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Depression and mania symptoms mediate the relationship between insomnia and psychotic-like experiences in the general population.

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ABSTRACT

Psychotic-like experiences (PLEs) are subclinical forms of psychosis commonly experienced in the general population. The nature of PLEs has yet to be clearly defined, yet mood and sleep disturbances may be two predictors. Sleep disturbance increases paranoia and hallucinations in experimental studies, and insomnia is considered a longitudinal precursor of psychosis. Mood disturbances including depression and mania, which can be induced by insomnia, can also result in psychotic symptoms. However, whether insomnia may predict PLEs via the mediation of mood disturbance has yet to be fully clarified. To advance this field, the aim of this study was to investigate the mediation role of depression and mania symptoms on the relationship between insomnia and PLEs.

1.086 community members (28.32±9.04 years, 58.1% females) cross-sectionally completed self-reported measures of insomnia severity, depression/mania symptoms, and PLEs. Bivariate correlations, hierarchical multiple regressions and mediation analyses with bootstrap approach were performed. Insomnia and mood disturbances (depression/mania) were significantly associated with PLEs (β = 0.06, p < .05; β = 0.225, p < .001, respectively). Mediation analysis revealed a significant indirect effect between insomnia and PLEs mediated by mood disturbance through bootstrap approach (β=0.13, se= 0.02, 95% CI: 0.10 - 0.17). Our results support the view of insomnia and mood disturbances as predictors of PLEs in the general population and foster the replication of these findings using longitudinal designs.

Introduction

Psychotic-like experiences (PLEs) may be defined as sub-threshold, non-clinical forms of psychosis such as persecutory ideation, bizarre experiences, and perceptual abnormalities that occur in up to 27% of the general population in industrialised western countries [1–2]. While PLEs are common in community samples, they are more often reported in individuals with mental disorders, suggesting that PLEs lie on a continuum ranging from one single PLE to a clinical diagnosis of psychotic disorder [3]. PLEs seem to emerge from complex interactions between genetic predisposition, environmental variables such as childhood adverse experiences, stress exposure, and cognitive-behavioural factors such as cognitive biases and substance use [4–5]. Moreover, psychosocial factors such as poverty, social isolation, being part of minorities or being single, widowed, divorced, or separated, have all been reported as risk factors for PLEs [2].

Accumulating evidence suggests that sleep disturbance may be a clinical variable causally associated with the onset of psychosis and PLEs. For instance, poor sleep and insomnia are common features of the prodromal period preceding the first psychotic episode in youth [6]. Moreover, a recent meta-analysis showed that individuals with psychotic symptoms showed greater impairments in objective sleep continuity (i.e., sleep onset and sleep maintenance difficulties) and sleep depth (i.e., reduced slow wave sleep) compared to healthy controls [7]. Also, large longitudinal surveys in the general population showed that chronic insomnia, which is the most prevalent sleep continuity disorder [8], was associated with three to four times greater odds of reporting hallucinations [9] and paranoid thinking [10] compared to healthy sleep conditions. Additionally, an experimental study conducted in healthy individuals showed that three nights of partial sleep deprivation resulted in increases in paranoia, hallucinations, and cognitive disorganization [11] and similar results emerged in patients with psychosis [12]. More recently, a 14-day ambulatory assessment study [13] showed that shorter objective sleep time and negative subjective dream valence predicted paranoid thinking, whereas feeling less rested and dream recall predicted hallucinatory experiences.

