UNIVERSITÀ
DEGLI STUDI
DELL'AQUILA

DISCAB
Dipartimento di Scienze
Cliniche Applicate
e Biotecnologiche

UNIVERSITY OF L'AQUILA

Department of Biotechnological and Applied Clinical Sciences

PhD in Experimental Medicine

Curriculum: Basic and Clinical Neuroscience

XXXVI cycle

Understanding the role of REM sleep fragmentation on emotional memory and emotional reactivity through a new methodological approach

S.S.D: M-PSI/02

PhD Candidate

Lorenzo Viselli

Course coordinator

Prof. Mariagrazia Perilli

Supervisor

Prof. Michele Ferrara

a.y. 2022/2023

Contents

Summary

Sleep is fundamental to allow proper memory and emotional processing. In particular, rapid eye movement (REM) sleep duration and continuity are supposed to be crucial for ensuring adequate encoding and consolidation of emotional events. However, how REM sleep acts on the emotional reactivity associated with the emotional information needs to be clarified. Different methodological limitations affect the studies in this field. Recently, studies on clinical populations highlighted that REM sleep fragmentation (manifested in cortical arousals and stage transitions that disrupt the continuity of REM periods) negatively interferes with emotional processing that is assumed to occur during REM sleep. Therefore, experimentally induced REM sleep fragmentation in healthy participants could be adopted as an alternative paradigm to investigate REM sleep's effects on emotional memory and emotional reactivity, overcoming existing methodological limitations. In this view, we aim to investigate the role of REM sleep in consolidating emotional memory and modulating emotional reactivity by inducing REM sleep fragmentation through cortical arousals elicited via a wristband delivering vibrotactile stimulations. Recruited participants underwent two experimental conditions (Fragmentation and Control) in a counterbalanced order. In each condition, the participants took part in: *i)* a baseline assessment of emotional reactivity followed by a stimulus encoding phase and a baseline assessment of emotional memory in the afternoon (at 18:00); *ii)* a nocturnal polysomnographic recording with 64-channel high-density EEG of the nocturnal sleep with (Fragmentation) or without (Control) vibrotactile stimulation; *iii)* a post-sleep emotional memory and emotional reactivity assessment one hour after the final

awakening, (iv) and a follow-up assessment of emotional reactivity and emotional memory after 48 hours. The effects of the experimental manipulation of REM sleep continuity on emotional memory and emotional reactivity for negative and neutral stimuli were evaluated by collecting behavioural (old/new paradigm), self-report (self-assessment manikin scale), and physiological (skin conductance responses and heart rate deceleration) measurements. We demonstrated that experimentally induced REM sleep fragmentation led to a lack of psychophysiological habituation to previously encountered emotional stimuli in healthy participants without affecting their subjective emotional reactivity ratings and emotional memory consolidation. Moreover, we revealed that re-exposing participants to emotional stimuli after a night of experimental REM sleep fragmentation did not allow this information to be correctly processed during two subsequent nights of regular sleep. Finally, we proved that our new methodological approach of experimentally induced REM sleep fragmentation is able to hinder REM sleep continuity, minimising macrostructural sleep alteration.

Altogether, our data support the importance of unperturbed REM sleep continuity in promoting a proper psychophysiological reaction to known emotional events.

1. Sleep and emotional processing

Sleep is a regular and recurring behavioural state that alternates with wakefulness and is characterised by significant central and peripheral physiological modifications (Kryger, Roth & Dement, 2010). During sleep, people undergo an increased response threshold to sensory input, a decreased motor output, and a modified state of consciousness. Nonetheless, sleep can be distinguished from other states of quiescence due to its rapid reversibility towards wakefulness through sufficient stimulation (Lesku, Martinez-Gonzalez & Rattenborg, 2009).

Sleep is a fundamental component of human health (Lowe, Satati & Hall, 2017). A healthy adult's recommended amount of sleep falls between 7 and 9 hours. Generally, sleeping less than 7 hours per night is associated with adverse negative health outcomes such as weight loss or obesity, type 2 diabetes, heart disease, hypertension, stroke, reduced immune function, lowered muscular strength, and an elevated risk of mortality (Craven et al., 2022; Watson et al., 2015).

Although there is a focus on the quantity of experienced sleep, it is acknowledged that sleep quality is also crucial for optimal psychophysical well-being (Scott et al., 2021). Sleep quality, partially dependent on duration, also relies on sleep latency, the number of nocturnal awakenings, the time spent awake after sleep onset, waking up too early compared to the desired time, the amount of arousal during the night, and the subjective satisfaction of the sleep experience (McCarter et al., 2022; Nelson, Davis & Corbett, 2022).

While sleep may outwardly appear as a homogeneous state, it is, in reality, a complex process comprised of distinct stages. Sleep comprises rapid eye movement (REM) sleep and non-rapid eye movement (NREM) sleep. NREM sleep is, in turn, subdivided into three stages (NREM1, NREM2, and NREM3) depicting different degrees of sleep depth (Berry et al., 2015). All these stages intricately delineate the essence of sleep, each possessing unique characteristics and fulfilling a specific role (Rasch & Born, 2013). For instance, the NREM stage primarily consolidates declarative and semantic memories. At the same time, REM sleep is associated with consolidating procedural memories and seems involved in emotional processing (Diekelmann & Born, 2010; Diekelmann, Wilhem & Born 2009).

The deleterious effects of inadequate sleep are not solely confined to physical health. Inadequate sleep primarily influences psychological functioning, significantly compromising cognitive performance. For instance, recent meta-analyses show how inadequate sleep compromises the ability to learn, consolidate, and remember information and is also associated with reduced attention, distorted perception, and impaired executive functions (Lo et al., 2016; Newbury & Monaghan, 2019; Newbury et al., 2021; Qin et al., 2023).
While inadequate sleep adversely affects cognitive functions, poor sleep quality and quantity have an even more significant impact on mood and emotional functioning (Pilcher & Huffcutt, 1996).

Among the several negative consequences on human health related to poor sleep quality, the present work focuses on the role of disturbed human sleep on emotional processing, starting by outlining the role of sleep in mood and emotions.

1.1 Sleep, mood and emotions

As highlighted above, sleep is crucial for optimal psychophysiological functioning, but emotions also influence our entire lives. For instance, emotions affect decision-making ability, social interactions, learning capacity, and health (Engen & Anderson, 2018; Ferrer & Mendes, 2018; Skurvydas et al., 2021). Numerous pieces of evidence emphasise how sleep influences our emotional functioning and how our emotions affect our sleep (Kahn et al., 2014; Tempesta et al., 2018).

Theoretically, there are six basic emotions: anger, happiness, sadness, disgust, surprise, and fear (Ekman, 1970). Sleep research has yet to investigate the relationship between basic emotions and sleep directly, except for a few exceptions, but has focused more on some aspects of these emotions. For example, many studies have explored the relationship between sleep and irritability, tension, pleasure, anxiety, depression, hostility, empathic abilities, calmness, and confusion. Furthermore, many studies have investigated sleep effects on the more general concept of mood, encompassing positive and negative aspects or adopting broader concepts such as overall distress (a composite measure of anger, depression and anxiety), bringing together emotions and mood in relation to sleep. Despite mood and emotions being distinct constructs (Gray & Watson, 2001), their relationship with sleep appears clear and coherent for both concepts. Therefore, we will address mood and emotions in relation to sleep together in this context.

Regarding negative mood and emotions, numerous studies support a primary role for sleep. To go into detail, Hruska, Anderson & Barduhn (2022) reported anger levels that were 18 to 35% higher in emergency medical services

professionals who reported poor sleep quality relative to colleagues with good sleep quality. Moreover, irrespective of their usual sleep quality, workers denounce higher levels of anger when they face poorer sleep than usual (Hruska, Anderson & Barduhn, 2022). Recently, the relationship between perceived sleep quality and anger was evaluated in a large sample of university students (Arbinaga Joaquin-Mingorance & Fernández-Cuenca, 2019). After assessing sleep quality and anger through validated questionnaires, the authors reported a remarkable relationship between these variables. Specifically, participants with a sleep efficiency < 85% reported greater state-anger. Likewise, subjects who reported sleeping < 7 hours per night scored higher on both state-anger and trait-anger. Similarly, state-anger and trait-anger are higher in participants with higher sleep latencies. In addition, a study based on a larger (N=4695) and older (age range 20–69) sample reported similar results (Shin et al., 2005). Here, the authors highlighted a positive significant association between trait-anger levels and sleep disturbances. In particular, difficulty initiating and maintaining sleep and early morning awakenings were associated with more significant trait-anger levels. It should be noted that sleep and anger are not associated only in the adult population. Many studies emphasise how this relationship is already present in children and adolescents. In a sample of N=8950 preschool children (4 years old), Scharf et al. (2013) found that children who sleep less than 9.45 hours have higher odds of exhibiting anger, aggression, impulsivity, overactivity, tantrums, and annoying behaviours relative to their peers who sleep longer. Likewise, adolescents (range age 14–18) report greater anger levels following one night of total sleep deprivation (TSD) (Short & Louca, 2015). Similar findings were reported by a

study with a larger (N=2767, range age 12–16) sample, in which adolescents with reduced or borderline total sleep time relative to the suggested one (7 hours) reported higher feelings of anger and more norm-breaking behaviour (e.g., stealing, drug use, violence et cetera) (Bauducco et al., 2016).

Research investigating the relationship between sleep and feelings of fear is scarce. Generally, researchers explore the link between sleep and fear in terms of fear conditioning and extinction learning rather than as a direct correlation between sleep and fear feelings. Nevertheless, some authors assessed the relationship between sleep and fear by focusing on the ability to recognise fearful facial expressions under sleep deprivation/restriction conditions. Overall, these studies report that inadequate sleep does not compromise the ability to identify fearful facial expressions (Cote et al., 2014; Huck et al., 2008; Killgore et al., 2017). The authors of these studies posit that the ability to recognise fear is an evolutionary advance, making this skill more resistant to the deleterious effects of sleep loss. Despite this, inadequate sleep compromises the ability to recognise happiness and sadness (Huck et al., 2008; Killgore et al., 2017). Researchers have conducted few studies on the relationship between disgust, surprise and sleep. Indeed, studies in this field have primarily evaluated recognition abilities for the visual expression of these emotions. Overall, studies indicate the absence of a sleep effect on the ability to recognise disgust and surprise (Beattie et al., 2015; de Almondes et al., 2016; Killgore et al., 2017). Moreover, these results are confirmed also in clinical populations. For instance, Crönlein et al. (2016) compared the recognition ability of the six basic emotions among insomniac subjects, subjects with sleep apnoea, and healthy controls. They found no significant differences in recognising emotions of disgust,

surprise, anger and fear between healthy and clinical participants. Nonetheless, clinical subjects exhibited a reduced ability to recognise the affective state of happiness and sadness compared to the control cohort. Similar results were also highlighted in healthy subjects under sleep deprivation conditions (Killgore et al., 2017). Interestingly, several authors attribute the lack of results in this type of experiment to the methodological approach (e.g., tasks with low ecological validity), emphasising that, in most studies, tasks involve static images of faces expressing emotion presented on the screen for a brief period (up to 5 seconds). Indeed, some authors have sought to investigate this relationship using more ecological methodologies. For instance, Holding et al. (2017) substituted emotional video clips for static images, expanding the range of emotions to be recognised to 12 (fear, pleasure, disgust, despair, anxiety, sadness, relief, pride, happiness, interest, irritation, and anger). Furthermore, the study was composed of two different experiments: in the first one, sleep was subjectively assessed through the Karolinska Sleep Diary, while in the second one, sleep variables were objectively categorised via actigraphy. Despite the methodological precautions, the authors found only a positive correlation between self-reported sleep quality and accuracy in recognising disgust. No other sleep variables, whether self-reported or objectively assessed, were associated with emotion recognition. Additionally, Sack, Brier & Anders (2019) evaluated if recognition ability under a sleep deprivation condition could be affected by different stimulus presentation times (2s vs 4s vs 8s vs 10s stimuli duration). In this study, the authors highlighted no significant differences in the ability to recognise emotions when the exposure time of the image ranged from 2s to 4s between subjects in normal sleep and sleep

deprivation conditions. Surprisingly, when stimulus exposure times reached 8/10s, sleep-deprived participants showed better emotion recognition abilities than subjects in the regular sleep group. Thus, these studies emphasise how subtle variations in the methodology can bring out differences that would otherwise go unnoticed. Furthermore, other authors attempted to evaluate if inadequate sleep could differently impact the ability to recognise the valence of the facial expressions of emotion (e.g., if the face shows a negative or positive or neutral emotion) and the ability to recognise the valence of the emotional content of words (e.g., if the written word is associated with a negative, positive or neutral emotion) (Maccari et al., 2014). Here, the proficiency in recognising the emotional content of the words deteriorated during sleep deprivation conditions for all the valences. Conversely, the ability to recognise the emotional valence of the facial expression was compromised only when the facial expression was neutral during sleep deprivation. Overall, the inconsistent results in these studies underscore the importance of a correct methodological approach in evaluating the relationship between sleep and emotions to prevent conflicting results due to confounding effects.

In contrast, the results of the literature appear more coherent regarding the relationship between sadness and sleep. In addition to compromising the ability to recognise the facial expressions of sadness and happiness (Killgore et al., 2017), inadequate sleep is associated with higher levels of sadness the following day (Patapoff et al., 2022). Furthermore, sleep-deprived individuals also visually appear sadder. Sundelin et al. (2013) asked participants to evaluate face pictures of individuals after regular sleep or after 31 hours of TSD on 12 aspects (including looking sad). Among the others, the faces of subjects

in the sleep deprivation condition were rated significantly sadder. As mentioned earlier, the relationship between sleep and emotion is bidirectional. In addition to the effect of sleep on sadness, it is acknowledged that sadness emerges as a significant predictor of poor sleep quality and lower total sleep time (Kahn et al., 2014; Kalmbach et al., 2014). The relationship between sleep and sadness is also solid in the younger population. For instance, Garnow et al. (2021) found a positive relationship between sadness and sleep difficulties in a large sample (N=1489) of adolescents (age range 15–17). Similarly, Settineri et al. (2012) found a positive association between daytime sleepiness and sadness in a sample of approximately 2000 adolescent/young adults (age range 16–22). Finally, Otsuka et al. (2020) reported comparable results on a broader sample of Japanese adolescents (N=64329, age range 12–19, 53,9% males), highlighting higher levels of sadness associated with poor sleep quality.

Although emotions are divided into six basic emotions, restricting the investigation of the relationship between sleep and emotion solely based on these constructs is limiting. Various aspects and behaviours contribute to forming the six basic emotions. We can conceptualise emotions broadly by categorising them as positive or negative. Furthermore, emotions represent a momentary state in response to an event not encompassing the more enduring concept of mood, which represents the individual's general affective disposition (again, positive or negative) that influences their attitude towards life (Gray & Watson, 2001). Indeed, sleep researchers have not limited their study of the relationship between sleep and emotions to predetermined categories, such as the six basic emotions. Here, the literature is broader and highlights the detrimental effect of inadequate sleep on emotional functioning. Acheson et al.

(2007), using the Profile of Mood States (McNair et al., 1971) (a standardised questionnaire providing both a general measure of total mood disturbance and scores on six different subscales: anxiety, depression, anger, vigour, fatigue, confusion), highlighted an increase in levels of anger, anxiety, depression, and confusion in a sample (N=20, age range 18–45) subjected to 24 hours of TSD. Different studies have replicated similar results, which show that TSD causes a general decline in mood while also increasing levels of depression, anxiety, stress, and fatigue (Babson et al., 2010; Ben Simon et al., 2020; Campbell, Feldner & Leen-Feldner, 2022; Sagaspe et al., 2006; Scott, McNaughton & Polman, 2006; Vardar et al., 2007). Moreover, Thompson et al. (2022) highlighted compromised emotional and cognitive functioning with higher levels of depression, anxiety, and anger, accompanied by altered biomarkers of inflammation and cortisol levels after a single night of TSD. Studies that extended beyond a single night of TSD also reported similar results. For example, Skurvydas et al. (2021) reported a significant worsening of mood alongside reduced motivation in a sample of young adults (N=36, mean age 21.2 years) after two nights of TSD. Similarly, healthy participants (N=25, age range 20–35) subjected to 56 hours of TSD reported increased pathological symptomatology relative to baseline. Specifically, participants reported greater depression, anxiety, paranoia, and somatic complaints as assessed by the Personality Assessment Inventory (Morey, 2014). Interestingly, studies of prolonged sleep deprivation reveal large interindividual variability in the deleterious effects of sleep deprivation (Floros et al., 2021; Kuna et al., 2012; Rupp et al., 2012). For instance, Beutler et al. (2003), assessing healthy subjects (N=48, age range 18–25) after 30 and 60 hours of sleep deprivation,

found worsened depressive symptomatology. However, looking at the results carefully, the authors highlighted individual differences in response to sleep deprivation. Specifically, they were able to distinguish three different subgroups of participants regarding the effects of sleep deprivation on depressive symptoms. Most of the participants (n=25) showed a significant, albeit modest, worsening of depressive symptomatology; other participants (n=11) showed a massive worsening of depressive symptomatology, while the remaining participants (n=12) showed a significant improvement in depressive symptomatology. Furthermore, the participants with the worst response showed worsening symptomatology throughout measurements. In contrast, the worsening/improvement in symptomatology in the other participants was evident after 30 hours of deprivation and remained stable at 60 hours.

Sleep restriction studies also found deleterious effects of inadequate sleep on negative affect. For example, Kahn et al. (2014) subjected a sample of adults (N=61, age range 20–29) to three different experimental conditions: one night of normal sleep, one night of sleep restriction (4 hours), and one fragmentation night (awakenings induced every 90 minutes lasting 10/15 minutes). Here, in both sleep manipulation conditions, participants reported an increase in levels of depression, fatigue, and confusion compared to control. Again, even when sleep is restricted to 5 hours for a single night, there seems to be a general impairment in mood accompanied by a worsening in depression, anger, confusion, vigour, and fatigue levels (Romney et al., 2016). Furthermore, in conditions of partial sleep restriction, it seems that the deleterious effects of sleep deprivation on mood are cumulative. Reducing sleep duration by 33% (relative to the average obtained during a typical week of sleep monitored via

actigraphy), mood appears significantly worse after the first day of sleep restriction, but the impairment continues to rise, resulting in a significantly worse mood on the fourth day relative to the first one of sleep restriction (Cote et al., 2008). Similar results were also reported in the young population. Lo et al. (2017) subjected healthy participants (N=47, age range 15–19) to two cycles of sleep restriction (5 hours per night for five nights in each sleep restriction cycle) interspersed with two nights in which participants were allowed to sleep 9 hours per night. The results showed a cumulative worsening in mood, cognitive functions, and subjective alertness over time. Interestingly, the authors highlighted that these neurobehavioral functions did not fully recover after the two recovery nights. Moreover, the second cycle of sleep restriction accelerates the deterioration of neurobehavioral functions. It is worth noting that participants who took a 1-hour nap in the afternoon experienced a slower decay in both cognitive performance and mood levels (Lo et al., 2017). Other studies in younger populations reported similar results. For instance, Baum et al. (2014) studied the effects of chronic sleep restriction (6.5 hours per night for five nights) on mood and emotional regulation in a sample of adolescents (N=50, age range 14–17). Through a cross-over design, participants were subjected to chronic sleep restriction and a regular sleep condition (10 hours of bedtime for five nights) and monitored via actigraphy. The authors report that adolescents in the sleep restriction condition are significantly more depressed, anxious, and irritable. Furthermore, McMakin et al. (2016) involved an even younger population (age range 11.5–15) investigating the effects of two nights of sleep restriction (4 hours per night) versus two nights of normal sleep (10 hours per night) on social interactions in a peer social context and self-reported mood.

When subjected to sleep restriction, early adolescents report increased negative affect and greater negative affective behaviour in a peer social context. In addition, some authors assessed if different amounts of sleep restriction were able to induce different effects among younger people. Booth et al. (2021) recruited a sample of adolescents (N=34, age range 15–17) that underwent nine nights of sleep with 9.5 hours in bed per night before being randomly assigned to one of the following three groups: 5 hours of sleep for five nights, 7.5 hours of sleep for five nights, and 10 hours of sleep for five nights. The authors report worsening levels of depression, anxiety, and confusion only in the 5-hour sleep restriction condition. Moreover, happiness levels increased in the group of subjects who were allowed to sleep for 10 hours compared to the baseline. Finally, in this experiment, each group was allowed two recovery nights (10 hours of sleep) before being reassessed on the study variables. The authors highlight how negative emotions in the 5-hour group did not return to baseline after recovery nights (Booth et al., 2021). It is interesting to note the consistency of the deleterious effects of sleep on emotional functioning. As reported above, studies with varying sleep deprivation methodologies, samples, and methods to assess participants' emotional states obtained nearly identical results.

It should be noted that our emotional life is not composed solely of negative emotions. Happiness or joy represents another fundamental emotion included in the six basic emotions. As previously reported, sleep deprivation compromises the ability to recognise the facial expression of this emotion (Killgore et al., 2017), but it also compromises the ability to experience happiness or joy (Fuligni & Hardway, 2006; Jackowska et al., 2011; Ong et al.,

2017; Troxel et al., 2009). For example, Jackowska et al. (2011) highlighted a positive association between sleep efficiency and happiness. Furthermore, other authors showed how happiness experienced during the day is positively associated with subsequent sleep duration (Fuligni & Hardway, 2006).

However, focusing solely on joy as a basic emotion would limit the review of the effects of sleep on positive affect. Sleep is known to impact the positive emotional domain in a broader sense negatively. Kaida & Niki (2013) highlighted how a single night of TSD, in addition to increasing negative affect, also reduces positive affect. Moreover, these effects are also observed in conditions of sleep restriction. For example, Rossa et al. (2014) showed that a single night of sleep restriction to 4 hours significantly reduced positive affect. Similarly, Saksvik-Lehouillier et al. (2020) showed that lowering the habitual sleep time by 2 hours for three consecutive nights had a negative impact on positive affect. Furthermore, Cote et al. (2009), restricting sleep to 3 hours for two nights, highlighted a dose-dependent effect of sleep restriction on positive mood. Participants exhibited a significant reduction in positive affect after the first night of sleep restriction relative to baseline. Furthermore, the impairment in the positive effect was significantly higher after the second night of sleep restriction than the first (Cote et al., 2009). Some authors evaluated whether the younger population is more sensitive to the deleterious effects of sleep loss on positive mood. Schwarz et al. (2019) conducted a study comparing a sample of young adults (18–30 years, N=63) and older adults (60–72 years, N=47) to see if deprivation would have a more negative impact on the younger adult group. Both groups were subjected to 30 hours of TSD and a night of normal sleep. After TSD, younger subjects reported higher levels of stress and a

significantly greater reduction in positive affects. Moreover, the other variables investigated (affect, depression, confusion, tension, anger, fatigue, total mood disturbance, and irritability) showed a significant worsening only in the young adult population, indicating that older adults are more resilient to the deleterious effects of sleep on mood. In this regard, it is worth citing the study by Talbot et al. (2010), which also included an adolescent population (early adolescence N=20 age range 10–13; mid-adolescence N=24 age range 13–16; and adulthood N=20 age range 30–60). Here, a significant worsening of positive effects associated with increased anxiety was observed in all the groups investigated, regardless of age. Notwithstanding that, when presented with emotional stimuli, the early adolescents evaluated these stimuli as significantly more threatening than other age groups (Talbot et al., 2010). The deleterious effects of sleep deprivation and restriction in adolescents have been repeatedly confirmed by others (e.g., Dagys et al., 2012; Lo, Ong, et al., 2016). Recently, the effects of sleep restriction in prepubertal children have also been investigated (N=53, age range 7 – 11), confirming a worsening of positive affect after inadequate sleep even in this population (Alfano et al., 2020). Notably, these evidences have recently been included in an in-depth meta-analysis by Palmer et al. (2023). The authors investigated the effects of different experimental sleep manipulation methods on emotions. Specifically, they reviewed the effects of sleep deprivation, restriction, and fragmentation on emotions. The authors emphasise that the most deleterious effects of sleep on emotions concern the domain of positive emotions, with larger effects given by TSD. However, effects also emerge following sleep restriction and sleep fragmentation. Small effect sizes for the negative affect domain were reported

only for sleep deprivation and restriction studies. Similarly, medium effect sizes emerge for general mood disturbances only for sleep deprivation and restriction studies. However, the authors emphasise that fragmentation studies' results should be considered cautiously. The literature in this area is limited, and the assessed emotional aspects are very different, limiting the statistical power of meta-analytical procedures.

In conclusion, a robust bidirectional relationship between sleep and the emotional domain is evident. Sleep can influence our emotional functioning, not only in terms of our ability to experience certain emotions but also to recognise them in others, not merely as a visual expression of emotions but also our ability to empathise with others' emotional experiences (Amicucci et al., 2021). Inadequate sleep compromises our emotional functioning broadly and dose-dependently. The greater our sleep alteration, the greater the emotional repercussions we experience. Furthermore, it should be emphasised that our sleep influences our emotional functioning, and our emotional experiences condition our subsequent sleep. This effect is not only limited to an impairment of our sleep due to the experience of negative emotions, but it also extends to the beneficial effects of positive emotions on our sleep.

Now, we will see how the relationship between sleep and emotions goes beyond the emotional experiences, influencing our ability to manage the emotions we endure based on the quality of our sleep.

1.2 Sleep and emotional regulation

Emotional regulation refers to the process through which we attempt to influence our emotions, when we have them, how we experience them, and how we express them (Gross et al., 1998). It can lead to changes in the latency, magnitude, and duration of emotion at both behavioural and experiential/physiological levels (Gross, 2002). Emotional regulation applies to positive and negative emotions, can be either an automatic or conscious process, and is adaptively based on the context. In other words, a specific emotion regulation strategy can be functional in some contexts and dysfunctional in others (Gross et al., 1998). The most widely recognised and accepted theoretical model of emotion regulation is Gross's Process Model of Emotion Regulation (Gross et al., 1998). This model differentiates five families of emotional regulation strategies along the emotion-generative process, assuming that individuals can employ emotional regulation strategies at different stages. The first type of applicable strategies are the situation selection strategies. These strategies are implemented to avoid or approach specific places, individuals, or things to regulate the emotion that might arise from those situations before it is experienced. Another type of applicable strategies are the situation modification strategies. These strategies encompass modifying a situation to reduce/change the emotional impact it might have. The third type of strategy in the model are attentional deployment strategies, which involve choosing which aspects of the situation to focus attention on. Such strategies include distraction, concentration, and rumination. The fourth type of strategy in the model, cognitive changes, encompasses strategies to alter the interpretation of the emotion-eliciting experience. Generally, this appraisal

modification helps decrease the magnitude of the emotional response. The last emotional regulation strategy is implemented after the onset of emotion and, thus, after generating response tendencies. These strategies refer to attempts to influence the emerged emotional response tendency. Researchers categorise the five families of emotional regulation strategies as antecedent-focused and response-focused (McRae & Gross, 2020; Palmer & Alfano, 2017). Antecedent-focused strategies are the strategies that we use before our emotional response tendencies affect our behaviour and peripheral physiology. Response-focused strategies refer to the strategies an individual uses after their emotion have already started and they have generated a set of response tendencies (Gross, 2002; McRae & Gross, 2020; Palmer & Alfano, 2017).

Generally, emotional regulation strategies belonging to cognitive change (e.g., reappraisal) are considered inherently functional, compared to other families of emotional regulation strategies (e.g., situation selection) that are considered inherently dysfunctional (Aldao & Nolen-Hoeksema, 2012). More importantly, a strategy's “functionality” nature may depend on the context in which it occurs (Aldao et al., 2014). It is worth noting that individuals who habitually employ inherently adaptive emotional regulation strategies tend to exhibit greater psychological well-being. Conversely, those habitually utilising inherently maladaptive emotional regulation strategies tend to show poorer psychological health (Aldao & Nolen-Hoeksema, 2012; Conklin et al., 2015; Navas-Casado et al., 2023). Besides the habitual use of adaptive/maladaptive emotion regulation strategies, flexibility in using these strategies based on contextual aspects is another fundamental feature of psychological well-being (Aldao et al., 2014; Aldao & Nolen-Hoeksema, 2012).

This brief description of emotion regulation strategies shows how properly regulating emotions plays a significant role in psychological health. Similarly, experiencing adequate sleep is another fundamental aspect of psychological well-being. Indeed, it is not surprising that researchers have investigated the relationship between sleep and emotional regulation. Researchers sustain that emotional regulation links psychological well-being and mental health (Palmer & Alfano, 2017) since sleep can alter our ability to use emotion regulation strategies (Lollies et al., 2022; Palmer & Alfano, 2017; Tomaso et al., 2021). A recent meta-analysis highlighted that inadequate sleep is associated with a reduced ability to use adaptive emotional regulation strategies without altering maladaptive emotional regulation strategies (Tomaso et al., 2021). These results were also confirmed in adolescent and child populations (Lollies et al., 2022).

Beyond the concept of intrinsically adaptive or maladaptive emotional regulation strategies, we can consider the effects of inadequate sleep on emotional regulation strategies by examining how sleep influences certain behaviours associated with the five families of emotion regulation strategies proposed in Gross's model (Gross et al., 1998).

Cognitive change and response modulation are the most studied families of emotion regulation strategies in relation to sleep. Concerning cognitive change, reappraisal is the primary strategy in this category, and many researchers have examined the effects of inadequate sleep on the functional capacity to adopt this emotional regulation strategy. A critical study conducted on 156 adults (mean age 43.5, age range 26–60) highlighted that poor sleep quality is associated with a reduced ability to regulate emotions through cognitive

reappraisal. This result held even after controlling for important potential confounding variables (e.g., sleep duration, caffeine use, time of day, verbal intelligence, working memory capacity, mood, anxiety, stress, emotional reactivity, age, and gender), suggesting that the relationship between poor sleep quality and the inability to use cognitive reappraisal properly is not due to third-variable confounds (Mauss, Troy & LeBourgeois, 2013). Results replicated in different sleep deprivation studies revealed that individuals under sleep deprivation conditions are less able to regulate their emotional experiences through cognitive reappraisal (Li et al., 2023; Stenson et al., 2021; Zhang, Lau & Hsiao, 2019). However, some authors do not report the deleterious effects of inadequate sleep on cognitive reappraisal (Boon et al., 2023; Reddy et al., 2017; Shermohammed, Kordyban & Somerville, 2019). For instance, Reddy et al. (2017) investigated the ability to use reappraisal to decrease negative emotional responses in a group of adolescents (N = 42; age range 13–17), comparing a night of sleep restriction (4 hours) with a night of regular sleep (9.5 hours). Despite participants under sleep restriction reporting higher levels of anxiety and negative mood, as well as a greater response to negative emotional stimuli, the use of cognitive reappraisal was able to downregulate the negative emotional response in both conditions. Similarly, Shermohammed, Kordyban & Somerville, (2019) investigated the ability to use cognitive reappraisal in a group of adults (N=34, age range 18–30), comparing a regular night of sleep with a night of TSD, finding no differences in the ability to use cognitive reappraisal between the two conditions. Despite contrasting findings in the literature, a recent meta-analysis (Tomaso et al., 2021) highlighted that inadequate sleep significantly reduces the ability to use

adaptive emotional regulation strategies (including cognitive reappraisal) following inadequate sleep.

Regarding the effects of sleep on response modulation, it is essential to consider that this family of strategies is more complex and broader than others. Firstly, this type of response involves altering the emotional, behavioural, and physiological response to an emotional event. Therefore, these strategies can range from "simple" techniques, such as the voluntary suppression of emotional experiences, to complex behaviours, such as substance abuse, used as a strategy to mitigate the psychophysiological reaction to an emotional event. Therefore, studying sleep's effects on these emotional regulation strategies is challenging. However, expressive suppression, an emotional regulation strategy within the family of response modulation strategies, has been extensively studied. Expressive suppression is considered a maladaptive emotional regulation strategy and involves the active effort to inhibit/suppress the emotional response elicited by the situation. The ability to use expressive suppression as a mechanism for emotional regulation is unaffected by sleep deprivation/fragmentation (Boon et al., 2023; Zhang et al., 2019). Furthermore, it is interesting to note that insomnia patients adopt expressive suppression as their preferential emotion regulation strategy (Harvey, 2001; Mojsa-Kaja & Ivcevic, 2023). In addition, insufficient sleep can impair an individual's behavioural functioning, leading to maladaptive behaviours such as substance use that may serve as dysfunctional emotional regulation strategies (Gross et al., 1998; López-Muciño et al., 2022; Valentino & Volkow, 2020).

Regarding the other emotion regulation strategies (situation selection, situation modification, attentional deployment), studies have yet to explicitly focus on

how sleep impacts their use. However, studies investigating the relationship between sleep and specific behaviours that underlie these emotion regulation strategies provide insights into the effect of sleep on these strategies. For example, concerning situation selection, sleep loss significantly compromises individuals' likelihood of engaging in positive emotional situations. Individuals with inadequate sleep generally participate less in social activities than good sleepers, reporting lower social well-being (Gordon et al., 2017). However, such behaviours may also be associated with automatic mechanisms, as evidenced by decreased motivation to participate in social and enjoyable activities. For instance, it is known that sleepiness is associated with reduced motivation to engage in social activities (Axelsson et al., 2020). These effects could automatically lead individuals to avoid potential emotional contexts without an active and conscious intent of emotion regulation. For the situation modification strategies, studies indicate that inadequate sleep compromises the behavioural abilities underlying these emotion regulation strategies. For instance, inadequate sleep impairs decision-making skills (Harrison & Home, 2000; Salfi et al., 2020). Moreover, sleep deprivation makes individuals more impulsive, compromising the ability to choose one situation over another (Liu et al., 2022). Furthermore, empathic abilities (Amicucci et al., 2021; Guadagni et al., 2014), self-monitoring, and social interaction abilities (Christian & Ellis, 2011; Harrison & Home, 2000) are altered following inadequate sleep, compromising the ability to modify an event to reduce/alter its emotional impact. Finally, attentional allocation involves internal modifications rather than external environmental changes like the previous strategies. As mentioned above, most of these strategies refer to distraction, rumination, or focused attention on specific

aspects of the experience to limit the emotional experience. As is well-known, sleep deprivation compromises our attentional functioning (Alhola & Polo-Kantola, 2022; Hudson, Van Dongen & Honn, 2020; Shenfield et al., 2020). Sleep deprivation compromises the ability to shift attention from one thing to another (Couyoumdjian et al., 2010; Whitney et al., 2017). Moreover, sustained attention, a fundamental concentration characteristic, is significantly compromised under sleep deprivation/restriction conditions (Shenfield et al., 2020). Therefore, sleep firmly impairs emotional regulation abilities based on attentional deployment due to its deleterious effects on attentional functioning.

