Nature and Science of Sleep

ORIGINAL RESEARCH

Prevalence, Clinical Correlates, Gender Differences, and Predictive Factors of Paradoxical Insomnia in Chinese Adults with Chronic Insomnia: A Cross-Sectional Study

Mengmeng Wang[1,2,*], Wenjun Zhu[1,3,*], Yunliang Sun[4], Zhaoyan Feng[1], Yanyan Hou[1], Huanhuan Wang[1], Bing Zhou[1], Fang Han[1], Fulong Xiao[1]

[1]Division of Sleep Medicine, Peking University People's Hospital, Beijing, People's Republic of China; [2]Nursing Department, Sir Run Run Shaw Hospital, School of Medicine, Zhejiang University, Hangzhou, Zhejiang, People's Republic of China; [3]Department of Respiratory and Critical Care Medicine, The Second Affiliated Hospital of Nanchang University, Nanchang, People's Republic of China; [4]Department of Respiratory and Critical Care Medicine, Binzhou Medical University Hospital, Binzhou Medical University, Binzhou, People's Republic of China

*These authors contributed equally to this work

Correspondence: Fang Han; Fulong Xiao, Division of Sleep Medicine, Peking University People's Hospital, Beijing, 100044, People's Republic of China, Email hanfang1@hotmail.com; xiaofulong1681@163.com

Objective: This study aimed to determine the prevalence of paradoxical insomnia (PI) in Chinese adults with chronic insomnia, compare clinical and sleep-related characteristics between PI and non-paradoxical insomnia (non-PI) groups, explore gender-specific differences, and identify factors associated with PI, with particular focus on anxiety and depression.

Methods: A total of 862 adults with chronic insomnia were involved. All participants completed 7-day sleep diaries and validated questionnaires, including the Hamilton Depression Rating Scale (HAMD; clinician-rated depression), Hamilton Anxiety Rating Scale (HAMA; clinician-rated anxiety), and Insomnia Severity Index (ISI; subjective insomnia severity). All participants underwent two consecutive nights of polysomnography (PSG), and biochemical testing. Group differences were tested using independent samples *t*-tests or chi-square tests. Subjective-objective sleep associations were examined using Pearson correlations. Multivariable logistic regression was conducted to identify factors associated with PI.

Results: PI was present in 30.5% of participants (n = 263). Compared with non-PI, the PI group showed a higher proportion of females and exhibited greater depressive symptom severity on the HAMD and somatic anxiety on the HAMA, alongside lower perceived insomnia severity on the ISI (all $p < 0.05$). Gender-stratified analyses suggested distinct profiles, including higher perceived insomnia severity among females with PI and higher apnea-hypopnea index (AHI) among males with non-PI. In multivariable analyses, older age, male gender, heightened somatic symptoms (one subscale of HAMD), and lower subjective insomnia severity remained independently associated with PI (all $p < 0.05$).

Conclusion: Nearly one-third of Chinese adults with chronic insomnia meet the criteria for PI. Depression, particularly somatic symptoms, male gender, older age, and lower subjective insomnia severity are key predictors of PI. The findings highlight that incorporating objective sleep assessment and considering gender-specific clinical features may reduce diagnostic misclassification and facilitate more individualized management.

Keywords: paradoxical insomnia, sleep state misperception, depressive symptoms, anxiety, gender differences

Introduction

Insomnia, a prevalent sleep disorder characterized by persistent difficulties in initiating or maintaining sleep, affects approximately 10–30% of adults worldwide, with chronic insomnia (duration ≥3 months) accounting for nearly half of these cases.[1] Beyond subjective distress, chronic insomnia is strongly associated with adverse health outcomes, including increased risk of depression, cardiovascular disease, cognitive impairment, and reduced quality of life.[2] Given its high

https://doi.org/10.2147/NSS.S570398
Received: 7 October 2025
Accepted: 4 January 2026
Published: 21 January 2026

burden, accurate diagnosis and subtyping of chronic insomnia are critical for guiding targeted interventions, yet this remains challenging for a distinct subtype known as paradoxical insomnia (PI).

Defined by the International Classification of Sleep Disorders, Third Edition (ICSD-3) as a marked discrepancy between subjective sleep complaints and objective sleep findings.[3] PI presents a unique clinical paradox: patients report severe insomnia (eg, prolonged sleep onset latency [SOL], insufficient total sleep time [TST]) yet exhibit near-normal sleep architecture on polysomnography (PSG), which is widely regarded as the gold standard for objective sleep assessment.[4,5] This dissociation between perception and physiology distinguishes PI from non-paradoxical insomnia (non-PI), where subjective complaints align with objective sleep impairment (eg, shortened TST, increased wake after sleep onset [WASO] on PSG).[6–8] Despite this distinction, PI remains underrecognized in clinical practice, largely due to two key barriers: first, the lack of a universal operational definition (eg, varying thresholds for subjective-objective SOL/TST discrepancy);[4,5] and second, overreliance on self-reported sleep measures (eg, sleep diaries) that fail to capture the physiological reality of PI.[4,5]

Existing research on PI has several critical limitations that constrain its clinical applicability and translation into routine practice. First, most studies are small-scale, leading to inconsistent estimates of PI prevalence within chronic insomnia.[4] This variability complicates efforts to quantify PI's true burden in clinical settings. Second, data on PI in Asian populations, particularly in China, remain scarce. While Western cohorts have explored PI's association with psychiatric symptoms (eg, anxiety, depression),[9] it is unclear whether these findings generalize to Chinese patients, who may differ in sleep-related beliefs, help-seeking behaviors, and cultural attitudes toward sleep complaints.[10] Third, gender differences in PI remain poorly understood. Although chronic insomnia overall shows a female predominance,[11,12] prior studies on PI have reported conflicting results: some studies report no gender bias,[13] and no consensus exists regarding how gender modulates sleep perception, symptom severity, or objective sleep parameters in PI.

Another unresolved question concerns the nature of the subjective-objective sleep discrepancy in PI. Although PI is defined by this dissociation, few studies have systematically compared the correlation between self-reported and objective sleep metrics across PI and non-PI groups. For instance, it remains unclear whether PI patients exhibit selective impairment in perceiving specific sleep stages (such as sleep onset vs total sleep duration) or whether the discrepancy is more generalized. Additionally, the relationship between PI and psychiatric comorbidities, particularly anxiety and depressive symptoms, requires further clarification. Prior work suggests that emotional distress may contribute to altered sleep perception,[14,15] yet it is unknown whether somatic anxiety, as a distinct dimension of anxiety, is more strongly associated with PI than depressive symptoms or other anxiety components. Clarifying these associations is clinically important because PI is frequently under-recognized, and failure to identify sleep state misperception may contribute to misdiagnosis and unnecessary escalation of pharmacological treatment in chronic insomnia.

To address these gaps, we conducted a large-scale, cross-sectional study of adults with chronic insomnia at a tertiary sleep medicine center in China. In addition to psychological and sleep-related factors, chronic insomnia has been linked to adverse metabolic profiles,[16,17] including dyslipidemia and elevated triglycerides,[18] which contribute to greater long-term cardiometabolic risk. Therefore, we assessed lipid-related biochemical parameters including triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) to explore potential metabolic correlates of PI and to account for participants' overall physiological health when examining factors associated with PI. Using rigorous diagnostic criteria (ICSD-3 for chronic insomnia) and comprehensive assessments (7-day sleep diaries, validated scales of insomnia severity, anxiety and depression, PSG and biochemical testing), we aimed to: (1) determine the prevalence of PI in a Chinese cohort of patients with chronic insomnia; (2) compare clinical characteristics, subjective sleep parameters, objective sleep parameters, and biochemical indices between PI and non-PI groups; (3) explore gender-specific differences in sleep perception, symptom severity, and objective sleep metrics within both PI and non-PI groups; and (4) identify factors independently associated with PI, including demographic variables, anxiety symptoms, depressive symptoms, and perceived insomnia severity.

We hypothesized that: (1) PI would be characterized by greater subjective-objective sleep discordance and a distinct pattern of clinical, PSG, and biochemical measures relative to non-PI; (2) gender-specific differences in sleep perception and symptom severity would be present within PI and/or non-PI groups; and (3) demographic characteristics, psychiatric symptoms, and perceived insomnia severity would show independent associations with PI in multivariable models. By addressing these hypotheses, this study seeks to refine clinical characterization of PI, improve diagnostic accuracy, and lay the groundwork for gender-tailored interventions in Chinese patients with chronic insomnia.

Methods

Participants

This was a cross-sectional study conducted in the Department of Sleep Medicine at Peking University People's Hospital. Adults presenting with complaints of insomnia were consecutively recruited between August 2019 and July 2023. All participants were required to abstain from any medications known to affect sleep for at least two weeks before enrollment in the study.

The inclusion criteria were as follows: (1) aged 18–64 years; (2) diagnosed with insomnia disorder according to the ICSD-3,[3] with: (a) complaints of difficulty falling and/or staying asleep for at least three months, (b) persistence of symptoms despite adequate sleep opportunities, and (c) daytime dysfunction (including mood disturbance, cognitive impairment, social or occupational difficulties). These criteria were further as confirmed by sleep diary reports, indicating >30 minutes of sleep onset latency (SOL) and wake time after sleep onset (WASO) occurring ≥3 nights per week;[19] (3) no cognitive impairment, defined as a Mini-Mental State Examination scores >24 (see Measures).[20] All Participants underwent a comprehensive screening evaluation, including a structured sleep history interview, two consecutive nights of polysomnography (PSG), routine blood tests (blood cell count), electrocardiogram and urine drug screening.