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In parallel, it has been theorised in influential models [14] and empirically demonstrated over the last decades that affective states and symptoms of mood disturbances may also be implicated in the onset of PLEs. The prevalence of major depression and bipolar disorders are three to four times higher in individuals reporting at least one PLE compared to those without PLEs [2]. Moreover, using experience sampling methods, lower mood has been shown to predict paranoia in the general population [15] and auditory verbal hallucinations in patients with psychotic disorders [16]. Similarly, longitudinal evidence showed that individuals with subclinical mania had a 17% risk of PLEs, compared with 2.3% in those without [17]. Importantly, variations in mood are also closely related to sleep. Insomnia and reduced need for sleep are respectively diagnostic criteria for major depression and mania episodes in international classifications [18]. Longitudinal studies showed that the presence of sleep disturbance is a precursor of mood alterations in depression and bipolar disorders [19–20]. Despite this evidence, the majority of the available studies investigated the role of sleep and mood disturbances as independent predictors of PLEs, and only few studies took into account both variables as determinants of PLEs. For instance, Sheaves and colleagues [8] showed that insomnia was associated with greater risk of hallucinations even after controlling for depression, while Reeve and colleagues [11–12] showed that negative affect (depression and anxiety symptoms) may be a potential mediator of the relationship between insomnia and PLEs. Similarly, preliminary evidence showed that sleep loss may induce mania, which in turn may lead to psychotic symptoms [21]. However, whether insomnia may be associated with PLEs taking into account the cumulative effect of both depressive and maniac symptoms has yet to be explored. Additionally, it has yet to be tested whether insomnia may be associated with PLEs through the mediation of both depressive and mania symptoms. To fill in this gap, we aimed to investigate the associations between insomnia, depressive and mania symptoms and PLEs in a large sample of the general population.

Materials and method

Participants

The study surveyed a convenience sample of 1.086 Italian community members. Age range varied from 18 to 60 years (28.32±9.04). Most of the sample were female (n = 643, 58.1%), had a high school diploma (n = 411, 37.7%), and were students (n = 377, 34.6%). With regard to marital status, 719 (65.4%) were unmarried, 251 (23%) were married, 99 (9.1%) divorced, and 11 (1%) were widow.

Measures

Insomnia

The presence and severity of insomnia in the preceding two weeks was assessed using the Insomnia Severity Index (ISI) [22]. The ISI is a brief seven-item self-report instrument assessing night-time and daytime symptoms of insomnia (e.g., “How noticeable of others do you think your sleeping problem is in term of impairing the quality of your life?” from 0 [*not at all noticeable*] to 4 [*very much noticeable*]). Scores range from 0 (insomnia absent) to 28 (very severe insomnia), with 7 as the cut-off for identifying individuals with subthreshold insomnia and 14 individuals with clinical insomnia. Cronbach’s alpha of the ISI in our sample was 0.89.

PLEs

The presence of PLEs was inspected using the psychosis items of the DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure [23], since this scale has been recently employed to study psychiatric comorbidity in individuals with insomnia [24]. The subscale is composed of two items assessing the presence of subclinical positive symptoms of psychosis including auditory hallucinations (i.e., “Hearing things other people couldn’t hear, such as voices even when no one was around?”) and paranoid thinking (i.e., “Feeling that someone could hear your thoughts, or that you could hear what another person was thinking?”). A 5-point scale from 0 (*none at all*) to 4 (*nearly every day*) is used to record responses and reflect how much or how often the person has been concerned by symptoms over the preceding two weeks. Cronbach’s alpha of the PLEs scale in our sample was 0.78.

Mood symptoms

The presence and severity of depression and mania symptoms was assessed using the subscale of the DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure [23], composed of four items assessing loss of interest, hopelessness (e.g., “Feeling down, depressed, or hopeless?”), involvement in new projects and risky behaviours (e.g., “Starting lots more projects than usual or doing more risky things than usual?”). A 5-point scale from 0 (*none at all*) to 4 (*nearly every day*) is used to record responses and reflect how much or how often the person has been concerned by symptoms over the preceding two weeks. Cronbach’s alpha of the mood disturbance scale in our sample was 0.62.

Procedure

This study is part of a larger survey on mental health in an Italian community sample. The data were collected through an online survey (conducted with Google Forms).

