Trends in Cognitive Sciences

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REVIEWS

Review

Sleep Loss and the Socio-Emotional Brain

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Are you feeling emotionally fragile, moody, unpredictable, even ungenerous to those around you? Here, we review how and why these phenomena can occur as a result of insufficient sleep. Sleep loss disrupts a broad spectrum of affective processes, from basic emotional operations (e.g., recognition, responsivity, expression), through to high-order, complex socio-emotional functioning (e.g., loneliness, helping behavior, abusive behavior, and charisma). Translational insights further emerge regarding the pervasive link between sleep disturbance and psychiatric conditions, including anxiety, depression, and suicidality. More generally, such findings raise concerns regarding society's mental (ill)health and the prevalence of insufficient and disrupted sleep.

Highlights

Sleep loss amplifies basic emotional reactivity, increasing negative mood states (e.g., anxiety, depression, suicidality), yet impairing the accurate recognition and outward expression of emotions.

Inadequate sleep further impacts higher-order, complex socio-emotional functioning, decreasing prosocial behaviors, increasing social withdrawal, triggering marital and workplace conflict, and enfeebling leadership skills.

The emotional dysfunction experienced by sleep-deprived individuals, such as loneliness or lack of work motivation, can be 'transmitted' to well-rested others who come in contact with an under-slept individual, reflecting viral contagion.

The underlying neural mechanisms include a loss of top-down prefrontal regulation of amygdala, aberrant cortical processing in the salience network, including insula and cingulate cortex, and sympathovagal changes in the body.

Consider a law-enforcement officer who irrationally reacts to a civilian, a nurse or doctor who responds without care to an inquiring patient or a boss who lashes out at a well-meaning employee. In these examples, improper socio-emotional behaviors are often metered out by under-slept individuals. Why is this?

Over the past years, a newly emerging field of sleep science has begun to examine how emotional processes of the human brain, and their consequential decisions and actions, become compromised when sleep gets short. The results, which we examine in this review, map out a sleep-deficient emotional phenotype that is remarkably diverse. Specifically, this profile of affective dysfunction is: (i) expressed at levels of basic emotional processing (e.g., recognition, responsivity, expression); (ii) similarly observed in more complex emotional states and during social interactions (e.g., loneliness, helping behavior, and abusive behavior); (iii) common across numerous clinical conditions (e.g., suicidality, depression, anxiety, addiction disorders); and (iv) robustly observed across different forms of sleep loss [e.g., **total sleep deprivation** (see Glossary), **sleep restriction**, and disrupted **sleep quality**].

While the current review concentrates on sleep loss, sleep and specific sleep stages/physiologies, it is important to note that circadian rhythms and circadian disruption also play a significant role in the regulation of emotional and mood states, as well as in several psychiatric disorders. These circadian effects on affective function have been reviewed in detail elsewhere [1–4].

Sleep Loss and Basic Emotional Function

At a fundamental level, sleep loss impacts basic affective processes of: (i) emotional reactivity, (ii) emotion evaluation, and (iii) emotion expression [5–9].

Sleep and Emotional Reactivity

Reacting to an emotional stimulus involves changes in physiology, subjective experience and consequential behavior. All are modulated by insufficient sleep [7,8].

Regarding subjective experience, sleep deprivation is associated with greater emotional volatility and irritability, together with lower positive mood [10–13]. Similar changes in mood states have been reported when sleep is restricted to less than 7 hours a night for several nights [14–17]. These affective state changes extend into clinical domains, where **disrupted sleep** increases

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depression and anxiety (Box 1) [18], as well as suicidal thoughts, suicide attempts, and tragically, suicide completion (Box 2) [19].

Teens appear to be as, if not more, susceptible to the consequences of inadequate sleep [20]. Similar mood changes are observed in adolescents limited to 6.5 hours across 5 nights or 4 hours of sleep for only 1 night, resulting in greater levels of anxiety, anger, confusion, irritability, and impaired emotion regulation [21,22]. This is also true of children aged 8–12 years following 1 hour of sleep reduction across 4 nights [23].

Interestingly, the impact of sleep loss on affect typically involves a greater reduction in positive mood relative to an increase in negative mood [14,24,25]. This dampening of positive mood (more than elevated negative mood) has been linked to the loss of **non-rapid eye movement (NREM)** slow-wave sleep [24,25]. Such findings offer clinical relevance in the context of mood disorders, particularly major depression, in which reductions in positive mood (anhedonia) and impairments in NREM slow-wave sleep co-occur (a topic we discuss in more detail in Box 1).

It is important to recognize that acute sleep deprivation and even partial sleep restriction can evoke stress associated with prolonged awakening, making interpretation of such data less clear (i.e., are effects due to the absence of sleep, the stress associated with staying awake, or some combination of the two?). Several approaches have been taken to address this issue.

Box 1. Sleep and Depression

Inadequate or disrupted sleep is a reliable symptom of, and risk factor for, bipolar and unipolar (major) depression [18]. At least 65% of patients suffering from major depression complain of disrupted sleep, including early morning awakenings and difficulties in initiating and/or maintaining sleep [142]. As a robust phenomenon, sleep disturbance forms an integral part of the diagnostic criteria for major depression. More than sleep disruption, the actual architecture of sleep stages across the night appears to be altered in major depression. This includes a faster entry into the first REM sleep episode, as well as an overall increase in the amount of REM sleep [145].

The strong comorbidity of disturbed sleep and major depression has raised the question of directionality: is sleep disruption a consequence of depression, or a contributing factor instigating the disorder? [18] Though evidence suggests both are likely true, a meta-analysis of 18 prospective epidemiologic studies across more than 3000 participants found that individuals with insomnia had a twofold greater risk for subsequent development of depression, relative to those without such sleep difficulties [146]. Similarly, individuals suffering sleep disruption as a result of obstructive sleep apnea have a twofold greater risk for future development of a depressive disorder a year later, a risk that was especially strong among women [147].

The question of directionality is also relevant in cases linking depression to dementia. Both anxiety and depression have been associated with higher risk of dementia in older age [148,149]. Independently, disturbed sleep, including insomnia and sleep apnea, significantly increases the risk of developing dementia later in life [150]. Given the high comorbidity of disturbed sleep in depression, it is possible (but to our knowledge, untested) that sleep disturbances represent one mediating mechanistic pathway through which mood disorders increase dementia risk.

