Disentangling Sleep’s Role in Emotion Processing

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# Abstract

Sleep plays a crucial role in emotion processing, with sleep disruptions contributing to emotion dysregulation and increased risk of mental illness. This review examines the relationship between sleep and three key aspects of emotion processing: emotional reactivity, cognitive emotion regulation, and emotional inertia. Evidence suggests that sleep deprivation heightens emotional reactivity, weakens the ability to regulate emotions adaptively, and increases the persistence of negative emotions over time. Neurobiological findings highlight the role of prefrontal-limbic circuitry in all of these processes, with sleep loss impairing top-down regulatory control over emotional responses. Furthermore, rapid eye movement sleep and slow-wave sleep appear to play distinct roles in restoring emotional balance. The findings from this review highlight the multifaceted pathways through which sleep disturbance gives rise to emotion dysregulation and, over time, increases vulnerability to mental illness.

# Keywords

Sleep, emotional reactivity, cognitive emotion regulation, emotional inertia, mental health, anxiety

# Introduction

Sleep disturbances often co-occur with psychiatric mood disorders and are a strong risk factor for both initial and recurrent episodes (Baglioni et al., 2010; Bi & Chen, 2022; Chellappa & Aeschbach, 2022; Freeman et al., 2017; Harvey, 2001; Palmer & Alfano, 2017). Reciprocally, improving sleep quality improves symptoms of depression, anxiety and stress (Kudrnáčová & Kudrnáč, 2023; Scott et al., 2021). A growing body of research suggests that sleep plays an important role in emotion processing, with insufficient sleep giving rise to emotional dysregulation and mental ill-health (Tempesta et al., 2018). Understanding the psychological and neurobiological mechanisms underpinning sleep’s role in emotion processing is therefore crucial for addressing the growing global burden of psychiatric disturbance. Furthermore, because emotion processing is broadly defined, complex and involves multiple temporal components, disentangling the impact of sleep across these components is central to such mechanistic understanding.

The scientific literature on sleep and emotion is generally focused on three overlapping processes (Figure 1). The first is concerned with how we initially respond to an aversive experience, termed emotional reactivity. The second relates to how we interact with our emotions and work to modify them using thought-based strategies, known as cognitive emotion regulation (CER). The third addresses the extent to which our emotions persist from one moment to the next, defined as emotional inertia. Previous reviews on sleep and emotion have typically addressed only one or two of these processes in isolation. In this review, we synthesise the literature linking sleep to all three processes and outline the overlapping psychological and neurobiological mechanisms.

# Emotional reactivity

Emotional hyper-reactivity is characteristic of a wide range of psychopathologies, including anxiety, depressive and psychotic disorders, for which sleep problems are also a central issue (Freeman et al., 2020). In this section, we will examine the effects of sleep deprivation on psychophysiological measures of emotional reactivity and self-reported evaluations of emotional affect. We will then explore the neurobiological mechanisms linking sleep to emotional reactivity.

Sleep deprivation intensifies psychophysiological responses to emotionally aversive stimuli. Across two studies, Franzen et al. (2008, 2009) showed that sleep-deprived individuals exhibited heightened pupillary responses to negative images compared to neutral and positive ones, whereas no such difference was observed in well-rested individuals. Others have observed amplified psychophysiological responses to stress following sleep loss. For instance, Liu et al. (2015) found that sleep deprivation intensified stress-induced increases in skin conductance responses during a challenging perceptual task with false feedback. Likewise, Minkel et al. (2014) reported elevated levels of the stress hormone cortisol, both at baseline and in response to a stressful speech task, in sleep-deprived individuals, whereas well-rested participants showed no such effect. Further evidence comes from van Leeuwen et al. (2018) who examined the effect of prolonged partial sleep deprivation on arousal responses. Participants were limited to 4 hours of time in bed per night for five consecutive nights, followed by three recovery nights with 8 hours of time in bed. Compared to a control group that maintained 8 hours of time in bed throughout the experiment, the partially sleep deprived group exhibited changes in heart rate variability consistent with heightened activity in the sympathetic nervous system.

