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Comparing primary insomnia to the insomnia occurring in major depression and general anxiety disorder

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ABSTRACT

Apart from possibly distinguishing the different clinical causes of insomnia, this article explores the subjective and objective sleep differences amongst primary insomnia, major depression with insomnia and general anxiety disorder with insomnia. Subjective sleep and objective sleep of the participants were evaluated by using the Pittsburgh sleep quality index and polysomnography, respectively. We found that major depression with insomnia exhibited higher daytime dysfunction than primary insomnia; showed significantly higher values of rapid eye movement (REM) periods, time of REM sleep and percentage of REM stage; and presented lower percentage of non-rapid eye movement stage compared with primary insomnia and general anxiety disorder with insomnia ($p < 0.05$). General anxiety disorder with insomnia showed lower awakening number (AN) than primary insomnia, and other objective and subjective sleep values of general anxiety disorder with insomnia and primary insomnia showed no significant difference ($p > 0.05$). Our findings showed that major depression with insomnia increased active REM sleep and severe daytime function, which could alert clinicians to the risk of depression. Major depression with insomnia and primary insomnia may be categorically different. However, general anxiety disorder with insomnia and primary insomnia might be a continuum of a disease rather than be categorically distinct.

1. Introduction

Insomnia is usually a subjective experience in patients who are dissatisfied with sleep quality and/or the time of sleep, which can influence daytime social function. Insomnia is a highly common disease in clinical sleep disorders, and its prevalence approximately ranges from 5% to 50% (Ohayon, 2002) due to various operationalisations. Insomnia with increasing tendency (Garland et al., 2018) is considered a main public health problem. Chronic insomnia not only decreases daily functions but also affects normal life and work and even causes bodily function disorders and systemic physical diseases (Foley et al., 2004; Taylor et al., 2003). Insomnia symptoms considerably increase the risk of various conditions, such as mental conditions, pain conditions with uncertain aetiology and chronic pain conditions (Sivertsen et al., 2009). After observing 3 million pregnant women for 6 years, researchers from the United States found that sleep disorders in pregnancy can significantly increase preterm birth, and the risk of preterm birth for pregnant women with insomnia increased by 30% (Felder et al., 2017). Insomnia is a severe burden to the family and society (Morin and Jarrin, 2013). In the United States, poor performance due to insomnia causes losses of $60 billion per year (Kessler et al., 2011). Major depression with the greatest burden of mental illness has a complex connection with insomnia. The prevalence of insomnia-related sleep disorders amongst major depression is approximately 90.9% (Seow et al., 2016). Sleep disorders can increase the risk and severity of major depression, and people with insomnia are almost 10 times more likely to suffer from major depression than those without insomnia (Taylor et al., 2005). As a common residual symptom of major depression, insomnia persists in half of patients treated with antidepressants (Nierenberg et al., 1999). Sleep disorders increase the risk of suicide by influencing the depressive symptoms (Weis et al., 2015); in the remission stage, insomnia negatively affects major depression by increasing the risk of recurrence (Ohayon and Roth, 2003). Compared with major depression, general anxiety disorder may exhibit a stronger interrelationship with insomnia (Uhde et al., 2009). Not all patients experience anxiety and insomnia at the same time, but anxiety and insomnia are generally observed in the same individuals. Approximately 75% of patients with general anxiety disorder report insomnia (Bélanger et al., 2004), which may be a trait marker for individuals at risk of developing anxiety disorders (Neckelmann et al., 2007). Patients with general anxiety disorder are more prone to suffer from insomnia than normal people with or without the accompanying

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depressive symptoms (Brenes et al., 2009). Amongst anxiety disorders, general anxiety disorder can predict insomnia, whereas obsessive-compulsive disorder, separation anxiety and social phobia are not significantly related to insomnia (Alvaro et al., 2014). The status of general anxiety disorder influences the relationship between insomnia and memory or concentration problems (Brownlow et al., 2017). Insomnia may be a common health condition amongst patients with general anxiety disorder and is associated with the severity of anxiety (Navarrete et al., 2017). Major depression, general anxiety disorder and insomnia are closely related, but the question of 'whether they are the same neurobiological abnormality or not' remains unclear.