Sleep deprivation can, however, offer a paradoxical antidepressant effect in approximately 45% of patients in randomized controlled studies [151]. Unfortunately, this benefit is transient in that more than 80% of these patients relapse back into depression following recovery sleep [152]. Possible mechanisms underlying this antidepressant sleep-loss benefit have included the normalization of default mode network hyperconnectivity [53], increases in striatal reward brain activity [58], as well as changes to serotonergic and dopaminergic tone following sleep loss [152,153].

Related, the suppression of REM sleep is a common feature of several antidepressant drugs, suggesting that manipulations of REM sleep may play a role in this antidepressant effect. Such a thesis is fitting with the above findings of a faster entry into REM sleep and greater amounts of REM sleep in depressed patients, which may be normalized by antidepressant medications. Alternatively, REM sleep abnormalities might not be depressogenic in their own right, but rather, indicate impairments in the homeostatic control of NREM sleep. That is, the underexpression of NREM slow-wave sleep may lead to a consequential overexpression of REM sleep and allow a faster entry into REM early in the night [152]. Fitting this possibility, individuals with major depression show diminished NREM slow-wave sleep and associated SWA [145].

Glossary

Cognitive behavioral therapy for insomnia (CBT-I): an evidence-based treatment of insomnia that consists of a multicomponent intervention designed to target the behavioral and cognitive underpinnings of insomnia.
Disrupted sleep: abnormal sleep that can be described in measures of deficient sleep quantity, structure (e.g., sleep-cycle architecture), and/or sleep quality [e.g., spectral electroencephalogram (EEG) power].
Functional connectivity: the statistical association between the fMRI time series of blood-oxygen-level-dependent signal in two or more anatomically distinct brain regions.
Impulsivity: acting without sufficient deliberation.
Non-rapid eye movement (NREM) sleep: a type of sleep that consists of sleep stages N1–3 (previously NREM 1–4). Each NREM sleep stage has distinct (electro)physiological characteristics. High amplitude, slow-frequency synchronized EEG oscillations predominate in stage N3 (previously NREM 3 + 4, also known as slow-wave sleep, or deep sleep), and reflecta homeostatic sleep process.
Rapid eye movement (REM) sleep: a unique phase of sleep characterized by high-frequency, low-amplitude desynchronized EEG oscillations, rapid eye movements, muscle paralysis, and vivid dreaming.
Sleep procrastination: going to bed later than intended due to self-choice.
Sleep quality: a metric of sleep evaluation often gauged from either subjective self-report (e.g., how satisfying and refreshing sleep was) or objective sleep features (e.g., sleep stage transitions, night-time awakenings/arousals, sleep stage amounts, and EEG sleep physiology).
Sleep restriction: the reduction (but not total absence) of sleep in the prior night or nights, usually ranging from 1 to 6 hours of sleep reduction, relative to the norm of 8 hours. Typically, sleep restriction is chronic if it persists for more than 24 hours.
Slow-wave activity (SWA): an electrophysiological signature of slow (typically 0.5–4.0 Hz), synchronized, oscillatory neocortical activity. SWA is maximally expressed during NREM sleep and intensifies as a function of prior wake duration.

Box 2. Disrupted Sleep and Suicidality

Sleep and suicide are intimately linked. Poor sleep quantity, quality, insomnia severity, and nightmares predict elevated risk for suicide ideation, suicide attempts, and suicide completion (for review, see [19]). Indeed, meta-analysis data indicate that sleep disturbance is associated with a 1.95 to 2.95 increased relative risk for suicide and suicide-related behaviors, including suicidal ideation and suicide attempts [154]. Importantly, these associations remain robust when accounting for anxiety and depression, as well as drug and alcohol use [154,155]. Such links with sleep disruption have now been observed in nonclinical and clinical populations [154], and across diverse age ranges (adolescent teens [156,157], early-midlife adults [158], and older-age adults [158,159]).

Interestingly, the strength of the relationship between suicidal behavior and nightmares exceeds those reported for disrupted sleep, with an odds ratio risk of 8.2 for nightmares [160]. A similar association has been observed during the longitudinal tracking of healthy individuals across 20 years, wherein the risk for suicide completion was twofold higher in those with frequent nightmares, compared with individuals without nightmares [161]. This may indicate the possibility of a dream-related 'mental biomarker' of suicide likelihood. Mechanistically, it has been postulated that nightmares reflect a failure to downregulate the emotional content of dreams, consequently increasing negative mood upon waking [162] and/or involve the overexpression of REM sleep physiology itself [163].

Although alterations in sleep are significantly associated with suicidal behavior, sleep disturbance appears to be a transdiagnostic feature common across many psychiatric disorders. Noted above, this includes depression (Box 1), affective mood disorders, anxiety disorders, autistic disorder, and schizophrenia [18,72,145]. A common binding relationship between disorders of the mind and disordered sleep therefore exists, the underlying physiology of which remains largely unclear.

Total sleep deprivation: the complete absence of sleep in the prior night or nights.

Viscerosensory: in humans, relating to corporeal signals communicated by the spinal cord to brain regions that enable homeostatic coordination of bodily functions and associated behaviors.

First, animal models that obviate physiological stress responses by way of adrenalectomy, still report significant impairments in emotional behavior following sleep deprivation, as do paradigms involving gentle handling that co-opt a milder implementation of sleep deprivation [26,27]. Second, prospective longitudinal studies in humans have demonstrated that even small, natural night-to-night variations in the quality of sleep are nevertheless associated with a significant worsening of next-day affective measures, including pain, anxiety as well as social withdrawal [28–30]. Third, and counter to the manipulation of acute sleep deprivation, the opposite causal approach of increasing sleep beyond habitual amounts, that is, sleep extension, can reduce measures of depression, negative mood, and emotional lability. Such effects have been observed in children [31], adolescents [32] and healthy adults [33]. Furthermore, sleep improvement by way of **cognitive behavioral therapy for insomnia (CBT-I)** is associated with corresponding decreases in both anxiety and depression [34]. These sleep extension findings represent a helpful adjunct to sleep deprivation studies as they indicate a causal sleep-manipulation that similarly results in socioemotional changes, but does not involve concerns associated with stress linked to experimental sleep loss.