Event-related potentials and especially the Late Positive Potential (LPP), an event-related potential with a centroparietal distribution that represents the brain’s attentional response to emotional stimuli, are also sensitive to sleep loss. For instance, Cote et al. (2015) found that LPPs were enhanced among sleep deprived individuals viewing negative compared to positive and neutral images, but not well rested participants. Similarly, Lustig et al. (2018) reported that partially sleep deprived individuals (4 hours of sleep) exhibited larger LPPs to positive versus neutral images relative to those who had slept normally (8 hours of sleep). In contrast, Alfarra et al. (2015) found LPPs to neutral images were greater than LPPs to emotional images following a night of sleep deprivation, as compared to normal sleep. While methodological differences may explain these varying results, the findings collectively suggest that sleep loss disrupts the attentional processing of emotional stimuli (Lustig et al., 2018).

  Other work has examined the effects of sleep deprivation on emotional reactivity using subjective measures of emotional arousal. Several studies have observed no significant change in self-reported arousal following sleep deprivation (Liu et al., 2015; Minkel et al., 2011; Schwarz et al., 2013; Tomaso et al., 2020), even when psychophysiological effects of sleep loss have emerged (Franzen et al., 2009). However, a recent meta-analysis by Palmer et al. (2024) concluded that sleep deprivation (total or partial) leads to an overall blunting of self-reported arousal in response to emotional stimuli. This suggests that, despite earlier inconsistencies, the broader literature now supports the conclusion that sleep loss reduces subjective arousal responses.

 This emerging consensus, however, underscores a persistent discrepancy between psychophysiological and self-reported markers of affective reactivity. Unlike subjective ratings, which reflect coarse, conscious evaluations of internal states, psychophysiological measures capture fine-grained autonomic responses (Bradley & Lang, 2007; Cunningham et al., 2014; Franzen et al., 2009; Tempesta et al., 2020). McCall et al. (2015) found that coherence between psychophysiological and subjective arousal measures was particularly strong among individuals with higher interoceptive accuracy. Given that sleep deprivation impairs interoceptive accuracy (Bynum & Brindle, 2025), sleep-deprived individuals may be less able to detect or report changes in their internal emotional states, potentially limiting the sensitivity of self-report measures to sleep-related affective changes.

 Turning to self-reported ratings of emotional valence (i.e., feelings of pleasantness), Reddy et al. (2017) found that a restricted 4-hour sleep opportunity intensified negative valence ratings to emotionally aversive images, as compared to a 9.5-hour sleep opportunity. However, a recent meta-analysis by Palmer et al. (2024) failed to find robust effects of sleep loss on valence ratings in response to emotional stimuli. In contrast, sleep loss appears to more reliably promote a negative bias in the processing of emotionally neutral stimuli. Tempesta et al. (2015) found that valence ratings for neutral images were more negative among people who reported having poor quality sleep, as compared to those reporting high sleep quality. Convergent findings come from studies that have used sleep deprivation protocols in the lab, finding that sleep deprived individuals are more likely to judge neutral stimuli as more negative than well-rested individuals (Tempesta et al., 2010; van der Helm et al., 2010).

Sleep deprivation has also been shown to lead to an overgeneralisation of emotional responses to non-threatening stimuli (Barber & Budnick, 2015; Zenses et al., 2020). Goldstein-Piekarski et al. (2015) found that sleep-deprived individuals rated significantly more non-threatening faces as threatening when compared to individuals who were well-rested. Moreover, stimulus-evoked changes in heart rate acceleration, a marker of threat sensitivity (van Honk et al., 2001), were amplified for threatening relative to non-threatening faces in well-rested individuals, whereas no significant cardiac discrimination was observed in sleep-deprived participants (Goldstein-Piekarski et al., 2015). Cote et al. (2014) found that sleep deprivation increased the N170 event-related potential component, a marker of emotional reactivity, when participants viewed fearful and angry faces. Interestingly, this effect was especially pronounced for faces that had a high degree of perceptual difficulty, suggesting that sleep loss had disrupted the specificity of emotional responses to negative stimuli.

Amplified emotional responding after sleep deprivation has been linked to hyperactivity in the brain’s emotion generating circuitry, including the amygdala and anterior cingulate cortex (Ben Simon et al., 2020; Goldstein et al., 2013; Simon et al., 2015; van der Helm & Walker, 2012; Yoo et al., 2007). Such hyperactivity is thought to arise from a breakdown of top-down limbic control by medial prefrontal cortex (Yoo et al., 2007). Consistent with this view, medial prefrontal engagement and connectivity with amygdala are reduced in sleep-deprived individuals (as compared to well-rested individuals) when viewing emotionally negative stimuli (Ben Simon et al., 2020; Goldstein & Walker, 2014; Simon et al., 2015; Yoo et al., 2007). This profile of exaggerated amygdala reactivity and reduced medial prefrontal cortex-amygdala connectivity when viewing aversive stimuli is also observed in people undergoing partial sleep deprivation protocols (Motomura et al., 2013; Tempesta et al., 2020).