Sleep evaluation mainly involves of the assessment of subjective and objective sleep differences. Sleep questionnaires assess the global subjective sleep quality and disturbances over a specific time period (Buysse et al., 2006). The Pittsburgh sleep quality index (PSQI) is a commonly used questionnaire (Buysse et al., 2006, 1989). Polysomnography (PSG) is the gold standard for sleep assessment; it monitors the continuity of sleep and comprehensive data on the sleep structure and is a widely used method for evaluating objective sleep. Different diseases exhibit varying characteristics of PSG. Compared with normal controls, sleep continuity of depressed subjects is often impaired with increased frequency of wakefulness, reduced sleep efficiency (SE) and total sleep time (TST), increased sleep onset latency, shortened rapid eye movement (REM) latency and decreased duration of the first REM period (Nutt et al., 2008). General anxiety disorder is mainly related to the difficulty in maintaining sleep and slightly related to the difficulty in falling asleep. No consistent conclusions have been found regarding non-rapid eye movement (NREM) and REM sleep structures (Monti and Monti, 2000).

The subjective and objective sleep involved in major depression and general anxiety disorder have been frequently studied. The majority of studies are concerned with the difference between these conditions and the normal population, and the differences amongst primary insomnia, major depression and general anxiety disorder are rarely reported. Kohn and Espie compared patients with primary and comorbid insomnia (comorbid with depressive or anxiety disorders) on several subjective and objective sleep variables; they suggested that primary and comorbid insomnia may be a continuum in insomnia severity rather than categorically distinct (Kohn and Espie, 2005). However, in their study, the group of comorbid insomniacs was not specified. Another study assessed the subjective sleep differences amongst patients with primary insomnia, insomniacs with comorbid mood disorder and insomniacs with comorbid anxiety disorder via self-report measures (van de Laar et al., 2015). Their study made a further but incomplete distinction of comorbid insomnia. Mood disorders include depressive and dysthymic disorder, whereas anxiety disorders include general anxiety, panic, post-traumatic stress, obsessive and anxiety disorders not otherwise specified and social phobia.

The two previously mentioned studies recruited participants from the perspective of insomnia. Firstly, the authors recruited participants who were diagnosed with insomnia and then further divided the insomniac patients into patients with primary insomnia and comorbid insomnia. In the present study, we recruited participants from the point of disease and we selected subjects who were diagnosed with major depression or general anxiety disorder and showed insomnia symptoms at the same time. We then compared the subjective and objective sleep differences amongst primary insomnia, general anxiety disorder with insomnia and major depression with insomnia. We aimed to explore the possibility of discriminating insomnia caused by different clinical causes and provide clues for the 'continuum or categorical' debate involving primary and comorbid insomnia.

2. Methods

2.1. Participants

All subjects were newly admitted inpatients recruited from the Psychiatric Department of the Second Xiangya Hospital of Central South University from October 2016 to November 2017. We completed all assessments before starting treatment on the first day of admission. The following inclusion criteria were applied. (1) All patients complained of insomnia with symptoms lasting for at least 1 month. Major depression with insomnia patients satisfied the diagnostic criteria for major depression of DSM-IV, general anxiety disorder with insomnia patients met the diagnostic criteria for general anxiety disorder of DSM-IV and primary insomnia patients satisfied the diagnostic criteria for primary insomnia of DSM-IV. (2) The participants must be aged between 18 and 65 years and (3) not taking medication or were subjected to a washout for more than 7 days. The following exclusion criteria were applied to all the patients: (1) severe physical disease, (2) suspected of sleep apnoea, (3) alcohol or drug abuse or dependence and (4) pregnant and lactating women and women during their menstrual period. Additionally, patients with evident anxiety symptoms with self-rating anxiety scale (SAS) scores of moderate or above anxiety (SAS $>$ 60) were excluded from the major depression with insomnia group to improve the comparability of anxiety and depression. Similarly, patients with evident depressive symptoms (self-rating depression scale, SDS $>$ 63) were excluded from the general anxiety disorder with insomnia group.