In addition to the effects of decreased sleep quantity, reductions in sleep quality also impact emotional well-being. Experimentally induced sleep fragmentation and self-reported poor sleep satisfaction are both associated with greater anxiety [11], worse mood [24,25,35], and impaired emotion regulation [36].

The neural mechanisms underlying the impact of inadequate sleep on emotional function are becoming increasingly clear (Figure 1). Early work demonstrated that 1 night of sleep deprivation resulted in a 60% amplification of amygdala reactivity to aversive images, relative to a full night of sleep [37]. A similar profile of exaggerated amygdala response has been reported following partial sleep restriction (4 hours of sleep per night for 5 nights [15]), and in those reporting poor sleep quality or short sleep duration [38,39]. Amplified amygdala responsivity following insufficient sleep is evident even if the emotional stimuli is not consciously perceived or attended to [40–42]. This would suggest a physiological (brain) impact of sleep loss on emotional reactivity that can be independent of conscious stimulus perception.

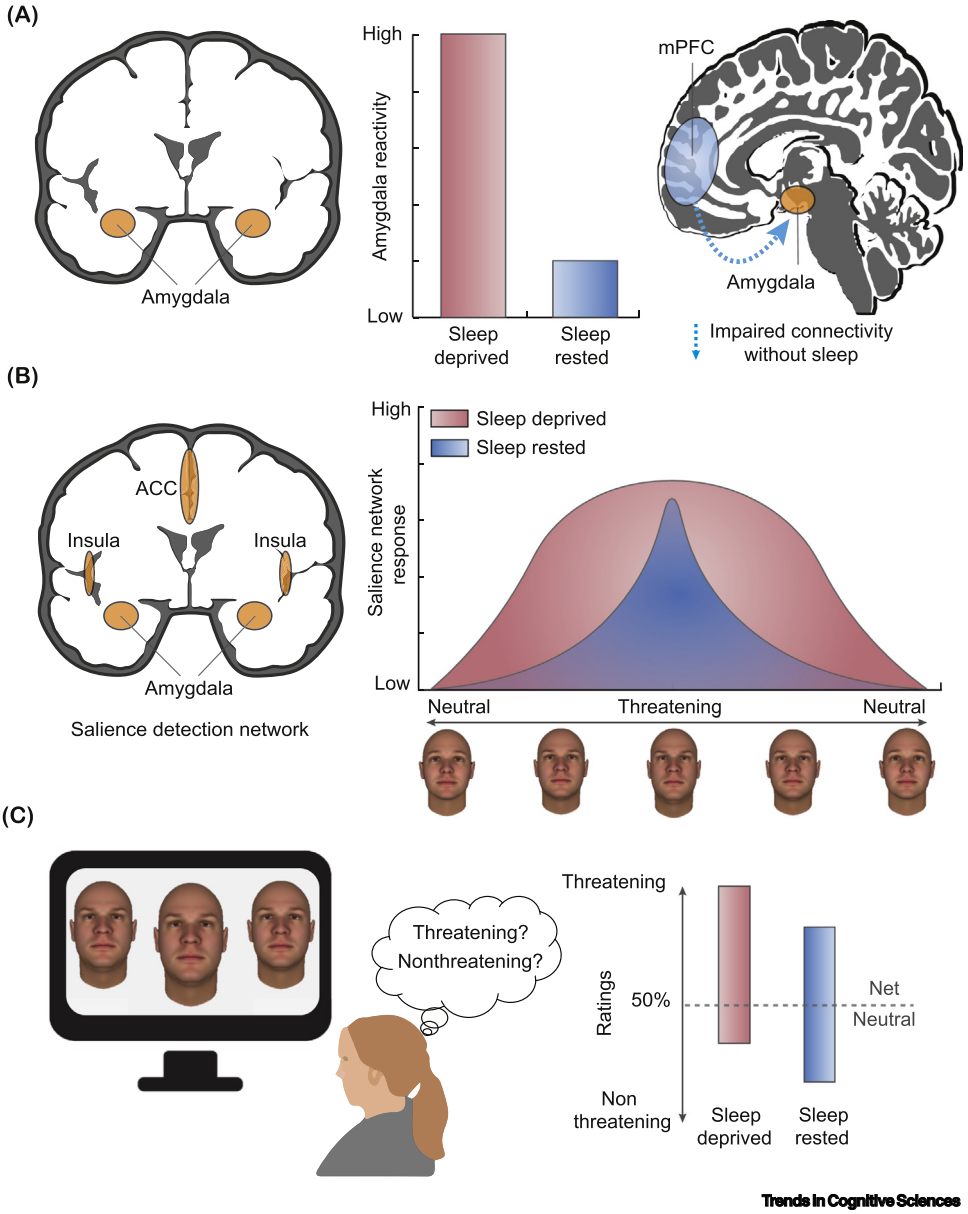

Figure 1. Sleep Loss and Basic Emotional Reactivity. (A) Sleep deprivation (red) amplifies emotional amygdala reactivity yet decreases associated medial prefrontal cortex (mPFC)-amygdala functional connectivity (broken line; modified from [37]), (B) Sleep deprivation alters emotional-stimulus mapping within the salience detection network [orange regions, left panel: amygdala, anterior cingulate cortex (ACC), and anterior insula] when processing aversive emotional signals (e.g., facial expressions). Under sleep-rested conditions (right panel, blue curve), a well-tuned range of detection sensitivity enables to accurately discriminate between degrees of emotional salience. However, sleep loss (red curve) triggers a saturated activity profile in the salience detection network. As a result, the ability to discriminate between degrees of emotional salience is impaired, including the ability to discern threatening from non-threatening stimuli. (C) A downstream behavioral consequence of the above central brain changes is a loss of 'net neutrality' in sleep-deprived participants. This is expressed as a negative perceptual-response bias (right panel) wherein individuals disproportionately weight otherwise neutral stimuli as aversive, resulting in a threat-dominant imbalance, rather than a net neutral balance present under sleep-rested conditions.

