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Altered Emotion Perception in Insomnia Disorder

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Study Objectives: Chronic insomnia is a prevalent sleep disorder that negatively affects daytime functioning and confers risk for the future development of psychiatric disorder. Patients with insomnia often report problems with emotion regulation and impaired social interactions. Moreover, experimental sleep loss in healthy adults is associated with altered reactivity to and interpretation of emotional information. In the current study, we investigated socioemotional processing in patients with chronic insomnia disorder relative to healthy good sleepers.

Design: Between-groups comparison.

Setting: Sleep Research Laboratory.

Participants: Patients with well-defined psychophysiological insomnia (PI; n = 16), free from psychiatric disorder, and an age- and sex-matched control group of good sleepers (GS; n = 15).

Interventions: N/A.

Measurement and Results: All participants completed a facial expression recognition task, between 18:00 and 19:00, requiring participants to categorize and rate the intensity of four emotional expression categories: anger, fear, happiness, and sadness. People with PI did not differ from GS with respect to categorization of facial expressions. However, in terms of intensity judgements, across all emotion categories, patients tended to rate faces as less emotionally intense (Cohen's *d* = 0.70). Specifically, they rated expressions displaying sadness and fear as significantly less emotionally intense than healthy GS (both P < 0.05; Cohen's *d* = 0.77 and 0.89, respectively). Measures of sleepiness (Psychomotor Vigilance Test, Karolinska Sleepiness Scale) or self-reported sleep were not reliably associated with emotional intensity ratings. However, anxiety and depression were negatively related to intensity ratings.

Conclusion: For the first time we show that chronic insomnia is associated with reduced ratings of emotion intensity for face expressions displaying sadness and fear. Further work is required to elucidate possible mechanisms and pathways underlying insomnia-related emotional impairment.

Keywords: Blunting, emotion, face expressions, insomnia, sleep

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INTRODUCTION

Insomnia disorder is characterized by persistent problems with the initiation and/or maintenance of sleep, resulting in impaired daytime functioning and quality of life.[1-3] Insomnia is highly prevalent, affecting approximately 10% of the adult population,[4] and is markedly persistent over time.[5] Although most research activity has concentrated on primary insomnia, persistent insomnia most commonly occurs in conjunction with comorbid physical and mental health conditions.[6,7] Indeed, sleep disturbances are characteristic of nearly all psychiatric disorders.[8] Importantly, however, longitudinal data also demonstrate insomnia to be a risk factor for the future development of psychopathology, notably depression,[9] anxiety,[10] and substance abuse.[11] Moreover, experiencing insomnia in the context of mood and anxiety disorders is associated with worse daytime functioning and enhanced disability.[12] Insomnia, therefore, is both a risk factor for, and potentiator of, psychological distress; this conclusion was reflected in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, as a paradigm shift away from primary and secondary insomnia, in favor of the more substantive and causal attribution-free term, insomnia disorder.[13]

Treatment seeking in patients with insomnia is typically prompted by impairment of daytime function, with the most common factors being fatigue, impaired work performance, cognitive complaints, emotional disturbance, and social/relationship dysfunction.[4,14,15] Although fatigue, work productivity, and cognitive performance have received considerable attention in the research literature,[3,16,17] there has been relatively little investigation of the direct socioemotional impact of insomnia. Preliminary work from our group reveals that impairment in social interactions and emotion regulation are salient concerns for those with insomnia.[15,18] This broad research area seems important to pursue for at least two related reasons. First, heightened emotional arousal is a putative etiological and maintaining factor for chronic insomnia;[19-21] and second, experimental sleep loss has recently been associated with disrupted next-day reactivity to, and interpretation of, emotional information, which may have implications for psychopathology.[22-28] Specifically, sleep deprived subjects exhibit exaggerated activity in limbic regions (the amygdalae), in association with reduced activity in prefrontal cortex toward emotional stimuli.[22,29-31] At the behavioral level, sleep deprived healthy controls present evidence of blunted behavioral performance[26,32,33] and have reduced emotional expressiveness.[25,34]

Although this work lays the foundation for understanding the importance of sleep in emotional brain homeostasis, the experimental context in which these studies were typically conducted