Given that sleep deprivation disrupts emotional reactivity, a converse question concerns the components of sleep that help to restore emotional brain function. Rapid eye movement (REM) sleep is thought to play an important role in the restoration of affective homeostasis. REM sleep is characterised by marked reductions in brain levels of noradrenaline (norepinephrine), a neurotransmitter that plays an essential role in the regulation of arousal and stress (Ouyang et al., 2004). Noradrenergic signalling is often disrupted in psychiatric conditions that are characterised by emotional disturbances, including depressive and post-traumatic stress disorder. Goldstein and Walker (2014) propose that reduced noradrenergic tone during REM sleep supports the restoration of top-down emotional control networks, and thus the recalibration of affective responding. Supporting this view, a growing body of work demonstrates an association between REM sleep physiology and the overnight dissipation of amygdala reactivity, coupled with increased medial prefrontal cortex-amygdala connectivity (Rosales-Lagarde et al., 2012; van der Helm et al., 2011; Walker & van der Helm, 2009).

Other research has further expanded our understanding of sleep’s role in emotional control by highlighting the importance of NREM slow-wave sleep. Specifically, reductions in both the duration of slow-wave sleep and the magnitude of its characteristic pattern of rhythmic brain activity (slow-wave activity, or SWA, <4 Hz) have been observed in individuals with anxiety disorders (Arriaga & Paiva, 1990; Baglioni et al., 2016; Forbes et al., 2008; Fuller et al., 1997). On the other hand, higher levels of SWA (and the duration of slow-wave sleep) have been linked to greater overnight reductions in state anxiety among healthy individuals (Ben Simon et al., 2020; Chellappa & Aeschbach, 2022). Interestingly, Ben Simon et al. (2020) also found that higher levels of sleep SWA were associated with stronger engagement of medial prefrontal cortex when participants viewed emotionally aversive stimuli. Given that impairments of top-down emotional control (alongside SWA disruptions) are often observed across anxiety disorders (Bishop et al., 2004; M. J. Kim et al., 2011), these findings may suggest that SWA supports the restoration of prefrontal mechanisms necessary for regulating anxiety.

In summary, sleep loss has been shown to increase psychophysiological reactivity to emotionally aversive stimuli. However, recent work suggests that sleep loss may blunt self-reported arousal responses to emotional stimuli. These inconsistencies may arise from the fact that self-report requires cognitive introspection, which may be less sensitive to the effects of sleep loss. Nonetheless, converging evidence indicates that sleep deprivation induces a negative bias in emotional responses and impairs threat detection. Functional neuroimaging studies suggests that sleep deprivation disrupts top-down control of amygdala by prefrontal cortex, leading to emotional hyperreactivity and impaired emotional discrimination. REM sleep and SWA appear to help restore this prefrontal-amygdala circuit in the service of mental health and wellbeing.

# Cognitive emotion regulation

Our ability to consciously regulate our emotions influences how we respond to affective experiences. The process model of emotion regulation (Gross, 2015) suggests that emotion generation involves encountering a relevant situation, focusing on key aspects of that situation, appraising the situation in relation to our goals, and then responding. People can use cognitive emotion regulation (CER) strategies to modify their emotional responses to situations (Aldao & Nolen-Hoeksema, 2010; Garnefski et al., 2001). These strategies can be categorised into five groups based on where they intervene in the emotion-generation process: 1) situation selection (e.g., avoiding the situation), 2) situation modification (e.g., taking action to change the situation), 3) attentional deployment (e.g., shifting attention away from the situation), 4) cognitive change (e.g., reinterpreting the situation) and 5) response modulation (e.g., suppressing outward emotional expression; McRae & Gross, 2020).