Mirroring limbic changes in the brain, 1 night of sleep loss increases peripheral sympathetic nervous system activity in response to emotional stimuli, relative to a sleep rested night [43–45]. Such commonality is fitting with a model of limbic hyperarousal associated with elevated adrenergic tone in the brain and body, that takes into account the added contribution of hypothalamic pituitary adrenal (HPA) axis changes [13,46,47].

One factor instigating limbic hypersensitivity appears to be lower negative coupling between the medial prefrontal cortex (mPFC) and the amygdala following sleep deprivation [7,48]. Impaired **functional connectivity** between control regions of the mPFC and amygdala have been observed following 1 night of total sleep deprivation, 4 hours of sleep restriction for 5 nights, and in those reporting less than 6 hours of nightly sleep [15,37,40,41,45,49]. Once again, this altered mPFC–amygdala connectivity signature due to insufficient sleep is not limited to adults but has been reported in under-slept children (ages 7–11 years) and adolescents (ages 13–18 years), with sleep duration measured using self-report [38,50].

When examining anatomical specificity in more detail, two subtly different impairments in amygdala–mPFC coupling can be observed as a result of sleep loss. First is a lessening of negative coupling between the amygdala and ventral regions of the mPFC (e.g., [33,49]). This is indicative of weaker top-down regulation of amygdala activity by the ventral mPFC. Second is a reduction in positive coupling between the amygdala and more dorsal regions of the mPFC (e.g., [41]), a regional interaction that typically supports emotional appraisal [51]. The latter would suggest that sleep loss compromises a dorsal mPFC–amygdala interaction necessary for the accurate evaluation of emotional experiences, a topic discussed in the next section. Independent of sleep, both mPFC communication failures are thought to be involved in depression and anxiety [51,52], highlighting the importance of this limbic circuit in the emotional impact of sleep loss.

Sleep-dependent changes to prefrontal connectivity are not specific to affective processing, however, having been reported in a variety of cognitive tasks not classically considered to be emotion-centric (see review [7]). Indeed, changes in PFC connectivity following sleep deprivation are observed in resting state fMRI data, independent of specific task engagement [53–55].Therefore, the signature of altered PFC function under conditions of sleep loss may be domain general, including robust deficits in the affective domain, but also beyond.

Changes in affective brain reactivity caused by a lack of sleep further unfold during the anticipation of an emotional experience in regions of the amygdala, anterior insula, and anterior cingulate cortex (ACC) [12]. Parenthetically, a similar neural profile is seen in clinical anxiety disorders that entail excessive anticipatory worry in combination with disrupted sleep. Linking these findings, 1 night of sleep loss in healthy individuals causally increases levels of anxiety to those observed in anxiety disorders, mirrored by a neural signature that is common in clinical anxiety, including reductions in mPFC activity and connectivity, yet hyperactivity in dorsal ACC and the amygdala in response to emotional stimuli [29].

Though the majority of studies to date have focused on aversive stimulus processing, excessive neural reactivity following sleep loss is not limited to one side of the emotional spectrum. Amplified limbic and striatal brain reactivity has been observed in response to rewarding stimuli following sleep deprivation [56–58]. This would indicate that the sleep-

deprived brain is not unidirectional in its change in emotional sensitivity. Rather, sleep loss leads to bi-directional alterations in affective brain reactivity: more extreme reactivity on both sides of the valence spectrum, positive and negative. We return to this theme in more detail in the following section.

Sleep and Emotion Evaluation or Expression

Beyond basic stimulus–response paradigms, more recent work has revealed an additional characteristic of the sleep-deprived human brain: impaired emotional evaluation. Sleep-rested individuals are able to accurately differentiate stimuli across an increased gradient of emotional strength. By contrast, these same participants when sleep deprived demonstrate a saturated and thus more inaccurate discrimination of emotions [59,60], especially when these emotional variations are subtle [61].

Safety detection appears to be especially impacted by sleep loss, degrading the ability to accurately decern threatening from affiliative facial expressions [46]. As a result, sleep loss leads to a biased perception of increased negative threat [61–63]. Similar findings have been reported following selective **rapid eye movement (REM) sleep** deprivation rather than total sleep deprivation, suggesting a role for REM sleep in preserving high-resolution emotional mapping (see Outstanding Questions) [64]. Negative emotional bias has similarly been observed in adults after 3 weeks of in-laboratory partial sleep restriction (4 hours of sleep per night for 5 nights) [65], as well as in adults and adolescents suffering from poor sleep quality, as assessed using self-report and actigraphy measurements [66,67].

Contributing to this prejudicial aversive sensitivity may be attentional bias. Sleep loss decreases the time participants spend attending to positive stimuli [25], while increasing that afforded to negative stimuli [68–70]. This may, in part, explain why those reporting poor quality sleep evaluate daily life events as more negative, relative to those that report better sleep quality [71].

Such findings would suggest that a sleep-loss bias toward heightened emotional sensitivity is maladaptive. However, a counter-argument can be made. Considering that the organism is in a compromised cognitive and physiological-body state when sleep deprived, shifting to a conservative (threat-overestimation) bias in emotional decision making may be adaptive, aiming to risk-mitigate sleep-loss-related vulnerabilities. Disambiguating between these two possibilities has not yet been made.

Returning to a maladaptive thesis, and considering that anxiety is a condition highly comorbid with sleep disruption [72], it is of clinical remark that high-anxious individuals display a very similar bias in emotional response profile leading to a failure to discriminate threat from safety signals [73]. This leads to the testable prediction that changes to threat sensitivity in clinical anxiety disorders may, in part, be mediated by inadequate sleep.

With respect to explanatory neural mechanisms, sleep-deprived individuals fail to show a normal graded emotional sensitivity in the anterior insula, ACC, and subcortical amygdala regions, relative to the same individuals following a full night of sleep [41,46], instead expressing a saturated (i.e., higher) emotional response profile [41,46,74]. This excessive and overly generalized neural activity may reflect a loss of 'net-neutrality' due to a narrowing of one's innate affective set point (Figure 1B). In this biased state, what may otherwise be regarded as a neutral stimulus is inaccurately registered as more emotionally negative by sleep-deprived participants. The normal

graded ability for accurate emotional brain discrimination is therefore replaced by a threat-perception bias (Figure 1C). Interestingly, a similar shift has been reported in the context of positive rewarding stimuli, leading to riskier and more hedonic-biased choices, discussed in detail in the section on complex decision making.