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(i.e., full night of sleep deprivation with healthy subjects) may not readily reflect the real-world effects of chronic nightly or near-nightly sleep impairment, which is more characteristic of patients with insomnia and psychiatric illness.[35] In the current study, we sought to investigate whether patients with psychophysiological insomnia (PI)—the most common primary insomnia phenotype, characterized by conditioned bedtime arousal, excessive sleep preoccupation, and voluntary sleep effort[1]—would perform differently, relative to healthy controls, on a task involving the categorization and appraisal of emotional face expressions. Facial expressions were chosen because they convey important social and biological information concerning an individual's internal state and motivational intentions, which may influence and guide social behavior.[36,37] Following stimulus presentation, the emotional significance of a face is appraised, which leads to an affective state and corresponding emotion behavior, subject to emotion regulation.[38] Such paradigms are sensitive to impairment in many psychiatric disorders, helping to shed light on the neurobiological underpinnings of disease phenomenology,[39,40] as well as putative treatment mechanisms.[41-43] Perceptual processing of faces involves a distributed network of cortical and subcortical regions.[44,45] Some of these regions have shown metabolic alterations post-sleep deprivation[46,47] and structural and/or functional impairment in insomnia patients relative to healthy controls.[48-51]

Patients with PI (n = 16) and an age- and sex-matched control group of good sleepers (GS; n = 15) completed the facial expression recognition task, requiring participants to categorize and rate the intensity of four emotional expression categories: anger, fear, happiness, and sadness. The recognition of these four basic emotion categories is typically high across cultures,[52] and the brain may have evolved to decode specific emotions.[53]

Extrapolating from the sleep science literature,[26,33] we hypothesized that patients with insomnia would not differ in gross categorization judgements but that they would show lower, more blunted ratings of emotional intensity, relative to GS controls. However, in light of research showing enhanced emotional reactivity in patients with insomnia toward sleep related emotional stimuli,[54] coupled with close links between sleep disturbance and depression, a competing hypothesis was that patients with insomnia may report increased ratings of emotion intensity. Because this is the first examination of face processing in insomnia, our study served to test both of these hypotheses in an exploratory manner.

In order to profile what factors may contribute to face task performance, we also assessed associations with sleep (self-reported), daytime functioning (anxiety, depression, fatigue) and sleepiness/vigilance (Psychomotor Vigilance Test [PVT], Karolinska Sleepiness Scale [KSS]), in both good and poor sleepers. In the absence of experimental manipulations of these variables in patient groups, examining such relationships may help determine the relative importance of associated daytime and sleep related factors.

METHODS

Participants

Sixteen thoroughly screened patients with PI were recruited to take part in the current study. Patients were all free from central nervous system-affecting medication and psychiatric or medical comorbidity (including occult sleep disorder pathology, see following paragraphs). A group of healthy age- and sex-matched good sleepers (n = 15) was recruited for comparative purposes.

Assessments and Procedure

Sleep Status

Patients with PI received a phone interview by an expert in behavioral sleep medicine to assess the absence of comorbidities and medication use, as well as the presence of insomnia, defined as satisfying the following criteria for subjective sleep impairment:

- Report of sleep disturbance for at least 3 nights per week for ≥ 6 mo
- Seep onset latency (SOL) and/or wake after sleep onset (WASO) > 30 min
- Total sleep time (TST) ≤ 6 h
- Sleep efficiency (SE) < 85%
- Daytime impairment attributed to disturbed sleep

The phone interview was based on guidelines described by Morin and Espie[55] and supplemented with a sleep disorders screening questionnaire.[56] Those deemed eligible were invited to attend a screening day, involving a thorough sleep and psychiatric interview with a licensed clinical psychologist (Mini-International Neuropsychiatric Interview),[57] trained in behavioral sleep medicine, and a medical assessment (electrocardiograraphy [ECG], blood chemistries, medical history, and drug screen) by a certified physician. Patients meeting Research Diagnostic Criteria for PI[58] and who met all other inclusion/exclusion criteria subsequently slept for 2 consecutive nights at the University of Glasgow Sleep Centre to undergo polysomnography (PSG). A standard PSG montage was used, involving electroencephalographic (EEG: Fp1 (neutral), C3, P3 (reference), O1, Fpz, Fz, Cz, Pz, Oz, F4, C4), electrooculographic (EOG: horizontal and vertical) and electromyographic (EMG; submental) recordings. On night 1, all participants were screened for sleep disordered breathing and periodic limb movements through monitoring of abdominal and thoracic effort, nasal airflow, oximetry, and bilateral tibialis anterior EMG. Sleep was recorded on a Lifelines Trackit™ ambulatory recorder and scored visually by two experienced scorers (> 90% inter-scorer reliability) according to criteria by Rechtschaffen and Kales.[59] For study inclusion, patients were required to have an apnea-hypopnea index (AHI) and periodic limb movements of sleep (PLMS) arousal index < 10 (see Table 1 for PSG parameters). Those subjects meeting criteria were subsequently invited to complete the experimental tasks.