All participants were informed about the purpose and content of the investigation, and about the voluntary nature of participation. They completed the survey anonymously and gave informed consent electronically before participating. Participants completed all Level 1 questions, and for the purpose of this study only the subscales of mood and psychotic symptoms were analysed. Respondents could leave the survey at any point. Privacy of the participants was guaranteed in accordance with the European Union General Data Protection Regulation 2016/679. The study was conducted in accordance with the 1964 Declaration of Helsinki and its later amendments. The study was approved by the Research Ethics Committee for Psychological Research of the University of Messina (no. 17758).

Data analysis

Data analysis was conducted with IBM SPSS 26 and Mplus 8.6. Data were checked for normal distribution, i.e., substantial departure from normality was considered as skew absolute value >2 and kurtosis absolute value >7 [25], and descriptive analysis of the sample was performed. Moreover, Pearson’s bivariate correlations between main study variables were conducted. To investigate the relationships between insomnia, mood symptoms and PLEs, a hierarchical multiple regression analysis was firstly conducted. In the latter, sociodemographic information (age, gender, and marital status) was inserted at the first step as control variables. Mood symptoms (depression and mania items) were inserted at the second step. Finally, insomnia (ISI scores) was inserted at the third step. Multicollinearity of the regression model was checked considering the variance inflation factor (VIF) values. VIF values < 10 are considered as reflecting absence of substantial multicollinearity [26]. Next, a mediation analysis was conducted on SPSS using Model 4 of PROCESS [27] macro at 5000 bootstrap sampling to test the mediation role of sleep disturbance in the relationship between insomnia and PLEs. In this model, age, gender, and marital status were all considered as covariates, given their role in influencing PLEs [2]. Lastly, the mediation analysis was repeated dichotomising both mood disturbance and PLEs. Following the Level 1 Cross-Cutting Symptom Measure recommendations [23], a mean score of 2 in the items composing the former and a mean score of 1 in the items composing the latter were used as cut-offs, respectively. To take into account the dichotomous feature of these variables, a probit model was implemented using Mplus 8.6. All details on this analysis are reported in the Supplementary Document 1.

Table 1
Descriptive statistics and zero-order correlations for study variables.

	Mean	Standard Deviation	Median	Interquartile range	Skewness	Kurtosis	1.	2.	3.	4.	5.
1. Age	28.32	9.04	25.00	6.00	1.9	3.06	1				
2. Gender $_{(0 = m\ 0.1 = f)}$	–	–	–	–	–	–	.34**	1			
3. PLEs	1.12	1.40	0	2.00	1.42	2.93	.31**	.37**	1		
4. Mood disturbances	1.81	.80	1.75	1.00	.18	.03	.10**	.16**	.40**	1	
5. Insomnia	10.51	6.02	10	8.00	.52	.25	.27**	.21**	.37**	.55**	1

Table 2
Hierarchical multiple regression analysis with PLEs as outcome.

	B	SE	β	t	Sign.	Tolerance	VIF
Step 1							
Constant	1.108	.155		7.166	<0.001		
Age	−0.023	.006	−0.150	−3.828	<0.001	.427	2.343
Gender	.535	.082	.188	6.531	<0.001	.793	1.262
Marital status	.911	.072	.527	12.712	<0.001	.383	2.610
R2= 0.287, F = 145.175, df=3, p<.001							
Step 2							
Constant	.094	.172	.545	.545	.586		
Age	−0.015	.006	−0.1	−2.659	.008	.421	2.378
Gender	.48	.078	.169	6.776	<0.001	.790	1.267
Marital status	.742	.069	.430	10.690	<0.001	.365	2.737
Mood disturbances	.495	.044	.285	11.296	<0.001	.926	1.080
R2= 0.362, R2Change= 0.075, F = 153.175, df=4, p<.001							
Step 3							
Constant	.055	.173		.319	.750		
Age	−0.016	.006	−0.101	−2.692	.007	.420	2.378
Gender	.479	.078	.168	6.172	<0.001	.790	1.267
Marital status	.714	.071	.413	10.093	<0.001	.351	2.848
Mood disturbances	.443	.051	.255	8.674	<0.001	.682	1.466
Insomnia	.015	.007	.062	2.02	.044	.619	1.615
R2= 0.365, R2Change= 0.002, F = 123.175, df=5, p<.001							