Of further mechanistic note, increased emotional reactivity following sleep loss still occurs in paradigms when the emotional stimulus itself is not consciously perceived [40–42]. This would once again suggest that a lack of sleep can amplifiy emotional responsivity independent of emotion perception, though both processes may still interact when conscious perception occurs.

In contrast to increases in emotional brain reactivity, sleep loss can blunt the downstream outward expression of emotions. This effect of decreased emotional expression has been observed in the face, in the voice [75–77], and verbally, as indexed by a reduction in the use of positive emotional words [78]. A similar blunting of emotional expressiveness is evident in infants who have not obtained a typical daytime nap [79], resulting in a 34% reduction in positive emotional face expressions, relative to infants that did nap.

In summary, a lack of sufficient sleep impairs the sensitive neural mapping and disambiguation of emotional stimulus differences, consistent with an affective state of poor signal-to-noise processing. Finally, sleep loss hinders the outward expression of emotions, that are nevertheless internally registered, and registered to a greater degree.

Sleep Loss and Complex Socio-Emotional Functioning

Higher-order socio-emotional functioning also suffers when sleep is insufficient, including aspects of prosocial behaviors, interpersonal and workplace relationships, as well as complex reward-based decision making.

Social Interactions, Interpersonal and Workplace Relationships

Poor sleep quality is associated with impaired understanding of relationship concerns between romantic partners. As a result, under-slept partners have greater interpersonal conflict [80], higher levels of marital aggression [81,82], and lower overall marital satisfaction [83,84]. Interpersonal conflicts are also evident in children and teens, where measures of poor sleep quality (subjective and objective) predict increases in conduct problems, violent behavior, disagreement with peers, and greater levels of hyperactivity [21,85–87].

Changes in emotional state within an under-slept individual may even be ‘transmitted’ to well-rested individuals. For example, independent judges who have viewed videos of sleep-deprived participants report feeling lonelier themselves, relative to how they felt after viewing the same participants in a sleep-rested state [28]. Furthermore, these judges consequently indicate that they would be significantly less likely to socially engage with sleep-deprived individuals [28,88]. Sleep deprivation may therefore be a state in which emotional contagion [89] is amplified. Due to such viral-like propagation, the ill-effects of a lack of sleep appear to spread to nearby social circles, affecting well-rested individuals that come into contact with a sleep-deprived other. Not only does this effect escalate the negative influence originating from the under-slept individual, but in return, ostracizes that individual due to conspecifics perceiving them as less socially desirable [28,88]. Whether sleep-loss-related increases in emotional contagion extend to other affective domains, such as anxiety or depressed mood, is currently unknown.

Mood-state alterations caused by disrupted sleep (experimentally or naturally induced) also impact interpersonal behavior in the workplace [90]. For example, sleep loss predicts lower levels of job satisfaction [91], reduces the perceived charisma of others [92], and decreases the extent of helping behavior among colleagues [93]. In addition, under-slept employees express greater overall hostility [94] and ethically mistreat their colleagues, such as taking credit for someone else's work or blaming someone else for their own mistakes [93,95]. Establishing bi-directionality between adequate sleep and workplace behaviors, improving the sleep of employees through an insomnia treatment program is associated with decreases in several of these negative interpersonal behaviors [96].

Lack of sleep can be especially impactful in business leaders due to their top-down, hierarchical influence on employees (Figure 2). A single night of self-reported poor sleep in a workplace manager results in greater levels of abusive supervision imposed on his or her subordinates [97]. This may be due, in part, to a reduction in emotional self-control, with under-slept leaders inappropriately venting frustrations toward subordinates in a hostile manner [97].

The viral transmission of emotional dysfunction caused by sleep loss is also observed in a workplace context. Employees working under a leader suffering from insufficient sleep report being less engaged at work themselves, irrespective of their own quality of sleep [97,98]. Moreover, employees under the duress of an under-slept superior report a reduction in the quality of their working relationship with that leader, relative to nights when their superior had sufficient sleep [97,98]. The effect works both ways: sleep-deprived subordinates express more aggression toward their leaders, which undermines the reported

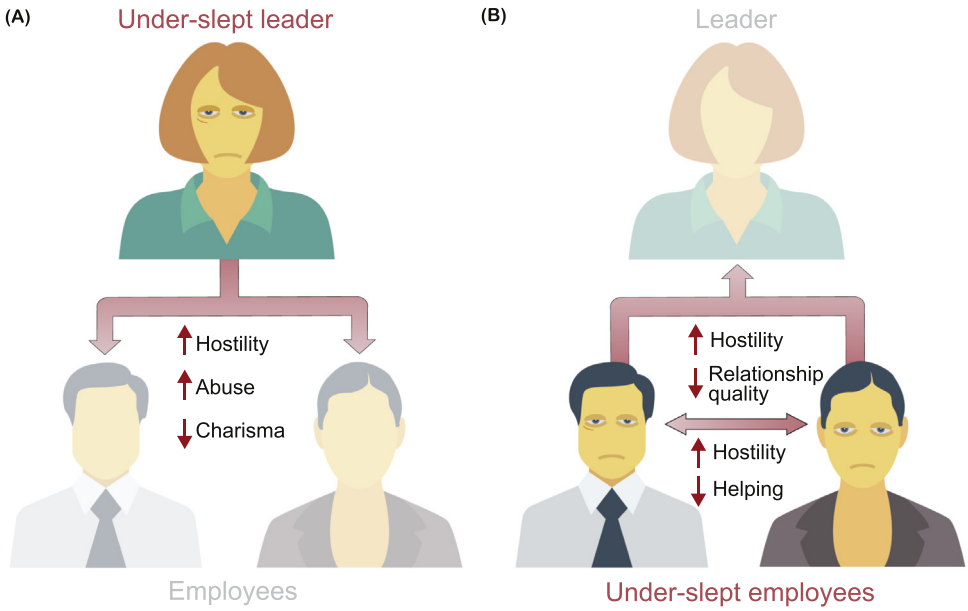

Figure 2. Insufficient Sleep Impacts Interpersonal Behavior in the Workplace. A single night of reported poor-quality sleep in a workplace manager results in greater levels of abusive supervision imposed on his or her employees. Furthermore, under-slept leaders are perceived as less charismatic and inspirational when communicating with subordinates (left panel). Similarly, under-slept employees are more hostile toward their colleagues and less likely to help each other in the workplace, further reflected in greater aggression toward leaders and overall lower quality of employee–leader relationships (right panel).

quality of the relationship by their workplace leader [98] (Figure 2). Interestingly, and perhaps concerningly, in both these contexts (employee and leader), the under-slept individual of focus was not aware of their expressed behavioral malpractice.