2.2. Pittsburgh sleep quality index (PSQI)

PSQI is a 19-item self-report measure of broad sleep disturbances over the past month that contains seven component scores (sleep quality, sleep latency, sleep duration, habitual SE, sleep disturbances, use of sleeping medication and daytime dysfunction). The results were added to obtain the total score. The score ranged from 0 to 21, and a score of 5 or higher indicated poor sleep (Buysse et al., 1989).

2.3. SDS and SAS

Depression and anxiety symptoms were measured separately by the Chinese versions of the SDS and SAS. These scales are valid measures for the Chinese population (Lee et al., 1994; Merz and Ballmer, 1984). Both scales consisted of 20 items rated on a 4-point scale. The summary scores ranged from 20 to 80, with high scores indicating elevated symptoms. The patients were considered to have clear depressive symptoms when the total SDS score was more than 63. Furthermore, the patients were considered to have clear anxiety symptoms when the total SAS score exceeded 60. These two tools can identify patients with pure depression or anxiety and increase the comparability amongst the three groups.

2.4. PSG

Standard PSG (Harmonie, Stellate Systems, Canada) was applied in accordance with the American Academy of Sleep Medicine criteria (Iber et al., 2007). The PSG montage included four EEG channels (C3-A2, C4-A1, C3-O1 and C4-O2), left and right EOG, chin EMG, right and left anterior tibialis EMG, EKG, nasal thermistor, oral thermistor, respiratory effort and pulse oximetry. PSG was carried out by experienced technicians, and results were analysed by sleep specialists. The recording time was at least 8 h. The sleep recordings were evaluated for the following parameters: total recorded time (TRT), total sleep period (TSP: time between lights out and final rising time), TST, wake after sleep onset (WASO), SE, sleep onset latency (SOL), awakening number (AN), sleep maintenance (SM: ratio of TST to TSP $\times$ 100%), REM sleep latency (RL), number of REM periods (NRP), time of sleep in stage 1

(N1), time of sleep in stage 2 (N2), time of sleep in stage 3 (N3), time of sleep in NREM sleep, time of REM sleep (RT) and percentage of every sleep stage (N1%, N2%, N3%, NREM% and RT%).

The study and its aims were explained to participants, and their informed consent was obtained. All participants completed PSQI, SAS and SDS on the test day. Diagnostic evaluation was completed by two senior clinicians, and the scales were assessed by a trained professional who did not know the participants' diagnosis. After completing the questionnaires, all participants spent 1 night in the sleep laboratory for PSG assessment. Finally, 27 patients with primary insomnia, 22 major depression with insomnia patients and 22 general anxiety disorder with insomnia patients were recruited.

2.5. Statistical analysis

All analyses were performed by IBM SPSS Statistics 22 for Windows. Differences in gender were analysed via the chi-square test. The data subject to normal distribution were analysed via one-way ANOVA, and Bonferroni was used for post hoc pairwise comparisons. Nonparametric Kruskal–Wallis H test was used for other data, and the Mann–Whitney U test was applied for post hoc pairwise comparison (Bonferroni correction alpha = 0.017). A significance level of 0.05 was used for each hypothesis.