Theory/Calculation

Previous cross-sectional and longitudinal evidence suggests that sleep disturbance [6–13] alongside affective states and mood symptoms [14–23] may be clinical variables causally associated with the onset of psychosis and PLEs. Moreover, robust longitudinal epidemiological studies showed that sleep disturbance may induce negative emotions and mood instability [19–20]. Accordingly, we hypothesise that mood disturbances such as depression and mania symptoms may be process variables linking sleep disturbances to PLEs in the general population.

Results

Descriptives of the sample

Descriptive statistics for study variables are reported in Table 1. Following the cut-offs used to dichotomise both mood disorder and PLEs, the proportions of the sample scoring under the cut-offs was 0.548 and 0.564, respectively.

Bivariate correlations

Results from correlation analysis are reported in Table 1. PLEs resulted significantly associated with age (r = 0.31, p<.001), gender (r = 0.37, p < .001), marital status (r = 0.50, p < .001), mood disturbance (r = 0.40, p <0.001), and insomnia (r = 0.37, p < .001). As largely expected, insomnia severity resulted significantly associated with mood disturbance (r = 0.55, p < .001).

Regression analysis

Results from the hierarchical multiple regression analysis are reported in Table 2. Results showed that the third model was significant (R2 = 0.365, F (5, 1085) = 123.985, p < .001) explaining for approximately 36% of the variance of PLEs. Sociodemographic variables were forced into the equation in the first step (R2 Change = 0.287, F Change (3, 1082) = 145.175, p < .001). Mood disorder was entered in the second step (R2 Change = 0.075, F Change (1, 1081) = 127.604, p < .001). Lastly, insomnia entered in the third step (R2 Change = 0.002, F Change (1, 1080) = 145.175, p = .044). In this final step, all the predictors included in the model were significantly associated with PLEs: gender (β=0.169, p<.001), age (β = −0.101, p = 0.007), marital status (β = 0.413, p <0.001), mood disturbance (β = 0.225, p < .001), and insomnia severity (β = 0.06, p < .05). Moreover, VIF values ranged from 1.262 to 2.848, reflecting absence of substantial multicollinearity. Thus, we proceeded in the implementation of the mediation model.

Mediation analysis

Results of mediation analysis (Fig. 1) showed that mood symptoms were significantly and directly predicted by insomnia severity (β=0.53, p <0.001), after controlling for the effects of age (β = −0.14, p <0.001) and marital status (β = 0.11, p <0.001). PLEs were significantly predicted by insomnia severity (β = 0.06, p = 0.04), and mood disturbances (β = 0.25, p <0.001) after controlling for the effects of age (β = −0.10, p <0.05), gender (β =0.16, p <0.001), and marital status (β=0.41, p <0.001). Moreover, a significant indirect effect between insomnia and PLEs mediated by mood symptoms emerged through 95% confidence intervals of bootstrap approach (β=0.13, se= 0.02, 95% CI: 0.10 - 0.17).