A lack of sleep has consequences that impact societal functioning, which move beyond interpersonal or work-place relationships (Figure 3). For example, sleep loss lowers the desire to socially

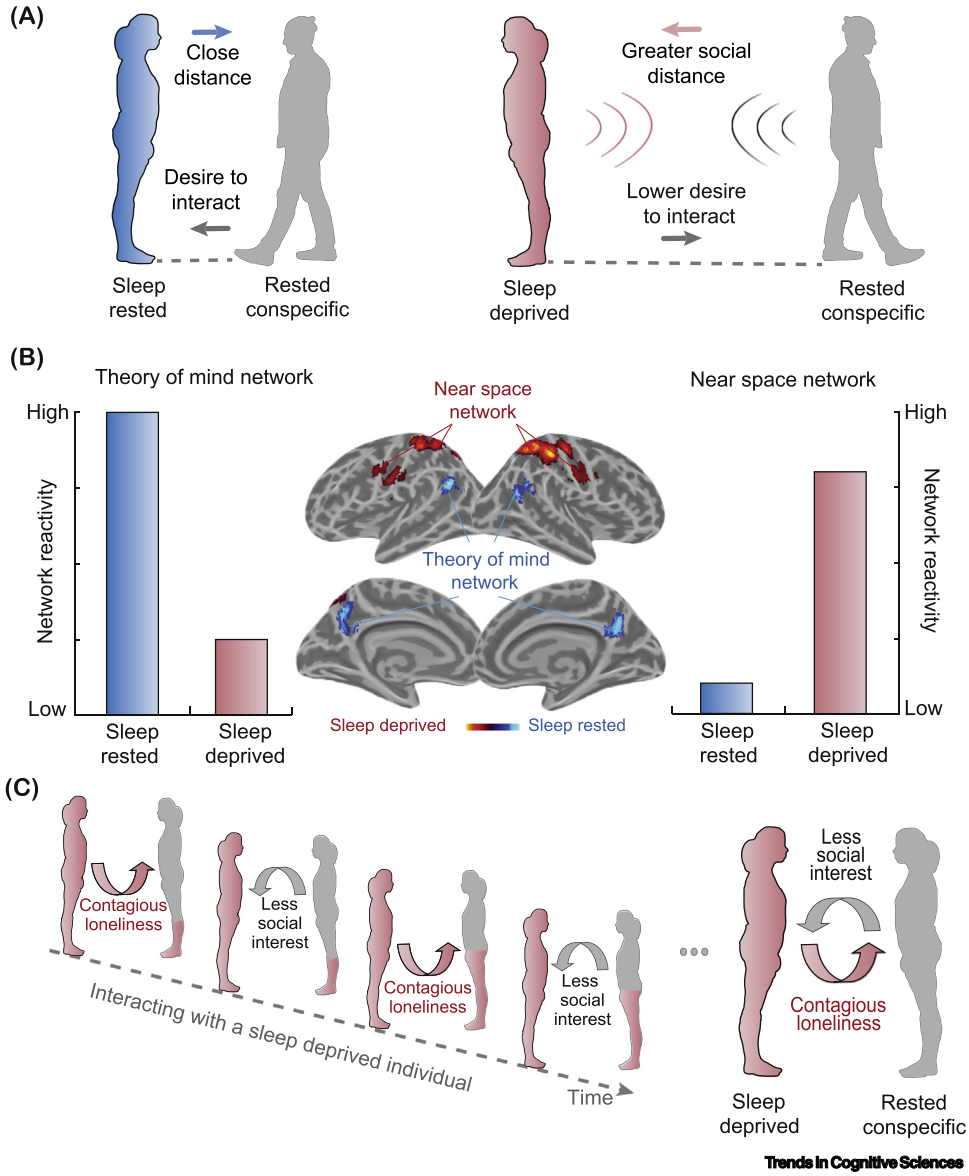

Figure 3. Sleep Loss and Increased Social Withdrawal. (A) Sleep deprivation increases the degree of physical social distance kept from others during a live social interaction (right panel; modified from [28]). (B) This asocial profile was further linked to amplified activity in regions of the near space network that regulate personal space (red, right bar graph), yet decreased activity in regions of the theory of mind network that encourage the understanding of others (blue; left bar graph; modified from [28]). (C) The mood-state changes experienced by sleep-deprived individuals (red) can be 'transmitted' to well-rested individuals (gray), such that feelings of loneliness originating from a sleep deprived individual (left panel) can increase feelings of loneliness in naive, well-rested conspecific (right panel).

interact with others [28] and increases the desire to be alone [28,99]. In addition, inadequate sleep degrades civic behaviors, including an individual's willingness to vote, to sign petitions, and donate to charities [100], potentially due to a reduction in the basic motivational drive to help others.

Candidate neural mechanisms are now emerging that may account for these varied and complex socio-emotional changes. One pertains to impaired prefrontal regulation of limbic activity following sleep deprivation leading to reduced impulse control [101], worse behavioral coping, and lower tolerance for frustration [102]. Such sleep-loss prefrontal disengagement is further implicated in impaired self-centered control necessary for outward prosocial altruistic behaviors [94,100], resulting in lower trust and raised levels of aggression toward others [103].

Recent neuroimaging studies of sleep loss and complex social behaviors have revealed alterations in two brain networks extending beyond basic subcortical limbic regions (Figure 3B): (i) the theory of mind (ToM) network, notably the temporal-parietal junction, mPFC, and precuneus, involved in comprehending the goals and motivations of others [104]; and (ii) the 'near-space' network, which regulates interpersonal space in both humans and primates (comprising regions of dorsal intraparietal sulcus and ventral premotor cortex [105]) .

