WD (2007) The effects of sleep deprivation on symptoms of psychopathology in healthy adults. Sleep Med 8(3):215–221

Kashani M, Eliasson A, Vernalis M (2012) Perceived stress correlates with disturbed sleep: a link connecting stress and cardiovascular disease. Stress 15(1):45–51

Kecklund G, Åkerstedt T (2004) Apprehension of the subsequent working day is associated with a low amount of slow wave sleep. Biol Psychol 66(2):169–176

Kenealy PM (1997) Mood state-dependent retrieval: the effects of induced mood on memory reconsidered. Q J Exp Psychol 50A(2):290–317. doi:10.1080/713755711

Killgore WD, Kahn-Greene ET, Lipizzi EL, Newman RA, Kamimori GH, Balkin TJ (2008) Sleep deprivation reduces perceived emotional intelligence and constructive thinking skills. Sleep Med 9(5):517–526

Kilpatrick L, Cahill L (2003) Amygdala modulation of parahippocampal and frontal regions during emotionally influenced memory storage. NeuroImage 20(4):2091–2099

Kim EJ, Dimsdale JE (2007) The effect of psychosocial stress on sleep: a review of polysomnographic evidence. Behav Sleep Med 5(4):256–278

Koniaris E, Drimala P, Sotiriou E, Papatheodoropoulos C (2011) Different effects of zolpidem and diazepam on hippocampal sharp wave-ripple activity in vitro. Neuroscience 175:224–234. doi:10.1016/j.neuroscience.2010.11.027

Lambert MT (2006) Aripiprazole in the management of post-traumatic stress disorder symptoms in returning global war on terrorism veterans. Int Clin Psychopharmacol 21(3):185–187

Lara Carrasco J, Nielsen TA, Solomonova E, Levrier K, Popova A (2009) Overnight emotional adaptation to negative stimuli is altered by REM sleep deprivation and is correlated with intervening dream emotions. J Sleep Res 18(2):178–187

Lauer C, Riemann D, Lund R, Berger M (1987) Shortened REM latency: consequence of psychological strain. Psychophysiology 24(3):263–271

Levin R, Nielsen T (2009) Nightmares, bad dreams, and emotion dysregulation: a review and new neurocognitive model of dreaming. Curr Dir Psychol Sci 18(2):84–88

Lewis PA, Critchley HD (2003) Mood-dependent memory. Trends Cogn Sci 7(10):431–433

Lewis P, Cairney S, Manning L, Critchley H (2011) The impact of overnight consolidation upon memory for emotional and neutral encoding contexts. Neuropsychologia 49(9):2619–2629

Lingenfelser TH, Kaschel R, Weber A, Zaiser-Kaschel H, Jakober B, Küper J (1994) Young hospital doctors after night duty: their task-specific cognitive status and emotional condition. Med Educ 28(6):566–572

Malcolm-Smith S, Koopowitz S, Pantelis E, Solms M (2012) Approach/avoidance in dreams. Conscious Cogn 21(1):408–412

Maloney KJ, Cape EG, Gotman J, Jones BE (1997) High-frequency γ electroencephalogram activity in association with sleep-wake