Despite the categorisation proposed by Gross's process model, emotion regulation strategies could be collapsed into adaptive (e.g., reappraisal) and maladaptive (e.g., expressive suppression) strategies. In a recent meta-analysis, Tomaso et al. (2021) assert that inadequate sleep compromises the ability to use adaptive emotional regulation strategies without interfering with maladaptive ones. Instead, particularly concerning dysfunctional behaviours due to sleep loss (e.g., impulsivity, aggression, substance abuse, attention deficits), maladaptive strategies seem to be used more frequently after inadequate sleep.

It is evident that healthy sleep can facilitate the use of more functional strategies for regulating emotional experiences. Furthermore, irrespective of the specific emotional regulation strategy used (adaptive vs. maladaptive), flexibility in employing different strategies plays a crucial role in psychological well-being (Aldao et al., 2014; Aldao & Nolen-Hoeksema, 2012). Additionally, it is interesting to note that similar to the relationship between sleep, emotions, and mood, the relationship between sleep and emotional regulation capacity is

bidirectional. Not only does inadequate sleep compromise our ability to use emotional regulation strategies flexibly, but also the inappropriate use of emotional regulation strategies can compromise our sleep quality (Mazzer et al., 2019; Thomsen et al., 2003; Vandekerckhove & Wang, 2018). For instance, Demichelis et al. (2023) highlighted that emotional regulation skills can protect against the deleterious effects of inadequate sleep. In their study, 740 participants (mean age = 34.77, age range = 18 – 65) completed a series of validated questionnaires to investigate sleep quality, stress, aggression, and difficulties in emotional regulation. The results, besides highlighting that poor sleep quality was associated with higher levels of stress, aggression, and worse emotional regulation capacity, revealed a mediation effect of emotional regulation skills on aggression levels. Specifically, emotional regulation partially mediated the relationship between sleep quality and verbal aggression, anger, and hostility while fully mediated the relationship between sleep quality and physical aggression.

Sleep's role in emotional processing is not confined to affecting our emotions nor to how sleep affects our ability to cope with our emotional experiences. As we will see in the next session, sleep also plays a fundamental role in emotional memory and reactivity.

1.3 Sleep, emotional memory, and emotional reactivity

Sleep plays a crucial role in memory formation. Older theories sustained that sleep facilitated memory formation by avoiding interference from external stimuli, reducing the likelihood of interfering stimuli exposure due to the

quiescent state sleep induces in individuals (Jenkins & Dallenbach, 1924). However, contemporary and widely accepted theories propose an active role of sleep in memory formation rather than a passive role merely involving disconnection from the external world. According to the synaptic homeostasis hypothesis (Cirelli & Tononi, 2022; Tononi & Cirelli, 2003, 2014, 2020), learning episodes during wakefulness strengthen synaptic connections in cortical networks, leading to an increase in the system's energy demands, reducing the learning capacity and affecting information processing. Consequently, during subsequent sleep, specifically during slow-wave sleep (SWS), the strength of these synaptic connections is linearly reduced, restoring cellular homeostasis. This active process of synaptic reduction indirectly consolidates memories by eliminating weak synaptic connections formed during wakefulness, improving the signal-to-noise ratio for more strongly encoded memory traces. Moreover, it allows the brain to renew its ability to acquire information during subsequent wakefulness (Cirelli & Tononi, 2022; Tononi & Cirelli, 2003, 2014, 2020). Other theories also emphasise the importance of REM sleep in memory formation. For instance, the two-process model of memory consolidation (Marshall & Born, 2007; Plihal & Born, 1997) suggests that REM and NREM sleep are involved in processing different information types. Specifically, NREM sleep, particularly SWS, is crucial for processing declarative memories, while REM sleep is essential for elaborating procedural and emotional memories. In addition, other authors emphasise the (uninterrupted) cyclic alternation of SWS and REM sleep for proper memory processing rather than differentiating the effects of sleep stages based on the type of information they are supposed to process. The sequential hypothesis (Giuditta, 2014; Giuditta et al., 1995) asserts that

SWS and REM sleep play sequential and complementary roles in processing memories acquired during wakefulness. Specifically, during SWS, unnecessary memories are eliminated or weakened, while relevant memories are preserved/strengthened. Then, relevant memories are integrated into pre-existing memory networks during the subsequent REM period. Another influential theory that partially integrates aspects of the previous theories is the active system consolidation hypothesis (Diekelmann & Born, 2010; Klinzing, Niethard & Born, 2019; Rasch & Born, 2013). According to this model, memory consolidation during sleep is an active process originating from repeated reactivation of newly encoded memory representations. During SWS, slow oscillations (SO, rhythmic electrical activity <1 Hz) guide the repeated reactivation of hippocampal memory representations in conjunction with hippocampal sharp-wave-ripples (SW-Rs, hippocampal neural events characterised by a fast depolarising wave superimposed by a ~200 Hz field potential named “ripple”) and thalamocortical spindles (waxing-and-waning waves with a frequency range between 10–15 Hz involved in inducing enduring plasticity changes in cortical areas) to redistribute and integrate neo-acquired hippocampal information into pre-existing cortical memory networks. This process induces qualitative changes in long-term memory stores that need stabilisation. The stabilisation process should occur during the subsequent REM sleep periods (Diekelmann & Born, 2010; Klinzing, Niethard & Born, 2019; Rasch & Born, 2013).

Overall, neural plasticity processes occurring during sleep actively ensure memory processing. These processes allow our brain to encode information without saturating, consolidate encoded information by integrating it into pre-

existing neural networks, and facilitate recalling the consolidated information when necessary. Indeed, inadequate sleep can compromise all these three stages of memory processing (Cousins & Fernández, 2019; Newbury et al., 2021; Rasch & Born, 2013).

On the other hand, what happens when the information to be processed is emotional? Emotional memories refer to memories of events able to elicit an emotional response (Faul & LaBar, 2020). These events are characterised by a psychophysiological reaction, namely the emotional reactivity, associated with the emotional experience. It is believed that the emotional reaction associated with the event allows for better consolidation of the emotional information. According to the memory modulation hypothesis (Roozendaal & Mcgaugh, 2011), significant emotional experiences activate brain and hormonal systems that modulate memory formation. Specifically, noradrenergic activation of the basolateral amygdala induced by the emotional event would regulate the consolidation of memories through interaction with different brain areas (e.g., hippocampus) responsible for the consolidation of recent experiences. Therefore, it is unsurprising that emotional memories have a privileged status in memory (LaBar & Cabeza, 2006). Indeed, emotional events are better and longer remembered than other types of information.

1.3.1. The impact of sleep on emotional memory encoding

Researchers sustain that sleep plays an active role in elaborating emotional information, similar to its involvement in other categories of information (Cunningham, Stickgold & Kensinger, 2022; Tempesta et al., 2018). Some

studies assessed sleep's role in encoding emotional information by manipulating sleep prior to a learning task. In two different experiments, Kaida et al. (2015) investigated the effects of TSD and selective REM sleep deprivation (REMD) on the ability to encode neutral and emotional stimuli compared to a regular sleep condition. Two groups of participants (TSD, n=14, mean age 21.4; REM n=18, mean age 22.0) underwent the experimental condition (TSD or REMD) and the regular sleep night in a counterbalanced cross-over design. After each condition, subjects underwent an emotional picture learning task, and their performance was evaluated immediately after the encoding phase (D1) and seven days later (D8). Participants' adherence to the TSD condition was monitored through actigraphy, while participants in the REMD condition slept in the laboratory on both REMD and control nights. REMD was induced by administering sounds at the onset of the first signs of REM sleep in the electroencephalogram (EEG) trace. Overall, the ability to encode stimuli was compromised after TSD but not following REMD or regular sleep, regardless of the emotional valence of the stimuli. Moreover, sleep manipulation by TSD and REMD did not influence the ability to evaluate the emotional reactivity associated with the pictures, assessed through self-reported valence and arousal ratings of the administered stimuli. Similarly, Tempesta et al. (2016) investigated the effect of one night of TSD on the ability to encode contextual and non-contextual emotional information. Contextual material involves retrieving specific details associated with the test item (e.g., the temporal order of events). In contrast, non-contextual memories involve only the ability to recognise if an item has been encountered before. Participants recruited in this study (N=48, mean age 21.6, age range 19–28)

were randomly assigned to a TSD or regular sleep night before a learning phase of emotional stimuli (6 film clips of different emotional valences, 2 neutral, 2 negative, and 2 positive). Subsequently, both groups were allowed two nights of regular sleep before assessing their mnemonic performance to avoid the potential confounding effects of sleep deprivation. The results highlighted a significantly compromised ability to encode contextual and non-contextual memory aspects following TSD. Moreover, participants in TSD performed equally to participants in regular sleep conditions in recognising non-contextual negative events, suggesting that the ability to encode negative information is more resistant to the deleterious effects of sleep deprivation (Tempesta et al., 2016). Walker & Stickgold (2006) reported a similar effect in subjects who learned words with negative, positive, and neutral valence after 36 hours of TSD. Participants were tested on their ability to recognise these stimuli after two nights of regular sleep. Participants who executed the learning task after TSD showed significantly worse performance in recognising positive stimuli than subjects in the control condition. Despite the TSD, recognition performance for negative and neutral stimuli was equal to that of subjects allowed to sleep before the encoding phase.

According to these studies, encoding emotional information requires adequate sleep. However, some events (e.g., adverse events) could be consolidated regardless of previous sleep.

1.3.2 The impact of sleep on emotional memory consolidation

After properly encoding, the newly acquired information must be adequately consolidated to avoid forgetting. Different studies have investigated the effects

of sleep on the ability to consolidate emotional information and the associated emotional reactivity. In this regard, Wagner and colleagues (Wagner, Gais & Born, 2001; Wagner et al., 2006) highlighted how post-learning sleep positively influences the retention of emotional memories even years later. In their first study (Wagner, Gais & Born (2001), participants had to learn text with negative or neutral emotional content before a re-test session after about 3 hours. During this interval, one group of participants could sleep normally (n=12), while another group had to stay awake (n=11). In the first experimental condition, participants in the sleep group could sleep from 23:00 to 02:00 after the learning phase to experience SWS-rich sleep before the memory performance re-test (around 02:30). A week later, in a within-subject design, participants in the sleep group could sleep from 03:00 to 06:00 to experience REM-rich sleep before the memory performance re-test (around 06:30). Here, memory performance was significantly better for emotional information relative to neutral one only after REM-rich sleep. Then, the authors point out that emotional memories benefit more from REM-rich sleep than SWS-rich sleep Wagner, Gais & Born, (2001). Four years later, the same participants were contacted and tested again on the previously learned material (Wagner et al., 2006). Despite the elapsed time, participants allowed to sleep after learning showed better performance for emotional stimuli than participants in the wake group, regardless of whether they belonged to the SWS-rich or REM-rich sleep condition. The beneficial effect of sleep on the consolidation of emotional information has been replicated in different studies. For example, Hu, Stylos-Allan & Walker, (2006) reported a positive and selective effect of post-learning sleep on the ability to consolidate negative emotional pictures. Similarly, (Cox et al., 2018) evaluated the impact

of post-learning sleep on the consolidation of emotional memories by subjecting participants to an emotional picture-encoding task and investigating the contextual memory of these stimuli (i.e., the ability to recognise the stimuli' location during the encoding phase). After the encoding phase and an immediate re-test, a 12-hour interval followed, including sleep or wakefulness. Participants in the sleep group (n=46) performed the encoding task with an immediate re-test in the evening and a delayed re-test in the morning. Task timing was reversed in the wake group (n=25). In this study, no significant differences emerged in the ability to recognise stimuli after 12 hours, regardless of the stimuli valence, between the two groups. Nevertheless, the contextual memory of negative stimuli was selectively consolidated only in the sleep group. Moreover, the contextual memory of neutral stimuli in the sleep group and the contextual memory for both categories of stimuli in the wake group were impaired after a 12-hour interval.

Other researchers have investigated the role of sleep in consolidating emotional memories not only through manipulating sleep itself but also by considering the subjective quality of participants' sleep. Tempesta et al. (2015) assessed the effects of sleep on emotional memory and emotional reactivity by comparing good sleepers (GS, n=31), poor sleepers (PS, n=30), and a group of participants undergoing one night of TSD (n=23). Sleep quality was determined through a validated questionnaire (Pittsburgh Sleep Quality Index, PSQI, Buysse et al., 1989; Curcio et al., 2013), and participants categorised as PS were monitored through actigraphy to objectively confirm their poor sleep condition. Participants in this study underwent an emotional picture encoding task (10:00, Day 1) followed by an immediate re-test (11:00, Day 1) before being

sleep deprived (TSD) or spending a regular night of sleep at home (GS and PS). Then, emotional memory and reactivity were tested again (11:00, Day 2). During the encoding task, participants were required to indicate the valence and arousal of the stimuli to assess the associated emotional reactivity. Valence and arousal values for the images were also assessed during the re-test to evaluate variations in emotional reactivity due to the condition. The results highlighted memory recognition performance impairment only after TSD. Meanwhile, PS had a memory performance equivalent to GS's. Moreover, TSD participants rated positive and neutral images more negatively than GS. Similarly, PS participants rated neutral images more negatively than GS. No differences were found in the arousal associated with stimuli between the groups after each condition. This study shows how TSD compromises the emotional memory consolidation process and predisposes individuals to perceive stimuli more negatively. It also highlights how the ability to consolidate emotional information is preserved in individuals with poor sleep quality, even though they report a more negative perception of stimuli (Tempesta et al., 2015). The same authors subsequently investigated the effects of sleep on the consolidation of emotional memories and associated reactivity through a more ecological paradigm using emotional film clips instead of static images (Tempesta et al., 2017). Participants recruited in this study were randomly assigned to a TSD condition or a regular sleep condition. Before each condition, participants performed an encoding task in which 6 video clips (2 positives, 2 negatives, and 2 neutrals) were shown. In this study, the re-test phase took place 48 hours after the encoding phase, allowing TSD subjects to sleep normally before the test. This recovery sleep night was granted to eliminate side effects of sleep deprivation

that could have biased the results, affecting the retrieval abilities. TSD participants reported significantly worse recognition performance than subjects in the regular sleep condition. The results highlighted how post-encoding sleep is crucial for properly consolidating information, regardless of the emotional valence of the stimuli to be encoded. Other authors have also adopted video clips as encoding material instead of static images to investigate the effects of sleep on the consolidation of emotional information. For example, Van Heugten-Van Der Kloet et al. (2015) subjected participants (N=56, mean age 20.7, age range 18–29) to a 36-hour TSD condition (n=28) or a regular sleep night (n=28) before assessing memory performance to emotionally negative, neutral, and positive cartoons video clips. Emotional memory performance was assessed through free recall, where participants were instructed to write down everything they remembered about each video clip. The results highlighted that after TSD, memory performance was significantly impaired only for positive stimuli. Meanwhile, the experimental condition did not affect the recall of negative and neutral information. Not limiting the evaluation to emotional memory but also considering the emotional reactivity associated with the stimuli, (Chambers & Payne, 2014) assessed the effects of sleep on the consolidation of emotional memories and emotional reactivity associated with positive stimuli. Participants recruited in this study were assigned to a sleep condition (n=37) or a daytime wakefulness condition (n=33). Before each condition, participants were shown video clips (15 seconds) of cartoons, after which participants had to evaluate mood, valence, arousal, and familiarity. The results highlighted a better ability to recall positive emotional information only in the sleep group, while memory for neutral stimuli was equivalent in both conditions. Additionally, emotional

reactivity associated with positive stimuli was reduced only in the sleep group. Thus, the authors emphasise how the effects of sleep on negative memories and emotional reactivity associated with this category of stimuli should also be extended to positive stimulus categories.

The number of studies that investigated the effects of sleep on the ability to consolidate emotional information is massive. Indeed, various reviews and meta-analyses have been recently carried out to systematically assess the results of previous research on the relationship between sleep and emotional memory (Cunningham, Stickgold & Kensinger, 2022; Davidson et al., 2021; Lipinska et al., 2019; Schäfer et al., 2020; Tempesta et al., 2018). In their review, Tempesta et al. (2018) focused on exploring the effects of sleep on emotional processing. Here, it is emphasised that sleep is crucial to ensure correct encoding and consolidation processes of emotional information. Furthermore, it is reported that sleep loss prevents consolidation processes, leading to the decay of emotional memory traces. Additionally, the authors emphasised that the beneficial effects of post-encoding sleep on emotional memory consolidation are present even after short periods of sleep (naps). This review also highlighted how sleep (especially REM sleep) appeared involved in the consolidation and extinction processes of fear memories. In a subsequent meta-analysis, Lipinska et al. (2019) investigated whether there was a preferential consolidation for emotional memories compared to neutral ones during sleep. In this context, preferential consolidation referred to a greater difference in performance in favour of emotional material compared to neutral material after a time interval containing sleep compared to wakefulness. However, the authors did not find a preferential consolidation effect for

emotional over neutral information. Specifically, the results showed that emotional information was better remembered than neutral information both after sleep and wakefulness. Moreover, the authors highlighted how the study methodology influenced the results, pointing out two methodological conditions that moderated the relationship between sleep and emotional memory performance. Specifically, the type of outcome measure (free recall vs. stimulus recognition) and controlling for baseline performance (subtracting baseline performance from post-experimental condition performance) moderated the results, leading to outcomes where the difference between emotional and neutral material in post-sleep performance was significantly greater than the wake condition (highlighting a preferential sleep effect for the consolidation of emotional information through different methodology). Furthermore, the authors emphasised that other methodological characteristics (e.g., a full night of sleep instead of a nap as a sleep condition, TSD instead of daytime waking as a control condition) or sample characteristics (e.g., age) should be carefully considered in future studies, as they could influence the results. Subsequently, another meta-analysis was conducted on the same topic (Schäfer et al., 2020) to investigate whether sleep selectively improved memories for emotional stimuli compared to neutral ones. Schäfer et al. (2020) confirmed that memory performance for emotional memories was higher than for neutral memories both after a period of sleep and after a period of wakefulness. They also highlighted that memory performance (both emotional and non-emotional) was always better after a period of sleep than after a period of wakefulness. However, as in Lipinska et al. (2019), the hypothesised selective effect of sleep on emotional information was not reported. Conversely, a substantial difference

in memory performance was found between emotional (better remembered) and neutral material in wake compared to sleep. According to the authors, this result suggests that consolidation processes during wakefulness are selective for emotional information, while sleep acts to consolidate information regardless of its emotional valence. Moreover, additional analyses regarding the specific effects of SWS and REM sleep on the consolidation of emotional information revealed that REM sleep selectively consolidates this type of information (Schäfer et al., 2020). Subsequently, two other reviews have investigated the relationship between sleep and emotional memories, reasserting how sleep was essential for consolidating memories regardless of their emotional valence. For example, Davidson et al. (2021), despite finding few studies that highlighted a preferential consolidation effect for emotional over neutral information, emphasised that most studies did not support this hypothesis. Indeed, in most of the reviewed works, sleep benefits memories regardless of their emotional valence. Finally, noteworthy is the recent review by Cunningham, Stickgold & Kensinger (2022) examining the effects of sleep and sleep loss on different memory stages of episodic emotional memory. Here, the authors emphasised how TSD compromised all stages of memory processing and how good sleep improved memory regardless of the stimuli's emotional valence compared to wakefulness. They also highlighted that the protocols used in studies investigating the relationship between sleep and emotional memories lead to confounding factors (e.g., circadian factors and different levels of sleep pressure) that made it challenging to interpret the results, especially when attempting to explore the differential effects of SWS and REM sleep.

Overall, sleep emerges as crucial to ensuring proper emotional memory consolidation, although adverse emotional events may be partially consolidated even under conditions of sleep deprivation.

Finally, less is known about the effects of the sleep on the third stage of emotional memory processing. Indeed, there has yet to be a specific human study on the effects of sleep on the ability to recall emotional information. Nevertheless, it is believed that sleep also plays a fundamental role in the ability to recall emotional information properly (Cunningham et al., 2022; Davidson et al., 2021; Lipinska et al., 2019; Schäfer et al., 2020) although this effect has been explicitly investigated only in an animal study where pre-retrieval sleep for consolidated emotional information was shown to be crucial for recall emotional information (Fernandes-Santos et al., 2012).

1.3.3 The effect of sleep on emotional reactivity

Different studies report decreased emotional reactivity after a night of regular sleep (Chambers & Payne, 2014; Cunningham et al., 2014; Pace-Schott et al., 2011). For example, (Cunningham et al., 2014) assessed the effects of sleep on emotional reactivity and memory by exposing participants to a regular night of sleep (n=18) or a diurnal wake condition (n=21). In the wake condition, participants performed an encoding task of negative and neutral emotional stimuli between 8:00 and 10:00, with a re-test after approximately 12 hours (between 20:00 and 22:00). In the nocturnal sleep condition, the timing of tasks was reversed. Additionally, emotional reactivity was assessed through subjective measures (self-reported arousal and valence ratings) and objective

measures (skin conductance response SCR, heart rate deceleration HRD) during both encoding and re-test phases. The results revealed a significant reduction in HRD and SCR only in the sleep condition, with no differences observed in HRD and SCR in the wake group. Furthermore, the authors reported a positive correlation between physiological activation levels during image viewing at encoding (HRD and SCR) and mnemonic performance for those stimuli during retrieval, but only in the sleep group. These results demonstrate that a sleep period is essential to ensure a downregulation of emotional reactivity associated with emotional events. Moreover, despite the belief that strong emotional activation enhances memory for that event, the authors emphasise that this process requires the intervention of a sleep period. Moreover, although some authors report an increase in emotional reactivity after a sleep period (Wagner et al., 2002), many researchers report the preservation of emotional reactivity after sleep (Ashton et al., 2019; Harrington et al., 2018; Prehn-Kristensen et al., 2017). For instance, in the Ashton et al. (2019) study, participants (N=48) underwent either a nocturnal sleep condition (n=24, mean age 20.04) or a diurnal wake condition (n=24, mean age 19.96). Before each condition, participants performed an encoding task of emotional stimuli in which emotional reactivity was assessed through SCR and HRD, as well as subjective ratings of valence and arousal. Measures of emotional reactivity were evaluated again after 12 hours during the memory performance task. The results showed maintenance of HRD and SCR values and self-reported valence and arousal after the sleep condition, indicating that sleep preserves the emotional reactivity associated with emotional stimuli.

Researchers have also explored the link between sleep and emotional reactivity in the context of sleep deprivation. Studies on sleep deprivation highlight how the lack of sleep increases emotional reactivity to negative events, decreases emotional reactivity to positive events, and alters the ability to interpret neutral events (Lipinska et al., 2022; Pilcher, Callan & Posey, 2015; Tempesta et al., 2010, 2015). For instance, Tempesta et al. (2010) reported that a night of TSD leads individuals to evaluate neutral stimuli more negatively, indicating a negative bias in event interpretation due to sleep loss. Furthermore, Zohar et al. (2005) reported that sleep loss significantly reduces emotional reactivity to positive daily events while significantly increasing how individuals respond to disruptive events. Even studies that adopt objective measures to assess emotional reactivity replicate these results. For example, Franzen et al. (2009) reported an increase in pupillary dilation (an indicator of affective and cognitive processing) in response to negative emotional stimuli in a group of participants subjected to TSD, while pupillary dilation in response to stimuli retained in subjects after a night of regular sleep.

As evident from the works mentioned above, outcomes regarding the role of sleep on emotional reactivity are contradictory, so it is necessary to clarify whether sleep reduces or preserves emotional reactivity. Nevertheless, researchers believe that sleep deprivation results in heightened emotional reactivity. Today, the scientific community widely accepts that REM sleep processes emotional information. According to the literature, REM sleep plays a role in processing emotional reactivity linked to emotional memories. As we will see in the next paragraph, studies on the role of REM sleep in emotional memory and emotional reactivity have led to the development of two opposing

hypotheses regarding the effects of this sleep stage on the emotional reactivity associated with the emotional event.

1.3.4 The role of REM sleep on emotional memory and emotional reactivity

As mentioned above, a growing body of literature emphasises the primary role of REM sleep in emotional processing (Schäfer et al., 2020; Schenker et al., 2021; Tempesta et al., 2018; Vandekerckhove & Wang, 2018). Furthermore, as early as 1997, it was hypothesised that REM sleep is directly involved in processing procedural and emotional information (Plihal & Born, 1997). Based on this hypothesis, various studies have sought to investigate the specific effects of REM sleep on emotional memory and the associated emotional reactivity (Baran et al., 2012; Carollo, Degasperi & Cellini, 2022; Groch et al., 2013, 2015; Kaida, Niki & Born, 2015; Lara-Carrasco et al., 2009; Morgenthaler et al., 2014; Pace-Schott et al., 2011; Wagner et al., 2002; Werner et al., 2021; Wiesner et al., 2015). Researchers primarily utilised the split-night design and the selective REM sleep deprivation paradigms to study this relationship. In the split-night design paradigm, one group of participants sleeps in the first part of the night (e.g., 00:00–03:00), experiencing SWS-rich sleep (and poor in REM sleep), while another group of participants (or the same group in a different session in a within-subject design) sleeps in the second part of the night (e.g., 04:00–07:00) theoretically experiencing REM-rich sleep (and poor in SWS). Alternatively, in the selective REM sleep deprivation (REMD) paradigm, participants are awakened (e.g., through acoustic stimulation) whenever they

enter the REM sleep stage during the night to minimise the time spent in this sleep stage.

By significantly reducing or almost eliminating the time spent in REM sleep throughout the night, these studies aimed to understand the role of REM sleep in the consolidation of emotional memories and associated psychophysiological reactivity.

1.3.4.1 Behavioural and psychophysiological studies about the role of REM sleep on emotional memory and emotional reactivity

In one of the early works in this field, the split-night paradigm was used, allowing participants 3 hours of sleep in either the first (SWS-rich sleep) or the last (REM-rich sleep) part of the night (Wagner, Fischer & Born, 2002). In this study, participants were asked to evaluate emotional reactivity through self-reported measures of valence and arousal for pictures presented before and after each condition. After REM-rich sleep, the images were rated as more negative and more arousing, while after SWS-rich sleep, the images were significantly rated as more positive. Therefore, the authors emphasised that emotional reactivity increased after REM-rich sleep. Likewise, Groch et al. (2013) investigated the effects of SWS-rich or REM-rich sleep on emotional memory and associated emotional reactivity using event-related potentials (ERPs). Participants recruited in this study (N=16, mean age 22.06, age range 18–26) underwent 3 hours of SWS-rich sleep and 3 hours of REM-rich sleep in counterbalanced order across participants, with conditions separated by a wash-out period of at

least two weeks. Before each condition (22:30 for SWS-rich sleep and 03:30 for REM-rich sleep), participants underwent an emotional stimulus encoding task while recording ERPs. Memory performance was assessed in participants approximately 45 minutes after awakening in each condition. To evaluate memory performance, authors adopted a memory recognition task (old-new paradigm) in which pictures presented during the encoding were shown again mixed with new images. Participants had to recognise whether they had encountered the stimuli during encoding, indicating "OLD" for pictures already seen and "NEW" for the new stimuli. Additionally, during the memory performance task, participants had to indicate the valence and arousal values of the images (subjective emotional reactivity), and ERPs were again recorded. Memory for emotional pictures was better after REM-rich sleep compared to SWS-rich sleep. This effect manifested in the late-sleep condition as an increased frontal Late Positive Potential (LPP, signal related to item recognition accuracy) 300-500 ms after stimulus presentation for images correctly categorised as "OLD" compared to those categorised as "NEW", reinforcing the assumption that REM-rich sleep enhances memory for emotional events. However, REM sleep did not alter valence and arousal ratings, which were equivalent between the two conditions. Thus, the authors concluded that REM sleep significantly contributes to emotional memory consolidation while preserving the associated emotional reactivity. Subsequently, the same authors (Groch et al., 2015) investigated the effects of SWS and REM sleep on emotional memory and source memory (defined as the ability to recall the spatial location of the item test and his encoding context, given by the frame colour in which the item was presented) using a split-night paradigm. The

authors reported a significantly increased memory for emotional stimuli only after REM-rich sleep and not after SWS-rich sleep. Furthermore, source memory for neutral memories was significantly better only after SWS-rich sleep. Therefore, the authors concluded by emphasising the crucial role of REM sleep in the preferential consolidation of emotional stimuli. Other studies that employed similar sleep design paradigms supported these results (Harrington et al., 2018; Sopp et al., 2018).

To further reduce the amount of REM sleep during the night and allow participants to experience longer sleep relative to the split-night paradigm, other authors have investigated the effects of REM sleep on emotional memory and emotional reactivity through the selective sleep stage deprivation paradigm. Lara-Carrasco et al. (2009) assessed the impact of REM sleep on emotional adaptation using a selective REMD paradigm. Participants (N=35) were randomly assigned to a REMD condition (n=17, mean age 26.4) or a control condition (n=18, mean age 23.7). Sleep was monitored through polysomnography (PSG) in both conditions. The adopted REMD paradigm was partial, as participants could normally experience the first and second REM periods. Starting from the third REM period, they were awakened every 5 minutes during REM sleep. Control group participants were awakened 25 minutes after the third REM period to control for the confounding effect of nocturnal awakenings. Experimentally induced nocturnal awakenings were limited to 6 to avoid excessive sleep fragmentation in both conditions. Before each condition, participants performed a task involving viewing emotionally negative and neutral images while rating valence and arousal for each image. The same task was administered one hour after awakening in each condition.

Results showed a higher level of emotional adaptation in participants with reduced %REM than those with higher %REM. The authors concluded that REM sleep preserves/increases emotional reactivity associated with known emotional stimuli. In addition to assessing emotional reactivity in REMD protocols, other authors have investigated concurrent emotional memory performance. For example, Morgenthaler et al. (2014) examined the effects of REM sleep on emotional memory by randomly assigning participants (N=29) to either REMD or regular sleep groups. Here, participants in each group underwent a control condition in which emotional memory was assessed during daytime wakefulness. As in other studies, REMD was applied through awakenings after the first epoch of REM sleep. Despite the correct application of the REMD protocol, no differences emerged between groups regarding memory performance, which was superior for negative images in both sleep conditions (REMD and regular) compared to the wakefulness condition. Therefore, the authors emphasised that REM sleep does not influence the consolidation of memories. However, other authors do not support this view. Wiesner et al. (2015) assessed the effects of REM sleep, SWS, and wakefulness on emotional memory and reactivity using a selective sleep stage deprivation protocol. Participants were assigned to either a REMD condition (n=21, mean age 23.5), a selective SWS deprivation condition (SWSD, n=20, mean age 23.5), or a daytime wakefulness condition (n=21, mean age 23.4). REMD and SWSD were applied by awaking the participant for at least one minute after the first epoch of REM or SWS. Before each condition, participants performed a task involving encoding emotional and neutral images followed by an immediate re-test of memory performance. Subsequently, memory

performance was assessed approximately 9 hours after the first re-test. Additionally, participants provided subjective valence and arousal ratings for each picture during each task to assess subjective emotional reactivity. In the SWSD group, significantly better performance for emotional than neutral stimuli was observed. This effect was supported by a positive correlation between time spent in REM sleep and memory for emotional stimuli in the SWSD group. However, no differences in emotional reactivity associated with stimuli were reported between conditions. Therefore, the authors emphasised that REM sleep selectively processes emotional memory without altering the associated emotional reactivity. Alternatively, other authors have investigated the effects of REM sleep on emotional memory and reactivity through daytime sleep (nap paradigm). In this manner, it is possible to manage nap duration to prevent (through naps of 30–60 minutes) or guarantee (through naps of 90–120 minutes) the presence of REM sleep without excessively manipulating sleep continuity (due to continuous awakenings in selective sleep stage deprivation paradigms). For instance, Pace-Schott et al. (2011) evaluated the effects of a nap on emotional reactivity to negative and neutral arousing stimuli. Participants in this study (N=43) were randomly assigned to a nap condition (n=22, mean age 20.1) or a wakefulness condition (n=21, mean age 21.3). Before each condition, an emotional stimulus viewing task was performed in which participants were asked to self-report valence and arousal ratings for each picture. Additionally, during the presentation of each stimulus, skin conductance response (SCR), heart rate, and the surface electromyographic response of the corrugator zygomatic muscle (zEMG) were recorded as objective indices of emotional reactivity. This was followed by a 2.5-hour interval

in which participants in the nap group had a 120-minute sleep opportunity. In contrast, the wakefulness group underwent 120 minutes of exposure to a non-arousing video. After this interval, subjects were exposed again to the stimulus presentation task. Half of the stimuli were completely new this time, and the other half were old stimuli already presented during the first task administration. No differences were found in self-reported emotional reactivity to stimuli after each condition. However, during the presentation of old stimuli, objective indices of emotional reactivity showed a habituation effect in the nap condition (SCR and zEMG). In contrast, the zEMG index of emotional reactivity was increased in the wakefulness group for the old stimuli. No differences were found in objective emotional reactivity for the new stimuli category. These results demonstrated a sleep psychophysiological dampening effect specific to familiar stimuli. Moreover, this study emphasised that the level of psychophysiological habituation to negative images for the zEMG signal was positively related to the SWS amount during the nap. In contrast, the amount of REM sleep was negatively associated with habituation in SCR response to negative images. Thus, the authors speculated that REM sleep may reinforce the emotional salience of stimuli by reducing habituation, while SWS promotes emotional homeostasis. Adopting a similar paradigm, Werner et al. (2021) assessed the effects of REM sleep on emotional reactivity and intrusive memories through a nap paradigm. Participants underwent an image evaluation task in which they were asked to rate the perceived aversiveness of negative and neutral stimuli. The aversiveness of pictures was tested again after a 90-minute nap opportunity. Additionally, from the last evaluation task until 48 hours later, participants were asked to report whenever the stimuli came to mind

during the day and rate the aversiveness of these intrusive memories. Regarding nap conditions, one group of participants was awakened before entering REM sleep (n=25), another group was awakened a few minutes after entering REM sleep (n=22), and a group was allowed to complete the REM sleep period before being awakened (n=29). No differences in aversiveness ratings were found between groups. However, regarding the image evaluation task performed immediately after the nap opportunity, results showed a positive correlation between time spent in REM sleep and high aversiveness ratings for negative images. Moreover, in the group allowed to complete the REM period, the aversiveness of intrusive memories assessed two days after the nap opportunity was significantly lower than in participants who were awakened before entering REM sleep. Therefore, the authors sustained that REM sleep preserves/increases emotional reactivity to negative stimuli immediately after the encoding but facilitates processing this information over time, reducing the reactivity to intrusive memories in the long term.