Reductions in ToM network activity have been reported as sleep-deprived participants view other individuals approaching them on screen, relative to those same participants when well-rested. Disengagement of the ToM network may further explain why sleep-deprived individuals exhibit decreases in empathetic sensitivity [106,107] and fail to accurately understand the emotional experience of their romantic partner during conflict [80].

Conversely, total sleep deprivation amplifies responsivity within the near-space network that alerts of incoming social approach [28]. This increased sensitivity within the near-space network further predicts the degree to which sleep-deprived individuals distance themselves from another person in real-life interactions, suggesting a neural mechanism by which sleep loss promotes social withdrawal (Figure 3). Similar findings have been reported as a result of very subtle night-to-night reductions in sleep quality, such that individuals who report modest nightly decreases in sleep quality felt lonelier and spent less time interacting with others the following day [28]. In this regard, it is worth mentioning that social isolation and loneliness increase mortality risk by 45% and, similar to sleep loss, are both linked to a broad collection of physical and mental comorbidities, including suicidality (Box 2) [108].

Complex Decision Making

There is a collection of occupations where sleep deprivation is commonplace yet individuals are required to make reasoned, logical decisions in a timely manner (e.g., medical and military personnel, airline pilots [109]). This can result in flawed choices, several of which carry marked repercussions.

Under-slept individuals are more likely to abandon their own beliefs and rely on the advice of others in a social judgmental task [6,110]. Of societal relevance to the judicial system and circumstances of state torture [111], 1 night of sleep deprivation increases the likelihood of signing a false confession by more than fourfold, relative to a sleep-rested state [112]. Reductions in nightly sleep also change the severity with which wrongdoing is judged. For example, individuals allowed to sleep only 6 hours a night for 4 nights believed a surgeon should be more severely punished, and a patient more greatly compensated, for a medical error relative to the estimates of well-rested individuals [113].

Sleep loss impacts emotion-driven lifestyle choices, coopting unhealthy behaviors such as binge-drinking, stimulants use, and snacking after dinner [114]. Furthermore, appetite and food intake both increase following sleep loss. This is despite overall physical activity often being reduced [115,116], resulting in an increased risk of metabolic disease and obesity [117].

The prevalence of unhealthy behaviors associated with sleep loss may be driven, in part, by increased **impulsivity**. Individuals who are high trait-impulsive have greater preference for instant gratification/gains, a phenomenon referred to as delay discounting. One observed consequence of this trait is the act of individuals forgoing their normal bedtime and thus short-changing their sleep amount, known as **sleep procrastination** [118,119]. Chronic insufficient sleep may, in turn, further exacerbate reward-related impulsive behaviors, ultimately leading to a self-fulfilling cycle of sleep procrastination and hence sleep deficiency. Supporting this bi-directional relationship, a recent cross-sectional study with a large sample (n = 1190) revealed that habitual short sleepers (≤6 hours) have increased delay discounting relative to a control group of average sleepers (7–9 hours) [120]. Still, experimental studies of delay discounting following partial or total sleep deprivation have so far yielded conflicting results [7,119].

Mechanistically, sleep-loss related changes in impulsivity and reward processing have been linked to generalized increases in reward sensitivity in the orbitofrontal cortex (OFC), areas of mPFC and anterior insula cortex. Similar to negative emotional processing, this hypersensitivity appears to impair the accurate coding of reward value, preventing the updating of accruing reward history/probability over time [7]. As a result, sleep-deprived participants consistently make less appropriate, riskier decisions, and assign greater weight to recent rewards, rather than the aggregated sum of their full experience overtime [121,122]. This may offer one neural and behavioral link between chronic sleep loss and the development and maintenance of addiction disorders [123,124]. Consistent with this possibility, 60% of patients with alcohol addiction who initially report insomnia, will relapse across a 5 month period, relative to 30% of patients who do not report such sleep difficulties [125].

Hedonically, sleep deprivation further increases sensitivity within the amygdala in response to rewarding food stimuli [56,57], yet impairs discriminatory mapping of these desirable food stimuli within the anterior insula, mPFC, and OFC. This can lead to a bias that disproportionately favors the choice of high calorie foods by sleep-deprived individuals, again indicative of generalized reward-gain sensitivity and shorter-term gratification [56]. Such a profile mirrors the inaccurate discrimination of negative emotional stimuli discussed earlier and indicates an affective impairment across the full valence spectrum of human emotional responsivity (Figure 1B).

Concluding Remarks and Future Perspectives

Sleep appears to be fundamental to the optimal social and emotional functioning of human beings. When sleep is insufficient, amplifications in basic aversive- and reward-reactivity emerge. Moreover, impairments in the accurate recognition of different types of emotions, their relative strength, and the outward communicative expression of emotions are also observed. Inadequate sleep further leads to a complex behavioral profile of higher-order emotion dysfunction. This may include social withdrawal, social isolation (loneliness) and the transmission (contagion) of these emotional states to other individuals. Additionally, insufficient sleep triggers maladaptive behaviors in the workplace, within interpersonal relationships, and even in choices that can influence the fabric of society.

Despite the illustrative collection of research to date, this field remains in its infancy. Looking to the future, we suggest at least four topics of research investigation that could prove fruitful in

Outstanding Questions

How does sleep loss impair the brain-body *inter*-relationships that map our embodied affective experiences, resulting in social and emotional dysfunction? In the context of sleep, current research has typically focused on measuring activity from either the brain or the body, yet concurrent assessments, within the same individual, are needed to resolve this embodied conundrum.

Can preemptive sleep extension provide emotional protection against unavoidable next-night sleep deprivation? If affirmed, this would reflect some capacity for "stored" sleep resilience in the context of affective functioning. Related, what is the time scale needed for recovery of different socio-emotional functions following bouts of sleep loss? This may be relevant for guidance in professions where long periods of continuous/semi-continuous work involve significant sleep loss.