Participants were excluded from the study if they met any of the following criteria: (1) a history of mental illness, depression or anxiety episodes (Hamilton Depression Scale scores ≥18 or Hamilton Anxiety Scale scores ≥14, see Measures) or presence of suicidal ideation (with a score of ≥3 on HAMD item 3), (2) comorbid other sleep disorders (eg, sleep apnea syndrome, restless-legs syndrome or narcolepsy), (3) use of medications known to effect sleep within two weeks prior to testing (eg, antidepressants, antipsychotics); 4) excessive alcohol or caffeine intake.[21]

This study was conducted in accordance with the principles of the Declaration of Helsinki and approved by Ethics Review Committee of Peking University People's Hospital (approve number: 2021PHA107-001).

Measures

Self-Reported Measures

Sleep Diaries

Participants completed sleep diaries[22] each morning during the baseline phase of the study (for at least 7 days). The sleep diaries recorded the following sleep parameters: (1) TST: total amount of time spent sleeping during the night, (2) SOL: time from lights-out to sleep onset, and (3) sleep efficiency (SE), calculated as TST divided by time in bed (TIB) × 100%. TIB represents the total amount of time spent in bed. Additionally, sleep onset difficulty and sleep quality were rated on a Likert scale, with higher scores indicating greater difficulty falling asleep and poorer sleep quality.

Questionnaires

Hamilton Rating Scale for Anxiety (HAMA): This 14-item scale assesses the severity of anxiety symptoms including two factors (psychic anxiety and somatic anxiety). Ratings are made on a 0–4 scale, with a total score ranging from 0 to 56. Higher scores indicate greater anxiety symptoms and a score of ≥14 suggests the presence of apparent anxiety.[23]

Hamilton Depression Rating Scale (HAMD): This 17-item scale measures the severity of depressive symptoms. Shafer et al[24] proposed a four-factor model comprising depression (Items 1, 2, 3, 7, 8), anxiety (Items 9, 10, 11, 15, 17), insomnia (Items 4, 5, 6), and somatic symptoms (Items 12, 13, 14, 16). Higher scores indicate greater depressive symptoms, with a score of ≥18 suggesting evident depressive symptoms. Item 3 evaluates suicidal behavior, with a score ≥3 indicating a higher suicide risk, leading to exclusion from the study.

Mini-Mental State Examination (MMSE):[25] The MMSE is a widely used screening instrument for assessing cognitive function, with a maximum score of 30. A score >24 indicates normal cognition.[20]

Insomnia Severity Index (ISI):[26] The ISI measures the self-reported severity and impact of insomnia symptoms. Each item is rated on a 5-point Likert scale (eg, 0 = no problem; 4 = very severe), covering aspects such as sleep onset and

 https://doi.org/10.2147/NSS.S570398

daytime dysfunction. Total scores range from 0 to 28, with higher scores indicating greater insomnia severity. The ISI was used to further support insomnia categorization.

Objective Measures

Two Consecutive Nights PSG Recordings

Participants who met the study criteria underwent two consecutive nights PSG recordings in the sleep laboratory. An initial adaptation night was conducted to familiarize participants with the laboratory environment and to exclude comorbid sleep disorders, such as obstructive sleep apnea (OSA; apnea-hypopnea index ≥15),[27] restless legs syndrome (RLS) or periodic leg movement index ≥15. During the study, patients were instructed to follow a regular bedtime (between 21:00 and 24:00) and maintain a consistent time in bed (6.5 to 9.0 hours). Each participant was monitored continuously using 16-channel polygraph to record electroencephalogram, electrooculogram, and electromyogram data. Additionally, respiration (via thermocouple and thoracic strain gauges), and oximeter data were collected. Sleep stages and events were visually scored by registered polysomnography technologists according to the American Academy of Sleep Medicine manual. The following PSG parameters were collected: TST, SE, SOL, WASO, rapid eye movement (REM) sleep latency (REMSL), sleep stage percentages (N1 stage, N2 stage, N3 stage, REM stage), arousal index (ArI), periodic leg movement index (PLMI), and apnea-hypopnea index (AHI).

Physical and Biochemical Parameters

Participants underwent a physical examination during their visit to the sleep laboratory, including measurement of height and weight to calculate body mass index (BMI, kg/m^2). Serum samples were collected between 6:00 and 8:00 a.m. and immediately transported to the hospital's laboratory center for analysis. Lipid profiles, TG, TC, LDL-C and HDL-C, were measured to assess lipid metabolism and cardiovascular risk.

Definition of PI

The definition of PI remains debated in clinical practice, but its primary feature is a marked discrepancy between subjective and objective sleep measures. In this study, PI was operationally defined by comparing subjective sleep data (averaged across a 7-day sleep diary) with objective PSG parameters collected across two consecutive nights. Specifically, PI was diagnosed in patients meeting ICSD-3 criteria for chronic insomnia who demonstrated substantial discrepancies between subjective complaints and PSG findings. The PSG-based diagnostic criteria for PI were defined as follows: 1) the TST ≥6.5 hours, 2) the SOL <30 minutes; or 3) the SE >85%.[4] Participants who fulfilled at least one of these PSG criteria despite reporting significant sleep difficulties in their diaries were classified as having PI.

Sample Size Considerations

The sample size was determined pragmatically by including all consecutive eligible patients who attended the sleep clinic during the study period, resulting in a final sample of 862 individuals. Previous study has reported that the prevalence of PI among patients with insomnia ranges from approximately 9.2% to 50%.[4] For a cross-sectional study estimating prevalence, the required sample size can be approximated by the standard formula $n = Z^2 \times \frac{P(1-P)}{d^2}$,[28] where Z is the Z value for a 95% confidence level (1.96), P is the expected prevalence, and d is the allowable error. Using an intermediate expected prevalence of 30% and a precision of ±5%, the minimum required sample size would be about 323 participants. Even under a conservative assumption of 50% prevalence, the required sample size would be approximately 384 participants. Our final sample of 862 thus substantially exceeds these theoretical requirements. In addition, around one-third of the sample met criteria for PI (≈260 outcome events). Given that the multivariable logistic regression models included 9 predictors (age, gender, HAMD subscales, HAMA subscales, and ISI), this corresponds to roughly 29 events per variable, which is well above the commonly recommended minimum of 10 events per variable for stable estimation of logistic regression coefficients.[29] These considerations indicate that the study had an adequate sample size to detect clinically meaningful associations between the examined predictors and PI status.

https://doi.org/10.2147/NSS.S570398

Data Analysis

All data were analyzed using R version 4.3.0 and R packages (eg, car, dplyr, tableone, ggplot2). Continuous data were presented as mean (standard deviation, SD), and categorical data were presented as numbers (percentage). Sociodemographic characteristics (age, gender, BMI) and sleep parameters were compared between the PI and non-PI groups using independent-samples *t* tests or chi-square tests, as appropriate. Pearson's correlation analyses were performed separately in the two groups to examine the relationships between self-reported and objective SOL and TST. Logistic regression analysis was used to examine factors associated with PI. PI status (1 = PI, 0 = non-PI) was treated as the dependent variable. Based on previous literature and clinical considerations,[14,15,30–32] age, gender, depressive and anxiety-related dimensions (HAMD and HAMA factors), and insomnia severity (ISI) were regarded as potential confounding or explanatory variables. In the multivariable model, age, gender, the four HAMD factor scores (depression, anxiety, insomnia, somatic symptoms), the two HAMA factor scores (psychic anxiety, somatic anxiety), and ISI total score were entered simultaneously as independent variables in order to estimate the independent association of each factor with PI while accounting for the others. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated from the regression coefficients. To explore the gender differences between non-PI and PI group, subgroup analysis was also conducted using independent *t*-tests or chi-square tests. All statistical tests were two-sided, and the level of significance was set at $p < 0.05$.

Results

Sample Characteristics and Prevalence of PI

A total of 862 individuals with insomnia, aged 19 to 64 years, were enrolled in this study, of whom 30.51% (n = 263) were categorized as PI. The majority of the participants were female (66.5%) and Han ethnicity (96.8%). Additional demographic and baseline characteristics are presented in Table 1.

