Facial Emotion Recognition in Insomnia: Mechanisms and Consequences
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1. Abstract
Insomnia is a sleep disorder that is closely related with various physiological and psychological problems. Facial emotion recognition (FER) is an important part of social function, and has been found impaired in individuals with insomnia. This paper aims to explore the mechanisms of the correlation between insomnia and FER impairment, and the possible consequences of impaired FER for people with insomnia. Impaired expression expressivity and amygdala dysfunction are proposed as potential mechanisms underlying the link between insomnia and impaired FER. FER impairment in insomnia patients might lead to overlooking danger and lower levels of vigilance and arousal, which could result in disastrous outcomes. Moreover, impaired FER could lead to social function disturbance, making insomnia patients vulnerable to bullying.

2. Introduction
Insomnia disorder has become increasingly prevalent due to several contributing factors, including rising levels of stress, addiction to smartphones, and shift work [1-4]. A bunch of evidence has demonstrated that insomnia disorder can lead to a range of physiological and psychological problems, such as obesity, depression, anxiety, and memory dysfunction [5-8]. Consequently, scholars have devoted much attention to studying insomnia disorder and its potential impacts.

Facial emotion recognition (FER) is an essential component of social function [9-11]. Correctly and quickly recognizing others’ facial expressions is crucial for daily social interactions. Impaired FER ability may lead to misinterpretation of emotional cues and inappropriate communication, resulting in unsatisfactory social interaction or even unexpected outcomes. Recently, a meta-analysis has revealed impaired FER ability in individuals with insomnia [12].

This present paper comprises two parts. The first part will focus on potential mechanisms that may explain the correlation between insomnia and FER impairment. The second part will explore the potential consequences of impaired FER for individuals with insomnia, as well as interventions to improve FER ability in this population.
3. Mechanisms of impaired FER in insomnia

Compared to healthy sleepers, individuals with insomnia show an attentional bias toward tired expressions, in other words, they tend to rate others’ tired faces as less tired but their own tired faces as more tired [13-15]. Furthermore, sleep disturbance can alter emotional expressivity [16], making people with sleep disturbance appear more fatigued, sadder, and less attractive than healthy sleepers [17-19]. These factors might contribute to insomnia patients’ impaired ability to correctly imitate others’ facial expressions, while the ability to imitate facial expressions is correlated with the FER ability [20]. This partially explains why people with insomnia have worse FER ability than healthy sleepers.

What is the brain basis of the FER impairment in people with insomnia? Although there is no specialized circuit in the brain for emotion processing[21], several brain structures, including the amygdala, hypothalamus, periaqueductal gray, ventral striatum, insula, orbitofrontal cortex, and medial PFC, have been found to be closely related to emotion processing [22]. Among these, the integrated systems of the amygdala, ventral striatum, and septum-hippocampus are particularly important for both evaluating and expressing emotions [21], with amygdala playing an important role in detecting danger [23]. A meta-analysis of 105 studies found that the amygdala was activated during the processing of happy, sad, and fearful expressions, while most sensitive during the processing of fearful expressions [24]. Research has found heightened amygdala responses to insomnia-related emotional stimuli in people with insomnia, compared with healthy sleepers [25]. This finding somehow puts light on the role of amygdala function during the processing of emotional information in people with insomnia. Additionally, atrophic changes in amygdala have been observed in people with chronic insomnia [26]. Thus, these findings may explain why the ability of people with insomnia to recognize fearful expressions is impaired, while their ability to recognize happy, sad, and angry expressions remains unaffected [12]. Research carried among such special population with impaired amygdala function, helps further provide behavioral evidence-based data for previous findings and assumption among healthy population, that amygdala is more engaged in stimuli with more ambiguity [23], and as fearful expression means unknown threat compared with angry expression, amygdala is more strongly evoked to fearful expression [27]. Surprised expressions, which also convey ambiguous information, are processed differently, with the parahippocampal region being more strongly evoked [28]. However, it is not yet known whether insomnia affects the parahippocampal region or the ability to recognize surprised expressions. Further research is needed to figure out these questions.

4. Consequences of impaired FER in insomnia

The aforementioned findings give rise to a legitimate concern that individuals with insomnia may face difficulties in rapidly and accurately detecting danger, leading to decreased levels of vigilance and arousal, which could result in catastrophic outcomes. In military operations, for instance, correctly identifying fearful expressions on others’ faces is critical for analyzing the environment and triggering an acute stress response [29,30]—fight or flight—that could increase the chances of survival. However, due to frequent high-intensity training, mild traumatic brain injury, deployments, insomnia is prevalent among military personnel [31,32], which could lead to a failure to recognize fear-related cues. Moreover, amygdala is not only involved in emotion processing, but also contributes to decision making [27,33,34]. Amygdala damage would lead to ‘elevated risk taking’ [27]. Therefore, individuals with insomnia may be more likely to overlook danger and engage in irrational coping strategies. Thus, insomnia and its deleterious consequences should be taken more seriously, particularly for people in high-risk occupations.