What is it about the beneficial presence of sleep that actively restores our emotional wellbeing: quantity, architecture, electrical quality, subjective quality, or some combination of these? Do different sleep stages uniquely predict the optimization of different socio-emotional processes and mood states? Answering these questions will aid the development of sleep therapy for mood disorders. Is the cumulative aggregation of sleep (quantity/quality) across many prior nights (e.g., the past 3, 7, or 30 nights) a far better predictor of how we will socially and emotionally operate on a given day, rather than simply the sleep we had the night before? If true, this would re-shape how we study the relationship between sleep and affective functioning.

advancing the field and that we offer as closing thoughts: (i) mechanism, (ii) sleep 'dose-response' and sleep recovery relationships, (iii) directionality, and (iv) the physiological benefits of sleep's presence (also see Outstanding Questions).

Mechanism

Though the impact of sleep loss on numerous affective functions is increasingly clear, the underlying biological mechanisms remain poorly characterized. We believe that any unifying framework attempting to explain this collection of effects will require consideration not of the brain, or of the body, but the organism as a whole: brain + body. Our affective experiences, behaviors, and interactions are influenced by almost every major homeostatic system of the body (for description, see [126]). This includes: (i) cardiorespiratory system, (ii) metabolic system, (iii) visceral system, (iv) thermoregulatory system, (v) immunological system, (vi) hormonal system, (vii) cutaneous system, and (viii) nociceptive system. These signals ascend and converge onto an extended viscerosensory network within the brain (strongly overlapping with the salience detection network [127]) and when accurately mapped, give rise to the affective states we call emotions. Those emotions, in turn, instigate motivated decisions and actions. That is, an embodied, closed-loop system.

Sleep deprivation has independently been demonstrated to impact every one of these eight major physiological systems within the body and, furthermore, reliably alters activity in most all regions of the extended viscerosensory network in the brain (e.g., insula, cingulate cortex, amygdala, and some key brainstem nuclei). Emerging from this recognition is the possibility that sleep loss alters the fundamental human homeostatic reference point used to register and differentiate emotions [7]. However, no studies to date have linked comprehensive disruptions of both the brain– and multiple systems within the body when seeking to explain the marked social and emotional dysfunction caused by a lack of sleep. Examples to date have typically associated changes in just one body measure (e.g., cardiovascular) with changes in the brain [45,46]. Therefore, an opportunity exists for research that takes a multisystems approach to the study of sleep and socio-emotional processing, one assessing the full plurality of body systems and brain networks. For example, do changes in autonomic activity trigger changes in thermoregulatory and immune function that, in turn, are registered by viscerosensory networks in the brain, which then instigate changes in mood? Only with such an embodied approach will a full mechanistic understanding emerge of how and why sleep loss significantly perturbs our emotional lives.

Sleep-Loss Dose and Recovery

We lack any nuanced understanding of how the human emotional brain is impacted by gradated forms, or 'doses' of sleep loss. This is similarly true in terms of the time window over which such different sleep-loss doses unfold. For example, do 8 days of 1-hour sleep restriction (8 hours sleep loss, total) result in the same socio-emotional impairments of 1 night (8 hours sleep loss, total) of sleep deprivation? Does the impact of these different 'flavors' of sleep loss change as a function of sex? And do we become more or less resilient to the socio-emotional effects of sleep loss as we age; pertinent considering that older adults suffer worse sleep quality and reductions in total sleep time as well as in selective NREM and REM sleep stages [128].

And what of recovery? We know least about if, or how quickly, a human being emotionally recovers from the effects of insufficient sleep, either acute or chronic. Related, do different socio-emotional functions recover at the same rate following different forms and doses of sleep loss? We also have a poor understanding of whether sleep extension, beyond habitual amounts, can beneficially improve socio-emotional functioning. Furthermore, could preemptive sleep extension confer emotional resilience to unavoidable upcoming bouts of sleep loss? Answering these

questions can offer guidance in professions where various doses of sleep loss are common practice.

Directionality

While most of the studies reviewed above focus on one specific directional association: sleep loss → affective functioning, less is known about the reverse associations: affective experience → subsequent sleep.

Emerging findings suggest a relationship between sleep and affective functioning that is bi-directional [129]. For example, levels of experienced daytime anxiety [130,131] predict consequential impairments in objectively measured sleep quality and increases in sleep fragmentation the following night, as do evening feelings of loneliness and regret [132,133]. Experimental manipulations of emotional states reveal similar findings [5,134]. Negative-mood induction at bedtime results in worse subsequent sleep efficiency and greater sleep fragmentation, relative to a baseline night [135]. In contrast, positive (happy) mood induction can hasten the speed with which participants fall asleep [136]. Extensive work in rodents further supports a detrimental impact of daytime stress on subsequent sleep [137]. Severe social stress [138] or foot-shock paradigms [139] result in significant sleep disruption and reductions in REM sleep amounts. Nevertheless, a detailed mapping of this bi-directional sleep ↔ emotional interrelationship is sorely absent.

Sleep Physiology

Finally, what is it about the presence of sleep that appears to be so necessary for our emotional well-being, beyond its simple absence? Do different sleep features (e.g., sleep quantity, sleep stages, electrophysiological sleep quality) uniquely predict optimization of different socio-emotional processes and mood states? If so, are similar relationships observed when it comes to recovery from sleep loss?

What little evidence there is suggests just such complexity. For instance, the quantity and quality of REM sleep has been associated with the overnight dissipation of emotional sensitivity, both in subjective emotional ratings and reported distress, together with objective measures of limbic brain reactivity [64,140,141]. This is fitting with a proposed model [48] in which REM sleep and its associated neurobiology can recalibrate appropriate affective brain activity, preventing excess and over-generalized emotional responsivity the next day.

In contrast to emotion responsivity, the homeostatic regulation of longer-lasting mood states, such as anxiety or depression, have been linked with reductions in NREM slow-wave sleep and associated **slow-wave activity (SWA)** (0.5–4 Hz) [142–144]. Notably, NREM-SWA has been associated with an overnight lowering of anxiety [29], a physiological sleep relationship that further predicts next-day (re)engagement of mPFC activity. However, these studies remain few in number, and still without consensus. A full understanding of the sleep stages/physiologies, or their combination, that service the many and varied emotions and mood states we experience, and that go awry in psychiatric conditions, remains unknown. This is yet another essential goal for the research field in years to come.

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