3. Results

Proportionately high numbers of women were observed in the three groups (primary insomnia group = 20 women/7 men, major depression with insomnia group = 14 women/8 men, general anxiety disorder with insomnia group = 12 women/10 men). However, this difference was not statistically significant ($\chi 2 = 2.045$, $p = 0.36$), and the average age per group was between 40 and 50 (Table 1). No significant differences in age and gender were found amongst the three groups. Thus, the confounding effects of these factors amongst group differences in sleep characteristics were likely (Table 1). Major depression with insomnia patients had no evident anxiety symptoms (SAS < 60) and showed higher SDS scores than general anxiety disorder with insomnia and primary insomnia patients. General anxiety disorder with insomnia patients had no evident depressive symptoms (SDS < 63) and showed higher SAS scores than major depression with insomnia and primary insomnia patients. The primary insomnia group showed no evident anxiety and depressive symptoms (Table 1).

Table 2 illustrates the differences amongst the three groups on the global and seven component scores of PSQI. The mean global PSQI scores of the three groups were 13.41, 13.68 and 12.93 for major depression with insomnia, general anxiety disorder with insomnia and primary insomnia, respectively; these values indicated moderate sleep disturbances. Major depression with insomnia patients exhibited more daytime dysfunction than those with primary insomnia ($p = 0.015$), which suggested that major depression with insomnia was accompanied with severe daytime function. No significant between-group differences for sleep quality, sleep latency, sleep duration, habitual SE, sleep disturbances and use of sleeping medication were observed.

The comparison of PSG variables amongst the three groups is shown in Table 3. Significant differences in AN, NRP, RT, RT% and NREM% amongst the three groups were found. Post hoc pairwise comparisons revealed that major depression with insomnia patients exhibited significantly higher NRP ($Z = -4.114$, $p < 0.001$), RT ($Z = -4.253$, $p < 0.001$) and RT% ($Z = -4.040$, $p < 0.001$) and lower NREM% ($Z = -4.040$, $p < 0.001$) and AN ($Z = -3.774$, $p < 0.001$) than primary insomnia patients. The general anxiety disorder with insomnia patients demonstrated lower AN ($Z = -2.953$, $p = 0.003$) than primary insomnia patients, and the major depression with insomnia patients showed higher NRP ($F = 2.682$, $p < 0.001$), SM ($Z = -2.408$, $p = 0.016$), RT ($Z = -4.455$, $p < 0.001$) and RT% ($Z = -2.606$, $p = 0.009$) and lower NREM% ($Z = -2.606$, $p = 0.009$) than the general anxiety disorder with insomnia patients. NRP, RT and RT% in the major depression with insomnia group were higher than those in the two other groups. Thus, the major depression with insomnia group demonstrated more active REM sleep than the other groups. No significant differences in TRT, TSP, SE and SOL were found amongst the three groups.

4. Discussion

Under the same subjective sleep quality, the major depression with insomnia group showed worse daytime function and active REM sleep than the general anxiety disorder with insomnia and primary insomnia groups. Regarding subjective sleep quality, no significant difference in the total and factor scores of PSQI between the general anxiety disorder with insomnia and primary insomnia groups was observed. In the PSG objective sleep index, significant differences between the general anxiety disorder with insomnia and primary insomnia groups were found in AN only. The primary insomnia and general anxiety disorder with insomnia groups exhibited similar sleep architectures and showed lower NRP, RT and REM% and higher NREM% than the major depression with insomnia group.

An item for daytime dysfunction is the 'lack of enthusiasm to get things done', which directly relates to the core symptoms of depression. Hence, daytime function must be closely related to depression. Depressed mothers during the early postpartum period show more daytime function than non-depressed mothers (Huang et al., 2004). Analyses of the specific PSQI component scores indicated that self-reported daytime dysfunction is most strongly related to the low ambulatory positive effect on the sleep components (Bower et al., 2010). Amongst community samples who received a body–mind–spirit intervention for coexisting sleep disturbances and depressive symptoms, daytime functioning at follow-up examinations is related more to changes in depressive symptoms than to changes in night-time sleep (Ji et al., 2018). In an elderly Asian population, Junhong YU found that the geriatric depression scale and geriatric anxiety inventory scores are both significantly correlated with sleep disturbance, whereas the geriatric depression scale score is uniquely associated with daytime dysfunction (Yu et al., 2016) . In addition, depressive patients show negative cognition and are overly concerned and sensitive with their own body, thereby leading to the patients' negative explanations about their insomnia and the increasingly severe manifestation of symptoms.