Considering the role of FER in the social function, it is reasonable to hypothesize that people with insomnia might experience social dysfunction and be more vulnerable to negative social events. Similar with insomnia patients, children with autism spectrum disorder (ASD) showed a remarkable reduction in processing the eye region in fearful face [35], and reduction in ability of recognizing negative expressions including fear, anger and disgust [36,37]. Meanwhile, bullying victims perform worse on rating the intensity of emotion in FER task than bullying perpetrators among adolescents with ASD [38]. At the same time, children with poor sleep are more likely to become victims of bullying [39], but to our best knowledge, no study has investigated whether if among insomnia population, worse FER ability would increase likelihood of becoming victims of bullying. In summary, it could be demonstrated that, FER ability is related with bullying behavior, at least in children with ASD. Bullying behavior would increase the risk of insomnia [40,41], which in turn would further impair FER ability, creating a vicious circle. Given that bullying is strongly linked to various mental health problems [42-43], finding effective treatments for insomnia and interventions to restore FER ability is crucial to prevent negative social outcomes.
Previous studies have shown a close relationship between insomnia and social anxiety or social phobia [44-46]. The association between insomnia and anxiety has been explained by Blake and his colleagues from three perspectives [45]: physiological factors, such as overlap of genes that may be involved in serotonin [47-49] and dopamine pathways [50,51]; psychological factors, such as attentional bias; and social factors, such as impaired social interaction, and for example, adolescents with sleep disturbance are more likely to inhibit self-expression [52]. Now, another possible social mechanism explanation for the correlation between insomnia and anxiety could be raised, that people with insomnia perform worse in FER, which is critical for social interaction, and are therefore more likely to receive negative social feedback and suffer from social anxiety.

5. Interventions to restore FER ability

Given the prevalence of insomnia as a chronic sleep disorder affecting a significant number of individuals worldwide [1], and the negative consequences associated with the impairment of FER ability in individuals with insomnia as discussed above, exploring effective treatments for this condition is of utmost importance.

A study has shown that stimulants, such as modafinil, dextroamphetamine, and caffeine, can significantly improve the ability to discern morphed expressions after sleep deprivation [53]. Morphed expressions are created by blending two different facial expressions to reveal subtler changes in facial expression perception. It is believed that the improvement in FER ability is partly due to the short-term enhancement of alertness and psychomotor vigilance speed provided by these stimulants [54,55]. However, it is worth noting that caffeine intake (600mg) was associated with more side effects, such as nausea, nervousness, abdominal pain, and excitation, compared to the other two stimulants [55]. These side effects could limit the use of caffeine as an intervention for improving FER ability.

Besides from taking stimulants, napping could also reverse the enhanced reactivity towards negative emotions (i.e., anger and fear) induced by extended wakefulness, while enhance ratings of positive emotion (i.e., happy) [56]. Although these studies focus on the effects of different methods to improve FER ability after sleep deprivation, they offer valuable insights for future researchers seeking to develop effective interventions for individuals with insomnia. It is worth mentioning, however, that insomnia patients often report more severe sleep disturbances than objectively measured [19,57-59]. Their sleep disturbance might fall somewhere between sleep-sated and sleep-deprived and can differ from that of individuals experiencing sleep deprivation [19].

In healthy people, recognizing different expressions relies on different facial parts [60]. Specifically, the eyes are more relied upon than the mouth for perceiving fearful and sad expressions, whereas the mouth is more important than the eyes for perceiving disgusted and happy expressions [60]. Akram and colleagues found that individuals with insomnia exhibit an attentional bias towards the eye region when recognizing tired expressions [61]. However, to our knowledge, no study has used an eye tracker to compare the differences in recognizing other expressions, such as fear, sadness, and anger, between individuals with insomnia and healthy sleepers. Therefore, further research is necessary to determine whether the attentional bias of individuals with insomnia towards different facial parts contributes to their impaired FER ability.

By investigating the underlying mechanisms of FER impairment in individuals with insomnia and potential consequences of this condition, we help further understand the basis of insomnia’s effects on health. Several promising interventions to restore their FER ability are raised, and may help improve insomnia patients’ social functions.

6. Declaration of Interests

N/A.

6. References

27. Pessoa L. Emotion and cognition and the amygdala: from "what is it?" to "what's to be done?" Neuropsychologia. 2010;48(12):3416-3429. doi:10.1016/j.neuropsychologia.2010.06.038