Table 1 The Demographic and Clinical Characteristics of All the Participants

Variables	Total (n=862)	Non-Paradoxical Insomnia (n=599)	Paradoxical Insomnia (n=263)	t/x^2	P
Age, years	38.92 (12.11)	38.41 (12.06)	40.10 (12.14)	−1.889	0.059
Gender, n (%)				4.359	**0.037**
Male	289 (33.5)	187 (31.2)	102 (38.8)		
Female	573 (66.5)	412 (68.8)	161 (61.2)		
Ethnicity, n (%)				0.0003	0.986
Han	834 (96.8)	579 (96.7)	255 (97.0)		
Others	28 (3.2)	20 (3.3)	8 (3.0)		
BMI, kg/m²	22.25 (2.75)	22.29 (2.71)	22.09 (2.87)	0.755	0.451
HAMD	6.30 (2.51)	6.18 (2.37)	6.58 (2.81)	−2.050	**0.041**
Depression symptoms	0.57 (0.95)	0.53 (0.89)	0.66 (1.08)	−1.611	0.108
Anxiety symptoms	1.00 (1.17)	0.95 (1.15)	1.11 (1.23)	−1.882	0.060
Insomnia symptoms	4.33 (1.25)	4.35 (1.25)	4.27 (1.26)	0.795	0.427
Somatic symptoms	0.41 (0.72)	0.35 (0.66)	0.54 (0.82)	−3.331	**0.001**
HAMA	5.49 (2.60)	5.43 (2.50)	5.65 (2.81)	−1.117	0.265
Psychic anxiety	4.77 (1.94)	4.78 (1.89)	4.75 (2.04)	0.233	0.816
Somatic anxiety	0.73 (1.24)	0.65 (1.18)	0.90 (1.37)	−2.670	**0.008**
ISI	18.22 (3.26)	18.38 (3.16)	17.84 (3.46)	2.178	**0.030**
Biochemical parameters					
TG, mmol/L	1.19 (0.72)	1.19 (0.73)	1.21 (0.71)	−0.383	0.702
TC, mmol/L	4.79 (0.92)	4.80 (0.90)	4.76 (0.97)	0.548	0.584
HDL-C, mmol/L	1.41 (0.32)	1.42 (0.31)	1.40 (0.35)	0.689	0.492
LDL-C, mmol/L	2.83 (0.75)	2.84 (0.73)	2.81 (0.79)	0.505	0.614

Notes: Values are shown as mean (standard deviation) or number (percentage), and values in bold indicate P < 0.05.
Abbreviations: SD, Standard deviation; BMI, Body mass index; HAMD, Hamilton Depression Rating Scale; HAMA, Hamilton Rating Scale for Anxiety; ISI, Insomnia Severity Index; TG, Triglycerides; TC, Total cholesterol; HDL-C, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein.

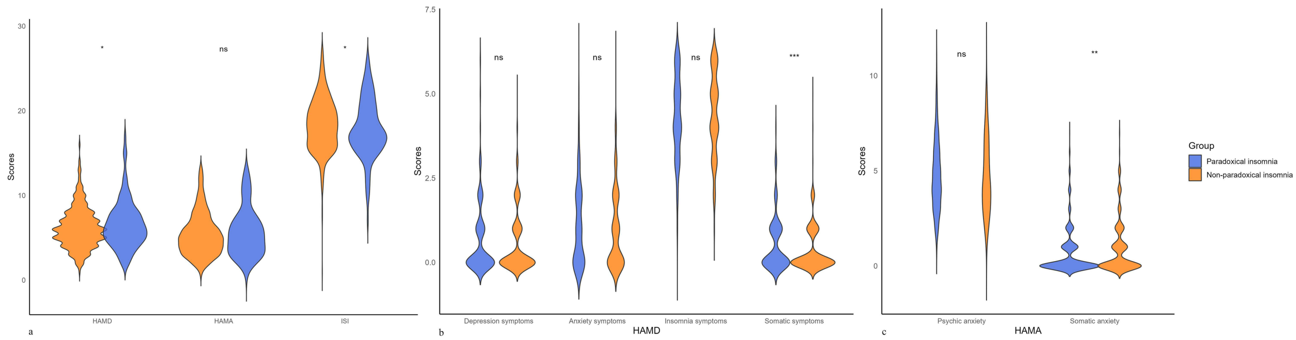

Figure 1 The comparison of depressive, anxiety and insomnia symptoms between paradoxical and non-paradoxical insomnia groups. (**a**) The depressive, anxiety and insomnia symptoms between paradoxical and non-paradoxical insomnia groups. (**b**) The subscales of HAMD between paradoxical and non-paradoxical groups. (**c**) The subscales of HAMA between paradoxical and non-paradoxical groups. The HAMD includes four subscales: depression, anxiety, insomnia and somatic symptoms. The HAMA includes two subscales: psychic anxiety and somatic anxiety. Statistical significance is indicated as follows: ***p < 0.001; **p < 0.01; *p < 0.05; ns p > 0.05.
Abbreviations: HAMD, Hamilton Depression Rating Scale; HAMA, Hamilton Rating Scale for Anxiety; ISI, Insomnia Severity Index.

Comparisons of Clinical, Subjective, Objective, and Biochemical Differences Between PI and Non-PI Groups

No significant difference in age was observed between the paradoxical and non-PI groups (p = 0.059). However, PI was more prevalent in males compared to the non-PI groups (p = 0.037) (Table 1). The severity of insomnia symptoms, as measured by the ISI, was greater in patients with non-PI compared to those with PI (18.38 ± 3.16 vs 17.84 ± 3.46, p = 0.030) (Figure 1a). Significant differences were identified in depressive symptoms (as measured by HAMD, p = 0.041) and in somatic anxiety (a subscale of HAMA, p = 0.008), with PI being associated with higher levels of depressive symptoms and somatic anxiety (Figure 1b and 1c). Biochemical parameters such as triglycerides and cholesterol levels, as well as BMI, showed no significant differences between the two groups.

The comparison of self-reported measure from sleep diaries revealed notable differences between paradoxical and non-PI groups. The PI group had a significantly shorter SOL compared to the non-PI group (62.41 ± 39.52 vs 73.75 ± 39.11, p < 0.001). Additionally, individuals with non-PI reported more difficulty falling asleep than those with PI (3.49 ± 0.59 vs 3.32 ± 0.78, p = 0.001), as well as longer self-reported sleep time than PI (423.81 ± 56.33 vs 413.13 ± 72.70, p = 0.035). Notably, no significant difference in self-reported sleep quality was detected between the groups (p = 0.560), indicating that participants in both groups perceived their overall sleep quality similarly.

For the objective measures based on PSG parameters, significant differences were found in TST, SOL, REMSL, WASO, SE, AHI, PLMI, REM stage percentage, N1 stage percentage and ArI (Table 2). Specifically, individuals with non-PI exhibited shorter TST, longer SOL, longer REMSL, longer WASO, lower SE, lower AHI, and lower PLMI

Table 2 The Comparison of Sleep Diaries and PSG Parameters Between Paradoxical and Non-Paradoxical Insomnia Groups

Variables	Total (n=862)	Non-Paradoxical Insomnia (n=599)	Paradoxical Insomnia (n=263)	t/x^2	P
Sleep diaries					
SOL, min	70.29 (39.56)	73.75 (39.11)	62.41 (39.52)	3.892	**< 0.001**
Sleep onset difficulty	3.44 (0.66)	3.49 (0.59)	3.32 (0.78)	3.285	**0.001**
Sleep quality	3.38 (0.68)	3.39 (0.65)	3.36 (0.75)	0.584	0.560
TIB, min	504.30 (40.56)	505.92 (38.06)	500.60 (45.61)	1.654	0.099
TST, min	420.56 (61.92)	423.81 (56.33)	413.13 (72.70)	2.117	**0.035**
SE, %	83.41 (10.37)	83.76 (9.15)	82.60 (12.72)	1.336	0.182

(*Continued*)

 https://doi.org/10.2147/NSS.S570398

Table 2 (Continued).

Variables	Total (n=862)	Non-Paradoxical Insomnia (n=599)	Paradoxical Insomnia (n=263)	t/x^2	P
PSG parameters					
TST, min	318.60 (67.16)	299.54 (66.13)	362.01 (46.02)	−15.944	**< 0.001**
SOL, min	67.09 (50.47)	86.61 (48.27)	22.64 (14.08)	29.688	**< 0.001**
REMSL, min	109.57 (52.68)	113.38 (54.34)	101.21 (47.90)	3.270	**0.001**
WASO, min	105.44 (53.09)	108.88 (55.01)	97.61 (47.61)	3.049	**0.002**
SE, %	66.54 (13.95)	62.59 (13.78)	75.52 (9.44)	−15.960	**< 0.001**
AHI, events per hour	2.81 (2.46)	2.64 (2.42)	3.21 (2.53)	−3.115	**0.002**
PLMI, events per hour	1.20 (1.96)	1.10 (1.80)	1.42 (2.28)	−2.047	**0.041**
Wake stage, min	111.93 (64.66)	120.53 (68.95)	92.26 (48.22)	6.896	**< 0.001**
REM stage, min	65.06 (24.04)	60.56 (23.15)	75.33 (22.88)	−8.683	**< 0.001**
N1 stage, min	44.83 (20.92)	43.77 (20.21)	47.26 (22.30)	−2.176	**0.030**
N2 stage, min	136.42 (41.80)	127.46 (39.95)	156.81 (38.70)	−10.153	**< 0.001**
N3 stage, min	72.47 (27.90)	67.91 (27.11)	82.86 (26.94)	−7.484	**< 0.001**
REM stage, %	19.99(5.83)	19.73(6.04)	20.59(5.27)	−2.096	**0.037**
N1 stage, %	14.72(8.21)	15.34(8.72)	13.31(6.7)	3.731	**< 0.001**
N2 stage, %	42.47(8.44)	42.21(8.63)	43.06(7.99)	−1.393	0.164
N3 stage, %	22.88(8.19)	22.78(8.42)	23.11(7.66)	−0.557	0.578
ArI, events per hour	3.98 (1.98)	4.09 (2.12)	3.76 (1.62)	2.500	**0.013**

Note: Values in bold indicate P < 0.05.
Abbreviations: PSG, Polysomnography; SOL, Sleep onset latency; TIB, Time in bed; TST, Total sleep time; SE, Sleep efficiency; REMSL, Rapid eye movement sleep latency; WASO, Wake after sleep onset; AHI, Apnea-hypopnea index; PLMI, Periodic leg movement index; REM, Rapid eye movement; ArI, Arousal index.

compared to those with PI. They also spent a greater proportion of sleep time in N1 sleep, a smaller proportion of sleep time in REM sleep, and experienced more wake time during the night, as reflected by a higher ArI.