Commonly used CER strategies include cognitive reappraisal (i.e., reframing the experience in a more positive light), acceptance (i.e., being aware of and accepting emotions without feeling the need to change them), and positive refocusing (i.e., shifting attention to neutral or positive matters; see Table 1; Gross & John, 2003). These strategies are often labelled as adaptive and tend to promote psychological wellbeing in the long-term (Kirschbaum-Lesch et al., 2021). Other commonly used strategies include rumination (i.e., immersing oneself in negative thoughts about a situation) and suppression (i.e., concealing expressions of true feeling; Clancy et al., 2020; Gross & John, 2003). These strategies are often labelled as maladaptive; they provide only short-term respite (Campbell-Sills & Barlow, 2007) and can even amplify affective disturbances in the long-term (Aldao & Nolen-Hoeksema, 2010; Garnefski et al., 2001; Nolen-Hoeksema et al., 2008; Sullivan et al., 2023). Recent research has shown that greater use of adaptive CER strategies is linked to reduced symptoms of depression and anxiety (Domaradzka & Fajkowska, 2018; Sullivan et al., 2023) whereas greater use of maladaptive CER strategies have been associated with greater depression and anxiety severity (Domaradzka & Fajkowska, 2018; Garnefski et al., 2001; Sullivan et al., 2023) and thus are a risk factor for mood and anxiety disorders.

| Strategy | Definition |
| --- | --- |
| Adaptive CER strategies  |  |
| Cognitive reappraisal | Re-evaluating an event as either more positive or less negative. |
| Refocus on planning | Thinking about the next steps and how to handle an event. |
| Positive refocusing | Turning thoughts towards joyful and pleasant matters.  |
| Putting into perspective | Downregulating the seriousness of an event and comparing it to other events. |
| Acceptance | Being aware of and accepting emotions without feeling the need to change them. |
| Distraction | Deployment of attention away from emotionally salient matters.  |
| Maladaptive CER strategies  |  |
| Rumination | Tendency to dwell on the negative feelings or thoughts associated with an event. |
| Self-blame | Blaming oneself for what has been experienced. |
| Other-blame | Blaming others for what has been experienced. |
| Catastrophising | Overemphasising the negative parts of an event. |
| Suppression  | Concealing expressions of internal states.  |

**Table 1.** Definitions of cognitive emotion regulation strategies.

Research indicates that poor sleep disrupts people’s ability to successfully use adaptive CER strategies, such as cognitive reappraisal. Sleep loss has been shown to impair the deployment of cognitive reappraisal in laboratory settings, where participants are explicitly instructed or encouraged to use this strategy in response to experimental stressors (Stenson et al., 2021; Tamm et al., 2019). For instance, Mauss et al. (2013) found that poor sleep quality weakened participants’ ability to reduce self-reported sadness when asked to reinterpret a negative picture as emotionally neutral. Similarly, Zhang et al. (2019) examined LPP amplitudes after a night of sleep or total sleep deprivation. Participants viewed negative images under two conditions: simply maintaining their emotional response (negative-maintain) or actively attempting to reframe the image more positively (negative-reappraise). In the sleep control group, LPP amplitudes were significantly smaller in the negative-reappraise condition compared to the negative-maintain condition, consistent with successful emotion regulation via reappraisal. However, in the sleep-deprived group, there was no significant difference in LPP amplitudes between negative-reappraise and negative-maintain conditions, suggesting unsuccessful regulation of stimuli-elicited negative emotions via reappraisal following sleep deprivation.

However, in Zhang et al. (2019), the effects of sleep deprivation on cognitive reappraisal were not reflected in subjective valence and arousal ratings, suggesting that objective neural measures (e.g., ERPs) may be more reliable indicators of emotion regulation success. Other studies have also reported null effects of sleep deprivation (Shermohammed et al., 2020), partial sleep deprivation (i.e., limiting sleep to 4 hours; Reddy et al., 2017), and sleep fragmentation (i.e., frequently awakening participants for short intervals during the night; Boon et al., 2023) on cognitive reappraisal ability assessed via subjective ratings. Likewise, subjective assessments of other adaptive CER strategies, such as distraction and acceptance, have not shown support for an effect of sleep loss (Boon et al., 2023; Zhang et al., 2019).

These conflicting findings may stem from the artificial nature of laboratory tasks, which often fail to reflect the salience of real-world experiences. In daily life, emotional events often arise unexpectedly and evolve over time, whereas in the laboratory settings, affective stressors (e.g., aversive images or videos) are presented only briefly. Moreover, participants in laboratory studies are often instructed in how to deploy reappraisal techniques, whereas people in the real world must regulate their emotions without explicit guidance. Hence, while laboratory research provides valuable insights into how poor sleep influences cognitive reappraisal, examining strategy use in real-world contexts is essential for a more comprehensive understanding.