Our study found that REM sleep was active in the major depression group; this finding was partially consistent with results of previous studies. Observational studies have emphasised that the change in REM

Table 1
Demographic, SDS, SAS characteristics of participants.

	Major depression with insomnia	General anxiety disorder with insomnia	Primary insomnia	F/χ2	p
Age (years)	42.23 ± 14.32	49.73 ± 8.18	42.78 ± 11.34	2.966[b]	0.058
Sex	male (8), female (14)	male (10), female (12)	male (7), female (20)	2.045[c]	0.360
SDS	68.55 ± 12.42	58.95 ± 6.89	50.37 ± 10.98	24.229[a]	<0.001
SAS	56.45 ± 7.98	60.18 ± 6.26	47.33 ± 8.90	17.495[b]	<0.001

SAS: self-rating anxiety scale; SDS: self-rating depression scale; a: Kruskal–Wallis H; b: one-way ANOVA; c: chi-square test.

Table 2
Pittsburgh sleep quality index (PSQI) estimates of sleep for each experimental group and results of ANOVA with post hoc analyses.

	Major depression with insomnia	General anxiety disorder with insomnia	Primary insomnia	F/χ2	*p*	post hoc
sleep quality	2.23 ± 1.15	2.64 ± 0.66	2.70 ± 0.72	3.311[a]	0.191	
sleep latency	2.23 ± 1.07	2.27 ± 0.88	2.33 ± 0.96	0.321[a]	0.852	
sleep duration	2.09 ± 1.15	2.27 ± 1.20	2.33 ± 1.00	0.637[a]	0.727	
habitual sleep efficiency	2.27 ± 1.08	2.45 ± 1.10	2.22 ± 1.15	1.466[a]	0.482	
sleep disturbances	1.36 ± 0.58	1.41 ± 0.67	1.11 ± 0.42	4.326[a]	0.115	
use of sleeping medication	2.18 ± 1.18	1.77 ± 1.31	1.63 ± 1.42	1.723[a]	0.423	
daytime dysfunction	1.09 ± 1.06*	0.91 ± 0.92	0.44 ± 0.80	6.494[a]	0.039*	MI > PI
global PSQI score	13.41 ± 3.39	13.68 ± 3.86	12.93 ± 2.83	0.323[b]	0.725	

[a] Kruskal–Wallis H;.
[b] one-way ANOVA;.
* $P < 0.05$.

sleep in patients with depression is not the only symptom of the disease (Modell and Lauer, 2007). Reduction in REM latency was once considered the most promising marker of 'endogenous' depression (Kupfer, 1976). REM latency in depressive patients does not change with the state of the disease (Giles et al., 1989). Moreover, REM sleep changes in depression can be observed in the remission state (Steiger et al., 1989), and the persistence of shortened REM latency in the remission period of major depression is associated with increased risk of recurrence (Giles et al., 1987). However, a meta-analysis of studies on the EEG features of various psychiatric disorders challenged the specificity of shortened REM latency for MDD, because it revealed that individuals with schizophrenia also exhibit this characteristic (Benca et al., 1992). Researchers then suggested that increased REM density, especially in the first REM period, may be a genetically transmitted biomarker of MDD (Pillai et al., 2011; Steiger and Kimura, 2010). A population-based study suggested that REM density is a marker of depressive symptoms in the general population (Luik et al., 2015). REM variables are strongly altered in major depression. Regardless of the episode state or being in early remission, recurrent unipolar major depression shows greater disturbances of REM sleep than single episodes (Jindal et al., 2002). Additionally, REM sleep changes in major depression are hereditary. A high-risk study of major depression relatives found that sleep patterns of identical twins show greater consistency than those of fraternal twins (Hori, 1986). Early theories suggested that central cholinergic activity and supersensitivity, which is responsible for the generation of REM, may be excessively increased in depression and represent a relevant neurobiological factor in the affect regulation (Christoph et al., 2006; Palagini et al., 2013). The association between REM sleep variables and affect regulation was also evidenced by neuroimaging studies (Nofzinger et al., 2004; Vanderhelm et al., 2011). Furthermore, increased REM activity and time are related to suicidal behaviours in individuals with depression (Sabo et al., 1991). Therefore, REM dysfunction is strongly related to major depression, and the differences between major depression and primary insomnia prove that major depression and primary insomnia may be categorically different.