Correlations Between Subjective and Objective Sleep Parameters in PI and Non-PI Patients

The correlation between self-reported and objective sleep parameters in the two groups revealed that, in the non-PI group, both self-reported SOL and TST were significantly correlated with their objective counterparts ($r = 0.152$, $p < 0.001$; $r = 0.139$, $p < 0.001$, respectively) (Figure 2a and Supplementary Table 1). In contrast, in the PI group, only self-reported TST showed a modest correlation with objective TST ($r = 0.145$, $p = 0.019$), while the correlation between self-reported and objective SOL was not significant ($r = 0.107$, $p = 0.083$) (Figure 2b and Supplementary Table 1).

Gender-Specific Differences Between PI and Non-PI Groups

A significant age difference was observed between males and females in both the PI groups (35.36 ± 11.22 vs 43.10 ± 11.76, $p < 0.001$) and non-PI (35.16 ± 10.59 vs 39.88 ± 12.41, $p < 0.001$) (Figure 3a). Concerning BMI, in both the paradoxical and non-PI groups, males had a slightly higher BMI than females (all $p < 0.05$) (Figure 3b). No gender differences in insomnia severity, as measured by ISI, were found in the non-PI group (18.33 ± 3.16 vs 18.50 ± 3.16, $p = 0.536$), whereas females had higher ISI scores compared to males (18.32 ± 3.08 vs 17.08 ± 3.88, $p = 0.007$) in the PI group (Figure 3c). For biochemical parameters, females exhibited higher HDL-C and higher TC levels compared to males, regardless of whether they were in the paradoxical or non-PI groups (all $p < 0.05$) (Figure 3d).

Gender differences in sleep diary metrics were minimal. There were no significant gender differences in SOL, TST, sleep quality, or sleep onset difficulty in either paradoxical or non-PI groups. However, in the non-PI group, males had shorter TIB compared to females (500.79 ± 34.48 vs 508.25 ± 39.39, $p = 0.020$). In contrast, the PI group did not show a significant difference in TIB between genders ($p = 0.581$). (Figure 3e).

 https://doi.org/10.2147/NSS.S570398

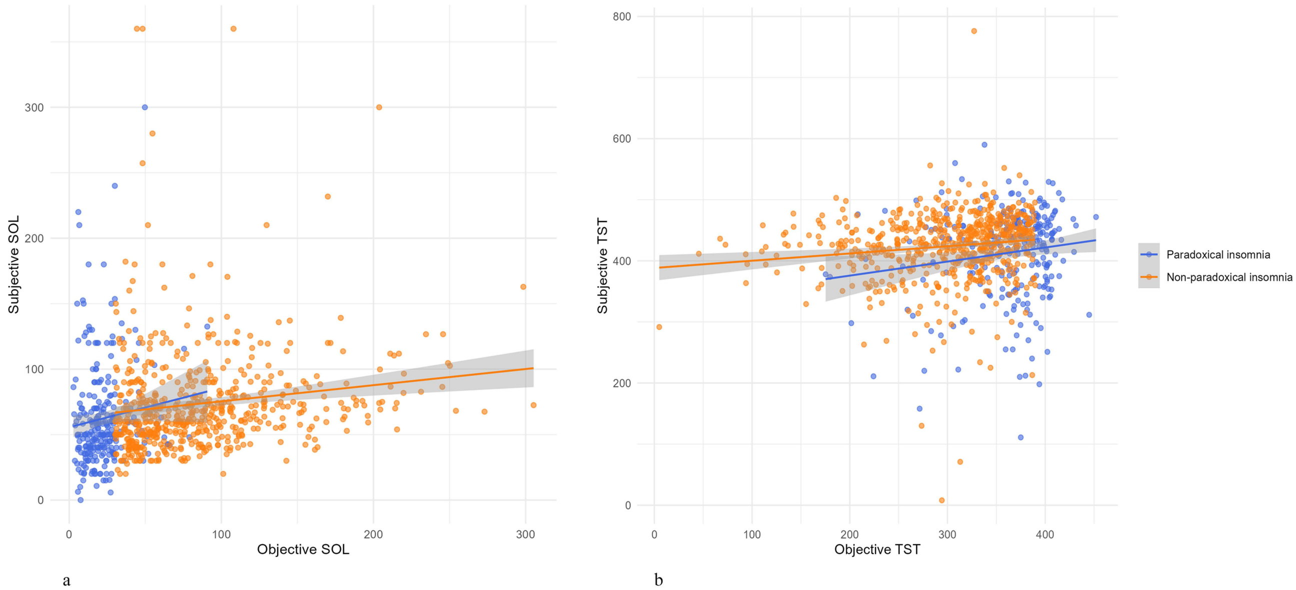

Figure 2 Correlation between self-reported and objective sleep parameters in the paradoxical and non-paradoxical insomnia groups. (**a**) Correlation between self-reported and objective sleep onset latency in the two groups. (**b**) Correlation between self-reported and objective total sleep times in the two groups. Self-reported SOL and TST refers to the data obtained from sleep diaries. Objective SOL and TST refers to the data obtained from polysomnography.
Abbreviations: SOL, Sleep onset latency; TST, Total sleep time.

PSG measures revealed some notable gender differences, particularly in the non-PI group, where males exhibited higher AHI than females (3.19 ± 2.43 vs 2.38 ± 2.37, $p < 0.001$) (Figure 3f). Moreover, males spent a significantly greater proportion of sleep time in N1 sleep compared to females (16.79 ± 10.83 vs 14.69 ± 7.49, p = 0.017) but a smaller proportion of time in N3 sleep (21.72 ± 8.31 vs 23.26 ± 8.44, p = 0.037). However, there were no significant gender differences in other stages of sleep (N2 stage or REM stage) in paradoxical or non-PI groups. More details were presented in Supplementary Table 2.

Multivariable Logistic Regression Analysis of Factors Associated with PI

Two-sample *t*-tests revealed that PI was associated with higher levels of depressive symptoms and somatic anxiety. Multivariable logistic regression analysis further identified key factors significantly associated with PI status. These included somatic symptoms (β = 0.318, p = 0.010), gender (β = −0.442, p = 0.007), age (β = 0.019, p = 0.004), and ISI scores (β = −0.054, p = 0.032) (Figure 4).

Discussion

PI, characterized by a marked discrepancy between subjective sleep complaints and objective sleep findings, remains a diagnostically challenging subtype of chronic insomnia due to its heterogeneous definitions and limited large-scale epidemiological data. In this cross-sectional study of 862 adults with chronic insomnia from a tertiary sleep medicine center, we examined the prevalence, clinical correlates, gender-specific differences, and independent predictors of PI. Our findings revealed that (a) PI was common, affecting almost one third of patients with chronic insomnia; (b) although women predominated in the overall insomnia sample, men were more likely than women to be classified as having PI; and (c) older age, male gender, lower ISI scores, and higher scores on the somatic symptoms factor of the HAMD were independently associated with PI. Taken together, these results confirm the substantial clinical burden of PI and provide new insights into its links with depressive somatic symptoms, sleep perception patterns, and gender-related differences, with important implications for the diagnosis and management of this underrecognized condition.