 Evidence is also mixed as to whether poor sleep reduces the frequency with which adaptive CER strategies are deployed (in addition to diminishing the effectiveness of such strategies, as described above). For instance, Boon et al. (2023) reported an increase in the use of distraction after fragmented sleep, but no effect on cognitive reappraisal or acceptance. Parsons et al. (2021), however, found that high-quality sleep was associated with a greater use of emotion-focused (e.g., appreciating the moment), self-focused (e.g., feeling proud) and dampening strategies (e.g., believing negative feelings will pass). In our recent work, poor sleep quality was associated with reduced adaptive strategy use based on a combined index of adaptive strategies (Sullivan et al., 2023).

By contrast, evidence appears more consistent with regard to the association between poor sleep and increased use of maladaptive CER strategies. In their systematic review and meta-analysis, Clancy et al. (2020) found a small effect for the association between rumination use and shorter sleep duration, and a medium effect for the association between rumination and poor sleep quality. Furthermore, Boon et al. (2023) found that participants reported higher use of rumination after fragmented sleep, as compared to normal sleep. Our recent work also showed that poor quality sleep is associated with increased use of maladaptive strategies including rumination, self-blame, other-blame and catastrophising (based on a composite measure; Sullivan et al., 2023). These findings suggest that poor sleep may be a more reliable indicator of heightened maladaptive CER strategy use than of reduced adaptive CER strategy use.

Cognitive reappraisal is a demanding process that relies on a frontoparietal control network to maintain emotional goals, together with anterior cingulate and ventrolateral prefrontal inputs that support performance monitoring and inhibition, respectively (Ochsner et al., 2012). Given that sleep deprivation is known to disrupt activity in key regions of this reappraisal network (Drummond et al., 1999; Krause et al., 2017; Mograss et al., 2009; Nilsson et al., 2005; Skurvydas et al., 2020) and impair top-down inhibition of amygdala (Ben Simon et al., 2020; Yoo et al., 2007), it is perhaps unsurprising that adaptive strategies are impaired by sleep loss. Along the same lines, Minkel et al. (2012) found that poor quality sleep, as measured by sleep medication use, was associated with reduced activation in the dorsomedial and dorsolateral prefrontal cortex during cognitive reappraisal, highlighting how sleep problems undermine the higher-order cognitive processes on which emotional control relies.

In summary, sleep disturbances appear to disrupt people’s ability to deploy adaptive CER strategies like cognitive reappraisal, although there are some inconsistencies among studies employing self-report measures. However, much of this work was performed in unnaturalistic laboratory settings, where participants were explicitly instructed to use specific CER strategies and/or respond to artificial stressors. By contrast, studies examining CER strategy use in real world contexts have found that poor quality sleep is consistently associated with an increased use of maladaptive CER strategies, and more inconsistently associated with decreased use of adaptive CER strategies. Complementing these behavioural findings, neuroimaging research has shown that sleep loss reduces prefrontal engagement during cognitive reappraisal, disrupting the executive functions necessary to maintain emotional control.

# Emotional inertia

Emotional inertia refers to the persistence of affective states over time (Koval et al., 2016; Kuppens et al., 2010). It is typically operationalised by regressing a person’s emotion state at a given timepoint (*t*) onto their emotion state at the previous time point (*t* − 1), with more positive slopes indicating more persistent affect, or higher inertia (Koval et al., 2021).

High emotional inertia reflects emotional rigidity, such that affective experiences remain stable over time. This high self-predictability suggests that intermediate events and internal regulatory efforts have had little impact on one’s emotion state, potentially reflecting an affective system that has become inflexible (Koval & Kuppens, 2024). In contrast, low emotional inertia suggests emotional flexibility—the capacity to adjust affective responses in line with environmental demands and to effectively return to baseline following perturbations through regulatory efforts (Koval & Kuppens, 2024). Consistent with this, high emotional inertia, particularly of negative emotions, has been robustly associated with depressive (Kuppens et al., 2010, 2012; Minaeva et al., 2021) and anxiety disorders (Bosley et al., 2019; Gilbert et al., 2019; Seidl et al., 2023), as well as neurotic traits (Koval et al., 2016; Suls et al., 1998; Waugh et al., 2017).