GS similarly received a phone interview to assess inclusion/exclusion criteria, defined as the absence of sleep, psychiatric or (unstable) medical disorder, and the endorsement of good quality, restorative sleep, in addition to the following:

- SOL and WASO < 15 min
- Number of nighttime awakenings ≤ 2
- TST > 6 h
- SE > 85%
- Stable sleep period between 22:00 and 08:00

All study participants completed a 7-day sleep diary,[55] in the week prior to completing the face task, to assess sleep

continuity and quality and help rule out circadian phase problems. Participants also completed the Flinders Fatigue Scale (FFS)[60] to index fatigue-related impairment, the Insomnia Severity Index (ISI),[61] a sensitive measure of insomnia severity, and the Hospital Anxiety and Depression Scale (HADS),[62] probing emotional distress in the past week.

It should be noted that matching between patients and controls was initiated on a subject-by-subject basis, with each patient matched with a corresponding GS in terms of sex and age ± 2 y. Successful one-to-one matching was achieved for 14 of 16 patients.

Table 1—Polysomnographic sleep parameters for patients with psychophysiological insomnia (n = 16)

PSG parameters	Mean (SD)
SOL (min)	9.7 (8.9)
WASO (min)	28.2 (18.4)
No. Awak	4.8 (2.6)
TST (min)	396.4 (41.6)
SE (%)	89.6 (5.3)
Stage 1 (%)	7.9 (4.3)
Stage 1 (min)	31.9 (18.0)
Stage 2 (%)	51.6 (6.4)
Stage 2 (min)	228.3 (35.8)
Stage SWS (%)	8.5 (5.8)
Stage SWS (min)	37.9 (26.0)
Stage REM (%)	22.8 (5.1)
Stage REM (min)	98.2 (22.3)
AHI[a]	2.1 (3.2)
PLMS index[a]	1.1 (2.0)

AHI, apnea-hypopnea index; No. Awak, number of awakenings; PLMS, periodic limb movements of sleep; PSG, polysomonography; REM, rapid eye movement; SD, standard deviation; SE, sleep efficiency; SOL, sleep onset latency; SWS, slow wave sleep; TST, total sleep time; WASO, wake after sleep onset. [a]Assessed on PSG night 1 (screening)

Face Task

Testing took place between 18:00 and 19:00. The stimuli in the emotional face task consisted of the validated and widely used Ekman and Friesen[63] faces displaying anger, fear, happiness, and sadness. These four emotional expressions, commonly used in studies probing psychopathology[64,65] and sleep-wake functioning,[66] were posed by five male and five female individuals, and repeated twice, to give a total of 80 stimuli. Faces were presented in a random order, with a break at the midpoint. The task was programmed in SuperLab (http://www.superlab.com/). All faces were presented in grayscale at a size of 3.7 × 5.0 cm with a resolution of 72 ppi, cropped to remove extraneous visual cues, and the average luminance and contrast of all face images were equated. An initial practice session aided familiarity with task demands. This practice session consisted of labeling four emotional words (anger, fear, happy, sad, repeated four times), using the middle and index fingers of each hand, with participants self-correcting errors. Following this, participants categorized and rated the intensity of four emotion faces (not used in the task, repeated four times), in order to familiarize subjects with the experimental task. The main experiment then started whereby participants were instructed to categorize the emotion shown by pressing the corresponding button as quickly and accurately as possible, and faces were presented until response. Subsequently, and after a 500-ms pause, participants were asked to rate the intensity of the expressed emotion,[67-69] on a seven-point Likert scale, anchored at 'not very intense' and 'extremely intense'. Following this, a blank screen was presented for 750 ms, followed by a fixation cross for 750 ms before the next trial began. Our task was designed to permit separation of categorization accuracy from intensity judgements; both of which are typically confounded in tasks where participants are asked to appraise emotion in (morphed) faces displaying different levels of emotion intensity.