Finally, the relationship between REM sleep, emotional memory, and emotional reactivity has been investigated by comparing nocturnal sleep and daytime wakefulness, correlating sleep stages duration with emotional reactivity and emotional memory performance. Baran et al. (2012) assessed the association between sleep, memory, and emotional reactivity using an incidental learning task (participants were unaware that their memory performance would be evaluated) administered before a night of sleep (n=54, sleep monitored with PSG) or daytime wakefulness (n=28). Memory performance was better after the sleep condition than after wakefulness for all stimulus categories (negative and neutral). However, the performance was superior for emotional stimuli

relative to neutral ones. Moreover, a significant reduction in the associated emotional reactivity was observed in the wake group, while emotional reactivity was preserved in the sleep group. Furthermore, preserved emotional reactivity was positively associated with the time spent in REM sleep during the night. Thus, the authors conclude by emphasising the beneficial effect of sleep on memories (regardless of their emotional valence) and a REM sleep "protective" effect on emotional reactivity associated with emotional memories. Additionally, Jones et al. (2016), in a study involving young adults (age range 18–30) and older adults (age range 50–80), found an association between the percentage of REM sleep in the third quarter of the night and preserved emotional valence ratings for stimuli. Furthermore, a recent study was the first to highlight an interesting correlation between the percentage of REM sleep the night before encoding emotional stimuli and memory performance for negative images in the immediate post-encoding memory performance assessment, emphasising the importance of REM sleep for proper emotional stimuli encoding (Carollo, Degasperi & Cellini, 2022).

The above-reported results primarily refer to behavioural studies, and only some authors have employed objective methodologies to assess emotional reactivity (e.g., SCR, ERPs, zEMG). However, some works in this field have utilised neuroimaging techniques to investigate the effects of REM sleep on the functioning of cortical and subcortical areas involved in emotional processing.

1.3.4.2 Neuroimaging studies about the role of REM sleep on emotional memory and emotional reactivity

Crucial for emotional processing is the activity of different cortical areas and subcortical structures such as the amygdala, insula, striatum, hippocampus, anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), ventromedial prefrontal cortex (vmPFC), and dorsolateral prefrontal cortex (dlPFC) (Altena et al., 2016). Regarding emotional reactivity, the amygdala is generally considered of primary importance. The amygdala is activated in response to an emotional event, influencing memory formation through reciprocal connections with other subcortical structures and with frontal and temporal cortical areas. Specifically, through connections with the medial temporal lobe memory systems, the amygdala influences the hippocampus, the entorhinal cortex, and the perirhinal cortex, conditioning memory formation (Faul & LaBar, 2020; LaBar & Cabeza, 2006; Roozendaal & Mcgaugh, 2011). Additionally, through connections with the hypothalamic-pituitary-adrenal axis (HPA), the amygdala triggers hormonal release capable of affecting memory trace formation for extended time intervals. Moreover, the activity of the amygdala under these conditions can be modulated (e.g., reduced/maintained) by different areas of the prefrontal cortex (PFC) (Faul & LaBar, 2020; LaBar & Cabeza, 2006; Roozendaal & Mcgaugh, 2011). Studies quantifying brain metabolism during sleep show that global brain metabolism is comparable to wakefulness during REM sleep, while it is reduced during NREM sleep (Buchsbaum et al., 2001; Dang-Vu et al., 2010). Specifically, some cortical areas exhibit hyperactivity, and others exhibit hypoactivity. Specifically, during REM sleep, there is hyperactivation of the pontine tegmentum, thalamus, amygdala, hippocampus, ACC, occipital and

temporal areas, and the basal forebrain. Conversely, areas showing reduced activation include the inferior parietal cortex, dlPFC, posterior cingulate gyrus, and precuneus (Buchsbaum et al., 2001; Dang-Vu et al., 2010).

Therefore, it is clear that areas involved in emotional processing are activated (similarly to wakefulness) during REM sleep. Moreover, positron emission tomography (PET) studies also emphasised the role of REM sleep in memory processing. Maquet et al. (2000) highlighted that specific cortical areas activated in wakefulness during a learning task were reactivated during subsequent REM sleep only in subjects engaged in that task. These results emphasise the involvement of REM sleep in memory processing (Maquet et al., 2000).

The reactivation of cortical structures involved in emotional processing during wakefulness in the course of REM sleep, along with imaging and behavioural studies that emphasise the role of REM sleep in memory processing, has increasingly prompted researchers to directly investigate the relationship between REM sleep, emotional memories, and emotional reactivity through neuroimaging techniques. Rosales-Lagarde et al. (2012) assessed the effects of REM sleep on emotional reactivity through functional magnetic resonance imaging (fMRI) in a sample of subjects during REMD. The REMD protocol was applied by awakening participants (n=12) each time they entered REM sleep during the night. As a control, another group of participants (n=8) underwent the same number of awakenings during NREM sleep (NREM-I) to control for the effects of repeated awakenings and total sleep time reduction. Participants performed an emotional reactivity task in fMRI after a baseline night and after the sleep manipulation night (REMD or NREM-I). During the task, participants

were asked to imagine themselves as part of the scene shown and decide whether to intervene by defending themselves (reaction indicated by pressing a button). Trials where participants decided to intervene were interpreted as high emotional reactivity trials. The results showed a significant behavioural and emotional reactivity increase only after REMD. Additionally, cortical areas involved in emotional processing showed significantly reduced activation compared to baseline only in the NREM-I condition. Participants undergoing REMD showed activation levels in prefrontal and temporal regions comparable to baseline. Specifically, the ventrolateral prefrontal cortex, involved in top-down emotion regulation, exhibited activation levels identical to those at baseline in trials categorised as high emotional reactivity. Additionally, occipital and temporal areas involved in evaluating emotional stimuli showed activation levels similar to baseline only in the REMD group. The authors conclude that the lack of REM sleep is associated with increased emotional reactivity at behavioural and neural levels. Therefore, the authors emphasised the specific role of REM sleep in the homeostasis of neural substrates involved in emotional reactivity. Similarly, Van Der Helm et al. (2011) investigated whether REM sleep decreased behavioural and amygdala reactivity to previously encountered emotional stimuli and how it influenced the functional connectivity between the amygdala and the medial prefrontal cortex in response to them. Participants recruited in this study (N=34, age range 18–30) underwent two separate fMRI scans separated by 12 hours containing either daytime wakefulness (n=16) or a regular night of sleep (n=18). During fMRI scans, participants performed an emotional stimulus presentation task where emotional reactivity was assessed through self-reported valence and arousal ratings. The task was identical in

both sessions. In the sleep group, there was a reduction in amygdala activity associated with increased functional connectivity between the amygdala and vmPFC compared to the wake condition. Furthermore, this decrease in amygdala activity in the sleep group was associated with a reduction in self-reported emotional reactivity to the stimuli, which decreased only in the sleep group. Additionally, a positive correlation emerged between decreased overnight amygdala activity and reduced prefrontal gamma (>30 Hz) EEG activity during REM sleep (gamma EEG activity during REM sleep is an index of unsuppressed adrenergic activity during this sleep stage). Moreover, the same correlation was also reported between reduced behavioural and emotional reactivity and reduced prefrontal gamma EEG activity during REM sleep. Therefore, the authors sustain that REM sleep is associated with the dissipation of the psychophysiological reactivity to previously experienced emotional events. Furthermore, Glosemeyer et al. (2020) assessed the effects of selective REMD on affect, perception of social exclusion, emotion regulation through cognitive reappraisal, and the neural bases of these aspects. Forty-two subjects (mean age 23.76) were randomly assigned to one of three study conditions: REMD (n=17), SWSD (n=10), and control (n=15). Both deprivation conditions involved awakening participants every time they entered the target sleep stage during the night. Results showed a negative correlation between the amount of REM sleep and negative affect. However, there was no influence of REMD on the subjective emotional response to the social exclusion task, nor on the ability to use cognitive reappraisal. Despite this, amygdala neural activity was increased, and its functional connectivity with the ACC was altered when participants experienced social exclusion during the task only in the REMD

group. Therefore, the results support the fundamental role of REM sleep in ensuring correct emotional processes, especially regarding functional connectivity and the proper activation of cortical structures.

Given the reported findings, REM sleep seems crucial for ensuring proper encoding and consolidation processes of emotional events. Furthermore, the deprivation/reduction of REM sleep alters the activity of various brain structures involved in emotional processing and the functional connectivity between them. Nevertheless, how this sleep stage acts on the intrinsic emotional reactivity associated with emotional information needs to be clarified. Specifically, whether REM sleep acts to reduce, preserve, or even enhance the emotional reactivity associated with the emotional event is still to be determined.

The conflicting results in the literature regarding the effects of REM sleep on emotional memory and the associated emotional reactivity have led to the formulation of two conflicting theoretical hypotheses. Although both hypotheses agree that REM sleep has a beneficial effect on emotional memory consolidation, they postulate opposed effects of this sleep stage on the psychophysiological reactivity associated with this type of memory. Wagner et al. (2002) claim that while REM sleep consolidates and improves emotional memory traces, it also *strengthens/preserves* the associated emotional reactivity. Based on this hypothesis, Wagner et al. (2002) suggest that selective REMD could be used therapeutically to prevent the formation of traumatic memories and to reduce the psychophysiological activation associated with these memories. According to this view, REMD could be used preventively against phobic disorders or post-traumatic stress disorder (PTSD). This theory has been challenged by the subsequent hypothesis of Walker & van der Helm,

(2009). According to the "sleep to forget, sleep to remember" hypothesis (SFSR) (Walker & van der Helm, 2009), REM sleep leads to the consolidation of emotional information while simultaneously *reducing* the psychophysiological activation associated with these memory traces. Moreover, they sustain that the psychophysiological reduction associated with the emotional event will be more significant when more REM cycles intervene after stimulus encoding. Thus, psychophysiological reactivity will weaken over the days following the consolidation process. The authors attribute this dissociated effect (enhancing emotional memory while reducing emotional reactivity) to the reactivation of cortical areas involved in emotional and memory processing during REM sleep while the brain has a neuro-biochemical state that favours memory formation without consolidating the associated psychophysiological activation. Specifically, during the REM stage, there would be a condition of elevated levels of acetylcholine coupled with increased activity in limbic structures (i.e., hippocampus and amygdala) that would facilitate the formation of emotional memories. Simultaneously, the locus coeruleus (LC) activity is reduced in the REM phase, leading the brain to reduced levels of noradrenaline (associated with physiological arousal). Therefore, in an environment that promotes emotional memory formations, the brain would not have the opportunity to consolidate the activation associated with this information over time, retaining that memory trace free from the psychophysiological reactivity previously associated with it. Thus, according to this theory, experiencing REM sleep after traumatic events would be therapeutic because it would allow the dissociation of the psychophysiological reaction associated with the traumatic event (Walker & van der Helm, 2009).

Despite the considerable body of research assessing the relationship between REM sleep, emotional memory, and emotional reactivity, this literature presents various conflicting results. Therefore, neither of the two theoretical assumptions can be confirmed. However, these contradictory results stem from methodological limitations of the studies investigating the effects of REM sleep on emotional memory and emotional reactivity.

2. The methodological challenge

The above-mentioned studies investigating REM sleep's role in emotional memory and reactivity primarily utilised split-night and selective REMD study paradigms. However, these research paradigms suffer from significant methodological limitations.

2.1 Intrinsic limitations of the studies on the role of REM sleep on cognition and emotions

The fundamental assumption of split-night and selective REMD study paradigms is to minimise or eliminate REM sleep throughout the night and assess how the absence of this sleep stage influences cognitive and emotional functioning. Generally, as a control condition in REMD protocols, participants can experience *i)* a night of regular sleep, *ii)* multiple nocturnal awakenings during another sleep stage (e.g., NREM2 or SWS), or *iii)* selective SWSD. Concerning the split-night paradigm, the control condition involves participants sleeping in the second half of the night (e.g., 04:00–07:00) and thus experiencing REM-rich sleep. However, these experimental designs inevitably introduce confounding variables affecting the interpretation of the outcomes. Specifically, in REMD protocols, participants must be awakened numerous times and kept awake for several minutes throughout the night to prevent REM sleep. Despite repeated awakenings potentially compromising daily functioning (Aakre et al., 2023; Wilckens et al., 2014), REMD studies often do not report the number of induced nocturnal awakenings (e.g., Kaida, Niki & Born, 2015; Morgenthaler et al., 2014). Moreover, during these enforced awakenings,

participants are often engaged in cognitive tasks (e.g., mental arithmetic calculations) to reduce the likelihood of subsequent REM sleep onset. These continuous and prolonged awakenings inevitably reduce total sleep time (TST) and increase wake after sleep onset (WASO). Indeed, the time spent awake in these studies varies from approximately 70 to 90 minutes (e.g., (Kaida et al., 2015; Morgenthaler et al., 2014), and sleep efficiency (SE) values are lower than deemed normal (i.e., 80%) (Desjardins et al., 2019; Ohayon et al., 2004). For instance, Wiesner et al. (2015) reported a SE of 68% in subjects undergoing REMD.

Due to these reasons, some authors have arbitrarily set a maximum limit on nocturnal awakenings during the REMD protocol (e.g., a maximum of 6 awakenings in Lara-Carrasco et al., 2009). However, this deprivation method led to a partial REMD, as participants still experienced a significant amount of REM sleep during the night. Nevertheless, REM sleep is partially experienced in all existing REMD protocols as depriving subjects of REM sleep inevitably requires waiting for participants to enter REM sleep itself.

Split-night paradigms halve TST, as participants can only sleep in the first or second half of the night. Moreover, the assessment phase in the REM-rich sleep and SWS-rich sleep groups in these protocols occurs at different times of the day (e.g., participants in the SWS-rich sleep group are generally tested at 22:00 and 03:00, while participants in the REM-rich sleep group are tested at 03:00 and 08:00). Therefore, the evaluations are affected by significant circadian influences. Moreover, participants in SWS-rich conditions even experience a small amount of REM sleep. Instead, participants in the REM-rich sleep condition go to sleep in the second half of the night experiencing partial sleep

deprivation (e.g., they are allowed to sleep at 4:00), resulting in a rebound of SWS in the second half of the night. Thus, REM and NREM2 sleep do not exclusively characterise participants' sleep during REM-rich sleep conditions since SWS will also be present. Some authors (Groch et al., 2013; Wagner, Fischer & Born, 2002) have modified the split-night paradigm to overcome this issue. In this modified version of the split-night paradigm, participants in the REM-rich sleep group start to sleep at a regular time (e.g., 23:00, like the SWS-rich group) while the behavioural assessment is maintained at mid (e.g., 03:00) and at the end of the night (e.g., 08:00). Consequently, this modified version of the split-night paradigm avoids a rebound of SWS and a TST reduction. However, it generates a significantly different REM-rich sleep condition from the SWS-rich sleep condition, which serves as a control. Specifically, despite the post-task sleep period of approximately 3–4 hours in both conditions, participants in REM-rich sleep have a TST of 7–8 hours, while participants in SWS-rich condition sleep only 3–4 hours. Therefore, despite the correct theoretical assumptions, the split-night paradigm is highly limiting in its practical application.

As highlighted in the introduction (see section 1.3.4.1), some authors have utilized the nap paradigm to overcome the methodological limitations characterizing REMD and split-night studies (Cellini et al., 2016; Pace-Schott et al., 2011; Werner et al., 2021). Indeed, the nap paradigm partially compensates for the confounding effects of reduced TST, increased WASO, and nocturnal awakenings. Additionally, it eliminates the circadian confounding factor due to different assessment timing in SWS-rich and REM-rich sleep, which are intrinsic to the split-night paradigm. However, the nap paradigm's

main limitation is the involvement of daytime sleep. Although daytime sleep comprises NREM and REM sleep, it often lacks SWS and is not aligned with the natural circadian rhythm (Mantua & Spencer, 2017). Consequently, the nap paradigm is not a resolutive solution to understanding REM sleep's role in emotional/cognitive functioning.

From these observations, it is clear that study paradigms investigating the effects of REM sleep on cognitive/emotional functioning are strongly affected by confounding variables. Specifically, these protocols are affected by a significant reduction in TST and SE, a substantial increase in WASO and in the number of nocturnal awakenings relative to a regular night of sleep. All these potential confounding variables could explain conflicting results in studies attempting to investigate the effects of REM sleep on emotional memories and associated emotional reactivity.

Moreover, sleep studies in this field are affected by small sample sizes and the use of convenience samples (Davidson et al., 2021; Tempesta et al., 2018).

The existence of methodological limitations in this literature field suggests the necessity of developing an alternative study paradigm. Sleep fragmentation studies could be a strategy for investigating the effects of REM sleep on emotional/cognitive functioning without excessively affecting sleep macrostructure.

2.2 Sleep fragmentation as an alternative paradigm

Sleep fragmentation refers to a condition in which sleep is frequently interrupted by awakenings, changes in sleep stage toward lighter sleep (e.g., from NREM2

to NREM1; from NREM3 to NREM2; and from REM to NREM1), body movements, and arousals (brief and sudden changes in EEG frequency that include alpha, theta, or frequencies greater than 16 Hz, but not spindles, lasting at least 3 seconds and preceded by at least 10 seconds of stable sleep; Berry et al., 2017) that compromise sleep continuity (Bhagavan & Sahota, 2021; Mezick et al., 2009). However, the term sleep fragmentation often refers to only some of these aspects, depending on the research methodology applied in the study. Indeed, sleep fragmentation can indicate only nocturnal awakenings and body movements during sleep in studies that recorded sleep through actigraphy (Lim et al., 2013). Furthermore, some authors considered sleep fragmentation only in terms of the number of nocturnal awakenings (Gott et al., 2020; Iacovides et al., 2017). For instance, in the protocol adopted by Iacovides et al. (2017), sleep fragmentation was realised through enforced awakenings of a minimum duration of 20 minutes. Additionally, other authors considered shifts to light sleep as sleep fragmentation only when these transitions were towards NREM1 (Morrell et al., 2000). Given the lack of agreement in the variables to include in the construct of sleep fragmentation and the fact that its assessment strictly depends on the adopted methodology (e.g., actigraphy, self-reported nocturnal awakenings in sleep studies without PSG), sleep fragmentation can be considered as referring to a multitude of factors that negatively affect the micro- and macro-structure of sleep (Bhagavan & Sahota, 2021; Mezick et al., 2009). Moreover, sleep fragmentation can be due to external (e.g., stimulations, noises) and internal (e.g., apnoea, limb movements) events (Bhagavan & Sahota, 2021), and is often present in clinical conditions. For example, fragmented sleep is observed in individuals with sleep apnoea, insomnia,

depressive disorder, and PTSD (Benjamins et al., 2017; Murphy & Peterson, 2015; Pesonen et al., 2019; Riemann et al., 2020; Van Liempt, 2012). In these clinical conditions, sleep fragmentation seems to interfere with the normal physiological processes occurring during sleep, compromising sleep functions. For instance, sleep fragmentation in PTSD compromised fear memory extinction, synaptic plasticity, and physical recovery, establishing a cycle of reciprocal influences where these impairments affected PTSD symptomatology, which in turn impaired sleep continuity (Van Liempt, 2012). Furthermore, in patients with sleep apnoea, sleep fragmentation appears to be associated with adverse cardiovascular consequences (Staats et al., 2020). Indeed, sleep fragmentation in patients with sleep apnoea is associated with dyslipidaemia, potentially contributing to cardiovascular diseases in this population (Qian et al., 2016). Moreover, Bonnet & Arand (2003) highlighted that in patients with periodic limb movements and respiratory-event-induced arousals, sleep fragmentation was the leading cause of daytime sleepiness. Finally, in the elderly population, sleep fragmentation is associated with increased cognitive decline and an increased incidence of Alzheimer's disease (AD) (Lim et al., 2013). This effect has also been highlighted in mice (Vasciaveo et al., 2023), where experimentally induced sleep fragmentation accelerates Alzheimer's progression in a mice model of AD and compromises learning and memory abilities in wild-type mice (Joiner, 2019; Vasciaveo et al., 2023).

The effects of sleep fragmentation have also been investigated in the healthy population. As observed in individuals with sleep apnoea (Staats et al., 2020), sleep fragmentation increased systolic blood pressure levels in healthy subjects during wakefulness, constituting a risk factor for cardiovascular disease (Morrell

et al., 2000). Moreover, awakening participants two times during the night (the first time for 1 hour and the second time for 20 minutes) to experimentally induce sleep fragmentation was sufficient to reduce positive mood in healthy subjects (Finan et al., 2015). Similarly, Iacovides et al. (2017) highlighted that experimentally induced sleep fragmentation reduced sleep quality, overall mood, and morning alertness and increased subjects' pain sensitivity. In this study, sleep fragmentation was induced for two consecutive nights by awakening participants for a minimum of 20 minutes and a maximum of 1 hour, allowing them to sleep for a maximum of 280 minutes each night (Iacovides et al., 2017). Furthermore, sleep fragmentation alters hormonal balance. For instance, it can alter cortisol and adrenocorticotropic hormone levels (Bonnet & Arand, 2003). Moreover, it alters leptin and ghrelin levels, affecting feelings of hunger and satiety (Baranwal et al., 2023). Indeed, sleep fragmentation seems to promote obesity in mice (Wang et al., 2014).

Nowadays, there are only two literature reviews of sleep fragmentation's effects on healthy subjects' daytime functioning (Bonnet & Arand, 2003; Stepanski et al., 2002). In these reviews, the authors reported the results of studies in which sleep fragmentation was experimentally induced at different intensities, either by nocturnal awakenings (e.g., 1 awakening every minute of sleep, 1 awakening every 5 minutes of sleep, 1 awakening every 10 minutes of sleep) or by arousal induction (e.g., 1 arousal every 30 seconds of sleep, 1 arousal every 60 seconds of sleep, 1 arousal every 90 seconds of sleep). These reviews showed that sleep fragmentation can negatively affect daytime functioning by increasing daytime sleepiness, compromising cognitive functioning and decreasing mood. Furthermore, in their review, Bonnet & Arand, (2003)

emphasised that the differences between sleep fragmentation, sleep deprivation, and sleep restriction depend on the degree to which sleep is compromised. Indeed, sleep fragmentation induced by an arousal/awakening rate of one every 30s/one per minute of sleep led to effects comparable to one night of TSD.

In light of these results, it seems that sleep fragmentation can compromise the physiological processes occurring during sleep that allow the brain to function properly the following day. Moreover, it should be emphasised that sleep fragmentation minimally affects sleep macrostructure compared to sleep deprivation/restriction protocols or split-night paradigms. Specifically, obtaining sleep fragmentation through arousals and sleep stage transitions toward lighter sleep (rather than through multiple awakenings) allows unchanged TST, WASO, and nocturnal awakenings compared to sleep deprivation/restriction protocols or split-night paradigms (Benkirane et al., 2022; Short & Banks, 2014). Thus, sleep fragmentation could be used as an alternative study paradigm to assess the effects of sleep on cognitive/emotional functioning since it appears capable of limiting the influence of confounding variables compared to commonly used sleep deprivation/restriction protocols.

Finally, it is interesting to note that recent studies on sleep fragmentation showed that it affects REM sleep in some clinical conditions (PTSD, depression, insomnia) (Colvonen et al., 2019; Pesonen et al., 2019; Wassing et al., 2019; Wu et al., 2021).

2.2.1 REM sleep fragmentation studies

According to Feige et al. (2023), REM sleep fragmentation, named REM sleep instability, is characterised by increased arousal number associated with a slight reduction in REM sleep duration and occurs in individuals with insomnia, leading them to perceive REM sleep as wakefulness. Riemann et al. (2012) first proposed REM sleep fragmentation as a different mechanism to explain the discrepancies between objective and subjective sleep reports in insomniac individuals. Specifically, the authors speculated that the mismatch between the mild objective alterations detected through PSG in sleep parameters and the significant self-reported sleep impairment in insomnia patients may be attributed to REM sleep instability. Several studies highlighted how REM sleep in individuals with insomnia is characterised by numerous micro- and macro-arousals. Moreover, these patients reported concerns about poor sleep quality, especially in the pre-sleep phases. According to Riemann et al. (2012), REM sleep fragmentation may make sleep concerns more accessible to consciousness, memory storage, and retrieval during REM sleep, resulting in non-restorative sleep. Furthermore, this work highlighted how REM sleep fragmentation negatively impacts the functionality of the cortical and subcortical structures involved in emotional processing (as reported in section 1.3), potentially contributing to cognitive and emotional alterations that may increase the risk of emotional disorders in insomniac people (Riemann et al., 2012). In addition, other authors emphasised the role of REM sleep fragmentation also in individuals with PTSD. Specifically, Saguin et al. (2021) hypothesised that REM sleep fragmentation compromised traumatic experience processing, thus playing a crucial role in maintaining PTSD symptoms (Saguin et al., 2021). A

previous study evaluating military personnel with PTSD highlighted a relationship between earlier-life traumatic events and REM sleep fragmentation (Insana et al., 2012). This study also emphasised that increased REM sleep fragmentation is associated with the development of disruptive nocturnal behaviours (Insana et al., 2012). REM sleep fragmentation role in PTSD is also emphasised in studies on animal models (Grafe et al., 2024; Pace-Schott et al., 2015). Aimed at analysing whether REM sleep fragmentation could have a mechanistic role in maintaining PTSD, Marshall et al. (2014) assessed the association between fear conditioning, safety learning, and REM sleep fragmentation in healthy subjects. Here, REM sleep fragmentation was referred to as reduced REM sleep latency, lower REM %, and increased number of transitions to waking/NREM stages during the REM period. In this study, the authors reported a positive correlation between consolidated REM sleep and learning levels. Specifically, REM sleep fragmentation compromised safety learning and the ability to discriminate between safety and threatening stimuli. Therefore, the findings supported a mechanistic role of REM sleep fragmentation in PTSD (Marshall et al., 2014). Lipinska & Thomas, (2019) investigated the effects of REM sleep fragmentation on sleep-dependent emotional memory consolidation by comparing subjects with PTSD, trauma-exposed non-PTSD individuals, and healthy subjects. REM sleep fragmentation was defined as the total number of arousals during this sleep stage. The authors highlighted a negative relationship between REM sleep fragmentation and memory performance for emotionally arousing stimuli (Lipinska & Thomas, 2019). Furthermore, in a recent narrative review (McCall & Watson, 2022) discussing the relationship between obstructive sleep apnoea (OSA) and

PTSD, it was emphasised an elevated comorbidity rate between these two disorders and the presence of fragmented sleep due to OSA-induced arousal-like features affecting PTSD. Specifically, it was highlighted that OSA-induced REM sleep fragmentation may interfere with emotional processing, potentially playing a role in the development and maintenance of PTSD (McCall & Watson, 2022).

The presence of fragmented REM sleep has been reported also in depressive disorder. For instance, Pesonen et al. (2019) emphasised that REM sleep fragmentation in the adolescent population is associated with depressive symptoms and genetic risk for somatic complaints. Moreover, Wu et al. (2021) recently highlighted that REM sleep fragmentation is positively related to depressive symptom levels in individuals with insomnia.

Since REM sleep fragmentation has a significant role in insomnia and PTSD, some authors attempted to reduce REM sleep fragmentation experimentally (Maurer et al., 2024; Vethe et al., 2022). For instance, Maurer et al. (2024) tried reducing REM sleep fragmentation by submitting insomniac individuals to chronic sleep restriction or through time in bed regularisation. However, neither of the two methodologies was capable of reducing REM sleep fragmentation in insomnia patients. Conversely, Vethe et al. (2022), exposing healthy participants to a blue-depleted light environment (BDLE) in the evening, significantly reduced REM sleep fragmentation during the subsequent night compared to a standard light environment exposure before sleep. Here, REM sleep fragmentation refers to the total number of sleep transitions through wakefulness or NREM sleep during the REM period divided by the total duration of REM sleep (Vethe et al., 2022).

The investigation of Wassing et al. (2019) is noteworthy in this field. In this study, the authors assessed whether endogenous REM sleep fragmentation interfered with the nocturnal reorganisation of limbic structures activity in subjects with a wide range of insomnia severity. REM sleep fragmentation was quantified as the total number of arousals recorded during REM sleep and the number of transitions to NREM/wakefulness during the REM period divided by the total duration of the REM episode. Each participant underwent two fMRI scans to record the activity of limbic structures (particularly the amygdala) in response to induced emotional distress. Participants' sleep was recorded via PSG between the two fMRI recordings. The results highlighted a positive correlation between reduced amygdala activation and increased duration of undisturbed REM sleep (Wassing et al., 2019).

As reported in Chapter 1, REM sleep is accompanied by suppressed LC activity (Van Egroo et al., 2022). However, the occurrence of cortical arousals during REM sleep (that leads to REM sleep fragmentation) indicates a failure of LC activity suppression (Van Egroo et al., 2022). The silencing of LC activity during REM sleep is hypothesised to allow adequate emotional reactivity dampening, reducing the emotional reactivity associated with the memory trace (Swift et al., 2018; Walker & van der Helm, 2009). On the other hand, during wakefulness, LC activity maintains noradrenaline release at a level that prevents synaptic depotentiation (Pace-Schott & Hobson, 2002; Swift et al., 2018). Therefore, cortical arousals during REM sleep might interfere with emotional processing, compromising REM sleep function. Indeed, as we have seen, fragmented REM sleep is present in depressive disorders, PTSD, and insomnia (Colvonen et al., 2019; Pesonen et al., 2019; Wassing et al., 2019; Wu et al., 2021). However, to

date, the association between LC activation during sleep and cortical arousals has only been reported in animal studies. In contrast, no study has empirically assessed whether arousal during REM sleep inhibited LC silencing in humans.

These studies highlighted how REM sleep fragmentation could interfere with cognitive functioning. Specifically, REM sleep fragmentation is supposed to interfere with the emotional processing assumed to occur during REM sleep. However, these investigations are based on clinical populations that present other macrostructural sleep alterations, limiting the generalizability of the results (Baglioni et al., 2016; Bastien, 2011; Palagini et al., 2013; Steiger & Pawlowski, 2019). Nevertheless, these studies demonstrate that REM sleep fragmentation could compromise the psychophysiological processes occurring during REM sleep, affecting human functioning. Therefore, experimentally inducing REM sleep fragmentation in healthy subjects could be adopted as an alternative paradigm to investigate REM sleep's effects on emotional functioning, overcoming previous limitations of the studies in this field. Consequently, we aim to induce REM sleep fragmentation in healthy subjects to evaluate its effect on emotional memory and reactivity.

3. The experimental study

3.1 Reproducing REM sleep fragmentation in healthy subjects

The few studies that have experimentally fragmented participants' sleep have primarily employed multiple nocturnal awakenings, significantly altering sleep macrostructure (Finan et al., 2015; Gott et al., 2020; Iacovides et al., 2017). Thus, we aim to induce REM sleep fragmentation mainly inducing cortical arousals. To achieve this aim, we have chosen a tool that provides high sensitivity in delivering mild stimuli to the sleeping subject, thus avoiding the induction of nocturnal awakenings (i.e., cortical activations >15 seconds). Since the brain appears particularly sensitive to somatosensorial information during sleep (Wei & Van Someren, 2020), our interest has turned to vibrotactile stimulation as a method to fragment REM sleep. In the past, some authors have effectively induced cortical arousal during sleep by coupling vibrotactile and acoustic stimulation (Kato et al., 2003, 2004), with an arousal induction rate during REM sleep of approximately 85% (Kato et al., 2004). To this aim, we developed a semi-automatised vibrotactile stimulation device based on Arduino® technology to induce cortical arousals during REM sleep.

3.1.1 Development of a device to deliver vibrotactile stimulations

Our vibrotactile stimulation device comprises an elastic fabric wristband housing five 5V vibration motors (Figure 1) connected to an Arduino® based control system (Figure 2).

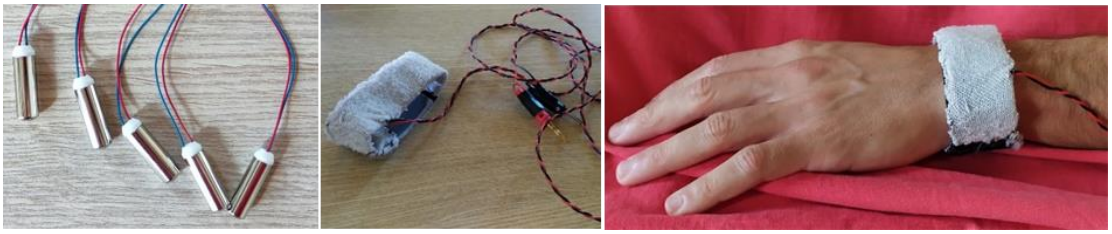

Figure 1. The left figure shows the five 5V vibration motors. The central figure shows the vibrotactile stimulation wristband housing the 5 vibration motors. In the right figure, the wristband is shown worn by a participant during an experimental session.

The control system hardware comprises an Arduino Uno Rev3 board (ARDUINO®, Italy) connected to a printed circuit board with a 5V Micro SD card module for Arduino®. These components are powered by a computer connection via a USB-B/USB-A cable and semi-automatically manage the activity of an externally powered 5V relay that supplies current to a power regulator, allowing for the vibration power control of the motors in the wristband. All these hardware components are enclosed in an ABS plastic module case, on which three buttons and the power regulator knob are positioned (Figure 2), allowing the handling of the stimulation during participants sleep.

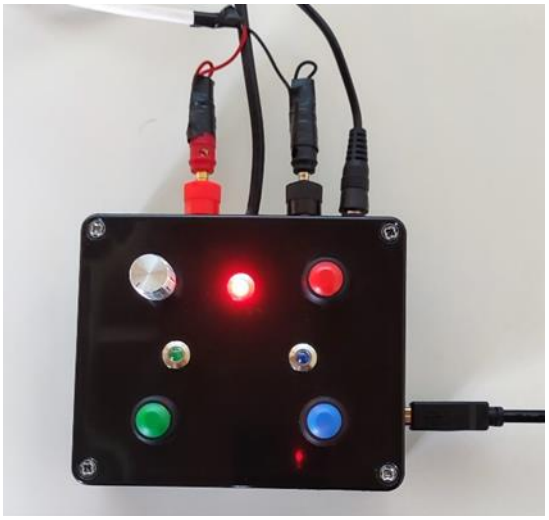

Fig. 2 The Arduino® based control system.
The green button generates a continuous automated stimulation-pause cycle, consisting of a 3 s stimulation followed by a 3 s pause. The red button can interrupt this cycle at the end of the 3 s stimulation. Instead, the blue button introduces a 40 s pause within the automated stimulation cycle, which will automatically resume at the end of the pause. Finally, the silver power regulator knob is used to manage the motor's stimulation power, ranging from a minimum of 0.8V to a maximum of 5V.