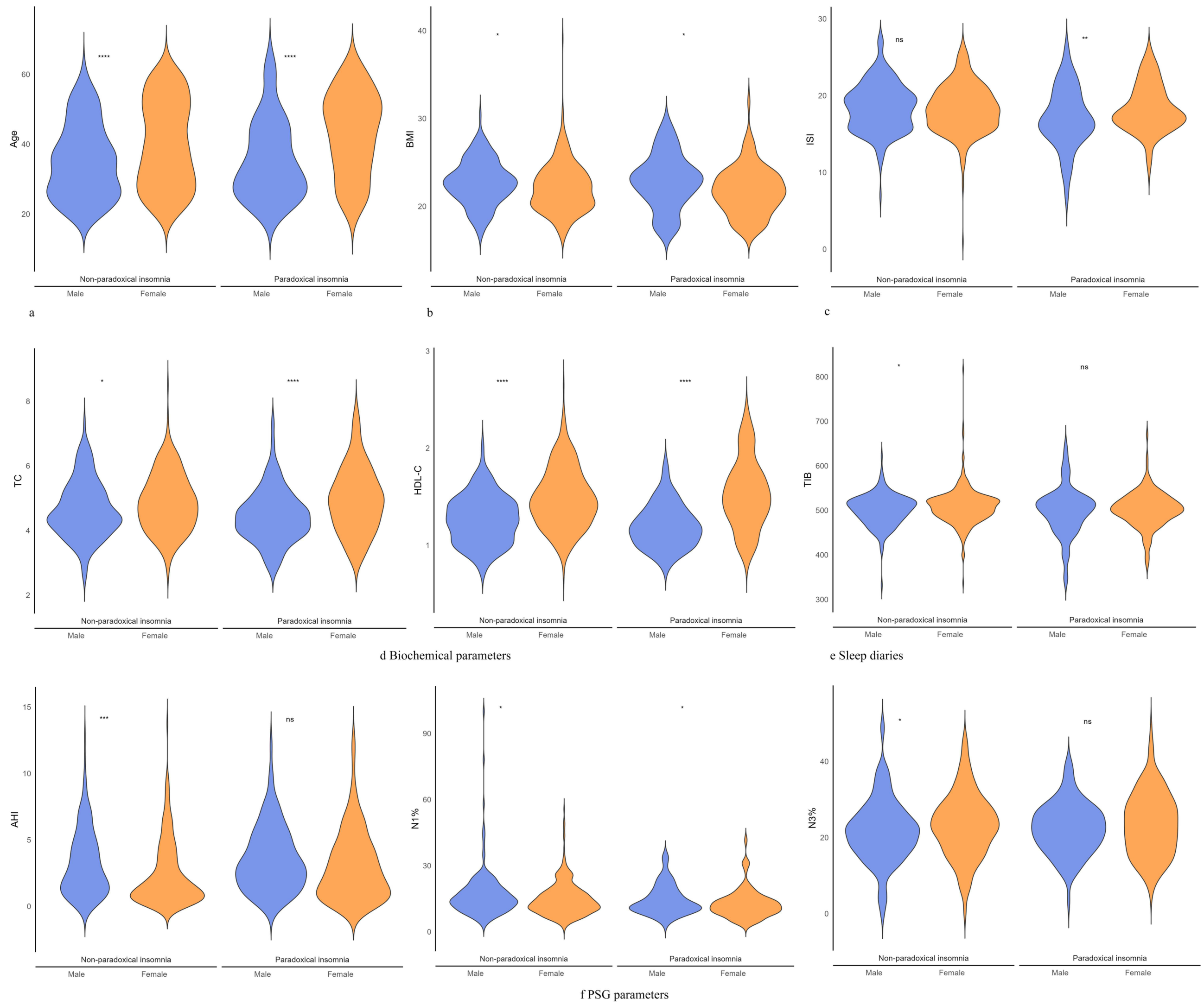

Figure 3 Gender differences in demographic, clinical characteristics, self-reported and objective sleep parameters between paradoxical and non-paradoxical insomnia groups. (**a**) Gender differences in age between paradoxical and non-paradoxical insomnia groups. (**b**) Gender differences in BMI between paradoxical and non-paradoxical insomnia groups. (**c**) Gender differences in ISI between paradoxical and non-paradoxical insomnia groups. (**d**) Gender differences in biochemical parameters between paradoxical and non-paradoxical insomnia groups. (**e**) Gender differences in sleep diaries between paradoxical and non-paradoxical insomnia groups. (**f**) Gender differences in PSG parameters between paradoxical and non-paradoxical groups. Statistical significance is indicated as follows: **** $p < 0.0001$; *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; ns $p > 0.05$.

Abbreviations: BMI, Body mass index; ISI, Insomnia Severity Index; TC, Total cholesterol; HDL-C, High-density lipoprotein cholesterol; TIB, Time in bed; TST, Total sleep time; PSG, Polysomnography; AHI, Apnea-hypopnea index.

Prevalence and Overall Clinical Profile of PI

In this tertiary sleep clinic cohort, PI was common, affecting almost one third of adults with chronic insomnia (30.5%). In Asia, another Korean study reported a prevalence of PI of approximately 26.4%, which is lower than that in our study.[6] This is broadly consistent with previous estimates of PI, which have ranged from approximately 9% to 50%[4] depending on diagnostic criteria, setting, and assessment methods. This study extends these observations to a large, well-characterized Chinese cohort. This high prevalence underscores that PI is not a rare variant of insomnia but a common clinical presentation, highlighting the need for routine objective sleep assessment (eg, PSG) to distinguish PI from non-PI, a distinction often missed when relying solely on subjective reports.

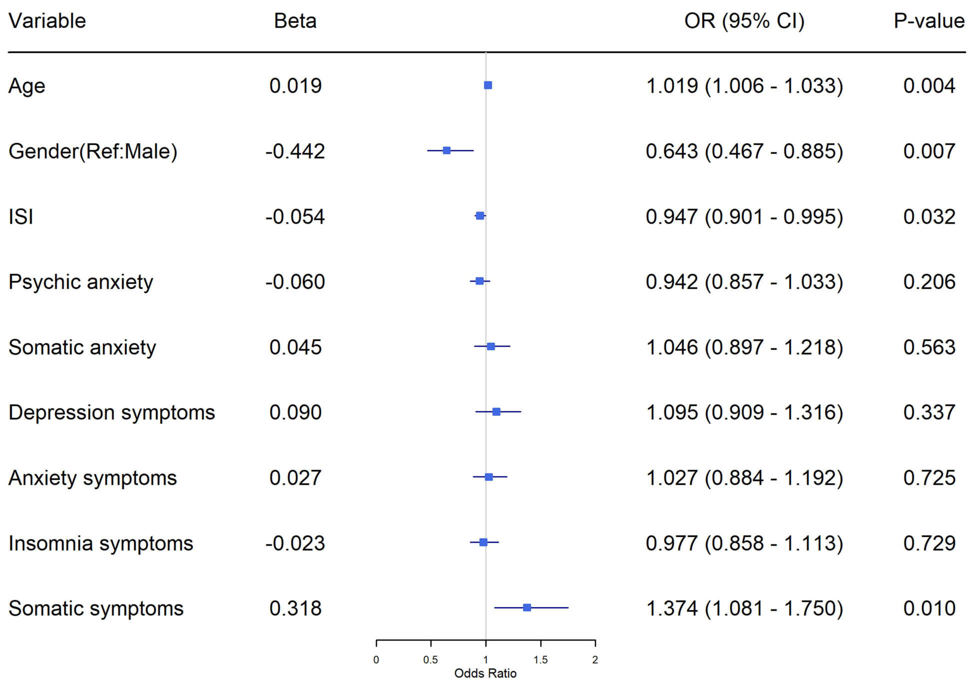

Figure 4 Logistic regression analysis of demographic and clinical factors on the risks of paradoxical insomnia. Paradoxical insomnia status (1 = present, 0 = absent) was the dependent variable, with age, gender (1 = female, 0 = male), HAMD subscales (depression, anxiety, insomnia, somatic symptoms), HAMA subscales (psychic anxiety, somatic anxiety), and the ISI as independent variables. Somatic symptoms were a subscale of Hamilton Depression Rating Scale. Somatic anxiety was a subscale of Hamilton Rating Scale for Anxiety.
Abbreviations: HAMD, Hamilton Depression Rating Scale; HAMA, Hamilton Rating Scale for Anxiety; ISI, Insomnia Severity Index; OR, odds ratio; CI, confidence interval.

Comparative Clinical, Psychological, and Sleep Characteristics Between PI and Non-PI

Consistent with prior reports[12,13] that insomnia is more frequent in women, two thirds of our overall sample were female. However, among patients with chronic insomnia, 35.3% of men (102/289) met criteria for PI, compared with 28.1% of women (161/573), and male gender remained an independent correlate of PI in the multivariable model. These findings suggest that although women are more likely to present with chronic insomnia, men with insomnia may be more prone to develop a PI phenotype.

Compared with patients with non-PI, those with PI showed a broadly similar demographic profile but a somewhat more pronounced symptom burden in specific domains. In line with previous work,[32] we found that patients with PI exhibited greater depression and anxiety emotional burden than those with non-PI. In univariable comparisons, PI was associated with higher total HAMD scores driven mainly by the somatic symptoms factor, and with higher HAMA somatic anxiety scores, suggesting that both depressive and anxiety-related distress are linked to sleep-state misperception,[15,33] which represents the clinical core feature of PI. Our multivariable analysis further refined this picture by showing that the somatic symptoms factor of the HAMD, rather than the core depressive mood or anxiety factors, remained independently associated with PI after adjusting for age, gender, other HAMD and HAMA dimensions, and ISI scores. This pattern implies that somatic manifestations of depression, such as fatigue, bodily discomfort, and general somatic complaints, may be particularly relevant to the development or maintenance of PI. Consistent with previous studies on anxiety and depressive disorders linking higher emotional distress to greater underestimation of sleep duration and overestimation of nocturnal wakefulness.[15,33] Furthermore, patients with PI may experience a heightened state of hyperarousal,[34] which likely contributes to this distorted perception of sleep. Our findings reinforce the notion that patients who are highly sensitive to bodily sensations and interpret them negatively tend to perceive their sleep quality as far worse than objective sleep parameters reflect.

From a clinical standpoint, these observations underscore the need to address emotional distress and somatic depressive symptoms when managing PI. Most studies of cognitive-behavioral therapy for insomnia (CBT-I) have focused on its effects on sleep parameters and daytime functioning in patients with insomnia,[27,35,36] whereas fewer have specifically examined changes in sleep misperception. Our results, together with previous evidence, suggest that incorporating targeted cognitive restructuring and psychoeducation about sleep perception may be particularly beneficial for patients with PI. For example, Dzierzewski et al reported that CBT-I improved the accuracy of sleep and wake perception and reduced subjective-objective sleep discrepancies in older adults with insomnia,[37] which may partly account for improvements in perceived sleep quality. Tailoring CBT-I components to challenge catastrophic interpretations of bodily sensations and to recalibrate expectations about sleep might therefore be a promising strategy for reducing sleep misperception in PI.

Interestingly, despite comparable or greater emotional and somatic symptom burden, PI patients reported slightly lower insomnia severity on the ISI than non-PI patients. This finding is intuitive: non-PI is defined by congruence between subjective complaints and objective sleep impairment (eg, shorter TST, longer SOL on PSG), so their self-reported severity (ISI) aligns with worse objective sleep. In contrast, PI patients' lower ISI scores may reflect a "disconnect" between their perceived sleep difficulty and relatively preserved objective sleep, suggesting that ISI alone is insufficient to distinguish PI from non-PI, and objective measures (eg, PSG) are critical for accurate diagnosis.