Sleepiness

Prior to completing the computerised tasks, participants completed the KSS,[70] a single-item measure of state sleepiness (1-9 scale; i.e. 1 = very alert, 5 = neither alert nor sleepy, 9 = very sleepy, great effort to keep awake, fighting sleep).

Psychomotor Vigilance Task

The PVT is a frequently used task in sleep research to assess the effect of sleep restriction or deprivation, or altered sleep timing on basic vigilant attention. The version of the PVT used in the current study has been applied in studies of insomnia and sleep perturbation.[71,72] In the task, participants are asked to respond with a left mouse click, as quickly as possible, to the presence of an asterisk located in the center of the computer screen. Interval onset for asterisks varied between 1 and 10 sec in duration, and there were 110 experimental trials. Participants completed five practice trials at the beginning to aid task familiarity. The PVT was programmed in E-prime (http://www.pstnet.com/eprime.cfm) and completed on a Dell laptop, at a viewing distance of 40 cm. Task duration was approximately 13 min. Testing took place at 18:00, prior to completing the face task. PVT reaction time (RT) [1/mean RT; lower scores indicate slowed reactions] was calculated as an index of sustained vigilance.[73]

Statistical Analyses

Independent *t*-tests were conducted to compare groups on demographic measures, self-report sleep, fatigue, PVT performance (RT), sleepiness (KSS), and emotional distress (Hospital Anxiety and Depression Scale, HADS). Analysis of variance (ANOVA) with group (PI, GS) as the between-subject factor and emotion (anger, fear, happiness, sadness) as within-subject factor was conducted for categorization accuracy and intensity judgements. Main effects and interactions were followed up with independent and paired *t* tests. Magnitude of group differences were quantified using Cohen's *d*: $(M1 - M2 / \delta\, pooled)$.[74] Means and standard deviations are presented in text unless otherwise stated. For face task dependent variables showing sensitivity to group, relationships between self-report sleepiness, vigilance, sleep parameters, and emotional distress were investigated using correlational analyses.

Table 2—Demographic, sleep and daytime functioning variables for patients with psychophysiological insomnia (PI) and good sleepers (GS)

	GS (n = 15)	PI (n = 16)
Age (SD)	47.1 (10.5)	47.1 (10.8)
Sex % (F:M)	66.7/33.3	62.5/37.5
ISI	0.8 (1.1)	17.8[a] (2.8)
HADS-A	2.1 (2.3)	6.4[a] (4.0)
HADS-D	0.9 (1.6)	4.0[a] (2.2)
FFS	1.9 (3.5)	13.4[a] (6.1)
KSS	2.8 (1.6)	4.8[a] (1.9)
PVT (RT)	3.03 (.36)	2.89 (.30)
SOL (min)	7.1 (7.9)	38.8[a] (32.4)
WASO (min)	6.8 (11.2)	62.6[a] (58.8)
No. Awak	1.2 (1.4)	2.1[b] (1.3)
TST (min)	449.9 (41.7)	338.7[a] (57.4)
TIB (min)	503.1 (51.0)	490.6 (66.8)
SE (%)	89.9 (6.3)	69.3[a] (12.3)
SQ (0-4)	3.3 (0.4)	1.7[a] (0.6)

FFS, Flinders Fatigue Scale; HADS-A/D, Hospital Anxiety and Depression Scale; ISI, Insomnia Severity Index; KSS, Karolinska Sleepiness Scale; No. Awak = number of awakenings; PVT, Psychomotor Vigilance Task (1/mean reaction time [RT], lower scores indicate poorer vigilance) SE, sleep efficiency; SOL, sleep onset latency; SQ = sleep quality TST, total sleep time; TIB, time in bed; WASO, wake after sleep onset. [a]P < 0.01 and [b]P < 0.10 for group comparison.