Mechanistically, emotional inertia is thought to arise from reduced responsiveness to both internal (e.g., regulatory efforts) and external inputs (e.g., changes in the environment). Theoretically, high emotional inertia may reflect impaired emotion regulation skills, including a diminished ability to deploy adaptive regulatory strategies in the wake of emotionally challenging events (Kuppens et al., 2010). Supporting this view, previous research has identified alterations or impairments in emotion regulation processes as a key mechanism underlying heightened emotional inertia (Koval, Brose, et al., 2015; Koval, Butler, et al., 2015).

Only a handful of studies have investigated the association between sleep and emotional inertia. Wen et al. (2020) used experience sampling to assess negative and positive affect at regular intervals throughout a seven-day study period and found that shorter sleep duration was associated with higher inertia of depressive mood. However, two other studies found no significant associations between sleep duration or quality and the persistence of negative emotion states (Frérart et al., 2023; Minaeva et al., 2021).

Such contrasting findings may be due to methodological differences between these studies. Whereas Wen et al. (2020) measured sleep with actigraphy, Minaeva et al. (2021) and Frérart et al. (2023) relied on self-report measures. Additionally, while Wen et al. (2020) measured emotional affect at five time points throughout the day, Minaeva et al. (2021) and Frérart et al. (2023) focused on changes in affect from evening to morning, which may have been influenced by circadian effects. Indeed, prior work has shown that evening types experience delayed peaks in positive affect, and generally report lower positive affect than morning-types on working days relative to non-working days (Miller et al., 2015). Therefore, evening types may indicate higher overnight inertia of negative emotions than morning types because they experience lower positive affect in the morning.

Although experience-sampling methodologies capture emotional responses in daily life, they provide no insight into the events that precipitate an emotional reaction (Koval et al., 2013; Kuppens et al., 2022). This prevents researchers from determining whether individual differences in emotional inertia are the result of environmental factors or internal differences in emotional regulation (Koole, 2009; Koval et al., 2013). To address this, other work has used mood-induction methods that expose participants to a fixed sequence of emotionally salient events in the laboratory, and thus controls for the events that participants experience within a pre-determined timescale. Participants are typically instructed to rate how they feel on various affect dimensions after viewing several film clips and again following an interval between each clip (Koval et al., 2013, 2016). Studies using this mood-induction paradigm have linked negative emotional inertia to depressive symptoms and rumination (Koval, Brose, et al., 2015; Koval et al., 2013, 2016).

We employed the mood-induction paradigm in recent work to examine the link between sleep quality and negative emotional inertia, using a composite score of negative affect that included sadness, depression, anxiety, and anger (Sullivan et al., 2024). Better self-reported sleep quality was associated with less persistent negative emotions, suggesting that high-quality sleep may help protect against mental health conditions by promoting faster recovery from negative emotion states.

The effect of sleep loss on emotional inertia likely arises from a breakdown of prefrontal regulatory control. Related to this idea, Waugh et al. (2017) measured changes in cerebral blood flow while participants viewed and rated the intensity of emotions elicited by self-relevant statements. They used these neural changes to predict emotional inertia in response to daily life events across seven days. Individuals exhibiting increased activation in the lateral prefrontal cortex−suggesting greater engagement of emotion-regulatory circuitry−showed lower positive and negative emotional inertia in their daily experiences, suggesting that lateral prefrontal engagement may support the inhibition of emotions arising from self-relevant statements (Waugh et al., 2017). Given that sleep loss reduces prefrontal engagement (Ben Simon et al., 2020; Yoo et al., 2007), these findings offer a plausible mechanism through which poor sleep may increase emotional inertia in daily life.

Building on these findings, Provenzano et al. (2018) examined how neural activation in response to positive and negative social feedback from peers during a laboratory task related to emotional inertia in daily life over two weeks. They found that greater activity in the right parahippocampal gyrus and the right lateral orbitofrontal cortex in response to negative feedback was associated with higher inertia of negative emotions, suggesting that increased activity in these regions may reflect impaired regulatory processing during socio-emotional experiences, leading to more persistent affective responses over time. Aberrant activation in these regions under conditions of sleep loss (Krause et al., 2017; Mullin et al., 2013) may likewise heighten the persistence of negative emotions.

To summarise, relatively few studies have investigated the relationship between sleep and emotional inertia, with experience-sampling protocols producing inconsistent results, potentially due to a lack of control over environmental factors. The use of the mood-induction procedure can control for this issue and demonstrates that high-quality sleep is associated with reduced negative affect inertia. Given that difficulties in top-down prefrontal control may underlie emotional inertia, sleep-related disruptions in affect regulation might also be a mechanism that gives rise to persistent emotional states.