The above-described version is the final version of the vibrotactile stimulation device. In the development phase, the initial version comprised a wristband housing four 3V vibration motors. However, this first version was not powerful enough to prevent habituation effects in participants, and it compromised the effectiveness of arousal induction during the night. Thus, we developed the more powerful version described above.

3.2. Aims of the study

The existing studies on the role of REM sleep in emotional processing have relied on methodologies that do not allow to define the specific role of REM sleep on emotional memory and emotional reactivity. As above reported in detail, these studies have been based on REMD and split-night protocols that

inevitably introduce confounding variables (increased WASO and nocturnal awakenings, reduced TST). Moreover, the role of REM sleep fragmentation in emotional processing has been reported only in clinical populations, limiting the results' generalizability. Lastly, REM sleep fragmentation due to cortical arousal interferes with LC silencing, potentially compromising REM sleep emotional processing.

In this view, we aim to investigate the effect of REM sleep fragmentation in consolidating the declarative component of the emotional memory trace and on the associated emotional reactivity via behavioural, self-report and psychophysiological measures in healthy subjects adopting a within-subjects design. We aim to induce, for the first time, REM sleep fragmentation by means of cortical arousals elicited via vibrotactile stimulation through our device.

3.3 Materials and Methods

3.3.1 Participants

Twenty-six students from the University of L'Aquila were recruited to participate in the study. From the recruited sample, a total of 9 participants were removed: 6 dropped out since they were unable to fall asleep during the night, 1 showed a sleep disorder not detected during the screening procedure, 1 exhibited restless REM sleep, 1 began to report flu symptoms before going to sleep and woke up with fever. Thus, our total sample comprised 15 subjects (mean age ± SD, 23.67 ± 3.94, age range 19–34, 3 males).

A screening test battery was administered before the enrollment to evaluate if the participants fulfilled all the recruitment criteria. The screening battery

encompassed the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989; Curcio et al., 2013), the Insomnia Severity Index (ISI; Bastien, 2011; Castronovo et al., 2016), and the Depression Anxiety Stress Scale (DASS–21; Bottesi et al., 2015; Lovibond & Lovibond, 1995). Moreover, through dedicated questions, we assessed if participants had a regular sleep schedule, a normal/correct to normal vision, if they used medications, and the presence of a sleep disorder and/or a skin disease.

Recruitment criteria encompassed: *i)* good sleep quality (PSQI global score < 6, participants mean ± SD, 3.73 ± 1.16), *ii)* no insomnia symptoms (ISI score < 7, mean ± SD, 2.60 ± 2.29), *iii)* absence of depression (DASS–21 Depression subscale score < 14, mean ± SD, 3.87 ± 3.89), stress (DASS–21 Stress subscale score < 19, mean ± SD, 9.07 ± 4.71), and anxiety symptoms (DASS–21 Anxiety subscale score < 10, mean ± SD, 1.87 ± 2.20), *iv)* regular sleep schedule, *v)* normal/correct to normal vision, *vi)* absence of medication intake potentially interfering with sleep architecture, *vii)* absence of sleep disorders (i.e., sleep bruxism, sleep apnoea, snoring, sleep talking, sleepwalking, periodic leg movement disorder), and *viii)* absence of skin disease.

Finally, all female participants were recruited at the end of their menstrual cycle to uniform the influence of hormonal fluctuations on the collected psychophysiological variables.

The study was approved by the institutional review board of the University of L'Aquila (protocol n. 49/2021) and was performed according to the principles established by the Declaration of Helsinki.

3.3.2 Procedure

The present study was conducted in the Laboratory of Sleep Psychophysiology and Cognitive Neurosciences of the University of L'Aquila.

We adopted a within-subject design in which the enrolled participant undergoes two experimental conditions (Fragmentation night - FRG- and Control night - CTR) in a counterbalanced order across participants, with a minimum washout period between each condition of three weeks (21 days).

In each condition, participants were required to arrive at the Laboratory at 17:00; here, we provided instructions for the emotional reactivity and emotional memory tasks (for details about the tasks, see section 3.1.3) and the electrodes for electrodermal activity (EDA) and heart rate (HR) recording were applied. At 18:00, the participants performed the emotional reactivity task, which lasted about 15 minutes. Before and after the emotional reactivity task, we acquired 5 and 3 minutes, respectively, of EDA and HR resting values while participants sat on an armchair. Subsequently, EDA and HR electrodes were removed. Then, as an active break between the two emotional tasks, participants underwent the sequential finger-tapping task (SFTT). Subsequently, they were exposed to the encoding phase of the emotional memory task, which lasted about 10 minutes. Then, participants had a 15-minute break before the first recognition phase of the emotional memory task, which lasted about 8 minutes. We refer to the above-reported testing phase as T0 for each task. Collectively, the T0 testing phase lasted about 1 hour.

After dinner, the participants were prepared for the PSG recording with the application of the 64-channel high-density EEG cap (BrainCap, Brain Products GmbH, Germany) and the electrodes for electrooculogram (EOG),

electromyogram (EMG), and electrocardiogram (ECG) recording. Then, 5 minutes of waking EEG with eyes open and closed were recorded while the participants sat on an armchair. Then, the participants could go to bed; here, they wore the vibration wristband, and two stimuli of different intensities were provided to make participants aware of the stimulation they might feel during the night. This procedure was performed to reduce the likelihood of awakening induction due to the novelty of the first stimulation. Participants' sleep was then monitored via video-PSG, and during FRG night, vibrotactile stimulation was provided by a sleep expert during REM sleep (for the REM sleep fragmentation procedure, see section 3.1.3.1).

During each night participants were allowed to sleep for 8 hours after the first NREM2 sleep epoch (Berry et al., 2017) before being awakened. Immediately after the final awakening, participants were asked to record their dream recall on a digital voice recorder (K9, Benjie, China). Moreover, within 15 minutes of the final awakening, they answered questions about the laboratory sleep night (see 3.2.2.1 for details). Participants were then allowed to have breakfast without consuming caffeine products (e.g., coffee, chocolate). Then, a 5-minute waking EEG recording was performed with eyes open and closed. Subsequently, the subjects were freed from the PSG equipment.

About one hour after the final awakening, participants performed the emotional reactivity task that lasted about 15 minutes. Again, we acquired 5 and 3 minutes of EDA and HR resting before and after the emotional reactivity task. Then, after removing EDA and HR electrodes, the participants underwent the SFTT. Finally, they performed the second recognition phase of the emotional memory task. Subsequently, the participants left the Laboratory and were asked to return 48

hours later to be evaluated after 2 nights of regular sleep at home. During this delayed assessment, as in the first-morning evaluation session, participants performed the emotional reactivity task (with pre- and post-task EDA/HR baseline recording), the SFTT, and the emotional memory recognition task. On the morning of the delayed assessment session, participants were required to have breakfast free from caffeine products (e.g., chocolate, coffee, tea). We refer to the testing phase participants underwent the morning after the night of laboratory sleep as T1 for each task and to the delayed assessment that participants performed after two nights of sleep at home as T2. Collectively, the T1 and T2 testing phase lasted about 30 minutes each. Participants' sleep was monitored via actigraphy from two days before the T0 session until T2 in each condition. During these days, participants were required to fill out a sleep diary each morning after the final awakening and to maintain a regular sleep schedule. Figure 6 shows a schematic representation of the study protocol.

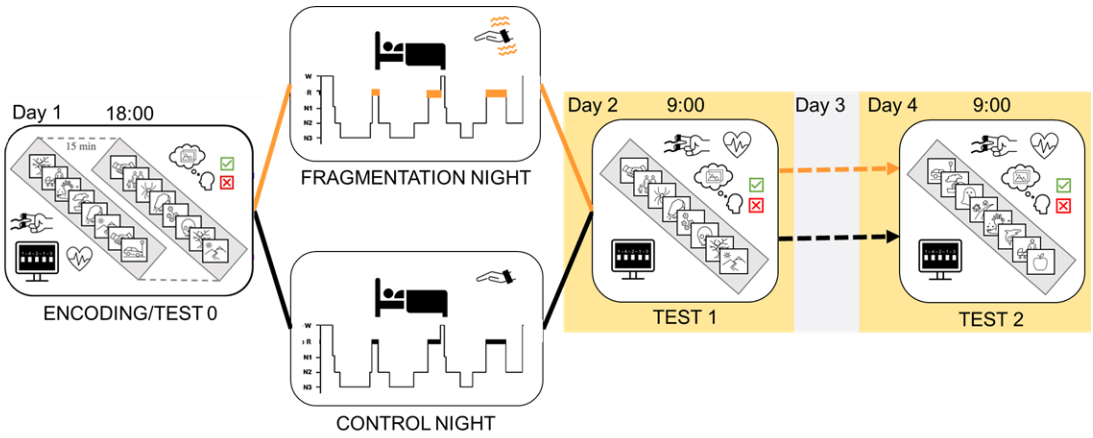

Figure 6. Schematic representation of the experimental protocol.
The hand icon with the index and middle fingers extended, and the heart icon represents the emotional reactivity task. The black screen monitor that shows a numerical sequence represents the SFTT. On Day 1, the grey bar on the left containing a sequence of images represents the encoding phase of the emotional memory task. Meanwhile, the grey bar with the sequence of images inside, accompanied by the head icon with a green checkmark and a red cross, represents the recognition phase of the emotional memory task. The open-hand icon with the wristband indicates that participants wear the vibrating bracelet at night. The orange saw line indicates vibration, and the orange bar overimposed to REM sleep in fragmentation night specifies that stimulation was provided during REM sleep.

3.3.2.1 REM sleep fragmentation procedure

During the FRG night, participants' REM sleep was fragmented according to the following protocol:

- the stimulation started at the minimum power after the first REM sleep epoch according to AASM® criteria (Berry et al., 2017);
- the stimulation lasted a maximum of 3 s or it was stopped before the 3 s in case of the appearance of a cortical arousal on any EEG trace;
- In case of the absence of response to the stimulation, the experimenter waited 3 s before delivering another stimulation with an increased vibration power;
- In the case of cortical arousal, the stimulation was turned off and:

- If the participant remained in REM sleep, the experimenter waited 30 s from the end of the cortical arousal and then provided another stimulation;
- If the participant exits from REM sleep, the experimenter waits until the participant returns to REM sleep.

The stimulation device was semi-automatised. Specifically, by pressing the green button of our Arduino®-based control system (see Figure 2, section 3.1.1) a continuous automated stimulation-pause cycle, consisting of a 3 s stimulation followed by a 3 s pause, was generated. This stimulation-pause cycle could be interrupted for 40 s by pressing the blue button of the control system. As an example, when the participant remained in REM sleep after a cortical arousal due to the stimulation, the experimenter should only suspend the stimulation until the device automatically delivered another stimulation 40 s after the end of the previous one.

3.3.3 Tasks

We developed the tasks utilised in our project through PsychoPy® coder v2020.2.10 and administered it with the same software on a 24-inch monitor (NILOX®, NXMMIPS240004) powered by a Mac mini (Apple® M1, 2020).

Before starting our experimental study, the emotional reactivity task and the emotional memory task described below were optimised by performing a pilot study.

Specifically, arousal and valence ratings were collected for sixty-eight images taken from the International Affective Picture System (IAPS; Lang et al., 2005)

and the Mnemonic Similarity Task (MST; Stark et al., 2019) to establish reference values for building the emotional reactivity task. For this purpose, 25 university students (mean age ± SD, 24.68 ± 2.40, 14 males) underwent an online picture evaluation task through Pavlovia software (Open Science Tools, Nottingham, UK). This data collection was carried out as images of the MST database lacked arousal and valence ratings.

For the emotional memory task, we aimed to create a task in which memory performance would decline over time, avoiding the ceiling effect that generally affects memory recognition tasks (Kopelman et al., 2007). We tested the first version of the emotional memory task on a different sample of 24 university students (mean age ± SD, 24.83 ± 2.40, 12 males) through Pavlovia software. However, this first version did not demonstrate a performance deterioration across the experimental sessions. Consequently, we developed a revised emotional memory task and evaluated this new version on 10 university students (mean age ± SD, 24.10 ± 2.64, 4 males). This second version emerged as reliable due to reduced recognition performance across the experimental sessions. Therefore, the second version of the emotional memory task was used in our study.

3.3.3.1 Emotional reactivity task

The emotional reactivity task was utilised to evaluate whether the experimentally induced REM sleep fragmentation altered the psychophysiological reactivity associated with the emotional stimuli. This task involved presenting the participants with emotionally negative and neutral

pictures while recording their EDA and HR. After each image presentation, participants were asked to rate the arousal and valence of the picture using a Likert scale ranging from 1 to 9 on a self-assessment manikin (SAM; Bradley & Lang, 1994; Lang, 1980).

The task consisted of 28 trials; each trial implied: *i)* the presentation of the emotional picture for 6 s, *ii)* 4 s Inter-Stimuli Interval (ISI), *iii)* the request to the participant to subjectively rate the arousal and valence of the image on the SAM, *iv)* 8 to 12 s (jitter time) Inter-Trial Interval (ITI). In Figure 3, a schematic representation of the task is reported.

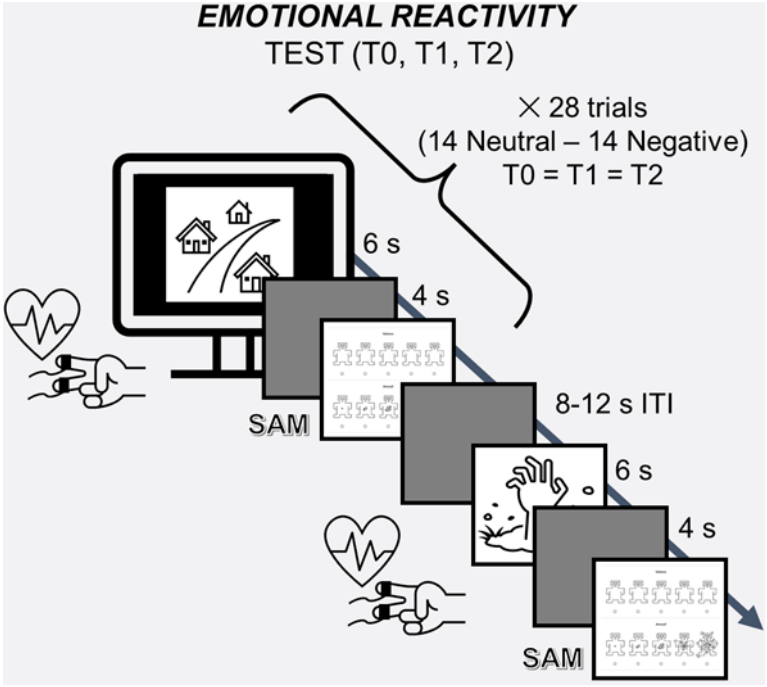

Figure *3*. Schematic representation of the emotional reactivity task.
The heart icon indicates HR recording, while the hand icon with black electrodes on the middle and index fingers indicates EDA recording during picture presentation. The "SAM" word near the manikin images represents the arousal and valence assessment after each picture is presented.

Fifty-six images (28 negative high-arousal, 28 neutral) were selected to build four task versions. The negative images are the most arousing and gruesome images from the IAPS (Lang et al., 2005), while the neutral ones are white background images of sports objects derived from the MST (Stark et al., 2019). The total pool of images was divided into 4 numerically identical blocks (E1_Neg, E2_Neg, E1_Neu, E2_Neu); within each emotional category, the blocks were balanced for arousal and valence scores (all $p \geq 0.780$, comparisons performed with independent sample T-test) derived from our pilot study (Table 1).

TABLE 1. Mean ± SD of valence and arousal value for each block of images of the emotional reactivity task.

				Valence		Arousal	
Block	Valence	Arousal	Comparison	t_{26}	p	t_{26}	p
E1_Neg	2.15 ± 0.39	6.36 ± 0.50	E1_Neg - E2_Neg	-0.02	0.99	0.04	0.96
E2_Neg	2.15 ± 0.49	6.36 ± 0.52					
E1_Neu	5.72 ± 0.25	2.48 ± 0.34	E1_Neu - E2_Neu	-0.28	0.78	-0.15	0.87
E2_Neu	5.74 ± 0.22	2.50 ± 0.34					

By combining the four blocks derived from the total pool of images, we designed 4 versions of the task (Version1.1 = E1_Neg+E1_Neu; Version2.2 = E2_Neg+E2_Neu; Version1.2 = E1_Neg+E2_Neu; Version2.1 = E2_Neg+E1_Neu). This task composition was chosen to minimise potential confounding effects due to pure randomisation of stimuli starting from the total pool of images (e.g., more arousing negative images in one condition than another for some participants and vice versa). Table 2 shows the task version presentation pattern adopted in our task for each participant.

TABLE 2. Emotional reactivity task version distribution for each participant.

Subject ↓	Emotional reactivity task version distribution					
Condition →	CTR/T0	CTR/T1	CTR/T2	FRG/T0	FRG/T1	FRG/T2
1	1-1	1-1	1-1	2-2	2-2	2-2
2	2-2	2-2	2-2	1-1	1-1	1-1
3	1-2	1-2	1-2	2-1	2-1	2-1
4	2-1	2-1	2-1	1-2	1-2	1-2
5	1-1	1-1	1-1	2-2	2-2	2-2
6	2-2	2-2	2-2	1-1	1-1	1-1
7	1-2	1-2	1-2	2-1	2-1	2-1
8	2-1	2-1	2-1	1-2	1-2	1-2
9	1-1	1-1	1-1	2-2	2-2	2-2
10	2-2	2-2	2-2	1-1	1-1	1-1
11	1-2	1-2	1-2	2-1	2-1	2-1
14	2-2	2-2	2-2	1-1	1-1	1-1
15	1-2	1-2	1-2	2-1	2-1	2-1
17	1-1	1-1	1-1	2-2	2-2	2-2
19	1-2	1-2	1-2	2-1	2-1	2-1

Notes: V1-1 = task Version1.1, V2-2 = task Version2.2, V1-2 = task Version1.2, V2-1 = task version 2.1.

3.3.3.2 Emotional memory task

The emotional memory task was adopted to assess the effects of REM sleep fragmentation on the declarative component of the emotional memory trace . This task consisted of a stimulus (emotionally negative/neutral pictures) encoding phase followed by stimuli recognition phases at three different time points (T0, T1, T2; for the encoding/assessment timing, see *Procedure*). During the encoding phase, the participant was asked to memorise the image. In the recognition phase, the participant had to discriminate between the stimuli presented during the encoding phase (i.e., “OLD”) and the new pictures shown during the recognition phase (i.e., “NEW”). The encoding phase consisted of 120 trials; each trial implied *i)* the presentation of the picture for 3s, *ii)* 1.5s ISI. The recognition phase consisted of 80 trials; each trial implied *i)* the

presentation of the image for 2.5s, *ii)* 0.5s ISI, *iii)* the request to the participant to indicate whether the picture was OLD or NEW, and *iv)* 1.5s ITI. In Figure 4, a schematic representation of the task is reported.

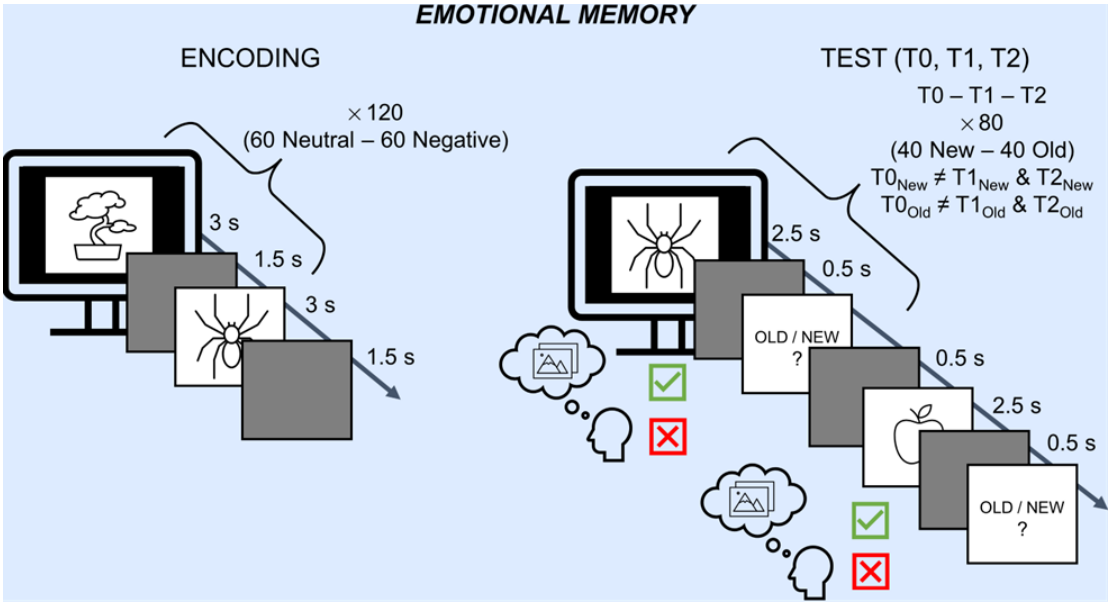

Figure 4. Schematic representation of the emotional memory task.
The schematic representation in the left part of the image refers to the task's encoding phase, indicating the number of stimuli presented for each emotional category. In the right part of the image, the schematic representation refers to the task's recognition (test) phase, specifying the number of pictures shown in each test, which had half of the images from the encoding phase and half completely new. It also specifies that all the New and Old images differed during each test phase.

To build the task, four-hundred-eighty images (240 negative, 240 neutral) were selected from the IAPS and Nencki Affective Picture System (NAPS, Lang et al., 2005; Marchewka et al., 2014). The total pool of images was divided into 8 numerically identical (60 stimuli each) macro-blocks (S1_Neg, S2_Neg, S3_Neg, S4_Neg, S1_Neu, S2_Neu, S3_Neu, S4_Neu) and each macro-block was divided again into 3 numerically identical (20 stimuli each) sub-blocks. For both valences, each macro-block and each sub-block were balanced for

valence and arousal score using standardised values Lang et al., 2005; Marchewka et al., 2014). The appropriateness of the above-reported balancing between macro-blocks for each emotional category was assessed by analysis of variance (ANOVA). The macro-block number (i.e., S1, S2, S3, S4) was adopted as a fixed factor for the macro-block comparisons (all p = 1.00). The same workflow was utilised for sub-block analysis (all p = 1.00). Table 3 shows the valence and arousal mean values for each macro-block.

TABLE 3. Mean ± SD of valence and arousal value for each macroblock of images of the emotional memory task.

Macroblock	Valence	Arousal
S1_Neg	2.50 ± 0.40	6.28 ± 0.58
S2_Neg	2.49 ± 0.37	6.28 ± 0.71
S3_Neg	2.50 ± 0.37	6.29 ± 0.59
S4_Neg	2.50 ± 0.34	6.29 ± 0.62
S1_Neu	5.02 ± 0.31	4.03 ± 0.87
S2_Neu	5.02 ± 0.26	4.03 ± 0.92
S3_Neu	5.02 ± 0.25	4.02 ± 0.90
S4_Neu	5.02 ± 0.25	4.03 ± 0.86

Subsequently, each macroblock was aggregated with its other valence's counterpart (S1_Neg+S1_Neu, S2_Neg+S2_Neu, S3_Neg+S3_Neu, S4_Neg+S4_Neu) to be used as stimuli set during the encoding phase. For the recognition phase, each sub-block of the encoding phase was intermixed with a sub-block taken from a different macroblock (e.g., macroblock S1 during encoding, sub-block S1A intermixed with sub-block S2B in the 1st recognition phase, sub-block S1C intermixed with sub-block S2A in the 2nd recognition phase, sub-block S1B intermixed with sub-block S2C in the 3rd recognition phase). This task composition was chosen to minimise the potential

confounding effects due to the pure randomisation of stimuli starting from the total pool of images. Table 4 shows the macroblocks/sub-blocks presentation pattern adopted in our task for each subject.

TABLE 4. Emotional memory task stimuli distribution for each participant.

Subject ↓	Emotional memory task version distribution							
Condition →	CTR/ENC	CTR/T0	CTR/T1	CTR/T2	FRG/ENC	FRG/T0	FRG/T1	FRG/T2
1	S1	S2A-S1A	S2B-S1B	S2C-S1C	S3	S4C-S3C	S4A-S3A	S4B-S3B
2	S4	S3A-S4A	S3B-S4B	S3C-S4C	S2	S1C-S2C	S1A-S2A	S1B-S2B
3	S2	S4A-S2A	S4B-S2B	S4C-S2C	S1	S3C-S1C	S3A-S1A	S3B-S1B
4	S3	S1A-S3A	S1B-S3B	S1C-S3C	S4	S2C-S4C	S2A-S4A	S2B-S4B
5	S3	S2B-S3B	S2A-S3A	S2C-S3C	S1	S4B-S1B	S4C-S1C	S4A-S1A
6	S4	S1B-S4B	S1A-S4A	S1C-S4C	S2	S3B-S2B	S3C-S2C	S3A-S2A
7	S1	S4B-S1B	S4A-S1A	S4C-S1C	S3	S2B-S3B	S2C-S3C	S2A-S3A
8	S2	S3B-S2B	S3A-S2A	S3C-S2C	S4	S1B-S4B	S1C-S4C	S1A-S4A
9	S4	S3C-S4C	S3B-S4B	S3A-S4A	S1	S2A-S1A	S2C-S1C	S2B-S1B
10	S2	S1C-S2C	S1B-S2B	S1A-S2A	S3	S4A-S3A	S4C-S3C	S4B-S3B
11	S1	S2C-S1C	S2B-S1B	S2A-S1A	S4	S3A-S4A	S3C-S4C	S3B-S4B
14	S3	S4A-S3A	S4C-S3C	S4B-S3B	S1	S2C-S1C	S2B-S1B	S2A-S1A
15	S1	S3A-S1A	S3C-S1C	S3B-S1B	S2	S4C-S2C	S4B-S2B	S4A-S2A
16	S4	S2A-S4A	S2C-S4C	S2B-S4B	S3	S1C-S3C	S1B-S3B	S1A-S3A
17	S1	S4B-S1B	S4C-S1C	S4A-S1A	S2	S3B-S2B	S3A-S2A	S3C-S2C
19	S2	S3B-S2B	S3C-S2C	S3A-S2A	S1	S4B-S1B	S4A-S1A	S4C-S1C

Notes: ENC = encoding phase.

3.3.3.3 Sequential finger-tapping task

The sequential finger-tapping task (SFTT) was utilised as a control measure to assess potential effects of REM sleep fragmentation on procedural memory performance. Moreover, it was useful as an active pause between the emotional reactivity task and the emotional memory task to mitigate potential interfering effects that may arise from performing two emotional tasks consecutively.

The SFTT required the participant to repeatedly digit a five-element numerical sequence as quickly and accurately as possible for 30-second epochs followed by 30-second breaks. The participant had to execute the task using the non-dominant hand's fingers (excluding the thumb). Participants were advised not

to correct occasional errors and to proceed with the task smoothly without interruptions. During the 30-second epochs of digiting, the sequence was always displayed on the screen to limit the working memory load. Below each number, a white rectangle was shown. Each key digit resulted in a circular red marker inside the white rectangle under the number, starting from left to right. Instantly, after tapping the last digit to complete the sequence, all markers vanished, signalling the start of a new sequence. During the 30-second pause, a fixation cross was displayed on the monitor. Figure 5 shows a schematic representation of the SFTT task.

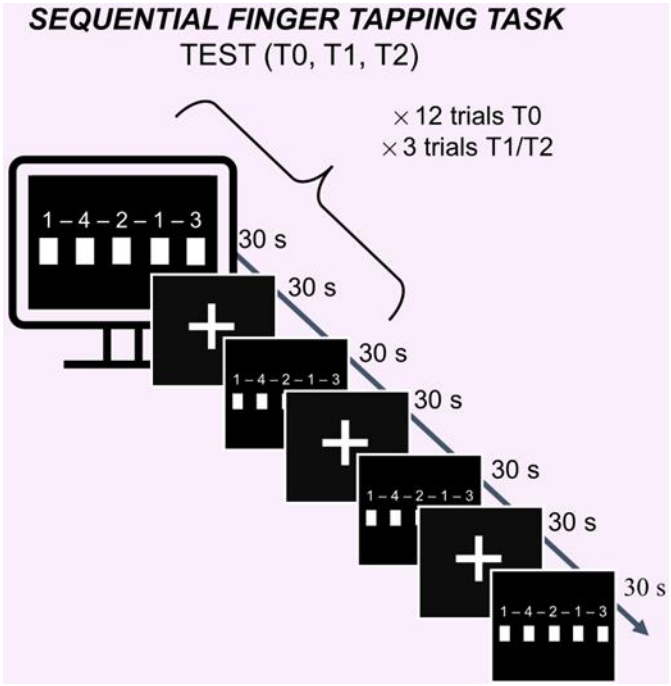

Figure 5. Schematic representation of the sequential finger-tapping task.

We adopted two different sequences in the SFTT (i.e., 1-4-2-1-3; 4-3-4-1-2). Each sequence was designed according to the following rules: *i)* the first digit of the sequence must be different from the last, *ii)* more than two sequential digits were not possible, neither in ascending (e.g., 2-3-4) nor in descending

order (e.g., 4-3-2), *iii)* each number could not appear twice sequentially, and *iv)* three consecutive digits of a sequence could not appear in the other sequence (Fischer et al., 2002; Pereira et al., 2015).

In our study, the participants performed the SFTT 3 times in each condition (T0, T1, T2; see the procedure for details). The first time (T0), the participants had to perform the SFTT executing 12 trials. Each SFTT trial comprised *i)* a 30-second epoch of sequence digiting and *ii)* a 30-second pause. At the T0, the first 9 trials were used as the learning phase, while the last 3 trials were taken as performance (see data analyses for details). In the other task sessions (T1, T2), the participants executed only 3 trials (all taken as performance). During each condition, the SFTT sequence was the same. The sequences adopted in each condition were counterbalanced across participants.

3.4 Data Acquisition and Pre-processing

3.4.1 Actigraphy data

All participants wore a GENEActiv accelerometer (Activinsights Ltd., Kimbolton, UK) on their non-dominant wrist from two days before the first laboratory sleep night until the delayed assessment session (T2) in each condition. The actigraphy devices were initialised through the GENEActiv PC software (version 3.3, Activinsights Ltd., Kimbolton, UK) with a measurement frequency of 50 Hz. Each participant wore the same device in both the CTR and FRG conditions. GENEActiv accelerometers were previously validated as reliable tools for adult sleep assessment (te Lindert & Van Someren, 2013).

Moreover, participants filled out a sleep diary every morning (within 15 minutes upon the final awakening), which has been used to support accelerometric data scoring.

In detail, actigraphic sleep parameters were calculated offline, matching accelerometric raw data with sleep diary data via CICADA software (version 0.10.4, beta, Australia). We derived the following variables: *Time in Bed* (TIB), which indicates the time window from bedtime to get-up time; *Sleep Onset*, which denotes sleep onset time; *Final Awakening*, which signals sleep end time; *Sleep Onset Latency* (SOL), which indicates the time participants took to fall asleep; *#awakenings*, which denotes the total number of awakenings during the sleep period; *Sleep Period*, which indicates the time between *Final Awakening* and *Sleep Onset*; *Wake After Sleep Onset* (WASO), which represents the total time participant spent awake during the sleep period; *Total Sleep Time* (TST), which denotes the total time participant spent sleeping at night (*Sleep Period − WASO*); *Sleep Efficiency* (SE), calculated as 100 * TST ÷ TIB; *#awakenings_Hour*, which denotes the number of awakenings per hour during the sleep period.

3.4.2 Behavioural and subjective data

3.4.2.1 Self-reported information about the laboratory nights

The morning after the laboratory nights, participants provided the following information: *Morning Sleepiness*, measured via a 10-point Likert scale from “extremely alert” to “extremely sleepy”; *Sleep Depth*, evaluated via a 10-point Likert scale from “very light” to “very deep”; *Sleep Calmness*, assessed via a

10-point Likert scale from “very agitated” to “very calm”; *Sleep Restfulness*, measured via a 10-point Likert scale from “very restless” to “very restful”; *Bracelet disturbance*, evaluated via a 10-point Likert scale from “not at all disturbing” to “very disturbing”; *#Perceived Stimulations*, assessed requiring participants to report the total number of perceived stimulation during the night; *Stimulation Disturbance*, measured via a 10-point Likert scale from “not at all disturbing” to “very disturbing”.

3.4.2.2 Subjective Emotional Reactivity

As reported in section 3.1.3.1, participants rated the arousal and valence levels of the stimuli during the emotional reactivity task using a 9-point Likert scale on the SAM (Bradley & Lang, 1994; Lang, 1980). The SAM scale depicted a cartoon-type manikin representing human emotional expressions from smiling and happy to frowning and unhappy for the valence rating scale. For the arousal evaluation scale, the SAM manikin represented expressions ranging from calm and relaxed to excited and wide-eyed. Participants' ratings were provided via numeric keypad on the keyboard. We derived *Valence* and *Arousal* variables to assess the subjective emotional reactivity (Baran et al., 2012; Wiesner et al., 2015).

3.4.2.3 Emotional Memory Performance

Emotional memory performance was evaluated by calculating the discrimination index (d-prime or d’).

The d' is a measure of sensitivity and reflects the ability to discriminate a target (old pictures) from non-target stimuli (new images) and is considered to be unaffected by response bias (Macmillan & Creelman, 2004; Stanislaw & Todorov, 1999). Higher d' values indicate better discrimination performance.

The d' index was obtained by calculating the Hit Rate (HR) and the False Alarm Rate (FAR). The HR refers to the Hits (i.e., the number of old pictures correctly identified as seen) divided by the total number of old pictures in the recognition task, namely $HR = Hits \div N_{OLD}$. The FAR refers to the False Alarm (i.e., the number of new images erroneously defined as seen) divided by the total number of new images in the recognition task, precisely FAR = False Alarm ÷ N_{NEW}. Then, HR e FAR values have been standardised (i.e., z-transformed), and the d' was determined using the formula: $d' = zHR - zFAR$. Considering that d' must not be determined when HR = 1 and FAR = 0, we substituted HR values of 1 with an $HR = 1 - 1\div(2N)$ and FAR values of 0 with a $FAR = 1\div 2N$, where N represents the number of targets (Macmillan & Creelman, 2004).