states and spontaneous behaviors in the rat. Neuroscience 76(2):541–555
Maquet P, Peters J, Aerts J, Delfiore G, Degueldre C, Luxen A, Franck G (1996) Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. Nature 383(6596):163–166
Marrosu F, Portas C, Mascia MS, Casu MA, Fà M, Giagheddu M, Imperato A, Gessa GL (1995) Microdialysis measurement of cortical and hippocampal acetylcholine release during sleep-wake cycle in freely moving cats. Brain Res 671(2):329–332
Marshall L, Kirov R, Brade J, Mölle M, Born J (2011) Transcranial electrical currents to probe EEG brain rhythms and memory consolidation during sleep in humans. PLoS One 6(2):e16905
Mauss IB, Troy AS, LeBourgeois MK (2013) Poorer sleep quality is associated with lower emotion-regulation ability in a laboratory paradigm. Cogn Emot 27(3):567–576
McGaugh JL (2004) The amygdala modulates the consolidation of memories of emotionally arousing experiences. Annu Rev Neurosci 27:1–28
McNamara P, McLaren D, Smith D, Brown A, Stickgold R (2005) A "Jekyll and Hyde" within aggressive versus friendly interactions in REM and non-REM dreams. Psychol Sci 16(2):130–136
Mikoteit T, Brand S, Beck J, Perren S, von Wyl A, von Klitzing K, Holsboer-Trachsler E, Hatzinger M (2012) Visually detected NREM stage 2 sleep spindles in kindergarten children are associated with stress challenge and coping strategies. World J Biol Psychiatry 13(4):259–268
Mikoteit T, Brand S, Beck J, Perren S, Von Wyl A, Von Klitzing K, Holsboer-Trachsler E, Hatzinger M (2013) Visually detected NREM stage 2 sleep spindles in kindergarten children are associated with current and future emotional and behavioural characteristics. J Sleep Res 22(2):129–136. doi:10.1111/j.1365-2869.2012.01058.x
Minkel J, Htaik O, Banks S, Dinges D (2010) Emotional expressiveness in sleep-deprived healthy adults. Behav Sleep Med 9(1):5–14
Minkel JD, Banks S, Htaik O, Moreta MC, Jones CW, McGlinchey EL, Simpson NS, Dinges DF (2012) Sleep deprivation and stressors: evidence for elevated negative affect in response to mild stressors when sleep deprived. Emotion 12(5):1015
Moscovitch M, Nadel L (1998) Consolidation and the hippocampal complex revisited: in defense of the multiple-trace model. Curr Opin Neurobiol 8:297–300
Motomura Y, Kitamura S, Oba K, Terasawa Y, Enomoto M, Katayose Y, Hida A, Moriguchi Y, Higuchi S, Mishima K (2013) Sleep debt elicits negative emotional reaction through diminished amygdala-anterior cingulate functional connectivity. PLoS One 8(2):e56578
Nishida M, Pearsall J, Buckner RL, Walker MP (2009) REM sleep, prefrontal theta, and the consolidation of human emotional memory. Cereb Cortex 19(5):1158–1166
Nofzinger EA, Mintun MA, Wiseman M, Kupfer DJ, Moore RY (1997) Forebrain activation in REM sleep: an FDG PET study. Brain Res 770(1):192–201
Ochsner KN, Gross JJ (2005) The cognitive control of emotion. Trends Cogn Sci 9(5):242–249
Pace-Schott EF, Shepherd E, Spencer R, Marcello M, Tucker M, Propper RE, Stickgold R (2011) Napping promotes inter-session habituation to emotional stimuli. Neurobiol Learn Mem 95(1):24–36
Pace-Schott EF, Verga PW, Bennett TS, Spencer R (2012) Sleep promotes consolidation and generalization of extinction learning in simulated exposure therapy for spider fear. J Psychiatr Res 46(8):1036–1044
Pallesen S, Johnsen BH, Hansen A, Eid J, Thayer JF, Olsen T, Hugdahl K (2004) Sleep deprivation and hemispheric asymmetry for facial recognition reaction time and accuracy. Percept Mot Skills 98(3 Pt 2):1305–1314