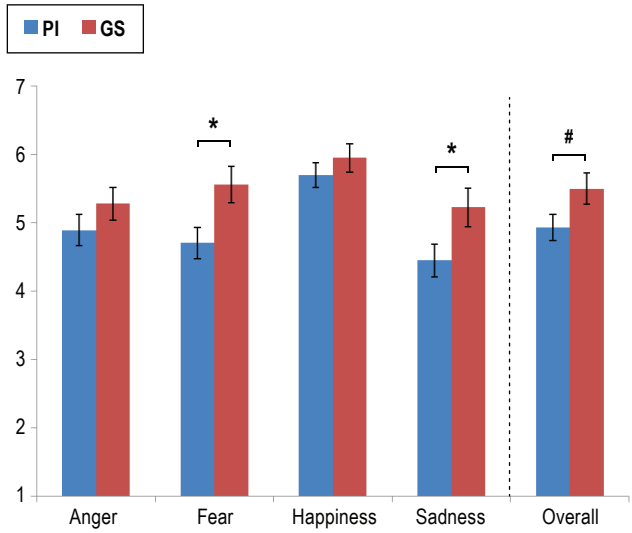

Figure 1—Emotional intensity ratings for patients with psychophysiological insomnia (PI) and good sleepers (GS) across the four facial expression categories (*P < 0.05, #P < 0.07 for focussed group comparison).

RESULTS

Participant Demographics

Sixteen patients (6 male; 10 female) with an average age of 47.1 y (standard deviation [SD] = 10.8 y) participated in the study. As a by-product of subject-by-subject matching, the control group had similar sex distribution and mean age (Table 2). Seven-day sleep diaries confirmed the presence of subjective sleep disturbance in patients and an absence of sleep complaints in those endorsing good-quality, restorative sleep. The PI group scored 17.8 (SD = 2.8) on the ISI, reflecting clinical levels of insomnia severity. As expected, the PI group reported greater levels of fatigue (FFS), anxiety, and depression (HADS). Of note, and consistent with the diagnosis of PI, anxiety and depression scores were in the mild range and approximate those found in large nonclinical samples.[75] During testing, patients reported greater levels of sleepiness (KSS), but there was no difference in objective vigilance levels as measured with the PVT.

Patients Versus GS

One participant from the PI group was removed from analysis due to extreme, below-chance scoring (i.e., 0% correct for labeling of emotion category). For categorization, ANOVA failed to reveal a significant effect of group [$F(1,28) = 1.33$, P = 0.26] or group × emotion interaction [$F(3,84) = 0.49$, P = 0.69], though a main effect of emotion was observed [$F(3,84) = 13.66$, P < 0.001]. Across all participants, categorization was highest for displays of happiness relative to all other categories (P < 0.05), whereas participants were least accurate for judging face expressions displaying anger (P < 0.05 for comparisons with happiness, fear, and sadness categories). Both groups performed at a similar level across emotion categories (Anger: PI = 80.3% [15.1] versus GS = 81.3% [15.2]; Fear: PI = 87.7% [13.5] versus GS = 92.3% [9.8]; Happiness: PI = 97.7% [7.8] versus GS = 99.0% [2.8]; Sadness: PI = 85.0% [17.9] versus GS = 91.7% [9.0]).

For intensity ratings, there was a trend toward a main effect of group [$F(1,28) = 3.67$, P = 0.066], a main effect of emotion [$F(3,84) = 22.84$, P < 0.001], and a near-significant group × emotion interaction [$F(2.8,77.8) = 2.70$, P = 0.056]. Across all participants, face expressions displaying happiness were rated as reliably more emotionally intense than all other categories (P < 0.001). Across all emotions, patients tended to rate facial expressions as less emotionally intense relative to good sleepers [4.93, SD = 0.73 versus 5.50, SD = 0.89; Cohen's $d = 0.70$; see Figure 1]. Follow-up group comparisons at the level of emotion category indicated that patients rated faces displaying fear [$t(28) = -2.44$, P = 0.021; Cohen's $d = 0.89$] and sadness [$t(28) = -2.10$, P = 0.045; Cohen's $d = 0.77$] as significantly less emotionally intense than healthy controls; this was not the case for facial expressions of anger [$t(28) = -1.17$, P = 0.25] or happiness [$t(28) = -0.92$, P = 0.36] (Figure 1). When examining the distribution of intensity ratings for facial expressions displaying fear and sadness, it became clear that, in line with average group differences, patients with insomnia were less likely to endorse ratings at the "high emotion intensity" end of the spectrum (scores of ≥ 5, on the 1-7 scale). We formally evaluated this in exploratory binary analyses, comparing proportion of participants in the PI group, scoring intensity ratings ≥ 5, relative to GS. For faces displaying sadness, 2 of 15 PI patients (13.3%) scored in the "high range" versus 10 of 15 (66.7%) GS ($X^2 = 8.89$, P = 0.003). For fear faces, 5 of 15 patients with PI (33.3%) scored in the "high range" compared with 12 of 15 GS (80%; $X^2 = 6.65$, P = 0.01). This was not the case for faces displaying happiness or anger (P > 0.05).