Depression was estimated to exceed 300 million in 2015 in the world's total population and ranked as the single largest contributor to global disability (Organization, 2017a). An effective treatment for moderate and severe depression is available, but the current treatment remains unsatisfactory. In high-income countries, nearly half of the people with depression remain untreated (Organization, 2017b). Only 18%–34% of young people with high levels of depression or anxiety symptoms seek professional help (Gulliver et al., 2010). The time interval between the initial symptoms of depression and treatment utilisation is rather long. About one-fourth of participants in Germany

Table 3
Comparison of polysomnography (PSG) sleep variables among three groups.

	Major depression with insomnia	General anxiety disorder with insomnia	Primary insomnia	F/χ2	*p*	Post hoc
TRT (min)	507.14 ± 32.76	520.02 ± 46.47	527.93 ± 31.89	1.900[a]	0.157	
TSP (min)	459.16 ± 50.23	482.41 ± 59.95	481.96 ± 68.25	4.159[a]	0.125	
TST (min)	364.93 ± 64.61	327.70 ± 74.25	345.98 ± 89.56	1.258[b]	0.291	
WASO (min)	138.25 ± 64.78	192.32 ± 84.55	181.94 ± 80.62	5.451[a]	0.066	
SE (%)	69.86 ± 13.89	63.36 ± 14.93	65.41 ± 15.79	3.031[a]	0.220	
SOL (min)	48.16 ± 38.33	37.61 ± 25.15	46.00 ± 46.63	0.656[a]	0.721	
AN	4.55 ± 2.58	5.36 ± 2.75	9.44 ± 5.31	16.532[a]	<0.001**	MI, GI < PI
NRP	3.86 ± 2.85	1.18 ± 0.40	1.41 ± 0.80	26.978[a]	<0.001**	MI > PI, GI
RL (min)	213.86 ± 88.70	235.95 ± 102.24	217.76 ± 119.01	0.281[b]	0.756	
SM	79.77 ± 12.69	68.56 ± 16.26	71.63 ± 15.48	7.539[a]	0.023*	MI > GI
N1(min)	55.32 ± 20.14	61.00 ± 26.35	56.07 ± 32.07	0.605[a]	0.739	
N1%	15.32 ± 5.85	18.60 ± 6.57	17.82 ± 11.58	3.956[a]	0.138	
N2 (min)	229.39 ± 56.05	204.05 ± 76.06	234.70 ± 80.85	1.187[b]	0.311	
N2%	63.20 ± 8.23	64.07 ± 9.60	66.20 ± 10.07	2.179[a]	0.336	
N3 (min)	43.34 ± 26.23	36.55 ± 14.93	39.02 ± 18.04	0.228[a]	0.892	
N3%	12.04 ± 6.91	12.05 ± 7.43	11.69 ± 5.74	0.012[a]	0.994	
RT (min)	33.70 ± 18.26	17.00 ± 5.77	16.19 ± 7.93	21.921[a]	<0.001**	MI > PI, GI
RT%	9.45 ± 5.19	5.28 ± 1.71	4.84 ± 2.35	17.938[a]	<0.001**	MI > PI, GI
NREM (min)	331.23 ± 64.93	310.70 ± 71.69	329.80 ± 87.90	0.508[b]	0.604	
NREM%	90.56 ± 5.18	94.72 ± 1.71	95.17 ± 2.36	17.985[a]	<0.001**	MI < PI, GI