3.4.2.4 Procedural Memory Performance

Procedural memory performance was evaluated through the SFTT by computing the following variables from each 30 s digiting epoch: correct sequences (CS), i.e., the number of correctly typed sequences; sequential reaction time (SRT), i.e., average digiting time (seconds) of the correctly completed sequences; and Accuracy (ACC), i.e., the number of correctly typed sequences relative to the total number of completed sequences.

3.4.3 Psychophysiological data

3.4.3.1 Electrodermal activity and heart rate

EDA and HR psychophysiological signals were acquired via the Biosignal Explorer (Biosignalsplux, PLUX wireless biosignals S.A., Lisbon, Portugal) using a sampling rate of 1000 Hz and a resolution of 16 bits. The Biosignal EDA sensor acquired signals from 0 to 24 μS, while the Biosignal ECG sensor acquired signals from -37.5 μV to + 37.5 μV.

After data acquisition, EDA raw data were handled in MATLAB (R2023a, Update 1, 9.14.0.2239454, The MathWorks Inc., Natick, Massachusetts) through the skin conductance analysis software Ledalab (MATLAB toolbox; Benedek & Kaernbach, 2010). The EDA raw signal was pre-processed by down-sampling it at 10 Hz, applying a low-pass second-order Butterworth filter at 1 Hz, and smoothing it with a Gauss window of 10. After the pre-processing phase, continuous decomposition analysis was performed.

We considered a skin conductance response (SCR) as a minimum EDA signal increase of 0.01 μS in a response window between 0.5 and 6 s after the stimulus onset (Braithwaite et al., 2013). Specifically, we extracted the following variables: *#SCR*, representing the number of SCR detected within the response window after the stimulus onset; and the $Latency_{SCR}$, corresponding to the time elapsed between the stimulus onset and the first SCR within the response window. EDA activity is directly regulated by the sympathetic branch of the autonomic nervous system (Dawson et al., 2000), and #SCR and LatencySCR are typically used as indices of emotional reactivity (Boucsein et al., 2012; Christopoulos et al., 2016; Herrero et al., 2020).

HR raw data were handled in Artiifact (Kaufmann et al., 2011) to extract the intra-bit interval (IBI) by applying the Bernston detection method as an artifact-correction procedure. HR artifact-corrected data taken from the emotional reactivity task were processed in MATLAB with Kardia software (Perakakis et al., 2010) to extract the phasic cardiac responses (PCR) to stimuli onset, allowing us to calculate heart rate deceleration (*HRD*) response. *HRD* was computed by subtracting the lowest heartbeat value collected during the 6 s post stimuli onset from the mean heartbeat recorded in the 2 s before stimulus presentation. HRD response reflects stimulus elaboration, and emotional salient information processing is associated with increased HRD compared to neutral stimuli (Cook et al., 1997; Horndasch et al., 2023; Pollatos et al., 2007).

3.4.3.2 PSG

The PSG encompassed EEG, EOG, EMG and ECG signal recording.

EEG and EOG signals were registered with a 64-channel BrainCap (Brain Products GmbH, Germany) connected to a BrainAmp MR amplifier (Brain Products GmbH, Germany) using a sampling rate of 500 Hz adopting a high cut-off frequency of 250 Hz, with a resolution of 0.5 μV acquiring a range of ± 16.384 mV. EMG and ECG signals were acquired with Ag/AgCl electrodes plugged into a bipolar amplifier (BrainAmp ExG MR, Brain Products GmbH, Germany) connected to the BrainAmp MR amplifier (Brain Products GmbH, Germany). EMG signal was acquired with a sampling rate of 500 Hz, a high cut-off frequency of 250 Hz, and a resolution of 0.1 mV, acquiring a range of ± 3.2768 mV. The ECG signal was recorded with a sampling rate of 500 Hz, a

high cut-off frequency of 1000 Hz, and a resolution of 10 mV, acquiring a range of ± 327.68 mV. Then PSG raw data were filtered during the acquisition with the following parameters: EEG and EOG, high-pass filter at 0.016 Hz, no low-pass filter and notch filter at 50 Hz; EMG, high-pass filter at 100 Hz, low-pass filter at 250 Hz and notch filter at 50 Hz; and ECG high-pass filter at 0.001 Hz, no low-pass filter and notch filter at 50 Hz.

The BrainAmp MR amplifier (Brain Products GmbH, Germany) was connected to a desktop computer (DESKTOP-C3LTGCK, Dell Technologies, Round Rock, Texas), and the BrainVision Recorder (Version 1.26.0101, Brain Products GmbH, Germany) was adopted as the acquisition software. Moreover, a trigger box (TriggerBox Plus, Brain Products GmbH, Germany) was connected to our Arduino®-based stimulation device to have a marker on EEG recording in conjunction with the start/finish of the vibrotactile stimulation.

During each laboratory sleep night, the PSG recording was shown on a 32-inch monitor (Samsung UE590, Seul, South Korea), and a display filter was applied to allow online sleep stage identification.

After each laboratory night, the EEG raw data were extracted in EDF+ format with BrainVision Analyser (version 2.2, Brain Products GmbH, Germany), and sleep scoring was performed according to the AASM® criteria (Berry et al., 2017) via Wonambi (version 7.11) software. Sleep scoring was performed by the same sleep expert for each participant on both laboratory sleep nights (CTR and FGR). Sleep macrostructure variables and sleep scoring data are reported in Table 5.

Table 5. Description of PSG macrostructure variables and sleep scoring data.

Variable	Definition
TST	Total Sleep Time (min)
SOL	Sleep Onset Latency (min)
WASO	Wake After Sleep Onset (min)
SE	Sleep efficiency (min)
N1	Time (min) in sleep stage NREM 1
N2	Time (min) in sleep stage NREM 2
N3	Time (min) in sleep stage NREM 3
REM	Time (min) in sleep stage REM
N1 %	% of the TST in NREM 1
N2 %	% of the TST in NREM 2
N3 %	% of the TST in NREM 3
REM %	% of the TST in REM
#awakenings	Number of nocturnal awakenings

Moreover, a series of variables were derived from the PSG to evaluate the effects of vibrotactile stimulations on a broad range of sleep characteristics (Table 6).

Table 6. Description of additional PSG variables focused on REM continuity, arousal, and body movements.

Variable	Definition
REM Sleep Fragmentation Index	Total bouts of NREM1/Wakefulness during the REM episodes divided by the total duration of the REM episodes (hrs)
Maximum unperturbed REM sleep duration	Maximum time of unperturbed REM sleep (min)
Mean unperturbed REM sleep duration	Averaged time of unperturbed REM sleep (min)
#spont. arousal	Number of spontaneous arousal during sleep
Spont. arousal duration	Averaged duration of spontaneous arousal during sleep
#BM	Number of Body Movement during sleep
BM duration	Averaged duration of Body Movement during sleep
#induced arousal	Number of induced arousal during sleep
Induced arousal duration	Averaged duration of induced arousal during sleep

3.5 Statistical Analysis

A paired-sample t-test (Students' t) was used to compare actigraphic data, self-reported information about the laboratory nights, and PSG sleep parameters between the CTR and FRG conditions.

Specifically, we evaluated potential differences between CTR and FRG conditions for the actigraphic variables on the 2 nights before the laboratory sleep nights and on the 2 nights preceding the T2 testing phase. Analyses were based on 13 participants for the days preceding the laboratory sleep nights, and on 14 participants for the days preceding the T2 testing session due to missing data caused by technical issues with the actigraphic recordings.

The comparisons on self-reported information about the laboratory nights aimed to assess potential subjective differences in morning sleepiness, sleep depth, calmness, and restfulness. Moreover, perception of the stimulation setting was also evaluated in both conditions.

Finally, we examined whether the experimental sleep fragmentation altered the participants' nocturnal sleep by comparing the laboratory PSG parameters in CTR and FRG conditions.

As regards t-test analyses, we decided not to correct paired t-test results for multiple comparisons, adopting a more conservative approach since correcting p-values would have increased the likelihood of confirming the assumptions of non-significant differences between conditions.

Linear mixed models (LMM) were used to compare subjective emotional reactivity indices, emotional memory performance, procedural memory performance, and objective emotional memory variables between the CTR and FRG conditions. For each model, the participant was used as a cluster variable, and a random intercept per participant was included to account for intraindividual variability and measure correlation within participants.

Specifically, for the subjective emotional reactivity indices, we performed LMM analyses with both Valence and Arousal ratings as dependent variables. Each

model comprised the factors *Condition* (CTR, FRG), *Session* (T0, T1, T2), *Stimulus Type* (Negative, Neutral), and their interaction as predictors. One subject was removed from the analyses due to unreliable responses caused by a misunderstanding of task instructions.

Regarding emotional memory performance, we carried out an LMM with d' as the dependent variable. The model included the factors *Condition* (CTR, FRG), *Session* (T0, T1, T2), *Stimulus Type* (Negative, Neutral), and their interaction as predictors.

For procedural memory performance, we performed LMM analyses with ACC, CS, and SRT as dependent variables. Each model comprised the factors *Condition* (CTR, FRG), *Session* (T0, T1, T2), and their interaction as predictors. Analyses performed on procedural memory performance variables were based on a sample of 12 participants due to missing data for technical issues with SFTT.

Finally, concerning the physiological indices of emotional reactivity, we performed LMM analyses with HRD and $Latency_{SCR}$ as dependent variables. On the other hand, a Generalized Linear Mixed Model (GLMM) with a Poisson distribution was fitted to predict #SCR due to the nature of the dependent variable (count data) (Coxe et al., 2009; Gardner et al., 1995).

For each model, the analysis comprised the factors *Condition* (CTR, FRG), *Session* (T0, T1, T2), *Stimulus Type* (Negative, Neutral), and their interaction as predictors. The analysis on HRD was based on 14 participants since data from 1 participant was removed because he was unable to follow the instruction of minimising movement to reduce recording artifacts. Analyses performed on EDA variables were based on 13 participants since 1 participant was

categorised as a *non-responder* during at least one of the T0 sessions (Gatti et al., 2018; Lonsdorf et al., 2019), and 1 participant did not follow the instruction of minimising movement to reduce artifacts.

For LMM and GLMM analyses, the interpretation of significant effects followed a hierarchical approach, in which significant main effects were subordinated to the absence of significant interaction effects involving the same factor. Similarly, the interpretation of interaction effects was conditioned by the absence of significant higher-order interaction effects.

In case of significant main effects, post-hoc comparisons with Bonferroni correction were computed. On the other hand, when significant interaction effects emerged, planned comparisons with Bonferroni correction were performed based on the study aims. This choice was made to reduce the overall number of contrasts to reduce the risk of Type II error when correcting for multiple comparisons. In particular, we tested potential differences between sessions within each condition (i.e., T0 vs T1, T0 vs T2, and T1 vs T2) to determine how the assessed variable evolved over time in the two conditions. Moreover, we were also interested in comparing CTR and FRG conditions at each time point to investigate possible baseline differences between conditions (i.e., CTR at T0 vs FGR at T0) and to assess if the condition differently affected the measured variable after the laboratory sleep nights (i.e., CTR at T1 vs FGR at T1) or in the long term (i.e., CTR at T2 vs FGR at T2).

All the above-reported analyses were performed in Jamovi (Version 2.3, The Jamovi Project, Sydney, Australia). All tests were two-tailed, and statistical significance was set at $p < 0.05$.

3.6 Results

3.6.1 Actigraphy

Actigraphic data analysis indicated that sleep parameters 2 days before the laboratory sleep night did not differ between CTR and FRG conditions (Table 7). Moreover, actigraphic sleep parameters on the day before the laboratory sleep night (Table 8) did not differ between the two experimental conditions.

Table 7. Mean ± SD of actigraphic sleep variables in the CTR and FRG conditions 2 days before the laboratory sleep nights and their statistical comparisons.

Variables	CTR	FRG	t_{12}	p
Sleep Onset	23:55 ± 01:10	23:45 ± 01:15	0.49	0.630
Final Awakening	08:20 ± 01:10	08:14 ± 01:00	0.26	0.798
SOL	6.08 ± 4.77	6.08 ± 5.20	0.00	1.000
#awakening	18.00 ± 3.96	19.08 ± 5.65	-0.68	0.507
WASO	62.35 ± 25.95	55.41 ± 28.36	0.78	0.448
TST	442.34 ± 59.44	454.20 ± 69.67	-0.52	0.611
Sleep Period	504.75 ± 69.80	509.65 ± 76.11	-0.22	0.828
SE	85.87 ± 4.21	87.33 ± 5.11	-0.91	0.382
#awakening_hour	2.18 ± 0.57	2.22 ± 0.40	-0.22	0.831

Table 8. Mean ± SD of actigraphic sleep variables in the CTR and FRG conditions the day before the laboratory sleep nights and their statistical comparisons.

Variables	CTR	FRG	t_{12}	p
Sleep Onset	23:45 ± 01:01	23:44 ± 01:11	0.03	0.975
Final Awakening	08:03 ± 00:43	08:04 ± 00:39	-0.10	0.923
SOL	3.85 ± 3.41	10.46 ± 22.62	-1.02	0.327
#awakening	18.85 ± 4.30	19.31 ± 4.89	-0.49	0.632
WASO	53.25 ± 21.57	56.04 ± 37.01	-0.26	0.802
TST	445.06 ± 58.15	443.88 ± 74.71	0.07	0.948
Sleep Period	498.22 ± 66.27	499.74 ± 84.81	-0.10	0.925
SE	87.54 ± 3.94	86.60 ± 6.22	0.52	0.612
#awakening_hour	2.265 ± 0.43	2.33 ± 0.54	-0.72	0.484

As far as the day after the laboratory nights is concerned, participants woke up significantly later in the FRG condition compared to the CTR condition. No other difference emerged between conditions (Table 9).

Table 9. Mean ± SD of actigraphic sleep variables in the CTR and FRG condition the day after the laboratory sleep nights, and their statistical comparisons. Significant comparisons are reported in bold.

Variables	CTR	FRG	t_{13}	p
Sleep Onset	00:00 ± 01:10	00:09 ± 01:33	-0.26	0.798
Final Awakening	**08:27 ± 01:01**	**09:19 ± 01:21**	**-2.43**	**0.030**
SOL	11.57 ± 17.70	4.28 ± 3.34	1.49	0.161
#awakening	17.71 ± 2.52	18.28 ± 4.32	-0.45	0.661
WASO	40.78 ± 21.52	53.79 ± 44.18	-0.94	0.366
TST	465.86 ± 77.39	496.64 ± 84.05	-1.91	0.078
Sleep Period	506.61 ± 86.10	550.62 ± 109.94	-1.86	0.085
SE	89.19 ± 5.02	89.42 ± 6.11	-0.10	0.920
#awakening_hour	2.13 ± 0.40	2.00 ± 0.31	1.51	0.154

Finally, actigraphic sleep parameters did not differ between conditions on the night before the T2 testing phase, except for Sleep Onset. Participants started to sleep (at home) earlier during the CTR condition relative to the FRG condition (Table 10).

Table 10. Mean ± SD of actigraphic sleep variables in the CTR and FRG condition the night before the T2 testing phase, and their statistical comparisons. Significant comparisons are reported in bold.

Variables	CTR	FRG	t_{13}	p
Sleep Onset	**23:39 ± 00:34**	**00:17 ± 00:44**	**-2.18**	**0.048**
Final Awakening	07:43 ± 00:31	07:40 ± 00:26	0.39	0.700
SOL	8.28 ± 8.04	5.86 ± 6.49	0.79	0.443
#awakening	17.86 ± 3.37	15.93 ± 3.43	1.56	0.143
WASO	46.95 ± 21.46	41.31 ± 28.49	0.76	0.463
TST	436.70 ± 44.52	401.26 ± 56.00	1.95	0.074
Sleep Period	483.46 ± 44.59	442.71 ± 61.35	2.12	0.054
SE	88.01 ± 5.10	88.74 ± 5.85	-0.40	0.695
#awakening_hour	2.23 ± 0.46	2.16 ± 0.39	0.45	0.657

3.6.2 Self-reported information about the laboratory nights

Paired-sample t-test results showed that participants reported similar morning sleepiness in CTR and FRG conditions. No differences emerged in self-reported sleep depth, calmness, and restfulness between conditions.

After the FRG night, participants reported higher bracelet disturbances, a greater number of perceived stimuli, and a significantly higher disturbance from the stimulations than the CTR condition. However, the number of perceived stimuli was significantly lower relative to the number of provided stimuli ($t = -12.50$, $p < 0.001$). Indeed, participants reported a mean (± SD) of 3.53 (± 1.68) perceived stimuli, while a mean of 71.73 (± 20.87) stimulations during FRG nights was delivered (Table 11).

Table 11. Mean ± SD of self-reported information about the laboratory night in the CTR and FRG condition and their statistical comparisons. Significant comparisons are reported in bold.

Variables	CTR	FRG	t_{14}	p
Morning Sleepiness	3.40 ± 1.92	4.00 ± 2.14	-1.04	0.315
Sleep Depth	5.40 ± 2.32	6.20 ± 1.52	-1.92	0.075
Sleep Calmness	5.93 ± 2.15	5.53 ± 2.29	0.95	0.361
Sleep Restfulness	6.27 ± 1.94	6.53 ± 1.88	-0.45	0.662
Bracelet disturbance	**0.80 ± 2.14**	**4.00 ± 2.85**	**-2.99**	**0.010**
#Perceived Stimulation	**0.00 ± 0.00**	**3.53 ± 1.68**	**-8.12**	**< 0.001**
Stimulation Disturbance	**0.00 ± 0.00**	**3.87 ± 2.36**	**-6.36**	**< 0.001**

3.6.3 PSG sleep parameters

As far as sleep macrostructure data are concerned, vibrotactile stimulation was associated with longer N1 sleep time and N1%, and reduced REM sleep time and REM% (Table 12). No other sleep parameters were affected by stimulation, suggesting a moderate effect of FRG condition on the overall sleep architecture.

Table 12. Mean ± SD of PSG macrostructure variables and sleep scoring data in the CTR and FRG conditions and their statistical comparisons. Significant comparisons are reported in bold.

Variables	CTR	FRG	t_{14}	p
TST	429.27 ± 29.76	427.67 ± 26.31	0.22	0.825
SOL	22.67 ± 15.49	22.20 ± 14.16	0.16	0.878
WASO	52.37 ± 34.38	45.27 ± 21.33	1.09	0.296
SE	85.27 ± 7.17	86.23 ± 4.26	-0.65	0.526
N1 (min)	**33.30 ± 7.31**	**63.27 ± 18.26**	**-7.51**	**< 0.001**
N2 (min)	225.23 ± 29.86	219.03 ± 29.49	0.82	0.426
N3 (min)	86.80 ± 17.50	93.57 ± 23.74	-1.82	0.091
REM (min)	**83.93 ± 17.69**	**51.80 ± 10.40**	**5.97**	**< 0.001**
N1 %	**7.77 ± 1.66**	**14.87 ± 4.53**	**-7.26**	**< 0.001**
N2 %	52.42 ± 5.77	51.10 ± 5.20	0.88	0.392
N3 %	20.30 ± 4.32	21.92 ± 5.45	-1.74	0.103
REM %	**19.50 ± 3.62**	**12.11 ± 2.29**	**6.73**	**< 0.001**
#awakenings	18.47 ± 7.16	21.80 ± 11.26	-1.30	0.216

On the other hand, during the FRG condition, participants showed a higher REM Sleep Fragmentation Index, reduced maximum unperturbed REM sleep duration, and shorter mean unperturbed REM sleep duration (Table 13), confirming the effectiveness of the experimentally induced REM sleep fragmentation.

Table 13. Mean ± SD of additional PSG variables focused on REM continuity, arousal, and body movements in the CTR and FRG conditions and their statistical comparisons. Significant comparisons are reported in bold.

Variables	CTR	FRG	t_{14}	p
REM Sleep Fragmentation Index (hrs)	**8.79 ± 2.65**	**24.02 ± 3.81**	**-14.30**	**< 0.001**
Maximum unperturbed REM sleep duration (min)	**15.07 ± 5.48**	**4.23 ± 1.49**	**7.11**	**< 0.001**
Mean unperturbed REM sleep duration (min)	**5.94 ± 2.06**	**1.30 ± 0.42**	**8.22**	**< 0.001**
#spont. arousal	46.93 ± 13.77	56.07 ± 21.51	-1.70	0.111
Spont. arousal duration (s)	7.62 ± 1.05	7.44 ± 0.60	0.92	0.373
#BM	28.47 ± 11.41	23.33 ± 11.92	1.49	0.159
BM duration (s)	11.61 ± 2.31	12.20 ± 1.48	-0.80	0.436
#induced arousal	**0.00 ± 0.00**	**58.67 ± 16.14**	**-14.08**	**< 0.001**
Induced arousal duration (s)		7.70 ± 1.12		

3.6.4 Behavioural variables

3.6.4.1 Emotional Memory Performance

Analysis on the d' variable highlighted a significant effect of the *Session* factor (Table 14). Post-hoc comparisons indicated that emotional memory performance decreased over time (Figure 8). However, emotional memory changes were not affected by the experimental condition or the emotional valence of the encoded material since the interactions involving those factors were not significant.

Table 14. Results of the LMM analysis performed on the d' variable. Significant effects are reported in bold.

Factor	F	p
Condition	3.20	0.075
Session	**81.09**	**< 0.001**
Stimulus Type	0.03	0.862
Condition x Session	0.21	0.811
Condition x Stimulus Type	0.02	0.891
Session x Stimulus Type	0.31	0.732
Condition x Session x Stimulus Type	1.32	0.269

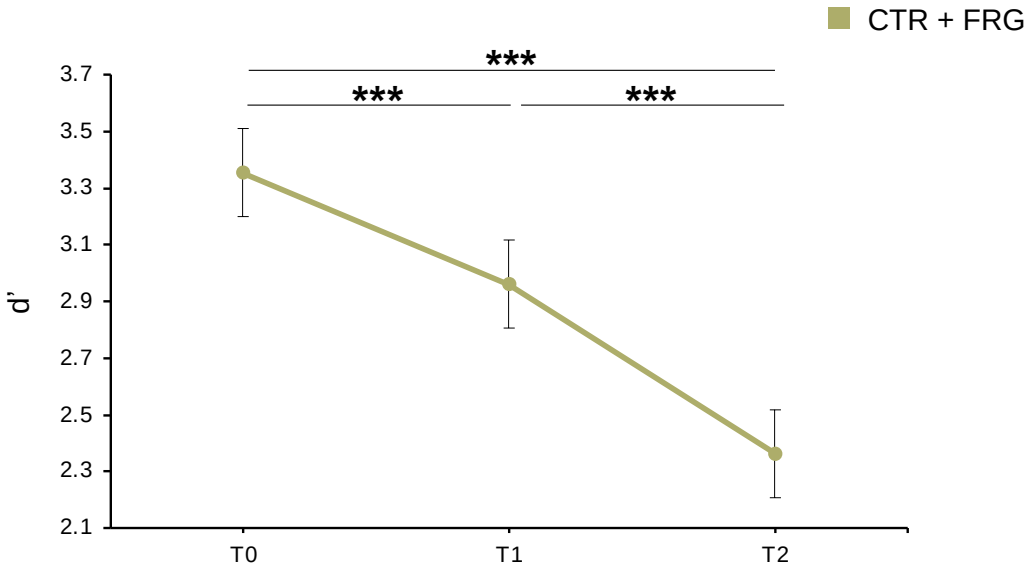

Figure 8. Mean and standard errors of d' values during the different assessment phases. Asterisks indicate significant differences (*** p < 0.001).

3.6.4.2 Subjective Emotional Reactivity

Results of LMM performed on the Valence ratings are reported in Table 15. Analysis revealed a significant main effect of the *Stimulus Type* factor. Negative pictures were rated lower on valence than the neutral images independently of session and condition (Figure 9). No other main effects or interactions were significant.

Table 15. Result of the LMM analyses performed on Valence ratings. Significant effects are reported in bold.

Factor	F	p
Condition	2.46	0.117
Session	2.54	0.079
Stimulus Type	**9017.91**	**< 0.001**
Condition x Session	2.17	0.115
Condition x Stimulus Type	3.24	0.072
Session x Stimulus Type	0.97	0.378
Condition x Session x Stimulus Type	2.35	0.095

As far as the Arousal rating variable is concerned, the analyses showed a significant effect of the *Stimulus Type* factor (Table 16), indicating that participants evaluated negative pictures as more arousing (Figure 9). No other significant main effect or interaction emerged.

Table 16. Result of the LMM analyses performed on Arousal ratings. Significant effects are reported in bold.

Factor	F	p
Condition	3.34	0.069
Session	1.67	0.189
Stimulus Type	**6663.81**	**< 0.001**
Condition x Session	0.82	0.441
Condition x Stimulus Type	0.12	0.727
Session x Stimulus Type	0.32	0.725
Condition x Session x Stimulus Type	0.91	0.403

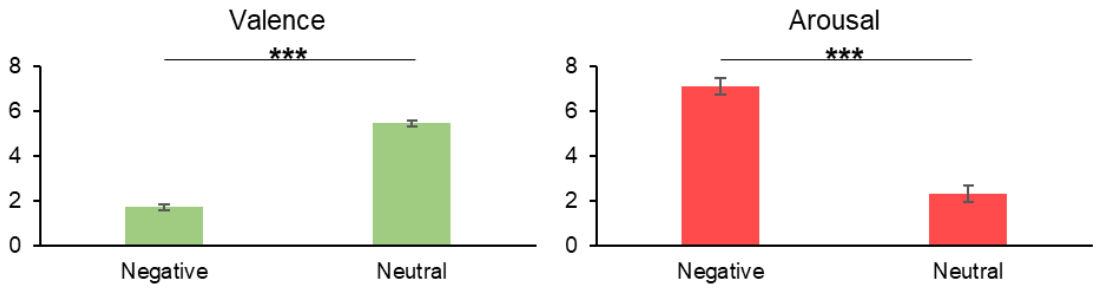

Figure 9. Mean and standard errors of Valence (left) and Arousal (right) ratings for Negative and Neutral pictures at the emotional reactivity task. Asterisks indicate significant comparisons (*** $p < 0.001$).

3.6.4.3 Procedural Memory Performance

LMM analyses carried out on the ACC variable reported significant main effects of the *Condition* and *Session* factors (Table 17). Regardless of the session, ACC was higher during CTR than during FRG condition (Figure 10 A). Moreover, post hoc comparisons revealed that ACC increased from T0 to T2 ($t = -3.29$, $p = 0.005$) (Figure 10 B). However, no significant *Condition* × *Session* interaction emerged.

Table 17. Result of the LMM analyses performed on the ACC variable. Significant effects are reported in bold.

Factor	F	p
Condition	**11.28**	**0.001**
Session	**5.58**	**0.006**
Condition x Session	0.15	0.862

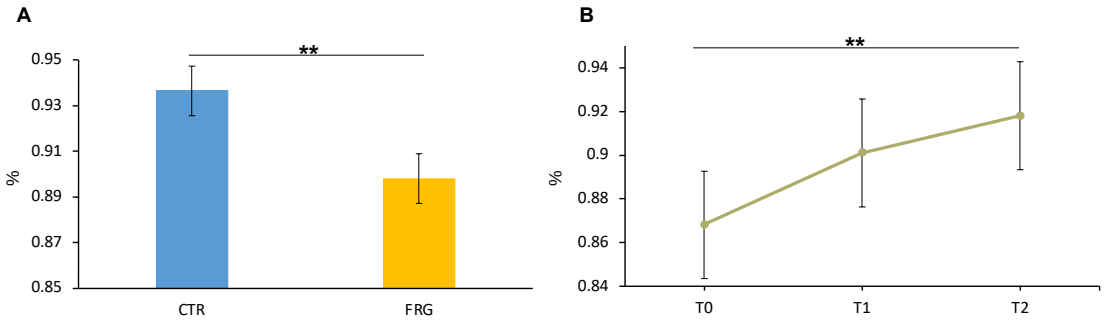

Figure 10. Mean and standard errors of ACC value during each condition (A) and assessment phase (B). The asterisk represents a significant comparison (** $p < 0.01$).

Analyses on the CS variable showed significant main effects of the *Condition* and *Session* factors (Table 18). Independently of the session, typed CS were higher during CTR than FRG condition (Figure 11 A). Furthermore, post hoc comparisons of the *Session* means indicated that the number of typed CS increased between T0 and T1 ($t = -3.79$, $p < 0.001$) and between T0 and T2 ($t = -5.83$, $p < 0.001$), remaining stable between T1 and T2 ($t = -2.03$, $p = 0.141$) (Figure 11 B). *Condition* × *Session* interaction was not significant.

Table 18. Result of the LMM analyses performed on the CS variable. Significant effects are reported in bold.

Factor	F	p
Condition	**10.53**	**0.002**
Session	**17.49**	**< 0.001**
Condition x Session	0.27	0.764

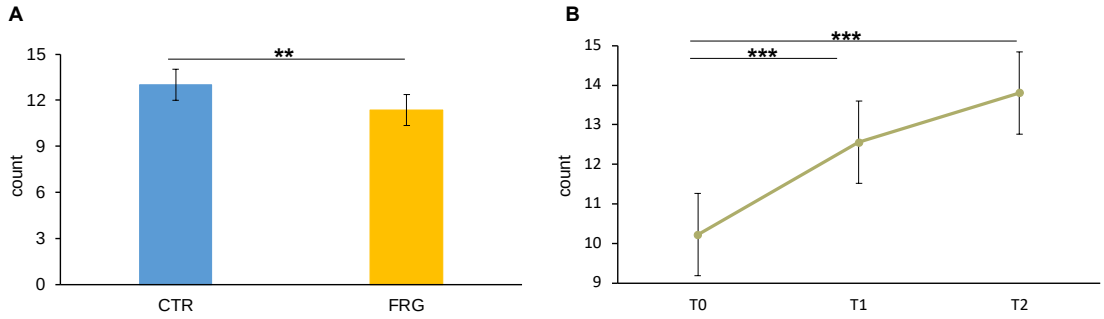

Figure 11. Mean and standard errors of CS value during each condition (A) and assessment phase (B). The asterisks indicate significative differences (** $p < 0.01$; *** $p < 0.001$).

As regards the SRT variable, the analysis indicated significant main effects of the *Condition* and *Session* Factors (Table 19). Regardless of the session, SRT values were lower during CTR than in the FRG condition (Figure 12 A). Furthermore, post hoc analyses of the *Session* means indicated that SRT decreased from T0 to T1 ($t = 3.53$, $p = 0.003$) and from T0 to T2 ($t = 5.01$, $p < 0.001$), remaining stable between T1 and T2 ($t = 1.48$, $p = 0.431$) (Figure 12 B). However, no significant *Condition* × *Session* interaction emerged.

Table 19. Result of the LMM analyses performed on the SRT variable. Significant effects are reported in bold.

Factor	F	p
Condition	**4.70**	**0.035**
Session	**13.27**	**< 0.001**
Condition x Session	0.04	0.960

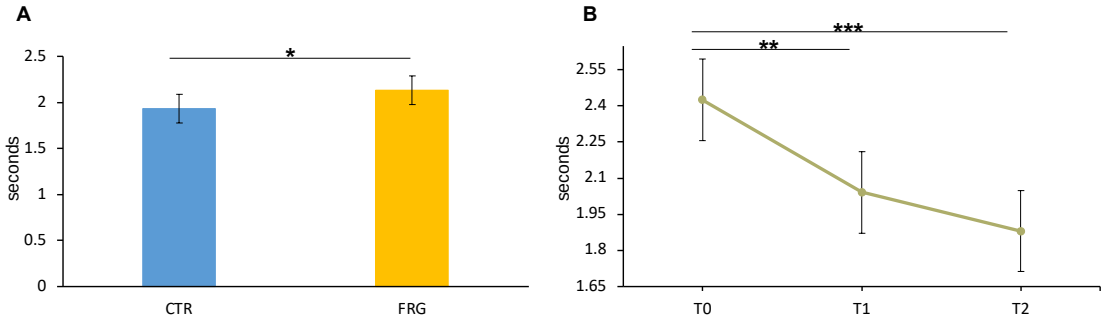

Figure 12. Mean and standard errors of SRT value during each condition (A) and assessment phase (B). The asterisks represent significative differences (* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$).

3.6.5 Psychophysiological data

3.6.5.1 Heart Rate Deceleration

The LMM analysis highlighted significant main effects of the *Session* and *Stimulus Type* factors and a significant *Condition* × *Session* interaction for the HRD index (Table 20). As far as the *Stymulus Type* effect is concerned, HRD was higher for negative stimuli compared to neutral ones (Figure 13).

Table 20. Result of the LMM analyses performed on the HRD variable. Significant effects are reported in bold.

Factor	F	p
Condition	0.68	0.411
Session	**4.10**	**0.017**
Stimulus Type	**7.11**	**0.008**
Condition x Session	**6.98**	**0.001**
Condition x Stimulus Type	0.39	0.531
Session x Stimulus Type	0.16	0.855
Condition x Session x Stimulus Type	0.04	0.963

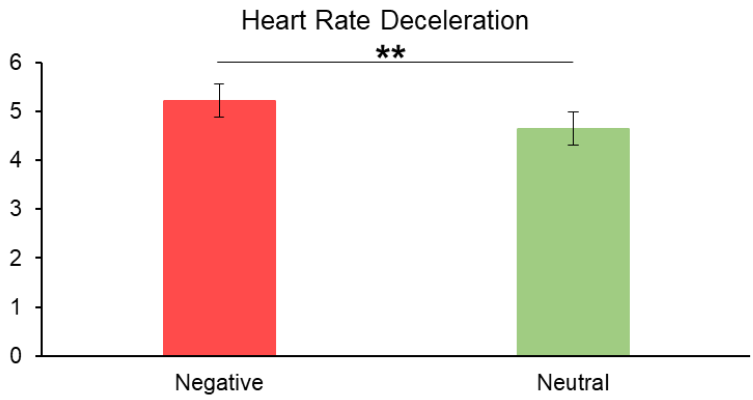

Figure 13. Mean and standard errors of HRD values for negative and neutral images. The asterisks indicate significative differences (** $p < 0.01$).

Moreover, planned comparisons for the *Condition* × *Session* interaction revealed that HRD decreased from T0 to T1 ($t = 3.64$, $p = 0.002$), stabilising at T2 ($t = 0.26$, $p = 1.000$) during the CTR condition. On the other hand, HRD remained stable in the FRG condition between sessions (all $p \geq 0.984$). Finally, HRD at T2 was significantly higher during FRG relative to the CTR condition ($t = 2.86$, $p = 0.039$) (Figure 14).