Table 3—Correlations between emotion intensity judgements and sleep diary, sleepiness and daytime functioning variables.

	Insomnia					Controls				
	Anger	Fear	Happiness	Sadness	Overall	Anger	Fear	Happiness	Sadness	Overall
HADS-A	-0.52[a]	-0.41	-0.74[a]	-0.38	-0.58[a]	-0.50	-0.28	-0.33	-0.65[a]	-0.49
HADS-D	-0.37	-0.29	-0.46	-0.64[a]	-0.52[a]	0.12	0.18	0.28	0.17	0.20
FFS	0.30	0.07	0.24	-0.06	0.15	-0.24	-0.28	-0.08	-0.28	-0.25
KSS	0.10	0.16	-0.31	-0.20	-0.06	0.13	0.07	0.37	0.37	0.25
PVT-RT	-0.28	-0.26	-0.22	-0.34	-0.33	0.13	0.27	0.28	0.19	0.24
SOL	-0.30	-0.26	-0.31	-0.19	-0.30	0.16	0.10	0.28	0.17	0.19
WASO	-0.10	0.13	-0.04	0.03	0.04	0.05	0.20	0.18	0.08	0.14
TST	-0.23	-0.15	-0.24	-0.45	-0.32	-0.14	-0.32	-0.03	-0.02	-0.14
SE	-0.06	0.01	-0.23	-0.17	-0.13	-0.24	-0.20	-0.46	-0.37	-0.34

[a]P < 0.05. FFS, Flinders Fatigue Scale; HADS-A/D, Hospital Anxiety and Depression Scale; KSS, Karolinska Sleepiness Scale; PVT, Psychomotor Vigilance Task (1/mean reaction time [RT], lower scores indicate poorer vigilance); SE, sleep efficiency; SOL, sleep onset latency; TST, total sleep time; WASO, wake-time after sleep onset.

Relationships Between Self-Reported Sleep, Sleepiness, Daytime Functioning Variables, and Emotion Intensity Judgements

We next investigated associations between emotional intensity judgements and self-reported sleep (diary values for SOL, WASO, SE, TST), sleepiness (KSS, PVT) and daytime functioning (HADS, FFS). There were no significant correlations between KSS scores or PVT performance (RT) and emotional intensity judgements, for either patients with PI or GS controls (Table 3). Similarly, diary reported values of SOL, WASO, TST, and SE were not significantly associated with emotional intensity judgements in either group.

Self-reported fatigue (FFS) was not associated with intensity judgements for either patients with PI or GS. However, both anxiety and depression were significantly (negatively) associated with intensity judgements for patients with PI, while anxiety was also moderately associated with intensity judgements for GS controls (Table 3). Specifically, anxiety was negatively associated with anger (r = -0.52), happiness (r = -0.74,) and overall (r = -0.58) emotion intensity judgements, for patients with PI, and negatively linked to ratings of sadness (r = -0.65) in GS controls. Depression was negatively associated with sadness (r = -0.64) and overall (r = -0.52) emotion intensity judgements in patients with PI.

DISCUSSION

In the current study, we investigated whether patients with PI, free from psychiatric disorder, would differ from GS on an emotional face task. The main findings suggest that groups do not differ with respect to broad categorization of emotion; that is, both groups performed well in matching facial expressions to emotion category. However, for intensity judgements, patients with PI tended to rate faces as being less emotionally intense. Focused group comparisons suggested that this emotional blunting was significant and pronounced for facial expressions displaying fear and sadness (with corresponding large effect sizes). Although the reliability of our correlational analyses must be interpreted with caution, due to the small sample size, markers of sleepiness (PVT performance, KSS) and subjective sleep parameters were not significantly related to task performance in patients with PI. Conversely, emotional distress (reflected in HADS scores) was significantly (negatively) associated with intensity judgements.