TRT: total recorded time; TSP: total sleep period; TST: total sleep time; WASO: wake after sleep onset; SE: sleep efficiency; SOL: sleep onset latency; AN: awakening number; NRP: number of REM; RL: REM sleep latency; SM: sleep maintenance; RT: time of REM sleep;.
[a] Kruskal–Wallis H;.
[b] one-way ANOVA;.
** $P < 0.01$,.
* $P < 0.05$.

stated that the time interval between the initial symptoms of depression and treatment utilisation is longer than 3 years, and only one-third said that they received treatment immediately or until 3 months after the initial symptoms of depression (Dietrich et al., 2016). This disappointing treatment status of depression is caused by many reasons, such as difficulty in early recognition of depression disorders. The first symptom that prompts many depression patients to seek help is insomnia. The identification of sleep structure abnormalities can alert clinicians to the risk of the onset of depression and application of preventive treatment. Considering the longitudinal association between insomnia and depression, treating sleep continuity difficulties at an early stage may interrupt the sequential process. Clinical studies have shown that the addition of cognitive behaviour therapy for insomnia in standardised intervention protocols of many mental disorders may improve the efficacy of such interventions (Rachel et al., 2011, 2008).

Sleep is considered a fundamental operating state of the central nervous system, and it is an important basic dimension of brain function and mental health (Harvey et al., 2011; Regier et al., 2012). Investigating the PSG sleep variables in mental disorders has the potential to reveal neurobiological mechanisms of specific disorders. In this study, the general anxiety disorder with insomnia and primary insomnia groups exhibited similar sleep architectures. Moreover, general anxiety disorder and primary insomnia groups demonstrated similar dysfunctional sleep beliefs (Tsai et al., 2013) and decreased resting-state connectivity (Pace-Schott et al., 2017). The similarities of the primary insomnia and general anxiety disorder with insomnia groups suggested that they might possess similar neurobiological mechanisms and similar neurobiological abnormalities but to varying degrees. Primary insomnia and general anxiety disorder with insomnia might be a continuum of a disease rather than be categorically distinct. The difference in AN between primary insomnia and general anxiety disorder with insomnia may be due to different degrees of disease severity. Further understanding of the neurobiological aspects of insomnia may help identify relevant pathophysiological pathways to insomnia and anxiety disorders. However, anxiety disorders, even severe ones, are not accompanied with sleep disorders from beginning to end. Certain patients with primary insomnia do not develop anxiety symptoms throughout the course of the disease. Therefore, the relationship between the two must be further studied.

Our study had the following limitations. Firstly, we used only one night of PSG in this study. The lack of an adaptation night may result in a possible ‘first night effect’, which is the tendency of individuals to sleep worse during the first night of PSG (Hirscher et al., 2015). This scenario likely caused low SE, long REM sleep latency and reduced amount of REM sleep in all groups of this study. Secondly, the lack of a healthy control group weakened the persuasiveness of the conclusions. Thirdly, the sample was relatively small, and the participants were predominantly women. Finally, a clinical interview instead of a structured interview was used in the diagnosis of mental disorders, so the accuracy of diagnosis may not be optimal.

In conclusion, this article selected subjects from the view of the disease and compared the subjective and objective sleep differences amongst patients with primary insomnia, major depression with insomnia and general anxiety disorder with insomnia. The major depression with insomnia group exhibited active REM sleep and severe daytime function, which could alert clinicians to the risk of depression. Major depression with insomnia and primary insomnia may be categorically different. However, the general anxiety disorder with insomnia and primary insomnia groups exhibited similar sleep architectures, which suggested than they might be a continuum of a disease rather than be categorically distinct. Their difference in AN might be caused by the varying degrees of disease severity.

Declaration of competing interest

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