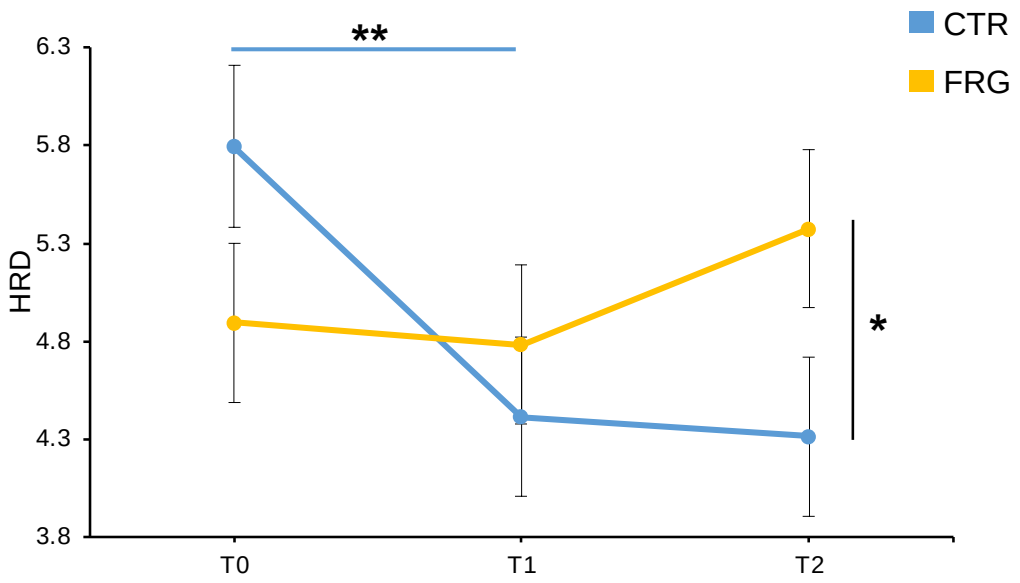

Figure 14. Mean and standard errors of HRD values in CTR and FRG condition at any assessment phase. Asterisks indicate significative differences (* $p < 0.05$; ** $p < 0.01$). The light blue bar shows the significant post-hoc comparison in the CTR condition. The black bar indicates the significant difference between the CTR and FRG conditions.

3.6.5.2 Electrodermal Activity Indices

Poisson GLMM performed on the #SCR variable revealed a significant main effect of the factor *Session* and a significant *Condition* × *Session* interaction (Table 21).

Table 21. Results of the GLMM analysis performed on the #SCR variable. Significant effects are reported in bold.

Factor	χ^2	p
Condition	0.23	0.634
Session	**57.22**	**<0.001**
Stimulus Type	0.00	0.999
Condition x Session	**10.1**	**0.006**
Condition x Stimulus Type	2.80	0.094
Session x Stimulus Type	1.56	0.458
Condition x Session x Stimulus Type	4.45	0.108

Planned comparisons indicated that the #SCR significantly decreased from T0 to T1 ($z = 4.73$, $p < 0.001$), stabilising at T2 ($z = -0.92$, $p = 1.000$) during the CTR condition. On the other hand, during the FRG condition, the #SCR significantly decreased from T0 to T1 ($z = 5.95$, $p < 0.001$) and increased from T1 to T2 ($z = -5.29$, $p < 0.001$), returning to T0 value at T2 ($z = 0.70$, $p = 1.000$). Moreover, #SCR at T2 was significantly higher during FRG relative to the CTR condition ($z = 2.83$, $p = 0.045$; Figure 15).

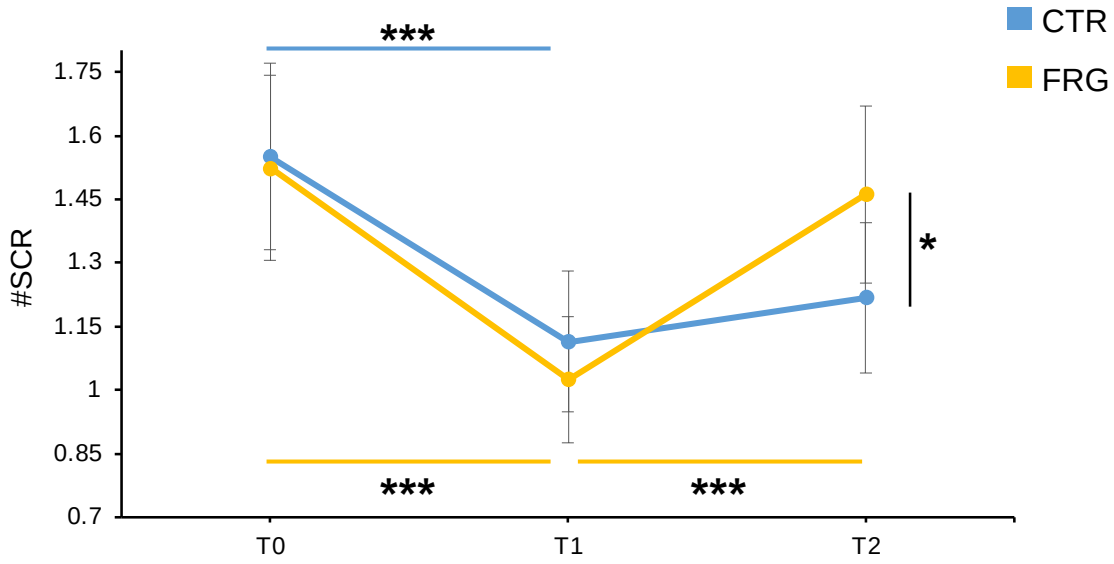

Figure 15. Mean and standard errors of #SCR values in CTR and FRG condition during each assessment phase. The asterisks indicate significant differences (* $p < 0.05$, *** $p < 0.001$). The light blue bar indicates the significant post-hoc comparisons in the CTR condition. Yellow bars refer to the significant post-hoc comparisons in the FRG condition. The black bar represents the significant post-hoc comparison between the CTR and FRG conditions.

LMM analyses carried out for the $Latency_{SCR}$ variable showed a significant main effect of the factor *Session*, a significant *Session* × *Stimulus Type* interaction, and a significant *Condition* × *Session* × *Stimulus Type* interaction (Table 22).

Table 22. Results of the LMM analyses performed on the $Latency_{SCR}$ variable. Significant effects are reported in bold.

Factor	F	p
Condition	0.13	0.723
Session	**3.43**	**0.033**
Stimulus Type	0.03	0.871
Condition x Session	0.30	0.738
Condition x Stimulus Type	0.00	0.958
Session x Stimulus Type	**3.68**	**0.025**
Condition x Session x Stimulus Type	**7.28**	**0.001**

Planned comparisons for the higher-order *Condition* × *Session* × *Stimulus Type* interaction showed that participants during the FRG condition became faster in reacting to the Negative stimuli at T2 compared to T0 ($t = 3.00$, $p = 0.025$) (Figure 16 A) and slower to respond to neutral images at T1 ($t = -3.53$, $p = 0.004$) and T2 ($t = -2.87$, $p = 0.037$) compared to T0 (Figure 16 B). On the other hand, no difference in $Latency_{SCR}$ emerged over sessions in the CTR condition for both negative (all $p = 1.000$) and neutral stimuli (all $p \geq 0.447$). Finally, $Latency_{SCR}$ did not differ between conditions at each time point for both negative (all $p \geq 0.305$) and neutral stimuli (all $p \geq 0.730$).

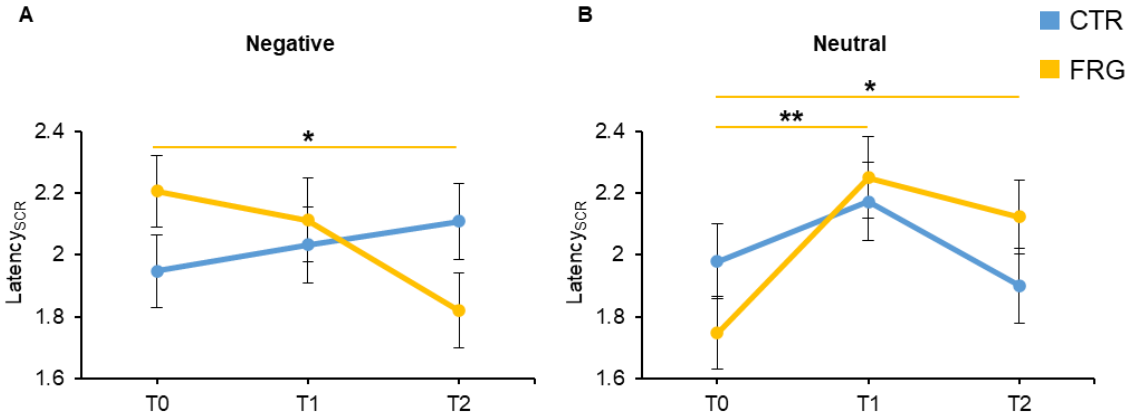

Figure 16. Mean and standard errors of $Latency_{SCR}$ values in CTR and FRG condition between sessions for negative (A) and neutral (B) stimuli. The asterisks depict significative Bonferroni planned comparisons (* $p < 0.05$; ** $p < 0.01$). Yellow bars refer to the significant post-hoc comparisons in the FRG condition.

3.7 Discussion

Intending to investigate the effects of REM sleep fragmentation on the declarative component of the emotional memory trace and on the psychophysiological reactivity associated with the emotional information previously encoded, we fragmented the REM sleep stage in healthy participants through vibrotactile stimulations. In accordance with Wassing et al. (2019), who assessed the effects of REM sleep fragmentation in insomniac individuals, we highlighted that experimentally induced REM sleep fragmentation through cortical arousals led to a lack of habituation in psychophysiological reactivity to emotional events in healthy participants. Thus, our results supported the hypothesised role of REM sleep in dampening the emotional reactivity associated with an emotional event (Walker & van der Helm, 2009). However, experimentally induced REM sleep fragmentation did not affect the subjective evaluation of emotional stimuli, similar to what is reported in REM sleep deprivation paradigms (Glosemeyer et al., 2020; Groch et al., 2013). Nevertheless, previous literature on this issue showed inconsistent results since other studies of REM sleep deprivation sustained that REM sleep led to a reduction (Lara-Carrasco et al., 2009; Rosales-Lagarde et al., 2012; Van Der Helm & Walker, 2011) or an increase (Baran et al., 2012; Pace-Schott et al., 2011; Wagner et al., 2002) in self-reported emotional reactivity. Finally, although the “Sleep to Forget, Sleep to Remember” hypothesis (Walker & van der Helm, 2009) is based on the presence/absence of REM sleep during the night, we revealed that REM sleep fragmentation did not compromise REM sleep effects on emotional memory. Thus, our results did not support the “Sleep to Remember” part of the “Sleep to Forget, Sleep to Remember” hypothesis

proposed by Walker & van der Helm (2009), at least in terms of REM sleep continuity. However, our result is in line with studies showing that emotional memories are consolidated regardless of REM sleep (Cellini et al., 2016; Lehmann et al., 2016) and with studies revealing that the brain activity during NREM sleep, which is fundamental for memory consolidation, is also essential for consolidating the declarative component of the emotional memories (Rodheim et al., 2023).

Therefore, our study highlighted that REM sleep fragmentation in healthy participants compromised the habituation processes in psychophysiological reactivity occurring during REM sleep without altering participants' evaluations of the emotional events and their consolidation.

As far as emotional memory is concerned, we reported that fragmentation of REM sleep following the encoding of emotional stimuli did not compromise the consolidation processes of the declarative component of the emotional information. However, it should be noted that REM sleep fragmentation did not eliminate REM sleep throughout the night. Indeed, despite REM sleep being continuously interrupted during the FRG night, the accumulation of short periods of undisturbed REM sleep led to about 12% of the total sleep time spent in this sleep stage. It has been reported in animal models that approximately 1 minute of REM sleep was sufficient to renormalise the neural firing rate in the neocortex after cortical activation (Almeida-Filho et al., 2018; Watson et al., 2016). Then, the accumulation of short periods of undisturbed REM sleep during the night may have been sufficient to ensure the consolidation of emotional information. Although literature reported that post-learning REM sleep fragmentation compromised emotional memory consolidation, this effect

has only been observed in clinical populations (Marshall et al., 2014; Saguin et al., 2021). For instance, Lipinska & Thomas (2019) reported memory performance deficits due to REM sleep fragmentation in individuals with PTSD but not in trauma-exposed non-PTSD participants who exhibited near identical levels of REM sleep fragmentation. Furthermore, as highlighted by Tempesta et al. (2015), inadequate sleep may not be sufficient to impair memory processing in healthy individuals. In their study, Tempesta et al. (2015) reported comparable memory performance between participants with poor and good sleep quality and observed memory performance deficits only in totally sleep deprived participants. Therefore, fragmented REM sleep may not be a sufficient condition to prevent memory consolidation processes occurring during REM sleep. This finding may seem at odds with the results of a recent meta-analysis that highlighted the role of REM sleep in emotional memory consolidation (Schäfer et al., 2020). However, this meta-analysis was based on a limited number of studies that directly compared NREM vs REM sleep (N=8), so the same authors claimed that the result should be investigated in greater detail (Schäfer et al., 2020).

In addition, it should be noted that other studies highlighted that sleep consolidated emotional memory regardless of REM sleep (Cellini et al., 2016; Lehmann et al., 2016; Morgenthaler et al., 2014). For instance, Cellini et al. (2016) reported no difference in memory performance between participants who experienced REM sleep during a nap compared to those who napped without experiencing REM sleep. In addition, using targeted memory reactivation, Lehmann et al. (2016) reported improved memory performance for emotional stimuli only when memory traces were reactivated during NREM

sleep. Recently, Rodheim et al. (2023) highlighted how the coupling of spindle activity and slow oscillations, considered crucial for the consolidation of non-emotional memories, may also be implicated in the consolidation of emotional information, reinforcing the idea of a significant role for NREM sleep even in emotional memory consolidation processes.

Finally, considering the lack of differences in memory performance for negative and neutral emotional stimuli and the similar memory performance between the CTR and FRG conditions, we cannot conclude if REM sleep fragmentation was unable to counteract the memory consolidation processes occurring during REM sleep or whether the declarative component of the emotional memory trace was processed during NREM sleep.

Regarding the effects on emotional reactivity, the experimentally induced REM sleep fragmentation led to a lack of habituation on the psychophysiological reactivity to emotional events. This effect was manifested by the maintenance of HRD and #SCR values over time to picture presentation at the baseline levels, which was associated with a reduced $Latency_{SCR}$ to negative pictures. Since this effect represents a faster activation of the sympathetic branch of the autonomous nervous system, it also indicates a greater emotional reactivity to negative stimuli after REM sleep fragmentation (Boucsein et al., 2012; Christopoulos et al., 2016). However, we reported that #SCR decreased when evaluated immediately after the FRG night, similar to what was observed in the CTR condition. This result may stem from an adaptive compensatory effect. Specifically, it has been reported that prefrontal cortical areas responsible for regulating amygdala activity (involved in the generation of SCR, Laine et al., 2009) maintain higher levels of activation, attempting to compensate for the

deleterious effects of REM sleep deprivation (Rosales-Lagarde et al., 2012). This compensatory effect could have conducted to a temporary psychophysiological reactivity habituation in the #SCR following the FRG night. Indeed, at T2, in the absence of compensatory strategies due to two nights of regular sleep, individuals reacted to stimuli as if they were entirely novel (#SCR value identical to baseline). Thus, the lack of psychophysiological habituation reported in the HRD index at T1 emerged only at the delayed assessment for the #SCR. It is interesting to note that, after REM sleep fragmentation, the reactivation of the memory traces by performing the same emotional reactivity task at T1 was not sufficient to allow the recovery of a proper emotional reactivity processing during the subsequent nights of regular sleep as indicated by the #SCR and HRD values, that were nearly identical to baseline at T2 during the FRG condition.

Physiological habituation to previously encountered stimuli demonstrated that they have been appropriately processed over time (Vila, 2004; Zimmer & Richter, 2023). Therefore, the lack of psychophysiological habituation to emotional pictures reported after REM sleep fragmentation interfered with the normal processing of emotional stimuli during REM sleep. Different studies reported that the lack of habituation for EDA and HR signals was present in participants with high levels of stress, anxiety, and depression, as well as in individuals with PTSD (Carson et al., 2007; Orr et al., 2012; Walker et al., 2019). Indeed, our results align with those on clinical populations in which dysfunctional emotional processing due to REM sleep fragmentation has been highlighted (Marshall et al., 2014; McCall & Watson, 2022; Saguin et al., 2021). For instance, Wassing et al. (2019) reported that REM sleep fragmentation was

associated with a lack of psychophysiological dampening of amygdala activity in response to known emotional events in insomnia participants. However, our study is the first to investigate experimentally induced REM sleep fragmentation in healthy participants and to show that a fragmented REM sleep can compromise the emotional processing occurring during REM sleep, leading to a lack of psychophysiological habituation to emotional stimuli. In this perspective, our results partially support the SFSR hypothesis (Walker & van der Helm, 2009). Specifically, they support only the assumption of the “Sleep to Forget” part of the hypothesis, which claims that the neurochemical milieu of the REM sleep stage, characterised by low levels of noradrenaline associated with high levels of acetylcholine, is essential to allow emotional memory traces to disengage from the associated emotional tone. Indeed, we showed that it is sufficient to induce cortical arousals during REM sleep, disrupting the continuity of this sleep stage, to interfere with the psychophysiological emotional reactivity dampening occurring overnight. This effect was possible since cortical arousals during REM sleep induced noradrenaline bursts from LC during REM sleep, preventing sleep-dependent emotional reactivity adaptation to emotional stimuli (Cabrera et al., 2024).

Therefore, our data underscore the importance of undisturbed REM sleep during the first night after experiencing emotional events to reduce the subsequent psychophysiological reaction associated with the event properly.

Regarding the effects on subjective emotional reactivity, REM sleep fragmentation did not alter participants' subjective evaluation of the emotional stimuli. However, in our study, even participants in the CTR condition did not show any reduction in self-reported arousal and valence ratings to emotional

stimuli. As reported in the first chapter, contrasting results exist on REM sleep effects on subjective emotional reactivity (see Tempesta et al., 2018 for a review). Indeed, several studies have reported psychophysiological habituation to emotional stimuli without finding differences in participants' subjective evaluations of the same events (Ashton et al., 2019; Cunningham et al., 2014; Franzen et al., 2009). For example, Franzen et al. (2009) found increased emotional reactivity to known emotional stimuli after total sleep deprivation, manifested as an anticipated and increased pupillary reactivity to negative stimuli, without observing variations in participants' arousal and valence ratings of the same stimuli. Similarly, Cunningham et al. (2014) reported a decrease in SCR and HRD value after a sleep condition relative to a similar period of diurnal wakefulness, highlighting a lack of psychophysiological habituation after wakefulness. Moreover, the authors (Cunningham et al., 2014) did not find significant differences between sleep and wakefulness conditions in self-reported valence and arousal ratings to these stimuli. Considering that, during an emotional experience, the brain structures involved in activating the body (providing a psychophysiological reaction) are different from those required to provide a cognitive response (Hariri et al., 2000, 2003; Lieberman et al., 2007; Malezieux et al., 2023), we can assume that REM sleep fragmentation was able to compromise the functioning of the subcortical structures that influence the psychophysiological reactions without altering the functionality of the neocortical structures involved in cognitively elaborated response (Wassing et al., 2019).

Furthermore, it is worth noting that the new sleep manipulation technology implemented in our study to induce REM sleep fragmentation allowed us to

minimise sleep macrostructure alterations. Specifically, the experimental induction of cortical arousals led to sleep fragmentation primarily through stage transition towards N1. Indeed, we observed a significant increase in N1 time during the FRG condition associated with a decreased REM sleep time. In addition, total sleep time, nocturnal awakenings, wake after sleep onset, and sleep efficiency were comparable in the CTR and FRG conditions. Moreover, our REM sleep parameters during the FRG night in healthy participants were similar to those reported in patients with fragmented REM sleep. Specifically, the mean REM sleep fragmentation index ranged from about 12 to 21 in studies on patients (Lipinska & Thomas, 2019; Maurer et al., 2024; Wassing et al., 2019), and we reported a mean REM sleep fragmentation index of 24. Similarly, REM% ranged from 11 to 19% in clinical populations (Feige et al., 2008; Lipinska & Thomas, 2019; Wu et al., 2021), while during the FRG night, we reported a REM% of 12%. Therefore, unlike REMD and split-night protocols, experimentally induced REM sleep fragmentation can hinder the continuity of REM sleep while minimising macrostructural sleep alterations, as highlighted by the lack of significant differences in the sleep macrostructure variables between the CTR and FRG nights.

Regarding the effects on procedural memory, the SFTT was primarily used as a control measure to evaluate the potential effects of the REM sleep fragmentation procedure on procedural memory. Our results revealed that participants performed better during the CTR condition than in the FRG condition and that performance improved between sessions in each condition for all the SFTT variables. However, we cannot assume that the lower

performance during the FRG condition derives from the experimental REM sleep fragmentation since the main effect of the *Condition* factor also encompasses the T0 assessment (antecedent to the FRG night), and the interaction *Condition* × *Session* was not significant. Therefore, considering the similar performance trend between sessions in both conditions, it is plausible that the main effect of the *Condition* factor is spurious. Thus, procedural memory was not affected by REM sleep fragmentation. As with emotional memory, it could be hypothesised that the accumulation of brief periods of undisturbed REM sleep during the night allowed for adequate processing of procedural information. Indeed, short periods of REM sleep could restore the typical neural discharge observed during the REM sleep stage in the neocortex after cortical activation (Almeida-Filho et al., 2018; Watson et al., 2016). However, unlike the hypothesised role of REM sleep in emotional memories, the role of REM sleep in procedural memories is more debated (Ackermann & Rasch, 2014). Indeed, despite many studies indicating that this information is primarily processed during REM sleep (Diekelmann et al., 2009; Diekelmann & Born, 2010), others argued that procedural memory processing occurs during NREM sleep (Ackermann & Rasch, 2014). For instance, nap studies revealed that NREM sleep was fundamental to enhancing procedural memory performance (Qian et al., 2022; Walker et al., 2002). Other authors highlighted that this effect was primarily attributable to N2 spindle activity (Nishida & Walker, 2007). Laventure et al. (2016) highlighted that targeted memory reactivation in N2 increased spindle activity, leading to better procedural performance the following day. Therefore, the lack of differences between the CTR and FRG

conditions in procedural memory performance could be attributable to the fact that this information is processed during N2.

Finally, the self-reported information about the characteristics of the laboratory nights highlighted that participants perceived some stimulations and referred that wearing the vibration bracelet was disturbing only during the FRG night. Nevertheless, it is interesting to note that participants perceived few stimulations (3.53 on average) relative to the delivered stimulations (71.73 on average).

In addition, the actigraphic data revealed that participants had identical sleep/wake rhythms between CTR and FRG conditions in the nights preceding the Laboratory sleep night. The actigraphic data also indicated that the day after the Laboratory sleep night, participants during the FRG condition woke up approximately one hour later compared to the CTR condition. Thus, participants exhibited an increased need for sleep following REM sleep fragmentation, as reported in different studies (Feige et al., 2023; Riemann et al., 2012). Moreover, we observed a difference in sleep onset time between CTR and FRG conditions the night preceding the delayed assessment (T2). Specifically, participants in the FRG condition went to bed approximately 30 minutes later. However, the two conditions had identical total sleep time, wake after sleep onset, and sleep efficiency. Therefore, it is unlikely that this difference could have influenced the results.

Altogether, these data suggest that participants showed an elevated adherence to their sleep schedule during both experimental conditions, reducing the likelihood that other confounding variables biased our results.

Nevertheless, some limitations of the study must be reported. For our study, a convenience sample of young university students was selected. Therefore, the generalizability of the result may be limited. Moreover, the memory recognition task, despite being essential for properly assessing the long-term effects of the experimental manipulation, may not have been sufficiently sensitive in detecting REM sleep fragmentation effects on emotional memory performance.

On the other hand, we strengthened our result by adopting a within-subject design and monitoring participants' sleep in a wide temporal window before and after the laboratory sleep nights.

3.8 Conclusion

Here, we showed that REM sleep fragmentation in healthy participants on the first night following exposure to emotional stimuli led to a lack of psychophysiological habituation to the emotional events without affecting memory consolidation and the subjective ratings of the emotional events. Furthermore, results suggest that if REM sleep is fragmented after an emotional stimuli exposure, 2 nights of undisturbed recovery sleep do not allow our brain to process this emotional information correctly. These results enrich the knowledge about the role of REM sleep in emotional processing, highlighting how microstructural alterations of REM sleep in the healthy population can lead to maladaptive emotional functioning. Specifically, we reproduced the REM sleep characteristics displayed by PTSD and insomnia patients (Lipinska & Thomas, 2019; Wassing et al., 2019), compromising the psychophysiological reactivity habituation in the healthy population. Our results could pave the way for other studies focused on increasing REM sleep continuity in insomnia and

PTSD patients to undermine the disruptive emotional experience associated with fragmented REM sleep in this population. Finally, REM sleep fragmentation by means of experimentally induced cortical arousals has proven to be a valid alternative to investigate the role of REM sleep without causing severe alterations of sleep macrostructure. Thus, this work lays the foundations for other studies aimed at deepening the functions of REM sleep by using vibrotactile stimulation as an alternative methodological approach to depict the stage-specific functions of sleep.

References

Aakre, J. A., Schulz, J., Ballard, C., Corbett, A., Bjorvatn, B., Aarsland, D., Creese, B., Hampshire, A., Brooker, H., & Testad, I. (2023). Self-reported sleep fragmentation and sleep duration and their association with cognitive function in PROTECT, a large digital community-based cohort of people over 50. *International Journal of Geriatric Psychiatry*, *38*(11). https://doi.org/10.1002/gps.6022

Acheson, A., Richards, J. B., & de Wit, H. (2007). Effects of sleep deprivation on impulsive behaviors in men and women. *Physiology and Behavior*, *91*(5), 579–587. https://doi.org/10.1016/j.physbeh.2007.03.020

Ackermann, S., & Rasch, B. (2014). Differential effects of non-REM and REM sleep on memory consolidation?. *Current neurology and neuroscience reports*, 14(2), 430. https://doi.org/10.1007/s11910-013-0430-8

Aldao, A., Jazaieri, H., Goldin, P. R., & Gross, J. J. (2014). Adaptive and maladaptive emotion regulation strategies: Interactive effects during CBT for social anxiety disorder. *Journal of Anxiety Disorders*, *28*(4), 382–389. https://doi.org/10.1016/j.janxdis.2014.03.005

Aldao, A., & Nolen-Hoeksema, S. (2012). The influence of context on the implementation of adaptive emotion regulation strategies. *Behaviour Research and Therapy*, *50*(7–8), 493–501. https://doi.org/10.1016/j.brat.2012.04.004

Alfano, C. A., Bower, J. L., Harvey, A. G., Beidel, D. C., Sharp, C., & Palmer, C. A. (2020). Sleep restriction alters children's positive emotional responses, but effects are moderated by anxiety. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *61*(10), 1150–1159. https://doi.org/10.1111/jcpp.13287

Alhola, P., & Polo-Kantola, P. (2022). *Sleep deprivation: Impact on cognitive performance*. https://doi.org/10.2147/ndt.s12160203

Almeida-Filho, D. G., Queiroz, C. M., & Ribeiro, S. (2018). Memory corticalization triggered by REM sleep: mechanisms of cellular and systems consolidation. *Cellular and molecular life sciences: CMLS*, 75(20), 3715–3740. https://doi.org/10.1007/s00018-018-2886-9

Altena, E., Micoulaud-Franchi, J. A., Geoffroy, P. A., Sanz-Arigita, E., Bioulac, S., & Philip, P. (2016). The bidirectional relation between emotional reactivity and sleep: From disruption to recovery. *Behavioral Neuroscience*, *130*(3), 336–350. https://doi.org/10.1037/bne0000128

Amicucci, G., Tempesta, D., Salfi, F., D'Atri, A., Viselli, L., De Gennaro, L., & Ferrara, M. (2021). The effect of 5 nights of sleep restriction on empathic propensity. *Journal of Sleep Research*, *30*(5), e13325. https://doi.org/https://doi.org/10.1111/jsr.13325

Arbinaga, F., Joaquin-Mingorance, M., & Fernández-Cuenca, S. (2019). Expression of anger in university students according to perceived quality of sleep. *Sleep Science*, *12*(1), 28–34. https://doi.org/10.5935/1984-0063.20190058

Ashton, J. E., Harrington, M. O., Guttesen, A. Á. V., Smith, A. K., & Cairney, S. A. (2019). Sleep Preserves Physiological Arousal in Emotional Memory. *Scientific Reports*, *9*(1). https://doi.org/10.1038/s41598-019-42478-2

Axelsson, J., Ingre, M., Kecklund, G., Lekander, M., Wright Jr, K. P., & Sundelin, T. (2020). Sleepiness as motivation: a potential mechanism for how sleep deprivation affects behavior. *Sleep*, *43*(6), zsz291. https://doi.org/10.1093/sleep/zsz291

Babson, K. A., Trainor, C. D., Feldner, M. T., & Blumenthal, H. (2010). A test of the effects of acute sleep deprivation on general and specific self-reported anxiety and depressive symptoms: An experimental extension. *Journal of Behavior Therapy and Experimental Psychiatry*, *41*(3), 297–303. https://doi.org/10.1016/j.jbtep.2010.02.008

Baglioni, C., Nanovska, S., Regen, W., Spiegelhalder, K., Feige, B., Nissen, C., Reynolds, C. F., & Riemann, D. (2016). Sleep and mental disorders: A meta-analysis of polysomnographic research. *Psychological Bulletin*, *142*(9), 969–990. https://doi.org/10.1037/bul0000053

Baran, B., Pace-Schott, E. F., Ericson, C., & Spencer, R. M. C. (2012). Processing of emotional reactivity and emotional memory over sleep. *Journal of Neuroscience*, *32*(3), 1035–1042. https://doi.org/10.1523/JNEUROSCI.2532-11.2012

Baranwal, N., Yu, P. K., & Siegel, N. S. (2023). Sleep physiology, pathophysiology, and sleep hygiene. *Progress in cardiovascular diseases*, 77, 59–69. https://doi.org/10.1016/j.pcad.2023.02.005

Bastien C. H. (2011). Insomnia: Neurophysiological and neuropsychological approaches. *Neuropsychology review*, 21(1), 22–40. https://doi.org/10.1007/s11065-011-9160-3

Bauducco, S. V, Flink, I. K., Jansson-Fröjmark, M., & Linton, S. J. (2016). Sleep duration and patterns in adolescents: correlates and the role of daily stressors. *Sleep Health: Journal of the National Sleep Foundation*, *2*(3), 211–218. https://doi.org/10.1016/j.sleh.2016.05.006

Baum, K. T., Desai, A., Field, J., Miller, L. E., Rausch, J., & Beebe, D. W. (2014). Sleep restriction worsens mood and emotion regulation in adolescents. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *55*(2), 180–190. https://doi.org/10.1111/jcpp.12125

Beattie, L., Kyle, S. D., Espie, C. A., & Biello, S. M. (2015). Social interactions, emotion and sleep: A systematic review and research agenda. Sleep medicine reviews, 24, 83–100. https://doi.org/10.1016/j.smrv.2014.12.005

Ben Simon, E., Vallat, R., Barnes, C. M., & Walker, M. P. (2020). Sleep Loss and the Socio-Emotional Brain. Trends in cognitive sciences, 24(6), 435–450. https://doi.org/10.1016/j.tics.2020.02.003

Benedek, M., & Kaernbach, C. (2010). A continuous measure of phasic electrodermal activity. *Journal of Neuroscience Methods*, *190*(1), 80–91. https://doi.org/10.1016/J.JNEUMETH.2010.04.028

Benjamins, J. S., Migliorati, F., Dekker, K., Wassing, R., Moens, S., Blanken, T. F., Te Lindert, B. H. W., Sjauw Mook, J., & Van Someren, E. J. W. (2017). Insomnia heterogeneity: Characteristics to consider for data-driven multivariate subtyping. Sleep medicine reviews, 36, 71–81. https://doi.org/10.1016/j.smrv.2016.10.005

Benkirane, O., Delwiche, B., Mairesse, O., & Peigneux, P. (2022). Impact of Sleep Fragmentation on Cognition and Fatigue. *International Journal of Environmental Research and Public Health*, *19*(23). https://doi.org/10.3390/ijerph192315485

Berry, R. B., Brooks, R., Gamaldo, C. E., Harding, S. M., Lloyd, R. M., Marcus, C. L., & Vaughn, B. V. (2015). *AASM | Scoring Manual Version 2.2 The AASM Manual for the Scoring of Sleep and Associated Events RULES, TERMINOLOGY AND TECHNICAL SPECIFICATIONS VERSION 2.2*. www.aasmnet.org

Berry, R. B., Brooks, R., Gamaldo, C. E., Harding, S. M., Lloyd, R. M., Quan, S. F., Troester, M. M., & Vaughn, B. V. (2017). *AASM | Scoring Manual Version 2.4 The AASM Manual for the Scoring of Sleep and Associated Events RULES, TERMINOLOGY AND TECHNICAL SPECIFICATIONS VERSION 2.4*. www.aasmnet.org

Beutler, L. E., Cano, M. C., Miró, E., & Buela-Casal, G. (2003). The role of activation in the effect of total sleep deprivation on depressed mood. *Journal of Clinical Psychology*, *59*(3), 369–384. https://doi.org/10.1002/jclp.10126

Bhagavan, S., & Sahota, P. (2021). Sleep Fragmentation and Atherosclerosis: is There a Relationship?. *Missouri Medicine*, *118*, 272–276.

Bonnet, M. H., & Arand, D. L. (2003). Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep medicine reviews*, 7(4), 297–310. https://doi.org/10.1053/smrv.2001.0245

Boon, M. E., van Hooff, M. L. M., Vink, J. M., & Geurts, S. A. E. (2023). The effect of fragmented sleep on emotion regulation ability and usage. *Cognition and Emotion*, *37*(6), 1132–1143. https://doi.org/10.1080/02699931.2023.2224957

Booth, S. A., Carskadon, M. A., Young, R., & Short, M. A. (2021). Sleep duration and mood in adolescents: an experimental study. *Sleep*, *44*(5), zsaa253. https://doi.org/10.1093/sleep/zsaa253

Bottesi, G., Ghisi, M., Altoè, G., Conforti, E., Melli, G., & Sica, C. (2015). The Italian version of the Depression Anxiety Stress Scales-21: Factor structure and psychometric properties on community and clinical samples. *Comprehensive Psychiatry*, *60*, 170–181. https://doi.org/10.1016/j.comppsych.2015.04.005

Boucsein, W., Fowles, D. C., Grimnes, S., Ben-Shakhar, G., Roth, W. T., Dawson, M. E., & Filion, D. L. (2012). Publication recommendations for electrodermal measurements. *Psychophysiology*, *49*(8), 1017–1034. https://doi.org/10.1111/j.1469-8986.2012.01384.x

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: the self-assessment manikin and the semantic differential. *Journal of Behavior Therapy and Experimental Psychiatry*, *25*(1), 49–59.