With respect to biochemical parameters, no significant differences in TG, TC, HDL-C, or LDL-C were observed between PI and non-PI. Although chronic insomnia has been linked to increased cardiometabolic risk in previous studies,[16,17] the absence of subtype differences in lipid profiles in our study may indicate that PI is not characterized by additional lipid abnormalities beyond those associated with chronic insomnia in general. This supports the view that the core features of PI lie primarily in the domain of sleep perception and psychological functioning rather than in distinct alterations of lipid metabolism. Future longitudinal studies with broader metabolic panels will be important to determine whether PI confers any specific long-term cardiometabolic risk or whether its impact on physical health is mediated indirectly through chronic stress and lifestyle factors.

These patterns establish a clear clinical profile for PI, which impacts nearly one-third of chronic insomnia patients as a considerable proportion that has historically been underrecognized. Compared with non-PI, patients with PI are characterized by slightly older age, a relatively higher proportion men (although women still constitute the majority of chronic insomnia cases), more prominent depressive symptoms and somatic anxiety, and somewhat lower perceived insomnia severity.

Subjective-Objective Sleep Discrepancies: A Core Feature of PI

The defining hallmark of PI, discrepancy between subjective and objective sleep parameters, was confirmed in our study, with distinct patterns between PI and non-PI groups. In non-PI group, self-reported SOL and TST showed weak but statistically significant correlations with their PSG counterparts, suggesting a relatively more accurate, although still imperfect, perception of sleep continuity. In contrast, among patients with PI, only subjective TST was modestly correlated with objective TST, whereas the association between subjective and objective SOL did not reach statistical significance. This pattern suggests that sleep misperception in PI may be particularly pronounced for sleep onset, while perception of overall sleep duration is somewhat better preserved.

Why might PI patients misperceive SOL but not TST? Several mechanisms may account for this selective distortion of SOL perception. Sleep onset is a is a brief and internally defined transition between wakefulness and sleep that depends heavily on the integration of sensory input and conscious awareness.[38] Patients with PI may engage in heightened "sleep monitoring" during this transition, consistent with neurocognitive models of psychophysiological insomnia,[39] leading them to overestimate the time spent awake before sleep. In line with this view, electroencephalographic studies have shown that individuals who markedly under- or overestimate their sleep duration exhibit more wake-like cortical activity during non-REM sleep, suggesting that subtle persistence of wake-like EEG patterns may contribute to distorted sleep perception.[40] In contrast, TST reflects a cumulative estimate of time asleep across the night, and even with impaired onset perception of sleep onset, individuals may still retain a rough sense of how much of the night was spent asleep versus awake, which could explain the modest TST correlations observed in both groups. Clinically, these findings suggest that discrepancies in SOL, rather than TST alone, may be a particularly sensitive marker of PI and should be routinely assessed using both sleep diaries and objective measures.

https://doi.org/10.2147/NSS.S570398

Objective PSG findings further reinforce the notion that PI is characterized by relatively preserved sleep physiology. Compared with non-PI, PI patients exhibited longer TST, markedly shorter SOL, higher sleep efficiency, and a lower arousal index, despite comparable or greater subjective complaints of poor sleep (Table 2). Non-PI patients, by contrast, exhibited the classic pattern of objective insomnia described in the ICSD-3: shorter TST, longer SOL/WASO, lower SE, and higher ArI, corresponding to "insomnia disorder with objective sleep disturbance".[3] These differences have important therapeutic implications. While patients with non-PI may benefit from interventions targeting sleep continuity, patients with PI may derive greater benefit from cognitive-behavioral approaches that focus on recalibrating sleep perception and modifying maladaptive monitoring and interpretation of nighttime experiences. Randomized studies demonstrate that CBT-I enhances subjective-objective perception accuracy in patients with chronic insomnia,[37,41] supporting its role as first-line intervention for PI and other conditions with prominent sleep misperception.

Gender-Specific Differences in Patients with PI

In the PI literature, detailed gender-stratified phenotyping within PI versus non-PI remains limited, despite the broader recognition that PI/sleep state misperception represents a subjective-objective discrepancy rather than a uniform PSG-defined disorder.[4] In our cohort, men were younger and had slightly higher BMI than women in both PI and non-PI groups, suggesting that these differences more likely reflect gender-related background characteristics and the structure of patients presenting to sleep services than PI-specific discriminators. Such patterns are compatible with sex- and age-related differences observed in sleep-center populations[42] and with broader evidence that insomnia is more prevalent in women across the life span, with heightened vulnerability at specific life stages.[43] Meanwhile, sex differences in body composition and fat distribution provide a biological basis for modestly higher BMI in men.[44,45]

As mentioned before, the comparison of biochemical parameters revealed no significant differences in TG, TC, HDL-C, or LDL-C levels between the PI and non-PI groups. Interestingly, females had higher HDL-C and TC than males in both groups. This is likely due to hormonal differences (eg, estrogen's protective effect on HDL-C).[44] Given prior evidence that chronic insomnia is associated with cardiometabolic risk, our findings suggest that PI may not be characterized by additional metabolic disturbance beyond that of chronic insomnia itself, and that its core mechanisms are more likely to be psychological and perceptual. Longitudinal studies incorporating broader metabolic indices are needed to determine whether PI has any distinct long-term cardiometabolic implications.

In the PI group, females reported higher ISI scores than males despite broadly comparable PSG indices, suggesting a greater subjective burden in women under conditions of relatively preserved objective sleep. This finding may be attributed to females' greater tendency to report emotional distress, which in turn exacerbates the subjective experience of sleep difficulty. This observation aligns with prior literature indicating that women often endorse more sleep complaints and distress than men.[43] In the non-PI group, males showed slightly higher AHI and a shift toward lighter sleep (higher N1, lower N3), consistent with established sex differences in sleep-disordered breathing and sleep architecture.[46,47] Notably, these PSG gender differences were attenuated within PI. Rather than implying that PI "eliminates" gender effects, a parsimonious explanation is that PI is defined by pronounced subjective-objective discrepancy with relatively preserved macrostructural PSG metrics, which may restrict variability in stage composition and respiratory indices, making typical gender patterns less apparent.

Factors Associated with PI

Multivariable logistic regression showed that four variables were independently associated with PI in this chronic insomnia cohort: higher somatic symptoms (HAMD somatic subscale), male gender, older age, and lower ISI scores. These findings may be interpreted as correlates of the PI phenotype. Higher somatic symptom on the HAMD were independently associated with PI, suggesting that PI may co-occur with more prominent bodily distress rather than reflecting symptom exaggeration alone.[4,48] Given our stringent eligibility criteria, which excluded clinically significant depressive/anxiety episodes, comorbid sleep disorders such as OSA and RLS, recent use of sleep-affecting medications, and excessive alcohol/caffeine intake, this association is unlikely to be driven simply by objectively fragmented sleep secondary to other sleep disorders. Instead, it may indicate a PI phenotype characterized by subthreshold somatic

https://doi.org/10.2147/NSS.S570398

hyperarousal and heightened monitoring of internal bodily signals. This interpretation is consistent with the hyperarousal framework of insomnia,[49] in which sustained cortical-autonomic activation can increase attention to brief arousals and bodily sensations, biasing sleep-wake state appraisal and amplifying subjective-objective discrepancy.

PSG/EEG evidence further supports a link between sleep misperception and arousal-related neurophysiology (eg, elevated fast-frequency activity around sleep initiation and autonomic correlates), compatible with the idea that bodily arousal and its salience may distort perceived sleep.[50] More broadly, interoception-based accounts propose that the salience and interpretation of internal sensations (including discomfort and autonomic cues) can shape conscious sleep experience and contribute to insomnia-related symptom perception,[51] providing a plausible cognitive-neurobiological context for the observed somatic–PI association. Clinically, these findings highlight the value of routinely assessing somatic distress and physiological arousal in suspected PI and, alongside standard CBT-I components, considering strategies that reduce somatic hyperarousal and recalibrate interpretations of bodily sensations (eg, relaxation-based techniques and cognitive work targeting somatic catastrophizing).

Male gender remained associated with PI after adjustment in our cohort. This pattern does not negate the well-established observation that insomnia overall is more common in women[42,43] and that women with insomnia symptoms may be more likely to seek professional help.[52] Instead, it suggests that men may be relatively overrepresented among patients in a clinical insomnia sample undergoing PSG-based phenotyping when their subjective complaints diverge from conventional PSG-defined sleep metrics. Because the PI/sleep-state misperception literature is still limited and definitions vary across studies,[4] gender-related findings in this area have not been consistently characterized, making clinic-based multivariable results like ours useful for hypothesis generation. One contributing consideration is cohort composition shaped by our design and clinical pathways: we excluded patients with comorbid sleep disorders such as OSA and RLS, conditions that show gender-skewed prevalence in both epidemiologic and sleep-center samples (eg, higher OSA in men and higher RLS in women).[42] Such exclusions may preferentially remove gender-specific sources of objectively disturbed sleep and thereby enrich a mismatch phenotype among those who remain for PSG-based phenotyping. In parallel, PI/sleep-state misperception has been linked to hyperarousal and dysfunctional sensory-gating processes[53] that can generate substantial subjective complaints without prominent abnormalities on conventional PSG summaries, providing a plausible pathway for subjective-objective discordance in either gender. Finally, socio-cultural influences on symptom framing and help-seeking may further shape which patients present for specialist evaluation and how distress is communicated,[43] potentially affecting the recognition of PI in clinical settings.