Pare D, Collins DR, Pelletier JG (2002) Amygdala oscillations and the consolidation of emotional memories. Trends Cogn Sci 6(7):306–314
Payne JD, Kensinger EA (2011) Sleep leads to changes in the emotional memory trace: evidence from fMRI. J Cogn Neurosci 23(6):1285–1297. doi:10.1162/jocn.2010.21526
Payne JD, Nadel L (2004) Sleep, dreams, and memory consolidation: the role of the stress hormone cortisol. Learn Mem 11(6):671–678
Payne JD, Stickgold R, Swanberg K, Kensinger EA (2008) Sleep preferentially enhances memory for emotional components of scenes. Psychol Sci 19(8):781–788
Peigneux P, Smith C (2010) Memory processing in relation to sleep. In: Kryger M, Roth T, Dement W (eds) Principles and practice of sleep medicine, 17th edn. Elsevier, Philadelphia, pp 335–347
Perogamvros L, Schwartz S (2012) The roles of the reward system in sleep and dreaming. Neurosci Biobehav Rev 36(8):1934–1951. doi:10.1016/j.neubiorev.2012.05.010
Petrovich GD, Canteras NS, Swanson LW (2001) Combinatorial amygdalar inputs to hippocampal domains and hypothalamic behavior systems. Brain Res Rev 38(1):247–289
Phelps EA, LeDoux JE (2005) Contributions of the amygdala to emotion processing: from animal models to human behavior. Neuron 48(2):175
Pilcher JJ, Huffcutt AI (1996) Effects of sleep deprivation on performance: a meta-analysis. Sleep 19(4):318–326
Pinter MM, Pogarell O, Oertel WH (1999) Efficacy, safety, and tolerance of the non-ergoline dopamine agonist pramipexole in the treatment of advanced Parkinson's disease: a double blind, placebo controlled, randomised, multicentre study. J Neurol Neurosurg Psychiatry 66(4):436–441
Popa D, Duvarci S, Popescu A, Léna C, Paré D (2010) Coherent amygdalocortical theta promotes fear memory consolidation during paradoxical sleep. Proc Natl Acad Sci 107(14):6516–6519
Prather AA, Bogdan R, Hariri AR (2013) Impact of sleep quality on amygdala reactivity, negative affect, and perceived stress. Psychosom Med 75(4):350–358
Revonsuo A (2000) The reinterpretation of dreams: an evolutionary hypothesis of the function of dreaming. Behav Brain Sci 23(6):877–901
Reynolds CF, Hoch CC, Buysse DJ, Monk TH, Houck PR, Kupfer DJ (1993) REM sleep in successful, usual, and pathological aging: the Pittsburgh experience 1980–1993. J Sleep Res 2(4):203–210
Rosales-Lagarde A, Armony JL, del Río-Portilla Y, Trejo-Martínez D, Conde R, Corsi-Cabrera M (2012) Enhanced emotional reactivity after selective REM sleep deprivation in humans: an fMRI study. Front Behav Neurosci 6. doi:10.3389/fnbeh.2012.00025
Rose M, Manser T, Ware JC (2008) Effects of call on sleep and mood in internal medicine residents. Behav Sleep Med 6(2):75–88
Schmidt C, Collette F, Cajochen C, Peigneux P (2007) A time to think: circadian rhythms in human cognition. Cogn Neuropsychol 24(7):755–789
Schredl M, Wittmann L (2005) Dreaming: a psychological view. Dreaming 484:92
Schwartz S (2004) What dreaming can reveal about cognitive and brain functions during sleep? A lexico-statistical analysis of dream reports. Psychol Belg 44(1/2):5–42
Schwartz S, Maquet P (2002) Sleep imaging and the neuro-psychological assessment of dreams. Trends Cogn Sci 6(1):23–30
Schwarz JFA, Popp R, Haas J, Zulley J, Geisler P, Alpers GW, Osterheider M, Eisenbarth H (2013) Shortened night sleep impairs facial responsiveness to emotional stimuli. Biol Psychol 93(1):41–44. doi:10.1016/j.biopsycho.2013.01.008
Scott JPR, McNaughton LR, Polman RCJ (2006) Effects of sleep deprivation and exercise on cognitive, motor performance and mood. Physiol Behav 87(2):396–408