To our knowledge, this is the first report to show that patients with insomnia differ with respect to their interpretation of emotional face information. What might contribute to these group differences? Three potential (nonexclusive) explanations may be relevant.

Altered Neural Processing of Emotional Information Due to Sleep Loss

It is well known that experimental sleep loss reduces cerebral metabolism in prefrontal regions,[47] and authors have suggested that such changes may help explain blunted ratings of emotion intensity in healthy sleep deprived controls.[27] However, sleep deprivation or restriction is not necessarily the same as chronic insomnia, where sleep tends to be only moderately perturbed relative to subjective perceptions[76] and from PSG data in the current study, sleep continuity parameters are not markedly impaired (Table 1). Moreover, and while still preliminary, we did not observe significant associations between markers of sleep loss (sleepiness, vigilance, sleep diary) and intensity judgements. Intriguingly, and although small and nonsignificant, we can see from Table 3 that PVT performance appears positively related to intensity ratings in healthy controls, yet negatively related in patients with PI (whereby lower levels of vigilance are linked to higher ratings of emotion intensity). This possible differential relationship may deserve attention in future studies.

tiredness correlated with intensity ratings in controls but not in insomnia?

It must be noted, however, that two studies using functional imaging—one using fludeoxyglucose-positron emission tomography (FDG-PET) and the other, functional MRI (fMRI)—have revealed evidence of daytime prefrontal hypoactivation in patients with insomnia,[48,50] similar to that observed in sleep deprived subjects. Indeed, the study by Nofzinger et al.[50] did not reveal differences between patients and controls with respect to objective sleep parameters. Functional deficit in prefrontal regions in insomnia patients—related or unrelated to sleep loss—may have implications for higher processing of

emotional states. This may manifest as attenuated mirroring or 'experiencing' off another's emotional state, subsequently affecting stimulus appraisal.[77]

It is also known that specific emotions are associated with partly dissociable neural structures.[78,79] Of relevance, the amygdalae, in functional imaging and lesion studies, have most often been associated with the processing of fear and sad facial expressions.[77,80,81] Indeed, amygdala activity has also been found to be positively related to increasing subjective perceptions of emotion intensity.[82] Although functional imaging studies in insomnia are limited, one recent study, using resting-state fMRI, revealed reduced amygdala connectivity with several brain regions involved in emotional processing (striatum, insula, thalamus), relative to normal sleepers.[83] It remains possible, therefore, that altered amygdala reactivity[23] and/or connectivity may contribute to the observed behavioral findings relating to face expressions displaying fear and sadness. From the current study design, of course, we cannot delineate underlying neural mechanisms behind group differences and future studies, combining assessments of sleep and neural responses to emotional stimuli, are required to address hypotheses arising from our data.

Psychological Distress

Although markers of sleep loss were not reliably associated with task performance, psychological distress (both anxiety and depression) tended to be significantly and moderately related to intensity judgements. Given that patients were psychologically healthy, beyond the presence of self-reported sleep disturbance, this association may reflect the daytime consequences of perceived sleep loss, perhaps suggesting a common mechanism linking subjective and objective daytime (emotional) dysfunction. Indeed, recent work probing neurobehavioral performance in primary insomnia also reveals significant associations with daytime functioning variables (including depression and anxiety), but no relationship with sleepiness or sleep diary parameters.[84] One clear extension of the current study is to consider how emotional intensity ratings are linked to affective reactions, induced by emotional stimuli. For example, Rottenberg and colleagues[85] have advanced the emotion context-insensitivity hypothesis of depression, whereby depressed patients exhibit attenuated emotion reactivity across both positive and negative stimuli. It is well known that passive viewing of emotional face expressions is associated with localized facial muscle responses,[86] and there is some evidence that these responses are altered in patients with depression[87] and possibly after sleep deprivation.[25] Given the negative relationship between affect and intensity ratings observed in the current study, triangulation of methodologies— involving facial EMG, introspective reports, and perceived expressivity—during an emotion reactivity task, may help further delineate mechanisms underlying group effects, and determine similarities with patients with major depression.