Braithwaite, J. J., Watson, D. P. Z., Jones, R. O., & Rowe, M. A. (2013). Guide for Analysing Electrodermal Activity & Skin Conductance Responses for Psychological Experiments. *CTIT Technical Reports Series*. https://api.semanticscholar.org/CorpusID:112717780

Buchsbaum, M. S., Hazlett, E. A., Wu, J., & Bunney, W. E., Jr (2001). Positron emission tomography with deoxyglucose-F18 imaging of sleep. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 25(5 Suppl), S50–S56. https://doi.org/10.1016/S0893-133X(01)00339-6

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4

Cabrera, Y., Koymans, K. J., Poe, G. R., Kessels, H. W., Van Someren, E. J. W., & Wassing, R. (2024). Overnight neuronal plasticity and adaptation to emotional distress. *Nature Reviews Neuroscience*, *25*(4), 253–271. https://doi.org/10.1038/s41583-024-00799-w

Campbell, R. L., Feldner, M. T., & Leen-Feldner, E. W. (2022). An experimental test of the effects of acute sleep deprivation on affect and avoidance. *Journal of Behavior Therapy and Experimental Psychiatry*, *77*. https://doi.org/10.1016/j.jbtep.2022.101770

Carollo, G., Degasperi, G., & Cellini, N. (2022). The role of sleep and wakefulness in the recognition of emotional pictures. *Journal of Sleep Research*, *31*(6), e13695. https://doi.org/https://doi.org/10.1111/jsr.13695

Carson, M. A., Metzger, L. J., Lasko, N. B., Paulus, L. A., Morse, A. E., Pitman, R. K., & Orr, S. P. (2007). Physiologic reactivity to startling tones in female vietnam nurse veterans with PTSD. *Journal of Traumatic Stress*, *20*(5), 657–666. https://doi.org/https://doi.org/10.1002/jts.20218

Castronovo, V., Galbiati, A., Marelli, S., Brombin, C., Cugnata, F., Giarolli, L., Anelli, M. M., Rinaldi, F., & Ferini-Strambi, L. (2016). Validation study of the Italian version of the Insomnia Severity Index (ISI). *Neurological Sciences*, *37*(9), 1517–1524. https://doi.org/10.1007/s10072-016-2620-z

Cellini, N., Torre, J., Stegagno, L., & Sarlo, M. (2016). Sleep before and after learning promotes the consolidation of both neutral and emotional information regardless of REM presence. *Neurobiology of Learning and Memory*, *133*, 136–144. https://doi.org/10.1016/J.NLM.2016.06.015

Chambers, A. M., & Payne, J. D. (2014). Laugh yourself to sleep: Memory consolidation for humorous information. *Experimental Brain Research*, *232*(5), 1415–1427. https://doi.org/10.1007/s00221-013-3779-7

Christian, M. S., & Ellis, A. P. J. (2011). Examining the effects of sleep deprivation on workplace deviance: A self-regulatory perspective. *Academy of Management Journal*, *54*(5), 913–934. https://doi.org/10.5465/amj.2010.0179

Christopoulos, G. I., Uy, M. A., & Yap, W. J. (2016). The Body and the Brain: Measuring Skin Conductance Responses to Understand the Emotional Experience. *Organizational Research Methods*, *22*(1), 394–420. https://doi.org/10.1177/1094428116681073

Cirelli, C., & Tononi, G. (2022). The why and how of sleep-dependent synaptic down-selection. *Seminars in Cell and Developmental Biology*, *125*, 91–100. https://doi.org/10.1016/j.semcdb.2021.02.007

Colvonen, P. J., Straus, L. D., Acheson, D., & Gehrman, P. (2019). A Review of the Relationship Between Emotional Learning and Memory, Sleep, and PTSD. *Current Psychiatry Reports*, *21*(1), 2. https://doi.org/10.1007/s11920-019-0987-2

Conklin, L. R., Cassiello-Robbins, C., Brake, C. A., Sauer-Zavala, S., Farchione, T. J., Ciraulo, D. A., & Barlow, D. H. (2015). Relationships among adaptive and maladaptive emotion regulation strategies and psychopathology during the treatment of comorbid anxiety and alcohol use disorders. *Behaviour Research and Therapy*, *73*, 124–130. https://doi.org/10.1016/j.brat.2015.08.001

Cook, E., Turpin, G., Lang, P. J., Simons, R. F., & Balaban, M. (1997). *Attention and orienting: Sensory and motivational processes*. Psychology Press, New York.

Cote, K. A., Milner, C. E., Osip, S. L., Baker, M. L., & Cuthbert, B. P. (2008). Physiological arousal and attention during a week of continuous sleep restriction. *Physiology and Behavior*, *95*(3), 353–364. https://doi.org/10.1016/j.physbeh.2008.06.016

Cote, K. A., Milner, C. E., Smith, B. A., Aubin, A. J., Greason, T. A., Cuthbert, B. P., Wiebe, S., & Duffus, S. E. G. (2009). CNS arousal and neurobehavioral performance in a short-term sleep restriction paradigm. *Journal of Sleep Research*, *18*(3), 291–303. https://doi.org/10.1111/j.1365-2869.2008.00733.x

Cote, K. A., Mondloch, C. J., Sergeeva, V., Taylor, M., & Semplonius, T. (2014). Impact of total sleep deprivation on behavioural neural processing of emotionally expressive faces. *Experimental Brain Research*, *232*(5), 1429–1442. https://doi.org/10.1007/s00221-013-3780-1

Cousins, J. N., & Fernández, G. (2019). The impact of sleep deprivation on declarative memory. *Progress in brain research*, 246, 27–53. https://doi.org/10.1016/bs.pbr.2019.01.007

Couyoumdjian, A., Sdoia, S., Tempesta, D., Curcio, G., Rastellini, E., De Gennaro, L., & Ferrara, M. (2010). The effects of sleep and sleep deprivation on task-switching performance. *Journal of Sleep Research*, *19*(1 PART. 1), 64–70. https://doi.org/10.1111/j.1365-2869.2009.00774.x

Cox, R., van Bronkhorst, M. L. V., Bayda, M., Gomillion, H., Cho, E., Parr, M. E., Manickas-Hill, O. P., Schapiro, A. C., & Stickgold, R. (2018). Sleep selectively stabilizes contextual aspects of negative memories. *Scientific Reports*, *8*(1). https://doi.org/10.1038/s41598-018-35999-9

Coxe, S., West, S. G., & Aiken, L. S. (2009). The Analysis of Count Data: A Gentle Introduction to Poisson Regression and Its Alternatives. *Journal of Personality Assessment*, *91*(2), 121–136. https://doi.org/10.1080/00223890802634175

Craven, J., McCartney, D., Desbrow, B., Sabapathy, S., Bellinger, P., Roberts, L., & Irwin, C. (2022). Effects of Acute Sleep Loss on Physical Performance: A Systematic and Meta-Analytical Review. *Sports Medicine*, *52*(11), 2669–2690. https://doi.org/10.1007/s40279-022-01706-y

Crönlein, T., Langguth, B., Eichhammer, P., & Busch, V. (2016). Impaired Recognition of Facially Expressed Emotions in Different Groups of Patients with Sleep Disorders. *PLOS ONE*, *11*(4), e0152754-. https://doi.org/10.1371/journal.pone.0152754

Cunningham, T. J., Crowell, C. R., Alger, S. E., Kensinger, E. A., Villano, M. A., Mattingly, S. M., & Payne, J. D. (2014). Psychophysiological arousal at encoding leads to reduced reactivity but enhanced emotional memory following sleep. *Neurobiology of Learning and Memory*, *114*, 155–164. https://doi.org/10.1016/j.nlm.2014.06.002

Cunningham, T. J., Stickgold, R., & Kensinger, E. A. (2022). Investigating the effects of sleep and sleep loss on the different stages of episodic emotional memory: A narrative review and guide to the future. *Frontiers in behavioral neuroscience*, 16, 910317. https://doi.org/10.3389/fnbeh.2022.910317

Curcio, G., Tempesta, D., Scarlata, S., Marzano, C., Moroni, F., Rossini, P. M., Ferrara, M., & De Gennaro, L. (2013). Validity of the Italian Version of the Pittsburgh Sleep Quality Index (PSQI). *Neurological Sciences*, *34*(4), 511–519. https://doi.org/10.1007/s10072-012-1085-y

Dagys, N., McGlinchey, E. L., Talbot, L. S., Kaplan, K. A., Dahl, R. E., & Harvey, A. G. (2012). Double trouble? The effects of sleep deprivation and chronotype on adolescent affect. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *53*(6), 660–667. https://doi.org/10.1111/j.1469-7610.2011.02502.x

Dang-Vu, T. T., Schabus, M., Desseilles, M., Sterpenich, V., Bonjean, M., & Maquet, P. (2010). Functional Neuroimaging Insights into the Physiology of Human Sleep. *Sleep*, *33*(12), 1589–1603. https://doi.org/10.1093/sleep/33.12.1589

Davidson, P., Jönsson, P., Carlsson, I., & Pace-Schott, E. (2021). Does Sleep Selectively Strengthen Certain Memories Over Others Based on Emotion and Perceived Future Relevance?. *Nature and science of sleep*, 13, 1257–1306. https://doi.org/10.2147/NSS.S286701

Dawson, M., Schell, A., & Filion, D. (2000). The electrodermal system. In *Handbook of Psychophysiology*. https://doi.org/10.1017/CBO9780511546396.007

de Almondes, K. M., Júnior, F. W. N. H., & Alves, N. T. (2016). Sleep deprivation and implications for recognition and perception of facial emotions. *Sleep and Biological Rhythms*, *14*(1), 13–22. https://doi.org/10.1007/s41105-015-0029-3

Demichelis, O. P., Grainger, S. A., Burr, L., & Henry, J. D. (2023). Emotion regulation mediates the effects of sleep on stress and aggression. *Journal of Sleep Research*, *32*(3). https://doi.org/10.1111/jsr.13787

Desjardins, S., Lapierre, S., Hudon, C., & Desgagné, A. (2019). Factors involved in sleep efficiency: a population-based study of community-dwelling elderly persons. *Sleep*, *42*(5), zsz038. https://doi.org/10.1093/sleep/zsz038

Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature reviews. Neuroscience*, 11(2), 114–126. https://doi.org/10.1038/nrn2762

Diekelmann, S., Wilhelm, I., & Born, J. (2009). The whats and whens of sleep-dependent memory consolidation. *Sleep medicine reviews*, 13(5), 309–321. https://doi.org/10.1016/j.smrv.2008.08.002

Ekman, P. (1970). Universal facial expressions of emotion. *California Mental Health Research Digest*, *8*(4), 151–158.

Engen, H. G., & Anderson, M. C. (2018). Memory Control: A Fundamental Mechanism of Emotion Regulation. *Trends in Cognitive Sciences*, *22*(11), 982–995. https://doi.org/10.1016/J.TICS.2018.07.015

Faul, L., & LaBar, K. S. (2020). Emotional Memory in the Human Brain. In *The Oxford Handbook of the Neurobiology of Learning and Memory*. Oxford University Press. https://doi.org/10.1093/oxfordhb/9780190069162.013.2

Feige, B., Al-Shajlawi, A., Nissen, C., Voderholzer, U., Hornyak, M., Spiegelhalder, K., Kloepfer, C., Perlis, M., & Riemann, D. (2008). Does REM sleep contribute to subjective wake time in primary insomnia? A comparison of polysomnographic and subjective sleep in 100 patients. *Journal of Sleep Research*, *17*(2), 180–190. https://doi.org/10.1111/j.1365-2869.2008.00651.x

Feige, B., Benz, F., Dressle, R. J., & Riemann, D. (2023). Insomnia and REM sleep instability. *Journal of sleep research*, 32(6), e14032. https://doi.org/10.1111/jsr.14032

Fernandes-Santos, L., Patti, C. L., Zanin, K. A., Fernandes, H. A., Tufik, S., Andersen, M. L., & Frussa-Filho, R. (2012). Sleep deprivation impairs emotional memory retrieval in mice: Influence of sex. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *38*(2), 216–222. https://doi.org/10.1016/J.PNPBP.2012.03.014

Ferrer, R. A., & Mendes, W. B. (2018). Emotion, health decision making, and health behaviour. *Psychology & Health*, *33*(1), 1–16. https://doi.org/10.1080/08870446.2017.1385787

Finan, P. H., Quartana, P. J., & Smith, M. T. (2015). The Effects of Sleep Continuity Disruption on Positive Mood and Sleep Architecture in Healthy Adults. *Sleep*, *38*(11), 1735–1742. https://doi.org/10.5665/sleep.5154

Fischer, S., Hallschmid, M., Elsner, A. L., & Born, J. (2002). Sleep forms memory for finger skills. *Proceedings of the National Academy of Sciences*, *99*(18), 11987–11991. https://doi.org/10.1073/pnas.182178199

Floros, O., Axelsson, J., Almeida, R., Tigerström, L., Lekander, M., Sundelin, T., & Petrovic, P. (2021). Vulnerability in Executive Functions to Sleep Deprivation Is Predicted by Subclinical Attention-Deficit/Hyperactivity Disorder Symptoms. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *6*(3), 290–298. https://doi.org/10.1016/J.BPSC.2020.09.019

Franzen, P. L., Buysse, D. J., Dahl, R. E., Thompson, W., & Siegle, G. J. (2009). Sleep deprivation alters pupillary reactivity to emotional stimuli in healthy young adults. *Biological Psychology*, *80*(3), 300–305. https://doi.org/10.1016/j.biopsycho.2008.10.010

Fuligni, A. J., & Hardway, C. (2006). Daily variation in adolescents' sleep, activities, and psychological well-being. *Journal of Research on Adolescence*, *16*(3), 353–378. https://doi.org/10.1111/j.1532-7795.2006.00498.x

Gardner, W., Mulvey, E. P., & Shaw, E. C. (1995). Regression analyses of counts and rates: Poisson, overdispersed Poisson, and negative binomial models. *Psychological Bulletin*, *118*(3), 392–404. https://doi.org/10.1037/0033-2909.118.3.392

Garnow, T., Einberg, E. L., Edberg, A. K., & Garmy, P. (2021). Sadness and other health complaints among Swedish adolescents: A cross-sectional study. *International Journal of Environmental Research and Public Health*, *18*(8). https://doi.org/10.3390/ijerph18083999

Gatti, E., Calzolari, E., Maggioni, E., & Obrist, M. (2018). Data Descriptor: Emotional ratings and skin conductance response to visual, auditory and haptic stimuli. *Scientific Data*, *5*. https://doi.org/10.1038/sdata.2018.120

Giuditta, A. (2014). Sleep memory processing: the sequential hypothesis. *Frontiers in Systems Neuroscience*, *8*. https://www.frontiersin.org/articles/10.3389/fnsys.2014.00219

Giuditta, A., Ambrosini, M. V., Montagnese, P., Mandile, P., Cotugno, M., Zucconi, G. G., & Vescia, S. (1995). The sequential hypothesis of the function of sleep. *Behavioural Brain Research*, *69*(1–2), 157–166. https://doi.org/10.1016/0166-4328(95)00012-l

Glosemeyer, R. W., Diekelmann, S., Cassel, W., Kesper, K., Koehler, U., Westermann, S., Steffen, A., Borgwardt, S., Wilhelm, I., Müller-Pinzler, L., Paulus, F. M., Krach, S., & Stolz, D. S. (2020). Selective suppression of rapid eye movement sleep increases next-day negative affect and amygdala responses to social exclusion. *Scientific Reports*, *10*(1). https://doi.org/10.1038/s41598-020-74169-8

Gordon, A. M., Mendes, W. B., & Prather, A. A. (2017). The Social Side of Sleep: Elucidating the Links Between Sleep and Social Processes. *Current Directions in Psychological Science*, *26*(5), 470–475. https://doi.org/10.1177/0963721417712269

Gott, J., Rak, M., Bovy, L., Peters, E., van Hooijdonk, C. F. M., Mangiaruga, A., Varatheeswaran, R., Chaabou, M., Gorman, L., Wilson, S., Weber, F., Talamini, L., Steiger, A., & Dresler, M. (2020). Sleep fragmentation and lucid dreaming. *Consciousness and Cognition*, *84*. https://doi.org/10.1016/j.concog.2020.102988

Grafe, L., Miller, K. E., Ross, R. J., & Bhatnagar, S. (2024). The importance of REM sleep fragmentation in the effects of stress on sleep: Perspectives from preclinical studies. *Neurobiology of Stress*, *28*. https://doi.org/10.1016/j.ynstr.2023.100588

Gray, E. K., Watson, D., Payne, R., & Cooper, C. (2001). Emotion, mood, and temperament: Similarities, differences, and a synthesis. *Emotions at Work: Theory, Research and Applications for Management*, 21–43.

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. *Neurobiology of Learning and Memory*, *99*, 1–9. https://doi.org/10.1016/j.nlm.2012.10.006

Groch, S., Zinke, K., Wilhelm, I., & Born, J. (2015). Dissociating the contributions of slow-wave sleep and rapid eye movement sleep to emotional item and source memory. *Neurobiology of Learning and Memory*, *122*, 122–130. https://doi.org/10.1016/j.nlm.2014.08.013

Gross J. J. (2002). Emotion regulation: affective, cognitive, and social consequences. *Psychophysiology*, 39(3), 281–291. https://doi.org/10.1017/s0048577201393198

Gross, J. J. (1998). The Emerging Field of Emotion Regulation: An Integrative Review. *Review of General Psychology*, 2(3), 271-299. https://doi.org/10.1037/1089-2680.2.3.271

Guadagni, V., Burles, F., Ferrara, M., & Iaria, G. (2014). The effects of sleep deprivation on emotional empathy. *Journal of Sleep Research*, *23*(6), 657–663. https://doi.org/10.1111/jsr.12192

Hariri, A. R., Bookheimer, S. Y., & Mazziotta, J. C. (2000). Modulating emotional responses: effects of a neocortical network on the limbic system. *Neuroreport*, 11(1), 43–48. https://doi.org/10.1097/00001756-200001170-00009

Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry*, *53*(6), 494–501. https://doi.org/10.1016/S0006-3223(02)01786-9

Harrington, M. O., Nedberge, K. M., & Durrant, S. J. (2018). The effect of sleep deprivation on emotional memory consolidation in participants reporting depressive symptoms. *Neurobiology of Learning and Memory*, *152*, 10–19. https://doi.org/10.1016/j.nlm.2018.04.013

Harrison, Y., & Home, J. A. (2000). The Impact of Sleep Deprivation on Decision Making: A Review. *Journal of Experimental Psychology: Applied*, *6*(3), 236–249. https://doi.org/10.I037//1076-8O8X.6.3.236

Harvey, A. G. (2001). I can't sleep, my mind is racing! An investigation of strategies of thoυght control in insomnia. *Behavioural and Cognitive Psychotherapy*, *29*(1), 3–11. https://doi.org/10.1017/S1352465801001023

Herrero, H., Tarrada, A., Haffen, E., Mignot, T., Sense, C., Schwan, R., EL-Hage, W., Maillard, L., & hingray, C. (2020). Skin conductance response and emotional response in women with psychogenic non-epileptic seizures. *Seizure*, *81*, 123–131. https://doi.org/10.1016/j.seizure.2020.07.028

Holding, B. C., Laukka, P., Fischer, H., Bänziger, T., Axelsson, J., & Sundelin, T. (2017). Multimodal Emotion Recognition Is Resilient to Insufficient Sleep: Results From Cross-Sectional and Experimental Studies. *Sleep*, *40*(11), zsx145. https://doi.org/10.1093/sleep/zsx145

Horndasch, S., Sharon, E., Eichler, A., Graap, H., Moll, G. H., & Kratz, O. (2023). Heart Rate as a Correlate for the Emotional Processing of Body Stimuli in Anorexia Nervosa. *Behavioral Sciences*, *13*(3). https://doi.org/10.3390/bs13030215

Hruska, B., Anderson, L., & Barduhn, M. S. (2022). Multilevel analysis of sleep quality and anger in emergency medical service workers. *Sleep Health*, *8*(3), 303–310. https://doi.org/10.1016/j.sleh.2022.02.005

Hu, P., Stylos-Allan, M., & Walker, M. P. (2006). *Sleep Facilitates Consolidation of Emotional Declarative Memory*.

Huck, N. O., Mcbride, S. A., Kendall, A. P., Grugle, N. L., & Killgore, W. D. S. (2008). The effects of modafinil, caffeine, and dextroamphetamine on judgments of simple versus complex emotional expressions following sleep deprivation. *International Journal of Neuroscience*, *118*(4), 487–502. https://doi.org/10.1080/00207450601125907

Hudson, A. N., Van Dongen, H. P. A., & Honn, K. A. (2020). Sleep deprivation, vigilant attention, and brain function: a review. In *Neuropsychopharmacology* (Vol. 45, Issue 1, pp. 21–30). Springer Nature. https://doi.org/10.1038/s41386-019-0432-6

Iacovides, S., George, K., Kamerman, P., & Baker, F. C. (2017). Sleep Fragmentation Hypersensitizes Healthy Young Women to Deep and Superficial Experimental Pain. *Journal of Pain*, *18*(7), 844–854. https://doi.org/10.1016/j.jpain.2017.02.436

Insana, S. P., Kolko, D. J., & Germain, A. (2012). Early-life trauma is associated with rapid eye movement sleep fragmentation among military veterans. *Biological Psychology*, *89*(3), 570–579. https://doi.org/10.1016/j.biopsycho.2012.01.001

Jackowska, M., Dockray, S., Hendrickx, H., & Steptoe, A. (2011). Psychosocial factors and sleep efficiency: Discrepancies between subjective and objective evaluations of sleep. *Psychosomatic Medicine*, *73*(9), 810–816. https://doi.org/10.1097/PSY.0b013e3182359e77

Jenkins, J.G., & Dallenbach, K.M. (1924). Obliviscence During Sleep and Waking. *American Journal of Psychology*, 35, 605.

Joiner W. J. (2019). Neuroscience: Sleep Fragmentation Impairs Memory Formation. *Current biology: CB*, 29(22), R1181–R1184. https://doi.org/10.1016/j.cub.2019.09.060

Jones, B. J., Schultz, K. S., Adams, S., Baran, B., & Spencer, R. M. C. (2016). Emotional bias of sleep-dependent processing shifts from negative to positive with aging. *Neurobiology of Aging*, *45*, 178–189. https://doi.org/10.1016/j.neurobiolaging.2016.05.019

Kahn, M., Fridenson, S., Lerer, R., Bar-Haim, Y., & Sadeh, A. (2014). Effects of one night of induced night-wakings versus sleep restriction on sustained attention and mood: A pilot study. *Sleep Medicine*, *15*(7), 825–832. https://doi.org/10.1016/j.sleep.2014.03.016

Kaida, K., & Niki, K. (2013). Total sleep deprivation decreases flow experience and mood status. *Neuropsychiatric Disease and Treatment*, *10*, 19–25. https://doi.org/10.2147/NDT.S53633

Kaida, K., Niki, K., & Born, J. (2015). Role of sleep for encoding of emotional memory. *Neurobiology of Learning and Memory*, *121*, 72–79. https://doi.org/10.1016/j.nlm.2015.04.002

Kalmbach, D. A., Pillai, V., Roth, T., & Drake, C. L. (2014). The interplay between daily affect and sleep: A 2-week study of young women. *Journal of Sleep Research*, *23*(6), 636–645. https://doi.org/10.1111/jsr.12190

Kato, T., Montplaisir, J. Y., Guitard, F., Sessle, B. J., Lund, J. P., & Lavigne, G. J. (2003). Evidence that Experimentally Induced Sleep Bruxism is a Consequence of Transient Arousal. *Journal of Dental Research*, *82*(4), 284–288. https://doi.org/10.1177/154405910308200408

Kato, T., Montplaisir, J. Y., & Lavigne, G. J. (2004). Experimentally induced arousals during sleep: A cross-modality matching paradigm. *Journal of Sleep Research*, *13*(3), 229–238. https://doi.org/10.1111/j.1365-2869.2004.00409.x

Kaufmann, T., Sütterlin, S., Schulz, S. M., & Vögele, C. (2011). ARTiiFACT: a tool for heart rate artifact processing and heart rate variability analysis. *Behavior Research Methods*, *43*(4), 1161–1170. https://doi.org/10.3758/s13428-011-0107-7

Killgore, W. D. S., Balkin, T. J., Yarnell, A. M., & Capaldi, V. F. (2017). Sleep deprivation impairs recognition of specific emotions. *Neurobiology of Sleep and Circadian Rhythms*, *3*, 10–16. https://doi.org/10.1016/j.nbscr.2017.01.001

Klinzing, J. G., Niethard, N., & Born, J. (2019). Mechanisms of systems memory consolidation during sleep. *Nature neuroscience*, 22(10), 1598–1610. https://doi.org/10.1038/s41593-019-0467-3

Kopelman, M. D., Bright, P., Buckman, J., Fradera, A., Yoshimasu, H., Jacobson, C., & Colchester, A. C. F. (2007). Recall and recognition memory in amnesia: Patients with hippocampal, medial temporal, temporal lobe or frontal pathology. *Neuropsychologia*, *45*(6), 1232–1246. https://doi.org/10.1016/J.NEUROPSYCHOLOGIA.2006.10.005

Kryger, M. H., Roth, T., & Dement, W. C. (2010). *Principles and Practice of Sleep Medicine - E-Book: Expert Consult - Online and Print*. Elsevier Health Sciences. https://books.google.it/books?id=3B52V4PnrVkC

Kuna, S. T., Maislin, G., Pack, F. M., Staley, B., Hachadoorian, R., Coccaro, E. F., & Pack, A. I. (2012). Heritability of Performance Deficit Accumulation During Acute Sleep Deprivation in Twins. *Sleep*, *35*(9), 1223–1233. https://doi.org/10.5665/sleep.2074

LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature reviews. Neuroscience*, 7(1), 54–64. https://doi.org/10.1038/nrn1825

Laine, C. M., Spitler, K. M., Mosher, C. P., & Gothard, K. M. (2009). Behavioral Triggers of Skin Conductance Responses and Their Neural Correlates in the Primate Amygdala. *Journal of Neurophysiology*, *101*(4), 1749–1754. https://doi.org/10.1152/jn.91110.2008

Lang, P. (1980). Behavioral treatment and bio-behavioral assessment: Computer applications. *Technology in Mental Health Care Delivery Systems*, 119–137.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2005). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual*. NIMH, Center for the Study of Emotion & Attention Gainesville, FL.

Lara-Carrasco, J., Nielsen, T. A., 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. *Journal of Sleep Research*, *18*(2), 178–187. https://doi.org/10.1111/j.1365-2869.2008.00709.x

Laventure, S., Fogel, S., Lungu, O., Albouy, G., Sévigny-Dupont, P., Vien, C., Sayour, C., Carrier, J., Benali, H., & Doyon, J. (2016). NREM2 and Sleep Spindles Are Instrumental to the Consolidation of Motor Sequence Memories. *PLoS Biology*, *14*(3). https://doi.org/10.1371/journal.pbio.1002429

Lehmann, M., Seifritz, E., & Rasch, B. (2016). Sleep benefits emotional and neutral associative memories equally. *Somnologie*, *20*(1), 47–53. https://doi.org/10.1007/s11818-015-0034-4

Lesku, J & Martinez, Dolores & Rattenborg, Niels. (2009). Phylogeny and Ontogeny of Sleep. https://doi.org/10.1016/B978-0-12-375073-0.50013-0

Li, Z. Q., Qin, Y., Cai, W. P., Deng, S. Q., Mao, X. F., Zhang, J. G., Hou, T. Y., Pan, Y., Cui, Y., Ge, Y. N., Dong, W., & Tang, Y. X. (2023). Sleep Deprivation Impairs Human Cognitive Reappraisal Ability: A Randomized Controlled Trial. *Nature and Science of Sleep*, *15*, 729–736. https://doi.org/10.2147/NSS.S414962

Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., & Way, B. M. (2007). Putting Feelings Into Words. *Psychological Science*, *18*(5), 421–428. https://doi.org/10.1111/j.1467-9280.2007.01916.x

Lim, A. S. P., Kowgier, M., Yu, L., Buchman, A. S., & Bennett, D. A. (2013). Sleep fragmentation and the risk of incident Alzheimer's disease and cognitive decline in older persons. *Sleep*, *36*(7), 1027–1032. https://doi.org/10.5665/sleep.2802

Lipinska, G., Austin, H., Moonsamy, J. R., Henry, M., Lewis, R., Baldwin, D. S., Thomas, K. G. F., & Stuart, B. (2022). Preferential consolidation of emotional reactivity during sleep: A systematic review and meta-analysis. *Frontiers in Behavioral Neuroscience*, *16*. https://www.frontiersin.org/articles/10.3389/fnbeh.2022.976047

Lipinska, G., Stuart, B., Thomas, K. G. F., Baldwin, D. S., & Bolinger, E. (2019). Preferential Consolidation of Emotional Memory During Sleep: A Meta-Analysis. *Frontiers in psychology,* 10, 1014. https://doi.org/10.3389/fpsyg.2019.01014

Lipinska, G., & Thomas, K. G. F. (2019). The Interaction of REM Fragmentation and Night-Time Arousal Modulates Sleep-Dependent Emotional Memory Consolidation. *Frontiers in psychology,* 10, 1766. https://doi.org/10.3389/fpsyg.2019.01766

Liu, J., Magielski, J., Glenn, A., & Raine, A. (2022). The bidirectional relationship between sleep and externalizing behavior: A systematic review. *Sleep Epidemiology*, *2*, 100039. https://doi.org/10.1016/J.SLEEPE.2022.100039

Lo, J. C., Groeger, J. A., Cheng, G. H., Dijk, D. J., & Chee, M. W. (2016). Self-reported sleep duration and cognitive performance in older adults: a systematic review and meta-analysis. *Sleep medicine*, 17, 87–98. https://doi.org/10.1016/j.sleep.2015.08.021

Lo, J. C., Lee, S. M., Teo, L. M., Lim, J., Gooley, J. J., & Chee, M. W. L. (2017). Neurobehavioral impact of successive cycles of sleep restriction with and without naps in adolescents. *Sleep*, *40*(2). https://doi.org/10.1093/sleep/zsw042

Lo, J. C., Ong, J. L., Leong, R. L. F., Gooley, J. J., & Chee, M. W. L. (2016). Cognitive performance, sleepiness, and mood in partially sleep deprived adolescents: The need for sleep Study. *Sleep*, *39*(3), 687–698. https://doi.org/10.5665/sleep.5552

Lollies, F., Schnatschmidt, M., Bihlmeier, I., Genuneit, J., In-Albnon, T., Holtmann, M., Legenbauer, T., & Schlarb, A. A. (2022). Associations of sleep and emotion regulation processes in childhood and adolescence - a systematic review, report of methodological challenges and future directions. *Sleep science* (Sao Paulo, Brazil), 15(4), 490–514. https://doi.org/10.5935/1984-0063.20220082

Lonsdorf, T. B., Klingelhöfer-Jens, M., Andreatta, M., Beckers, T., Chalkia, A., Gerlicher, A., Jentsch, V. L., Drexler, S. M., Mertens, G., Richter, J., Sjouwerman, R., Wendt, J., & Merz, C. J. (2019). Navigating the garden of forking paths for data exclusions in fear conditioning research. *ELife*, *8*. https://doi.org/10.7554/eLife.52465

López-Muciño, L. A., García-García, F., Cueto-Escobedo, J., Acosta-Hernández, M., Venebra-Muñoz, A., & Rodríguez-Alba, J. C. (2022). Sleep loss and addiction. In *Neuroscience and Biobehavioral Reviews* (Vol. 141). Elsevier Ltd. https://doi.org/10.1016/j.neubiorev.2022.104832

Lovibond, S. H., & Lovibond, P. F. (1995). Depression anxiety stress scales. *Psychological Assessment*.

Lowe, C. J., Safati, A., & Hall, P. A. (2017). The neurocognitive consequences of sleep restriction: A meta-analytic review. *Neuroscience & Biobehavioral Reviews*, *80*, 586–604. https://doi.org/10.1016/J.NEUBIOREV.2017.07.010

Maccari, L., Martella, D., Marotta, A., Sebastiani, M., Banaj, N., Fuentes, L. J., & Casagrande, M. (2014). Effects of sleep loss on emotion recognition: a dissociation between face and word stimuli. *Experimental Brain Research*, *232*(10), 3147–3157. https://doi.org/10.1007/s00221-014-3995-9

Macmillan, N. A., & Creelman, C. D. (2004). *Detection Theory*. Psychology Press. https://doi.org/10.4324/9781410611147

Malezieux, M., Klein, A. S., & Gogolla, N. (2023). *Annual Review of Neuroscience Neural Circuits for Emotion*. https://doi.org/10.1146/annurev-neuro-111020

Maquet, P., Laureys, S., Peigneux, P., Fuchs, S., Petiau, C., Phillips, C., Aerts, J., Del Fiore, G., Degueldre, C., Meulemans, T., Luxen, A., Franck, G., Van Der Linden, M., Smith, C., & Cleeremans, A. (2000). Experience-dependent changes in cerebral activation during human REM sleep. *Nature Neuroscience*, *3*(8), 831–836. https://doi.org/10.1038/77744

Marchewka, A., Żurawski, Ł., Jednoróg, K., & Grabowska, A. (2014). The Nencki Affective Picture System (NAPS): Introduction to a novel, standardized, wide-range, high-quality, realistic picture database. *Behavior Research Methods*, *46*(2), 596–610. https://doi.org/10.3758/s13428-013-0379-1

Marshall, A. J., Acheson, D. T., Risbrough, V. B., Straus, L. D., & Drummond, S. P. A. (2014). Fear Conditioning, Safety Learning, and Sleep in Humans. *The Journal of Neuroscience*, *34*(35), 11754. https://doi.org/10.1523/JNEUROSCI.0478-14.2014

Marshall, L., & Born, J. (2007). The contribution of sleep to hippocampus-dependent memory consolidation. In *Trends in Cognitive Sciences* (Vol. 11, Issue 10, pp. 442–450). https://doi.org/10.1016/j.tics.2007.09.001

Maurer, L. F., Sharman, R., Espie, C. A., & Kyle, S. D. (2024). The effect of sleep restriction therapy for insomnia on REM sleep fragmentation: A secondary analysis of a randomised controlled trial. *Journal of Sleep Research*, *33*(1). https://doi.org/10.1111/jsr.13982

Mauss, I. B., Troy, A. S., & LeBourgeois, M. K. (2013). Poorer sleep quality is associated with lower emotion-regulation ability in a laboratory paradigm. *Cognition and Emotion*, *27*(3), 567–576. https://doi.org/10.1080/02699931.2012.727783

Mazzer, K., Boersma, K., & Linton, S. J. (2019). A longitudinal view of rumination, poor sleep and psychological distress in adolescents. *Journal of Affective Disorders*, *245*, 686–696. https://doi.org/10.1016/J.JAD.2018.11.053

McCall, C. A., & Watson, N. F. (2022). A Narrative Review of the Association between Post-Traumatic Stress Disorder and Obstructive Sleep Apnea. *Journal of Clinical Medicine*, *11*(2). https://doi.org/10.3390/jcm11020415

McCarter, S. J., Hagen, P. T., St. Louis, E. K., Rieck, T. M., Haider, C. R., Holmes, D. R., & Morgenthaler, T. I. (2022). Physiological markers of sleep quality: A scoping review. *Sleep Medicine Reviews*, *64*, 101657. https://doi.org/10.1016/J.SMRV.2022.101657

McMakin, D. L., Dahl, R. E., Buysse, D. J., Cousins, J. C., Forbes, E. E., Silk, J. S., Siegle, G. J., & Franzen, P. L. (2016). The impact of experimental sleep restriction on affective functioning in social and nonsocial contexts among adolescents. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *57*(9), 1027–1037. https://doi.org/10.1111/jcpp.12568

McNair, D., Lorr, M., & Doppleman, L. (1971). *Manual for the Profile of Mood States*. Educational and Industrial Testing Service.