Siegel JM (2005) Clues to the functions of mammalian sleep. Nature 437(7063):1264–1271

Smith MR, Antrobus JS, Gordon E, Tucker MA, Hirota Y, Wamsley EJ, Ross L, Doan T, Chaklader A, Emery RN (2004) Motivation and affect in REM sleep and the mentation reporting process. Conscious Cogn 13(3):501–511

Solms M (2000) Dreaming and REM sleep are controlled by different brain mechanisms. Behav Brain Sci 23(6):843–850

Sterpenich V, Albouy G, Boly M, Vandewalle G, Darsaud A, Balteau E, Dang-Vu TT, Desseilles M, D'Argembeau A, Gais S, Rauchs G, Schabus M, Degueldre C, Luxen A, Collette F, Maquet P (2007) Sleep-related hippocampo–cortical interplay during emotional memory recollection. PLoS Biol 5(11):e282

Sterpenich V, Albouy G, Darsaud A, Schmidt C, Vandewalle G, Dang Vu TT, Desseilles M, Phillips C, Degueldre C, Balteau E, Collette F, Luxen A, Maquet P (2009) Sleep promotes the neural reorganization of remote emotional memory. J Neurosci 29(16):5143–5152

Strange BA, Dolan RJ (2004) β-Adrenergic modulation of emotional memory-evoked human amygdala and hippocampal responses. Proc Natl Acad Sci USA 101(31):11454–11458

Strauch I, Meier B (1996) In search of dreams: results of experimental dream research. State University of New York Press, Albany

Talamini LM, Bringmann LF, de Boer M, Hofman WF (2013) Sleeping worries away or worrying away sleep? physiological evidence on sleep–emotion interactions. PLoS One 8(5):e62480. doi:10.1371/journal.pone.0062480

Tempesta D, Couyoumdjian A, Curcio G, Moroni F, Marzano C, De Gennaro L, Ferrara M (2010) Lack of sleep affects the evaluation of emotional stimuli. Brain Res Bull 82(1–2):104–108. doi:10.1016/j.brainresbull.2010.01.014

Tucker AM, Whitney P, Belenky G, Hinson JM, Van Dongen HPA (2010) Effects of sleep deprivation on dissociated components of executive functioning. Sleep 33(1):47

van der Helm E, Walker MP (2009) Overnight therapy? The role of sleep in emotional brain processing. Psychol Bull 135(5):731–748. doi:10.1037/a0016570

van der Helm E, Gujar N, Walker MP (2010) Sleep deprivation impairs the accurate recognition of human emotions. Sleep 33(3):335

van der Helm E, Gujar N, Nishida M, Walker MP (2011) Sleep-dependent facilitation of episodic memory details. PLoS One 6(11):e27421. doi:10.1371/journal.pone.0027421

Vandekerckhove M, Cluydts R (2010) The emotional brain and sleep: an intimate relationship. Sleep Med Rev 14(4):219–226

Vandekerckhove M, Weiss R, Schotte C, Exadaktylos V, Haex B, Verbraecken J, Cluydts R (2011) The role of presleep negative emotion in sleep physiology. Psychophysiology 48(12):1738–1744

Vandekerckhove M, Kestemont J, Weiss R, Schotte C, Exadaktylos V, Haex B, Verbraecken J, Gross JJ (2012) Experiential versus analytical emotion regulation and sleep: breaking the link between negative events and sleep disturbance. Emotion 12(6):1415

Vriend JL, Davidson FD, Corkum PV, Rusak B, Chambers CT, McLaughlin EN (2013) Manipulating sleep duration alters emotional functioning and cognitive performance in children. J Pediatr Psychol. doi:10.1093/jpepsy/jst033

Wagner U, Gais S, Born J (2001) Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. Learn Mem 8(2):112–119

Wagner U, Fischer S, Born J (2002) Changes in emotional responses to aversive pictures across periods rich in slow-wave sleep versus rapid eye movement sleep. Psychosom Med 64(4):627–634

Wagner U, Degirmenci M, Drosopoulos S, Perras B, Born J (2005) Effects of cortisol suppression on sleep-associated consolidation of neutral and emotional memory. Biol Psychiatry 58(11):885–893

Wagner U, Hallschmid M, Rasch B, Born J (2006) Brief sleep after learning keeps emotional memories alive for years. Biol Psychiatry 60(7):788–790

Walsh JK, Hall-Porter JM, Griffin KS, Dodson ER, Forst EH, Curry DT, Eisenstein RD, Schweitzer PK (2010) Enhancing slow wave sleep with sodium oxybate reduces the behavioral and physiological impact of sleep loss. Sleep 33(9):1217

Wamsley EJ, Hirota Y, Tucker MA, Smith MR, Antrobus JS (2007) Circadian and ultradian influences on dreaming: a dual rhythm model. Brain Res Bull 71(4):347–354

Yoo SS, Gujar N, Hu P, Jolesz FA, Walker MP (2007) The human emotional brain without sleep—a prefrontal amygdala disconnect. Curr Biol 17(20):R877–R878

Yu SS, Rugg MD (2010) Dissociation of the electrophysiological correlates of familiarity strength and item repetition. Brain Res 1320:74–84

Zohar D, Tzischinsky O, Epstein R, Lavie P (2005) The effects of sleep loss on medical residents' emotional reactions to work events: a cognitive-energy model. Sleep 28(1):47–54