Trait-Level Emotion-Coping Strategies

In the absence of experimental manipulation, it is possible that our study findings may reflect trait-like characteristics of emotional processing in those with insomnia. Indeed, some have speculated that insomnia patients may internalize and suppress their emotions during the day,[20,88] preventing adequate 'processing', and potentially leading to a 'rebound' at sleep-onset or during the sleep period.[89,90] The cognitive model of insomnia hypothesizes that suppression of emotional material may be a strategy adopted by patients in order to manage excessive cognitive activity and anxiety.[20] This is particularly relevant to the current study where anxiety was found to be elevated in patients, relative to controls and negatively associated with intensity ratings. The relationship between emotion-regulation strategies, personality, and performance on emotion tasks would be worthy of future research.[54] Sampling of emotional performance after both good and poor nights of sleep—in keeping with night-to-night variability in insomnia symptoms[91]—may help inform on the stability of observed impairments. Finally, natural history studies assessing the trajectory of normal sleep, through to acute and chronic insomnia,[5] would also be useful in addressing potential trait-level explanations for our findings.

Strengths and Limitations

This work has several methodological strengths, including: rigorous participant screening and group matching; exclusion of occult sleep disorder pathology through PSG; fixed testing time to control for possible time-of-day effects; and measurement of sleepiness, general vigilance, and emotional distress to determine relationships with task performance. Nevertheless, several important limitations should be considered. Principally, while our sample was well defined, our total *n* was small. This may have limited our ability to detect interaction effects at the 5% alpha level. However, power calculations indicated that 15 cases per group were sufficient to detect a large effect for the between-subjects factor group and within-between interaction, with a power of 80%. Future studies should attempt to replicate and extend our work with larger and more varied samples (e.g., other primary insomnia phenotypes, comorbid insomnia presentations). Similar sample size caveats must be taken into account when interpreting results from our correlation analyses, particularly because we conducted a large number of tests without alpha-level adjustment. Regarding this point, several correlations reported in Table 3 are of small to moderate strength, yet fail to reach statistical significance. Correlational findings, therefore, must be viewed as preliminary. Future studies, with larger samples and experimental manipulations, will be important to determine the reliability of these associations, before more definitive conclusions can be drawn regarding the relationship between face task performance, sleep, and daytime functioning variables.

With respect to measurement, we did not increase the difficulty of our task by varying emotion intensity levels and so cannot determine where in the intensity spectrum impairments would be most likely observed. However, the high categorization performance of both groups using this particular stimulus set, supports the validity of our task, with the advantage that correct performance can be identified. Furthermore, for intensity analysis, we only selected those trials that were correctly identified, ensuring that impaired categorization accuracy was not a confounder. Ultimately, we recognize that this is the first assessment of facial expression appraisal in insomnia disorder and that other face task paradigms, routinely applied within the broader psychopathology field,[92] should similarly be investigated in future studies to further circumscribe the nature of and

contributors to insomnia-related emotional impairment. Finally, although we did conduct PSG on our patient sample, this was not completed for the night prior to performance testing. Future work would benefit from assessing associations between objective sleep parameters and next-day emotional performance, in both good and poor sleepers, and after experimental manipulation of sleep.

CONCLUSIONS

Overall, we show that patients with PI report reduced ratings of emotion intensity toward facial expressions, in particular for faces displaying fear and sadness. Although these findings would be considered partially consistent with the sleep deprivation literature, it must be noted that patients were not markedly sleep deprived in the current study, at least assessed through classic PSG parameters; and, although requiring replication, self-reported sleep and markers of sleep debt were not reliably linked to performance. However, depression and anxiety were negatively associated with intensity ratings. Future research attention should be directed toward assessing neural responses to emotional stimuli in patients with insomnia; characterization of how patients, relative to healthy controls, decode facial expressions; and the relationship between emotion task performance and emotion-coping strategies. Given the prevalence and burden of chronic insomnia, we hope this work will stimulate others to probe socioemotional processing in patients with chronic poor sleep and help shed light on the interplay between sleep disturbance and mental health.[35]

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