McRae, K., & Gross, J. J. (2020). Emotion regulation. *Emotion*, *20*(1), 1–9. https://doi.org/10.1037/emo0000703

Mezick, E. J., Matthews, K. A., Hall, M., Kamarck, T. W., Buysse, D. J., Owens, J. F., & Reis, S. E. (2009). Intra-individual variability in sleep duration and fragmentation: Associations with stress. *Psychoneuroendocrinology*, *34*(9), 1346–1354. https://doi.org/10.1016/J.PSYNEUEN.2009.04.005

Mojsa-Kaja, J., & Ivcevic, Z. (2023). Emotion regulation strategies and mental health symptoms during COVID-19: the mediating role of insomnia. *International Journal of Occupational Medicine and Environmental Health*, *36*(1), 151–159. https://doi.org/10.13075/ijomeh.1896.01977

Morey, L. C. (2014). The Personality Assessment Inventory. In *Personality assessment, 2nd ed.* (pp. 181–228). Routledge/Taylor & Francis Group.

Morgenthaler, J., Wiesner, C. D., Hinze, K., Abels, L. C., Prehn-Kristensen, A., & Göder, R. (2014). Selective REM-Sleep Deprivation Does Not Diminish Emotional Memory Consolidation in Young Healthy Subjects. *PLOS ONE*, *9*(2), e89849-. https://doi.org/10.1371/journal.pone.0089849

Morrell, M. J., Finn, L., Kim, H., Peppard, P. E., Safwan Badr, M., & Young, T. (2000). Sleep Fragmentation, Awake Blood Pressure, and Sleep-Disordered Breathing in a Population-based Study. *American Journal of Respiratory and Critical Care Medicine*, *162*(6), 2091–2096. https://doi.org/10.1164/ajrccm.162.6.9904008

Murphy, M. J., & Peterson, M. J. (2015). Sleep Disturbances in Depression. *Sleep Medicine Clinics*, *10*(1), 17–23. https://doi.org/10.1016/j.jsmc.2014.11.009

Navas-Casado, M. L., García-Sancho, E., & Salguero, J. M. (2023). Associations between maladaptive and adaptive emotion regulation strategies and aggressive behavior: A systematic review. *Aggression and Violent Behavior*, 71, 1–25. https://doi.org/10.1016/j.avb.2023.101845

Nelson, K. L., Davis, J. E., & Corbett, C. F. (2022). Sleep quality: An evolutionary concept analysis. *Nursing Forum*, *57*(1), 144–151. https://doi.org/https://doi.org/10.1111/nuf.12659

Newbury, C. R., & Monaghan, P. (2019). When does sleep affect veridical and false memory consolidation? A meta-analysis. *Psychonomic Bulletin and Review*, *26*(2), 387–400. https://doi.org/10.3758/s13423-018-1528-4

Newbury, C. R., Crowley, R., Rastle, K., & Tamminen, J. (2021). Sleep deprivation and memory: Meta-analytic reviews of studies on sleep deprivation before and after learning. *Psychological Bulletin*, 147(11), 1215–1240. https://doi.org/10.1037/bul0000348

Nishida, M., & Walker, M. P. (2007). Daytime Naps, Motor Memory Consolidation and Regionally Specific Sleep Spindles. *PLOS ONE*, *2*(4), e341-. https://doi.org/10.1371/journal.pone.0000341

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-Analysis of Quantitative Sleep Parameters From Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values Across the Human Lifespan. *Sleep*, *27*(7), 1255–1273. https://doi.org/10.1093/sleep/27.7.1255

Ong, A. D., Kim, S., Young, S., & Steptoe, A. (2017). Positive affect and sleep: A systematic review. *Sleep medicine reviews*, 35, 21–32. https://doi.org/10.1016/j.smrv.2016.07.006

Orr, S. P., Lasko, N. B., Macklin, M. L., Pineles, S. L., Chang, Y., & Pitman, R. K. (2012). Predicting post-trauma stress symptoms from pre-trauma psychophysiologic reactivity, personality traits and measures of psychopathology. *Biology of Mood & Anxiety Disorders*, *2*(1), 8. https://doi.org/10.1186/2045-5380-2-8

Otsuka, Y., Kaneita, Y., Itani, O., Jike, M., Osaki, Y., Higuchi, S., Kanda, H., Kinjo, A., Kuwabara, Y., & Yoshimoto, H. (2020). Skipping breakfast, poor sleep quality, and Internet usage and their relation with unhappiness in Japanese adolescents. *PLOS ONE*, *15*(7), e0235252-. https://doi.org/10.1371/journal.pone.0235252

Pace-Schott, E. F., Germain, A., & Milad, M. R. (2015). Sleep and REM sleep disturbance in the pathophysiology of PTSD: the role of extinction memory. *Biology of mood & anxiety disorders*, 5, 3. https://doi.org/10.1186/s13587-015-0018-9

Pace-Schott, E. F., & Hobson, J. A. (2002). The Neurobiology of Sleep: Genetics, cellular physiology and subcortical networks. *Nature Reviews Neuroscience*, *3*(8), 591–605. https://doi.org/10.1038/nrn895

Pace-Schott, E. F., Shepherd, E., Spencer, R. M. C., Marcello, M., Tucker, M., Propper, R. E., & Stickgold, R. (2011). Napping promotes inter-session habituation to emotional stimuli. *Neurobiology of Learning and Memory*, *95*(1), 24–36. https://doi.org/10.1016/j.nlm.2010.10.006

Palagini, L., Baglioni, C., Ciapparelli, A., Gemignani, A., & Riemann, D. (2013). REM sleep dysregulation in depression: state of the art. Sleep medicine reviews, 17(5), 377–390. https://doi.org/10.1016/j.smrv.2012.11.001

Palmer, C. A., & Alfano, C. A. (2017). Sleep and emotion regulation: An organizing, integrative review. *Sleep medicine reviews*, 31, 6–16. https://doi.org/10.1016/j.smrv.2015.12.006

Palmer, C. A., Bower, J. L., Cho, K. W., Clementi, M. A., Lau, S., Oosterhoff, B., & Alfano, C. A. (2024). Sleep loss and emotion: A systematic review and meta-analysis of over 50 years of experimental research. *Psychological bulletin*, 150(4), 440–463. https://doi.org/10.1037/bul0000410

Panel, C. C. (2015). Recommended Amount of Sleep for a Healthy Adult: A Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society. *Sleep*, *38*(6), 843–844. https://doi.org/10.5665/sleep.4716

Patapoff, M., Ramsey, M., Titone, M., Kaufmann, C. N., Malhotra, A., Ancoli-Israel, S., Wing, D., Lee, E., & Eyler, L. T. (2022). Temporal relationships of ecological momentary mood and actigraphy-based sleep measures in bipolar disorder. *Journal of Psychiatric Research*, *150*, 257–263. https://doi.org/10.1016/J.JPSYCHIRES.2022.03.055

Perakakis, P., Joffily, M., Taylor, M., Guerra, P., & Vila, J. (2010). KARDIA: A Matlab software for the analysis of cardiac interbeat intervals. *Computer Methods and Programs in Biomedicine*, *98*(1), 83–89. https://doi.org/10.1016/J.CMPB.2009.10.002

Pereira, S. I. R., Beijamini, F., Vincenzi, R. A., & Louzada, F. M. (2015). Re-examining sleep's effect on motor skills: How to access performance on the finger tapping task? *Sleep Science*, *8*(1), 4–8. https://doi.org/10.1016/J.SLSCI.2015.01.001

Pesonen, A. K., Gradisar, M., Kuula, L., Short, M., Merikanto, I., Tark, R., Räikkönen, K., & Lahti, J. (2019). REM sleep fragmentation associated with depressive symptoms and genetic risk for depression in a community-based sample of adolescents. *Journal of Affective Disorders*, *245*, 757–763. https://doi.org/10.1016/j.jad.2018.11.077

Pilcher, J. J., Callan, C., & Posey, J. L. (2015). Sleep deprivation affects reactivity to positive but not negative stimuli. *Journal of Psychosomatic Research*, *79*(6), 657–662. https://doi.org/10.1016/j.jpsychores.2015.05.003

Pilcher, J. J., & Huffcutt, A. I. (1996). Effects of Sleep Deprivation on Performance: A Meta-Analysis. *Sleep*, *19*(4), 318–326. https://doi.org/10.1093/sleep/19.4.318

Plihal, W., & Born, J. (1997). Effects of Early and Late Nocturnal Sleep on Declarative and Procedural Memory. *Journal of Cognitive Neuroscience*, *9*(4), 534–547. https://doi.org/10.1162/jocn.1997.9.4.534

Pollatos, O., Herbert, B. M., Matthias, E., & Schandry, R. (2007). Heart rate response after emotional picture presentation is modulated by interoceptive awareness. *International Journal of Psychophysiology*, *63*(1), 117–124. https://doi.org/10.1016/J.IJPSYCHO.2006.09.003

Prehn-Kristensen, A., Molzow, I., Förster, A., Siebenhühner, N., Gesch, M., Wiesner, C. D., & Baving, L. (2017). Memory consolidation of socially relevant stimuli during sleep in healthy children and children with attention-deficit/hyperactivity disorder and oppositional defiant disorder: What you can see in their eyes. *Biological Psychology*, *123*, 196–204. https://doi.org/10.1016/J.BIOPSYCHO.2016.12.017

Qian, L., Ru, T., He, M., Li, S., & Zhou, G. (2022). Effects of a brief afternoon nap on declarative and procedural memory. *Neurobiology of Learning and Memory*, *194*, 107662. https://doi.org/10.1016/J.NLM.2022.107662

Qian, Y., Yi, H., Zou, J., Meng, L., Tang, X., Zhu, H., Yu, D., Zhou, H., Su, K., Guan, J., & Yin, S. (2016). Independent Association between Sleep Fragmentation and Dyslipidemia in Patients with Obstructive Sleep Apnea. *Scientific Reports*, *6*(1), 26089. https://doi.org/10.1038/srep26089

Qin, S., Leong, R. L. F., Ong, J. L., & Chee, M. W. L. (2023). Associations between objectively measured sleep parameters and cognition in healthy older adults: A meta-analysis. *Sleep Medicine Reviews*, *67*, 101734. https://doi.org/10.1016/J.SMRV.2022.101734

Rasch, B., & Born, J. (2013). About Sleep's Role in Memory. *Physiol Rev*, *93*, 681–766. https://doi.org/10.1152/physrev.00032.2012.-Over

Reddy, R., Palmer, C. A., Jackson, C., Farris, S. G., & Alfano, C. A. (2017). Impact of sleep restriction versus idealized sleep on emotional experience, reactivity and regulation in healthy adolescents. *Journal of Sleep Research*, *26*(4), 516–525. https://doi.org/10.1111/jsr.12484

Riemann, D., Krone, L. B., Wulff, K., & Nissen, C. (2020). Sleep, insomnia, and depression. *Neuropsychopharmacology: official publication of the American College of Neuropsychopharmacology*, 45(1), 74–89. https://doi.org/10.1038/s41386-019-0411-y

Riemann, D., Spiegelhalder, K., Nissen, C., Hirscher, V., Baglioni, C., & Feige, B. (2012). REM sleep instability--a new pathway for insomnia?. *Pharmacopsychiatry*, 45(5), 167–176. https://doi.org/10.1055/s-0031-1299721

Rodheim, K., Kainec, K., Noh, E., Jones, B., & Spencer, R. M. C. (2023). Emotional memory consolidation during sleep is associated with slow oscillation–spindle coupling strength in young and older adults. *Learning & Memory*, *30*(9), 237–244. https://doi.org/10.1101/lm.053685.122

Romney, L., Larson, M. J., Clark, T., Tucker, L. A., Bailey, B. W., & LeCheminant, J. D. (2016). Reduced Sleep Acutely Influences Sedentary Behavior and Mood But Not Total Energy Intake in Normal-Weight and Obese Women. *Behavioral Sleep Medicine*, *14*(5), 528–538. https://doi.org/10.1080/15402002.2015.1036272

Roozendaal, B., & McGaugh, J. L. (2011). Memory modulation. *Behavioral neuroscience*, 125(6), 797–824. https://doi.org/10.1037/a0026187

Rosales-Lagarde, A., Armony, J. L., 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. *Frontiers in Behavioral Neuroscience*, *JUNE*. https://doi.org/10.3389/fnbeh.2012.00025

Rossa, K. R., Smith, S. S., Allan, A. C., & Sullivan, K. A. (2014). The effects of sleep restriction on executive inhibitory control and affect in young adults. *Journal of Adolescent Health*, *55*(2), 287–292. https://doi.org/10.1016/j.jadohealth.2013.12.034

Rupp, T. L., Wesensten, N. J., & Balkin, T. J. (2012). Trait-Like Vulnerability to Total and Partial Sleep Loss. *Sleep*, *35*(8), 1163–1172. https://doi.org/10.5665/sleep.2010

Sack, B., Broer, K., & Anders, S. (2019). Sleep deprivation selectively enhances interpersonal emotion recognition from dynamic facial expressions at long viewing times: An observational study. *Neuroscience Letters*, *694*, 225–230. https://doi.org/10.1016/J.NEULET.2018.10.035

Sagaspe, P., Sanchez-Ortuno, M., Charles, A., Taillard, J., Valtat, C., Bioulac, B., & Philip, P. (2006). Effects of sleep deprivation on Color-Word, Emotional, and Specific Stroop interference and on self-reported anxiety. *Brain and Cognition*, *60*(1), 76–87. https://doi.org/10.1016/j.bandc.2005.10.001

Saguin, E., Gomez-Merino, D., Sauvet, F., Leger, D., & Chennaoui, M. (2021). Sleep and PTSD in the Military Forces: A Reciprocal Relationship and a Psychiatric Approach. *Brain Sciences*, *11*(10). https://doi.org/10.3390/brainsci11101310

Saksvik-Lehouillier, I., Saksvik, S. B., Dahlberg, J., Tanum, T. K., Ringen, H., Karlsen, H. R., Smedbøl, T., Sørengaard, T. A., Stople, M., Kallestad, H., & Olsen, A. (2020). Mild to moderate partial sleep deprivation is associated with increased impulsivity and decreased positive affect in young adults. *Sleep*, *43*(10). https://doi.org/10.1093/sleep/zsaa078

Salfi, F., Lauriola, M., Tempesta, D., Calanna, P., Socci, V., De Gennaro, L., & Ferrara, M. (2020). Effects of total and partial sleep deprivation on reflection impulsivity and risk-taking in deliberative decision-making. *Nature and Science of Sleep*, *12*, 309–324. https://doi.org/10.2147/NSS.S250586

Schäfer, S. K., Wirth, B. E., Staginnus, M., Becker, N., Michael, T., & Sopp, M. R. (2020). Sleep's impact on emotional recognition memory: A meta-analysis of whole-night, nap, and REM sleep effects. *Sleep Medicine Reviews*, *51*, 101280. https://doi.org/10.1016/J.SMRV.2020.101280

Scharf, R. J., Demmer, R. T., Silver, E. J., & Stein, R. E. (2013). Nighttime sleep duration and externalizing behaviors of preschool children. *Journal of developmental and behavioral pediatrics: JDBP*, 34(6), 384–391. https://doi.org/10.1097/DBP.0b013e31829a7a0d

Schenker, M. T., Ney, L. J., Miller, L. N., Felmingham, K. L., Nicholas, C. L., & Jordan, A. S. (2021). Sleep and fear conditioning, extinction learning and extinction recall: A systematic review and meta-analysis of polysomnographic findings. *Sleep medicine reviews*, 59, 101501. https://doi.org/10.1016/j.smrv.2021.101501

Schwarz, J., Axelsson, J., Gerhardsson, A., Tamm, S., Fischer, H., Kecklund, G., & Åkerstedt, T. (2019). Mood impairment is stronger in young than in older adults after sleep deprivation. *Journal of Sleep Research*, *28*(4), e12801. https://doi.org/https://doi.org/10.1111/jsr.12801

Scott, A. J., Webb, T. L., Martyn-St James, M., Rowse, G., & Weich, S. (2021). Improving sleep quality leads to better mental health: A meta-analysis of randomised controlled trials. *Sleep medicine reviews*, 60, 101556. https://doi.org/10.1016/j.smrv.2021.101556

Scott, J. P. R., McNaughton, L. R., & Polman, R. C. J. (2006). Effects of sleep deprivation and exercise on cognitive, motor performance and mood. *Physiology and Behavior*, *87*(2), 396–408. https://doi.org/10.1016/j.physbeh.2005.11.009

Settineri, S., Gitto, L., Conte, F., Fanara, G., Mallamace, D., Mento, C., Silvestri, R., Tati, F., Zoccali, R., Cordici, F., Grugno, R., Polimeni, G., Vitetta, A., & Bramanti, P. (2012). Mood and sleep problems in adolescents and young adults: an econometric analysis. *The journal of mental health policy and economics*, 15(1), 33–41

Shenfield, L., Beanland, V., Filtness, A., & Apthorp, D. (2020). The impact of sleep loss on sustained and transient attention: An EEG study. *PeerJ*, *2020*(3). https://doi.org/10.7717/peerj.8960

Shermohammed, M., Kordyban, L. E., & Somerville, L. H. (2019). Examining the causal effects of sleep deprivation on emotion regulation and its neural mechanisms. *Journal of Cognitive Neuroscience*, *32*(7), 1289–1300. https://doi.org/10.1162/jocn_a_01555

Shin, C., Kim, J., Yi, H., Lee, H., Lee, J., & Shin, K. (2005). Relationship between trait-anger and sleep disturbances in middle-aged men and women. *Journal of Psychosomatic Research*, *58*(2), 183–189. https://doi.org/10.1016/j.jpsychores.2004.07.002

Short, M. A., & Banks, S. (2014). The functional impact of sleep deprivation, sleep restriction, and sleep fragmentation. In M. T. Bianchi (Ed.), *Sleep deprivation and disease: Effects on the body, brain and behavior* (pp. 13–26). Springer Science + Business Media. https://doi.org/10.1007/978-1-4614-9087-6_2

Short, M. A., & Louca, M. (2015). Sleep deprivation leads to mood deficits in healthy adolescents. *Sleep Medicine*, *16*(8), 987–993. https://doi.org/10.1016/j.sleep.2015.03.007

Skurvydas, A., Kazlauskaite, D., Zlibinaite, L., Cekanauskaite, A., Valanciene, D., Karanauskiene, D., Zuoziene, I. J., Majauskiene, D., Mickeviciene, D., & Satas, A. (2021). Effects of two nights of sleep deprivation on executive function and central and peripheral fatigue during maximal voluntary contraction lasting 60s. *Physiology and Behavior*, *229*. https://doi.org/10.1016/j.physbeh.2020.113226

Sopp, M. R., Michael, T., & Mecklinger, A. (2018). Effects of early morning nap sleep on associative memory for neutral and emotional stimuli. *Brain Research*, *1698*, 29–42. https://doi.org/10.1016/J.BRAINRES.2018.06.020

Staats, R., Barros, I., Fernandes, D., Grencho, D., Reis, C., Matos, F., Valença, J., Marôco, J., de Almeida, A. B., & Bárbara, C. (2020). The Importance of Sleep Fragmentation on the Hemodynamic Dipping in Obstructive Sleep Apnea Patients. *Frontiers in physiology*, 11, 104. https://doi.org/10.3389/fphys.2020.00104

Stanislaw, H., & Todorov, N. (1999). Calculation of signal detection theory measures. *Behavior Research Methods, Instruments, & Computers*, *31*(1), 137–149. https://doi.org/10.3758/BF03207704

Stark, S. M., Kirwan, C. B., & Stark, C. E. L. (2019). Mnemonic Similarity Task: A Tool for Assessing Hippocampal Integrity. *Trends in Cognitive Sciences*, *23*(11), 938–951. https://doi.org/10.1016/J.TICS.2019.08.003

Steiger, A., & Pawlowski, M. (2019). Depression and Sleep. *International journal of molecular sciences*, 20(3), 607. https://doi.org/10.3390/ijms20030607

Stenson, A. R., Kurinec, C. A., Hinson, J. M., Whitney, P., & Van Dongen, H. P. A. (2021). Total sleep deprivation reduces top-down regulation of emotion without altering bottom-up affective processing. *PloS one*, 16(9), e0256983. https://doi.org/10.1371/journal.pone.0256983

Stepanski E. J. (2002). The effect of sleep fragmentation on daytime function. *Sleep*, *25*(3), 268–276. https://doi.org/10.1093/sleep/25.3.268

Sundelin, T., Lekander, M., Kecklund, G., Van Someren, E. J. W., Olsson, A., & Axelsson, J. (2013). Cues of Fatigue: Effects of Sleep Deprivation on Facial Appearance. *Sleep*, *36*(9), 1355–1360. https://doi.org/10.5665/sleep.2964

Swift, K. M., Gross, B. A., Frazer, M. A., Bauer, D. S., Clark, K. J. D., Vazey, E. M., Aston-Jones, G., Li, Y., Pickering, A. E., Sara, S. J., & Poe, G. R. (2018). Abnormal Locus Coeruleus Sleep Activity Alters Sleep Signatures of Memory Consolidation and Impairs Place Cell Stability and Spatial Memory. *Current Biology*, *28*(22), 3599-3609.e4. https://doi.org/10.1016/j.cub.2018.09.054

Talbot, L. S., McGlinchey, E. L., Kaplan, K. A., Dahl, R. E., & Harvey, A. G. (2010). Sleep Deprivation in Adolescents and Adults: Changes in Affect. *Emotion*, *10*(6), 831–841. https://doi.org/10.1037/a0020138

te Lindert, B. H. W., & Van Someren, E. J. W. (2013). Sleep Estimates Using Microelectromechanical Systems (MEMS). *Sleep*, *36*(5), 781–789. https://doi.org/10.5665/sleep.2648

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 Research Bulletin*, *82*(1–2), 104–108. https://doi.org/10.1016/j.brainresbull.2010.01.014

Tempesta, D., De Gennaro, L., Natale, V., & Ferrara, M. (2015). Emotional memory processing is influenced by sleep quality. *Sleep Medicine*, *16*(7), 862–870. https://doi.org/10.1016/j.sleep.2015.01.024

Tempesta, D., Socci, V., Coppo, M., Dello Ioio, G., Nepa, V., De Gennaro, L., & Ferrara, M. (2016). The effect of sleep deprivation on the encoding of contextual and non-contextual aspects of emotional memory. *Neurobiology of Learning and Memory*, *131*, 9–17. https://doi.org/10.1016/j.nlm.2016.03.007

Tempesta, D., Socci, V., De Gennaro, L., & Ferrara, M. (2018). Sleep and emotional processing. *Sleep medicine reviews*, 40, 183–195. https://doi.org/10.1016/j.smrv.2017.12.005

Tempesta, D., Socci, V., Dello Ioio, G., De Gennaro, L., & Ferrara, M. (2017). The effect of sleep deprivation on retrieval of emotional memory: a behavioural study using film stimuli. *Experimental Brain Research*, *235*(10), 3059–3067. https://doi.org/10.1007/s00221-017-5043-z

Thompson, K. I., Chau, M., Lorenzetti, M. S., Hill, L. D., Fins, A. I., & Tartar, J. L. (2022). Acute sleep deprivation disrupts emotion, cognition, inflammation, and cortisol in young healthy adults. *Frontiers in behavioral neuroscience*, 16, 945661. https://doi.org/10.3389/fnbeh.2022.945661

Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination--relationship with negative mood and sleep quality. *Personality and Individual Differences*, 34(7), 1293–1301. https://doi.org/10.1016/S0191-8869(02)00120-4

Tomaso, C. C., Johnson, A. B., & Nelson, T. D. (2021). The effect of sleep deprivation and restriction on mood, emotion, and emotion regulation: three meta-analyses in one. *Sleep*, *44*(6), zsaa289. https://doi.org/10.1093/sleep/zsaa289

Tononi, G., & Cirelli, C. (2003). Sleep and synaptic homeostasis: a hypothesis. *Brain Research Bulletin*, *62*(2), 143–150. https://doi.org/10.1016/J.BRAINRESBULL.2003.09.004

Tononi, G., & Cirelli, C. (2014). Sleep and the price of plasticity: from synaptic and cellular homeostasis to memory consolidation and integration. *Neuron*, 81(1), 12–34. https://doi.org/10.1016/j.neuron.2013.12.025

Tononi, G., & Cirelli, C. (2020). Sleep and synaptic down-selection. *The European journal of neuroscience*, 51(1), 413–421. https://doi.org/10.1111/ejn.14335

Troxel, W. M., Buysse, D. J., Hall, M., & Matthews, K. A. (2009). Marital happiness and sleep disturbances in a multi-ethnic sample of middle-aged women. *Behavioral Sleep Medicine*, *7*(1), 2–19. https://doi.org/10.1080/15402000802577736

Valentino, R. J., & Volkow, N. D. (2020). Drugs, sleep, and the addicted brain. *Neuropsychopharmacology: official publication of the American College of Neuropsychopharmacology*, 45(1), 3–5. https://doi.org/10.1038/s41386-019-0465-x

van der Helm, E., & Walker, M. P. (2011). Sleep and Emotional Memory Processing. *Sleep medicine clinics*, 6(1), 31–43. https://doi.org/10.1016/j.jsmc.2010.12.010

Van Der Helm, E., Yao, J., Dutt, S., Rao, V., Saletin, J. M., & Walker, M. P. (2011). REM sleep depotentiates amygdala activity to previous emotional experiences. *Current Biology*, *21*(23), 2029–2032. https://doi.org/10.1016/j.cub.2011.10.052

Van Egroo, M., Koshmanova, E., Vandewalle, G., & Jacobs, H. I. L. (2022). Importance of the locus coeruleus-norepinephrine system in sleep-wake regulation: Implications for aging and Alzheimer's disease. *Sleep Medicine Reviews*, *62*, 1–11. https://doi.org/10.1016/j.smrv.2022.101592

Van Heugten-Van Der Kloet, D., Giesbrecht, T., & Merckelbach, H. (2015). Sleep loss increases dissociation and affects memory for emotional stimuli. *Journal of Behavior Therapy and Experimental Psychiatry*, *47*, 9–17. https://doi.org/10.1016/j.jbtep.2014.11.002

Van Liempt, S. (2012). Sleep disturbances and PTSD: A perpetual circle? *European Journal of Psychotraumatology*, *3*. https://doi.org/10.3402/ejpt.v3i0.19142

Vandekerckhove, M., & Wang, Y. L. (2017). Emotion, emotion regulation and sleep: An intimate relationship. *AIMS neuroscience*, 5(1), 1–17. https://doi.org/10.3934/Neuroscience.2018.1.1

Vardar, S. A., Oztürk, L., Kurt, C., Bulut, E., Sut, N., & Vardar, E. (2007). Sleep deprivation induced anxiety and anaerobic performance. *Journal of sports science & medicine*, 6(4), 532–537.

Vasciaveo, V., Iadarola, A., Casile, A., Dante, D., Morello, G., Minotta, L., Tamagno, E., Cicolin, A., & Guglielmotto, M. (2023). Sleep fragmentation affects glymphatic system through the different expression of AQP4 in wild type and 5xFAD mouse models. *Acta Neuropathologica Communications*, *11*(1), 16. https://doi.org/10.1186/s40478-022-01498-2

Vethe, D., Drews, H. J., Scott, J., Engstrøm, M., Heglum, H. S. A., Grønli, J., Wisor, J. P., Sand, T., Lydersen, S., Kjørstad, K., Faaland, P. M. P., Vestergaard, C. L., Langsrud, K., & Kallestad, H. (2022). Evening light environments can be designed to consolidate and increase the duration of REM-sleep. *Scientific Reports*, *12*(1), 8719. https://doi.org/10.1038/s41598-022-12408-w

Vila, J. (2004). Psychophysiological Assessment. *Encyclopedia of Applied Psychology, Three-Volume Set*, *3*, 197–203. https://doi.org/10.1016/B0-12-657410-3/00512-2

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. *Psychosomatic Medicine*, *64*, 627–634. https://doi.org/10.1097/01.PSY.0000021940.35402.51

Wagner, U., Gais, S., & Born, J. (2001). Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. *Learning and Memory*, *8*(2), 112–119. https://doi.org/10.1101/lm.36801

Wagner, U., Hallschmid, M., Rasch, B., & Born, J. (2006). Brief Sleep After Learning Keeps Emotional Memories Alive for Years. *Biological Psychiatry*, *60*(7), 788–790. https://doi.org/10.1016/j.biopsych.2006.03.061

Walker, F. R., Thomson, A., Pfingst, K., Vlemincx, E., Aidman, E., & Nalivaiko, E. (2019). Habituation of the electrodermal response – A biological correlate of resilience? *PLOS ONE*, *14*(1), e0210078. https://doi.org/10.1371/journal.pone.0210078

Walker, M. P., Brakefield, T., Morgan, A., Hobson, J. A., & Stickgold, R. (2002). Practice with Sleep Makes Perfect: Sleep-Dependent Motor Skill Learning. *Neuron*, *35*(1), 205–211. https://doi.org/10.1016/S0896-6273(02)00746-8

Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. *Annual review of psychology,* 57, 139–166. https://doi.org/10.1146/annurev.psych.56.091103.070307

Walker, M. P., & van der Helm, E. (2009). Overnight Therapy? The Role of Sleep in Emotional Brain Processing. *Psychological Bulletin*, *135*(5), 731–748. https://doi.org/10.1037/a0016570

Wang, Y., Carreras, A., Lee, S., Hakim, F., Zhang, S. X., Nair, D., Ye, H., & Gozal, D. (2014). Chronic sleep fragmentation promotes obesity in young adult mice. *Obesity*, *22*(3), 758–762. https://doi.org/https://doi.org/10.1002/oby.20616

Wassing, R., Lakbila-Kamal, O., Ramautar, J. R., Stoffers, D., Schalkwijk, F., & Van Someren, E. J. W. (2019). Restless REM Sleep Impedes Overnight Amygdala Adaptation. *Current Biology*, *29*(14), 2351-2358.e4. https://doi.org/10.1016/j.cub.2019.06.034

Watson, B. O., Levenstein, D., Greene, J. P., Gelinas, J. N., & Buzsáki, G. (2016). Network Homeostasis and State Dynamics of Neocortical Sleep. *Neuron*, *90*(4), 839–852. https://doi.org/10.1016/j.neuron.2016.03.036

Wei, Y., & Van Someren, E. J. W. (2020). Interoception relates to sleep and sleep disorders. *Current Opinion in Behavioral Sciences*, 33, 1–7. https://doi.org/10.1016/j.cobeha.2019.11.008

Werner, G. G., Schabus, M., Blechert, J., & Wilhelm, F. H. (2021). Differential Effects of REM Sleep on Emotional Processing: Initial Evidence for Increased Short-term Emotional Responses and Reduced Long-term Intrusive Memories. *Behavioral Sleep Medicine*, *19*(1), 83–98. https://doi.org/10.1080/15402002.2020.1713134

Whitney, P., Hinson, J. M., Satterfield, B. C., Grant, D. A., Honn, K. A., & Van Dongen, H. P. A. (2017). Sleep Deprivation Diminishes Attentional Control Effectiveness and Impairs Flexible Adaptation to Changing Conditions. *Scientific Reports*, *7*(1). https://doi.org/10.1038/s41598-017-16165-z

Wiesner, C. D., Pulst, J., Krause, F., Elsner, M., Baving, L., Pedersen, A., Prehn-Kristensen, A., & Göder, R. (2015). The effect of selective REM-sleep deprivation on the consolidation and affective evaluation of emotional memories. *Neurobiology of Learning and Memory*, *122*, 131–141. https://doi.org/10.1016/j.nlm.2015.02.008

Wilckens, K. A., Woo, S. G., Kirk, A. R., Erickson, K. I., & Wheeler, M. E. (2014). Role of sleep continuity and total sleep time in executive function across the adult lifespan. *Psychology and Aging*, *29*(3), 658–665. https://doi.org/10.1037/a0037234

Wu, D., Tong, M., Ji, Y., Ruan, L., Lou, Z., Gao, H., & Yang, Q. (2021). REM Sleep Fragmentation in Patients With Short-Term Insomnia Is Associated With Higher BDI Scores. *Frontiers in Psychiatry*, *12*. https://doi.org/10.3389/fpsyt.2021.733998

Zhang, J., Lau, E. Y. Y., & Hsiao, J. H. wen. (2019). Using emotion regulation strategies after sleep deprivation: ERP and behavioral findings. *Cognitive, Affective and Behavioral Neuroscience*, *19*(2), 283–295. https://doi.org/10.3758/s13415-018-00667-y

Zimmer, H., & Richter, F. (2023). Novelty detection and orienting: effects on skin conductance and heart rate. *Psychological Research*, *87*(4), 1101–1113. https://doi.org/10.1007/s00426-022-01735-2

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. https://doi.org/10.1093/sleep/28.1.47