Older age emerged as an independent factor associated with PI in our multivariable model. Prior work[6,54,55] on age and sleep-state misperception has been mixed, partly because "PI/sleep discrepancy" has been operationalized inconsistently across studies. A plausible interpretation in our cohort (18–64 years, with major comorbid sleep disorders such as OSA/RLS excluded) is that age is acting as a proxy for gradual changes in sleep neurophysiology and sleep-related information processing rather than simply reflecting comorbidity burden. This suggests that, even among otherwise "uncomplicated" insomnia patients, increasing age may signal greater vulnerability to subjective-objective mismatch, reinforcing the need to evaluate sleep discrepancy directly rather than inferring severity from symptoms alone.

PI was associated with less severe subjective insomnia (lower ISI), which contrasts with non-PI but aligns with PI's defining feature (subjective-objective discrepancy). This highlights that a "mild" ISI score does not rule out clinically significant insomnia, objective testing is still necessary. Lower ISI scores were independently associated with PI in our model. Although this may appear counterintuitive, the ISI operationalizes insomnia severity as a multidimensional patient-reported construct (eg, sleep difficulties, worry/distress, and daytime interference) and is intended to complement other components of insomnia assessment rather than replace them.[26,56] In a mismatch phenotype such as PI, where objective sleep can be relatively preserved despite prominent sleep complaints, patients may endorse less pervasive daytime impairment or global severity on the ISI, yielding lower total scores even when their perceived sleep is discordant with objective findings. This interpretation is consistent with the defining feature of PI/sleep-state misperception as a pronounced subjective-objective discrepancy rather than uniformly "more severe" insomnia.[4] This suggests that an ISI score in the mild-to-moderate range should not be used to exclude PI; when symptom reports seem disproportionate to diary patterns or when a discrepancy phenotype is suspected, evaluation should integrate sleep diaries and,

 https://doi.org/10.2147/NSS.S570398

when clinically indicated, objective measures (eg, actigraphy/PSG) to characterize mismatch and exclude alternative sleep disorders.

Limitation

This study has several limitations. First, the cross-sectional design limits causal inferences between PI and its associated factors. Longitudinal studies are needed to establish temporal relationships. Second, due to our stringent exclusion criteria, participants with psychiatric disorders (eg, anxiety or depression) and medication use were excluded from the sample, which may limit the generalizability of our findings. However, this rigorous approach aligns with our primary objective of exploring the relationship between PI and insomnia without confounding factors. Future studies using more diverse, community-based samples would help verify and extend these findings. Third, although gender differences were examined, the study did not measure potentially relevant biological factors, such as menopausal status or hormonal fluctuations, nor did it address cultural attitudes towards sleep perception. These factors should be explored in future studies to provide a more nuanced and comprehensive understanding of PI.

Conclusion

In a large cohort of Chinese adults with chronic insomnia, nearly one-third met criteria for PI. PI showed a distinct clinical profile, including gender-related presentation patterns, and in multivariable analyses was independently associated with higher HAMD somatic-symptom burden, male gender, older age, and lower ISI scores. These findings emphasize that PI cannot be reliably characterized by symptom severity questionnaires alone and that accurate identification benefits from integrating structured clinical assessment (including somatic/psychiatric symptom dimensions) with objective sleep evaluation (eg, actigraphy/PSG) when clinically indicated. Recognizing PI as a mismatch phenotype can help clinicians prioritize interventions targeting sleep perception, hyperarousal/somatic distress, and gender-sensitive symptom presentation, thereby improving management of this challenging insomnia subtype.

Abbreviations

AHI, Apnea-Hypopnea Index; ArI, Arousal Index; BMI, Body Mass Index; HAMA, Hamilton Rating Scale for Anxiety; HAMD, Hamilton Depression Rating Scale; HDL-C, High-Density Lipoprotein Cholesterol; ICSD-3, International Classification of Sleep Disorders, Third Edition; ISI, Insomnia Severity Index; LDL-C, Low-Density Lipoprotein Cholesterol; MMSE, Mini-Mental State Examination; PLMI, Periodic Leg Movement Index; PSG, Polysomnographic; REM, Rapid Eye Movement; REMSL, Rapid Eye Movement Sleep Latency; RLS, Restless Legs Syndrome; SE, Sleep Efficiency; SSM, Sleep State Misperception; SOL, Sleep Onset Latency; TG, Triglycerides; TIB, Time in Bed; TST, Total Sleep Time; WASO, Wake After Sleep Onset.

Data Sharing Statement

Data are available upon reasonable request to corresponding author Fulong Xiao (email: xiaofulong1681@163.com).

Ethics Approval and Consent to Participate

All participants provided written informed consent. This study was approved by Ethics Review Committee of Peking University People's Hospital, and all participants provided written informed consent (approval number: 2021PHA107-001).

Acknowledgments

The authors thank all participants for their time and excellent cooperation in this study.

Author Contributions

Mengmeng Wang: Data curation, Methodology, Formal analysis, Writing – original draft, Writing – review & editing. Wenjun Zhu: Data curation, Formal analysis, Investigation, Methodology, Formal analysis, Writing – review & editing. Yunliang Sun: Data curation, Investigation, Methodology, Writing – review & editing. Zhaoyan Feng: Data curation,

Investigation, Methodology, Writing – review & editing. Yanyan Hou: Data curation, Investigation, Writing – review & editing. Huanhuan Wang: Data curation, Investigation, Writing – review & editing. Bing Zhou: Data curation, Investigation, Writing – review & editing. Fang Han: Methodology, Supervision, Writing – review & editing Validation. Fulong Xiao: Conceptualization, Supervision, Formal analysis, Funding acquisition, Project administration, Writing – review & editing. All authors agreed to the final version submitted for publication; agreed to submit this work to Nature and Science of Sleep, and agree to be accountable for the content of this paper.

Funding

This study was supported by the National Natural Science Foundation of China (No. 82400114, No. 82470087, No. 82070091 and No. 82341243).

Disclosure

The authors declare no conflicts of interest related to this study.

References

1. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev*. 2002;6(2):97–111. doi:10.1053/smrv.2002.0186
2. Morin CM, Jarrin DC. Epidemiology of insomnia: prevalence, course, risk factors, and public health burden. *Sleep Med Clin*. 2022;17(2):173–191. doi:10.1016/j.jsmc.2022.03.003
3. Sateia MJ. International classification of sleep disorders-third edition. *Chest*. 2014;146(5):1387–1394. doi:10.1378/chest.14-0970
4. Rezaie L, Fobian AD, McCall WV, Khazaie H. Paradoxical insomnia and subjective-objective sleep discrepancy: a review. *Sleep Med Rev*. 2018;40:196–202. doi:10.1016/j.smrv.2018.01.002
5. Jansson-Fröjmark M, Alfonsson S, Bohman B, Rozental A, Norell-Clarke A. Paradoxical intention for insomnia: a systematic review and meta-analysis. *J Sleep Res*. 2022;31(2):e13464. doi:10.1111/jsr.13464
6. Moon HJ, Song ML, Cho YW. Clinical characteristics of primary insomniacs with sleep-state misperception. *J Clin Neurol*. 2015;11(4):358–363. doi:10.3988/jcn.2015.11.4.358
7. Edinger JD, Krystal AD. Subtyping primary insomnia: is sleep state misperception a distinct clinical entity? *Sleep Med Rev*. 2003;7(3):203–214. doi:10.1053/smrv.2002.0253
8. Manconi M, Ferri R, Sagrada C, et al. Measuring the error in sleep estimation in normal subjects and in patients with insomnia. *J Sleep Res*. 2010;19(3):478–486. doi:10.1111/j.1365-2869.2009.00801.x
9. Benbir Şenel G, Aydın Ö, Tanrıöver Aydın E, Bayar MR, Karadeniz D. Changes in sleep structure and sleep spindles are associated with the neuropsychiatric profile in paradoxical insomnia. *Int J Psychophysiol*. 2021;168:27–32. doi:10.1016/j.ijpsycho.2021.07.626
10. Yung KP, Chung KF, Ho FYY, Yeung WF, Ng THY. The experience of chronic insomnia in Chinese adults: a study using focus groups and insomnia experience diaries. *Behav Sleep Med*. 2016;14(4):406–428. doi:10.1080/15402002.2015.1017097
11. Zeng LN, Zong QQ, Yang Y, et al. Gender difference in the prevalence of insomnia: a meta-analysis of observational studies. *Front Psychiatry*. 2020;11:577429. doi:10.3389/fpsyt.2020.577429
12. Zhang B, Wing YK. Sex differences in insomnia: a meta-analysis. *Sleep*. 2006;29(1):85–93. doi:10.1093/sleep/29.1.85
13. Baldi E, Cerolini S, Meneo D, Baglioni C, Palagini L. Insomnia disorder: gender issues over the lifespan. *J Sleep Res*. 2025;34(5):e70110. doi:10.1111/jsr.70110
14. Yoon G, Lee MH, Oh SM, Choi JW, Yoon SY, Lee YJ. Negative and positive sleep state misperception in patients with insomnia: factors associated with sleep perception. *J Clin Sleep Med*. 2022;18(7):1789–1795. doi:10.5664/jcsm.9974
15. Kawai K, Iwamoto K, Miyata S, et al. A study of factors causing sleep state misperception in patients with depression. *Nat Sci Sleep*. 2022;14:1273–1283. doi:10.2147/NSS.S366774
16. Zhang Y, Jiang X, Liu J, Lang Y, Liu Y. The association between insomnia and the risk of metabolic syndrome: a systematic review and meta-analysis. *J Clin Neurosci*. 2021;89:430–436. doi:10.1016/j.jocn.2021.05.039
17. Kim JW, Jain AB, Khullar A. Insomnia and cardiometabolic health: bridging the gap between sleep deficit and disease prevention. *Can J Diabetes*. 2025;49(5–6):342–350. doi:10.1016/j.jcjd.2025.04.008
18. Jia F, Shi SY, Fei SF, Zhou M, Li JJ. Association of insomnia, lipid profile, and lipid-lowering medications: a narrative review. *Rev Cardiovasc Med*. 2025;26(1):24978. doi:10.31083/RCM24978
19. Lichstein KL, Durrence HH, Taylor DJ, Bush AJ, Riedel BW. Quantitative criteria for insomnia. *Behav Res Ther*. 2003;41(4):427–445. doi:10.1016/S0005-7967(02)00023-2
20. Zheng C, Liu Y, Xie S, Zeng S, Wu Q. The mediating role of depression and disability in the association between widowhood and cognitive function among elderly Chinese individuals. *J Psychiatr Res*. 2025;181:1–6. doi:10.1016/j.jpsychires.2024.11.026
21. Ren W, Zhang N, Sun Y, et al. The REM microarousal and REM duration as the potential indicator in paradoxical insomnia. *Sleep Med*. 2023;109:110–117. doi:10.1016/j.sleep.2023.06.011
22. Carney CE, Buysse DJ, Ancoli-Israel S, et al. The consensus sleep diary: standardizing prospective sleep self-monitoring. *Sleep*. 2012;35(2):287–302. doi:10.5665/sleep.1642
23. Matza LS, Morlock R, Sexton C, Malley K, Feltner D. Identifying HAM-A cutoffs for mild, moderate, and severe generalized anxiety disorder. *Int J Meth Psych Res*. 2010;19(4):223–232. doi:10.1002/mpr.323