# Future Directions

Emotion processing mechanisms do not operate in isolation but dynamically interact with one another. For example, heightened emotional reactivity is associated with increased use of maladaptive regulatory strategies (Kozubal et al., 2023), creating a feedback loop where ruminative thought patterns lead to increased negative emotional inertia (Blanke et al., 2022; Koval et al., 2012; Sullivan et al., 2024).

This dynamic interplay is further influenced by sleep quality. For instance, sleep deprivation increases emotional reactivity by amplifying sensitivity to both negative and neutral stimuli (Goldstein-Piekarski et al., 2015; Reddy et al., 2017; Tempesta et al., 2015) while simultaneously impairing prefrontal control processes necessary for adaptive cognitive emotion regulation strategies, such as cognitive reappraisal (Minkel et al., 2012; Ochsner et al., 2012). This impairment may increase the tendency to engage in maladaptive, ruminative thought patterns that contribute to a heightened state of negative emotional inertia (Koval, Brose, et al., 2015; Koval, Butler, et al., 2015).

These observations coalesce on a need to better understand the multifaceted relationships between sleep and affect. Future research should adopt integrative approaches, combining experience sampling with neuroimaging and/or psychophysiology, to better capture how sleep influences emotion processing. Longitudinal designs are particularly valuable for clarifying how sleep disruptions give rise to maladaptive cycles marked by heightened emotional reactivity, impaired emotion regulation, and persistent negative affect.

It is equally important to understand how emotional dysregulation influences sleep. For instance, heightened emotional reactivity has been linked to longer sleep latency, fragmented sleep, and reductions in SWS and REM sleep duration (Fairholme & Manber, 2015; E. J. Kim & Dimsdale, 2007). Likewise, inducing ruminative thought patterns reduces the quality of subsequent sleep, particularly among individuals with high trait rumination (Guastella & Moulds, 2007). More comprehensive insights into these relationships are vital for delineating the bidirectional relationship between sleep and mental health.

Finally, while studies employing total sleep deprivation protocols have provided important insights into the impact of sleep loss on emotion processing, they represent an acute and controlled manipulation that differs from many sleep disturbances encountered in the real-world. Many real-world disruptions are chronic and are characterised by either partial or fragmented sleep loss. For example, insomnia is characterised by pre-sleep hyperarousal and sleep fragmentation, rather than total sleep loss. Similarly, shift workers experience chronic circadian misalignment and irregular sleep-wake schedules (James et al., 2017). Although some progress has been made, particularly through studies using partial sleep deprivation paradigms (Lustig et al., 2018; Reddy et al., 2017; Tempesta et al., 2020; van Leeuwen et al., 2018), future studies should incorporate more representative models of sleep disturbance, such as shift work, partial sleep deprivation, and fragmented sleep.

# Conclusions

The current review identifies several mechanisms through which sleep supports emotion processing, including emotional reactivity, cognitive emotion regulation and emotional inertia. Sleep deprivation amplifies emotional reactivity, making individuals more sensitive to negative stimuli and impairs emotional discrimination, heightening responses to neutral and non-threatening events. Furthermore, insufficient sleep weakens prefrontal control of emotions, negating the use adaptive CER strategies (e.g., cognitive reappraisal) and, correspondingly, increasing the use of maladaptive CER strategies (e.g., rumination). Additionally, sleep loss contributes to the persistence of negative emotion states over time, reinforcing the patterns of negative affect that underlie poor mental health. Collectively the findings from this review highlight how sleep dysfunction affects various overlapping components of emotion processing and, over time, increases the risk of psychiatric disturbance. Future research should focus on reciprocal interactions across these various facets of emotion processing to gain a more comprehensive understanding of sleep’s role in mental health.

# Credit authorship contribution statement

**Emma C. Sullivan**: Writing – original draft, Investigation, Conceptualisation. **Cade McCall**: Conceptualisation, Writing – Review & Editing.  **Lisa-Marie Henderson**: Conceptualisation, Writing – Review & Editing. **Scott A. Cairney:** Conceptualisation, Writing – Review & Editing, Writing – original draft, Supervision.

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Authors report no conflicts of interest.

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No data was used for the research described in the article.

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