24. Shafer AB. Meta-analysis of the factor structures of four depression questionnaires: beck, CES-D, Hamilton, and Zung. *J Clin Psychol*. 2006;62 (1):123–146. doi:10.1002/jclp.20213
25. Tsoi KKF, Chan JYC, Hirai HW, Wong SYS, Kwok TCY. Cognitive tests to detect dementia: a systematic review and meta-analysis. *JAMA Intern Med*. 2015;175(9):1450–1458. doi:10.1001/jamainternmed.2015.2152
26. Morin CM, Belleville G, Bélanger L, Ivers H. The insomnia severity index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*. 2011;34(5):601–608. doi:10.1093/sleep/34.5.601
27. Sweetman A, McEvoy RD, Smith S, et al. The effect of cognitive and behavioral therapy for insomnia on week-to-week changes in sleepiness and sleep parameters in patients with comorbid insomnia and sleep apnea: a randomized controlled trial. *Sleep*. 2020;43(7):zsaa002. doi:10.1093/sleep/zsaa002
28. Arya R, Antonisamy B, Kumar S. Sample size estimation in prevalence studies. *Indian J Pediatr*. 2012;79(11):1482–1488. doi:10.1007/s12098-012-0763-3
29. Vittinghoff E, McCulloch CE. Relaxing the rule of ten events per variable in logistic and cox regression. *Am J Epidemiol*. 2007;165(6):710–718. doi:10.1093/aje/kwk052
30. Hermans LWA, van Gilst MM, Regis M, et al. Modeling sleep onset misperception in insomnia. *Sleep*. 2020;43(8):zsaa014. doi:10.1093/sleep/zsaa014
31. De las Cuevas C, Segovia M. The sleep expectation-reality gap: exploring discrepancies between perceived and ideal sleep duration in primary care patients. *Health Care*. 2025;13(6). doi:10.3390/healthcare13060650
32. Liao J, Zhu S, Li X. Anxiety and depression in paradoxical insomnia: a case-control study. *Neuropsych Dis Treat*. 2018;14:231–238. doi:10.2147/NDT.S156058
33. Liang Y, Zhao X, Zhang C, et al. Sleep misperception and associated factors in patients with anxiety-related disorders and complaint of insomnia: a retrospective study. *Front Neurol*. 2022;13:836949. doi:10.3389/fneur.2022.836949
34. Pérusse AD, Pedneault-Drolet M, Rancourt C, Turcotte I, St-Jean G, Bastien CH. REM sleep as a potential indicator of hyperarousal in psychophysiological and paradoxical insomnia sufferers. *Int J Psychophysiol*. 2015;95(3):372–378. doi:10.1016/j.ijpsycho.2015.01.005
35. Shaffer KM, Hedeker D, Morin CM, Ingersoll K, Thorndike F, Ritterband LM. Intraindividual variability in sleep schedule: effects of an internet-based cognitive-behavioral therapy for insomnia program and its relation with symptom remission. *Sleep*. 2020;43(12):zsaa115. doi:10.1093/sleep/zsaa115
36. Suh S, Nowakowski S, Bernert RA, et al. Clinical significance of night-to-night sleep variability in insomnia: exploring the variability of sleep schedules and insomnia symptoms. *Sleep Med*. 2012;13(5):469–475. doi:10.1016/j.sleep.2011.10.034
37. Dzierzewski JM, Martin JL, Fung CH, et al. CBT for late-life insomnia and the accuracy of sleep and wake perceptions: results from a randomized-controlled trial. *J Sleep Res*. 2019;28(4):e12809. doi:10.1111/jsr.12809
38. Andrillon T, Taillard J, Strauss M. Sleepiness and the transition from wakefulness to sleep. *Neurophysiol Clin*. 2024;54(2):102954. doi:10.1016/j.neucli.2024.102954
39. Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt JK. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *J Sleep Res*. 1997;6(3):179–188. doi:10.1046/j.1365-2869.1997.00045.x
40. Lecci S, Cataldi J, Betta M, Bernardi G, Heinzer R, Siclari F. Electroencephalographic changes associated with subjective under- and over-estimation of sleep duration. *Sleep*. 2020;43(11):zsaa094. doi:10.1093/sleep/zsaa094
41. Spina MA, Bei B, Rajaratnam SW, et al. Cognitive behavioural therapy for insomnia reduces actigraphy and diary measured sleep discrepancy for individuals with comorbid insomnia and major depressive disorder: a report from the TRIAD study. *Sleep Med*. 2024;114:137–144. doi:10.1016/j.sleep.2023.12.014
42. Pieroni I, Cerroni G, Velluto L, Sorbi S, Guarnieri B. Sex and age differences in sleep disorders: insights from a diverse cohort referred to a sleep center. *Neurol Sci*. 2025;46(8):3885–3897. doi:10.1007/s10072-025-08133-7
43. Suh S, Cho N, Zhang J. Sex differences in insomnia: from epidemiology and etiology to intervention. *Curr Psychiatry Rep*. 2018;20(9):69. doi:10.1007/s11920-018-0940-9
44. Palmisano BT, Zhu L, Eckel RH, Stafford JM. Sex differences in lipid and lipoprotein metabolism. *Mol Metab*. 2018;15:45–55. doi:10.1016/j.molmet.2018.05.008
45. Schorr M, Dichtel LE, Gerweck AV, et al. Sex differences in body composition and association with cardiometabolic risk. *Biol Sex Differ*. 2018;9 (1):28. doi:10.1186/s13293-018-0189-3
46. Zaw M, Hein L, Martinez AC, Ascher KB, Abreu AR, Chediak AD. Gender differences in sleep disordered breathing—a review of literature. *Curr Pulmonol Rep*. 2021;10(3):121–128. doi:10.1007/s13665-021-00278-y
47. Krishnan V, Collop NA. Gender differences in sleep disorders. *Curr Opin Pulm Med*. 2006;12(6):383–389. doi:10.1097/01.mcp.0000245705.69440.6a
48. Hamilton M. A rating scale for depression. *J Neurol Neurosur Ps*. 1960;23(1):56–62. doi:10.1136/jnnp.23.1.56
49. Riemann D, Spiegelhalder K, Feige B, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep Med Rev*. 2010;14 (1):19–31. doi:10.1016/j.smrv.2009.04.002
50. Maes J, Verbraecken J, Willemen M, et al. Sleep misperception, EEG characteristics and autonomic nervous system activity in primary insomnia: a retrospective study on polysomnographic data. *Int J Psychophysiol*. 2014;91(3):163–171. doi:10.1016/j.ijpsycho.2013.10.012
51. Wei Y, Van Someren EJ. Interoception relates to sleep and sleep disorders. *Curr Opin Behav Sci*. 2020;33:1–7. doi:10.1016/j.cobeha.2019.11.008
52. Liu Y, Zhang J, Lam SP, et al. Help-seeking behaviors for insomnia in Hong Kong Chinese: a community-based study. *Sleep Med*. 2016;21:106–113. doi:10.1016/j.sleep.2016.01.006
53. Joo EH, Altier HR, Selai C, et al. Neurobiological mechanisms of sleep state misperception in insomnia disorder: a theoretical review. *Sleep Med Rev*. 2025;81:102096. doi:10.1016/j.smrv.2025.102096
54. Dittoni S, Mazza M, Losurdo A, et al. Psychological functioning measures in patients with primary insomnia and sleep state misperception. *Acta Neurol Scand*. 2013;128(1):54–60. doi:10.1111/ane.12078
55. Valko PO, Hunziker S, Graf K, Werth E, Baumann CR. Sleep-wake misperception. A comprehensive analysis of a large sleep lab cohort. *Sleep Med*. 2021;88:96–103. doi:10.1016/j.sleep.2021.10.023
56. Bastien CH, Vallières A, Morin CM. Validation of the insomnia severity index as an outcome measure for insomnia research. *Sleep Med*. 2001;2 (4):297–307. doi:10.1016/s1389-9457(00)00065-4