Discussion

This study investigated whether insomnia severity and mood disturbances were associated with PLEs and whether the presence of mood symptoms mediated the relationship between insomnia and PLEs. As a first result, we showed that insomnia and mood symptoms were all associated with PLEs with moderate effects in both correlation and regression analyses. This finding is in line with previous longitudinal data

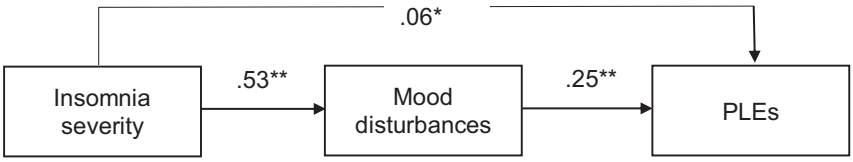

Fig. 1. Results of mediation model. All coefficients are standardised. Covariates: age, gender, marital status. * p <0.05; ** p <0.01. PLEs, psychotic-like experiences.

showing that sleep and mood symptoms may be associates with positive psychotic symptoms and PLEs in clinical and community samples [2–6]. Interestingly, both correlation and hierarchical regression analyses showed that the presence of mood symptoms explained a larger proportion of the variance of PLEs compared with insomnia. Notwithstanding, the presence of insomnia symptoms were still associated with PLEs after accounting for the effects of mood (depression and mania) symptoms, providing evidence for an independent association between insomnia and PLEs. Notably, in our sample the associations between insomnia and PLEs remained significant even after controlling for relevant demographic confounders such as marital status, age and gender [2]. To the best of our knowledge, this finding represents an advance of knowledge in the field, as no previous studies explored the relationships between sleep problems and PLEs taking into account both depression and mania symptoms. Moreover, we hypothesised and consistently showed that insomnia may be associated with PLEs through the mediation of mood symptoms. This hypothesis was drawn from meta-analytic and longitudinal evidence showing that insomnia may be causally implicated in the onset of depression and mania symptoms [19–20]. Our mediation results were also consistent with a previous investigation in patients with non-affective psychosis showing that insomnia may be associated with the onset of positive psychotic symptoms via increased negative affect [12] (depressive and anxiety symptoms), and with an experimental study conducted by the same group showing that in healthy individuals sleep deprivation impact PLEs through increased negative affect [11].

This study encompasses several limitations that should be acknowledged. First, although our hypotheses were theory-based [14] and supported by robust longitudinal and experimental literature [7,9,20,11,12], the cross-sectional nature of our data precluded to clearly establish the directionality of the associations between insomnia, mood symptoms and PLEs. In line with this consideration, it is plausible to hypothesise that a bidirectional link between insomnia and PLEs [12] that should be further explored using multiple assessments in longitudinal and ecological studies. Second, although the Self-Rated Level 1 Cross-Cutting Symptom Measure [23] has the strength of having been developed in the context of DSM-5 and employed in individuals with insomnia [24], the use of this instrument is not free of limitations. For instance, the Level 1 scale assesses only the presence of auditory hallucinations and paranoid thinking and neglects other aspects of PLEs such as social/emotional withdrawal and disorganisation. Thus, future studies would benefit from considering other more robust and comprehensive scales of PLEs [1]. Another measurement issue is that we combined two subscales of the Level 1 (depression and mania) into a single score; this allowed us to assess the contribution of mood disturbance (depression and mania combined) on PLEs. However, the mania items of the Level 1 scale detect activation and goal directed activity which may capture only partially mania experience. Thus, future studies are needed to replicate our findings using more robust measures of mood symptoms.

Third, other variables could impact the associations between insomnia, mood, and PLEs, and their role should be considered in future investigations. For instance, emotion dysregulation, which is frequently associated with insomnia and mood disturbances [28] may influence auditory hallucinations and affectivity [29]. Similarly, some forms of repetitive cognitions such as rumination as worry, which are conceptualised as exacerbating and maintaining factors of insomnia and mood disturbances [30–31] may also be associated with PLEs [32]. Finally, we limited this study on a measure of insomnia [22]. However, other sleep conditions, either pathological (e.g., somnolence, sleep apnoea, narcolepsy), and non-pathological (e.g., habitual short sleep), may also play a role and should be addressed in future studies. This may be particularly interesting since sleep restriction has been associated with mood enhancement in unipolar depression [7]. Similarly, we did not include any measures of daily activity; since daily activities and commitments may potentially influence the association between sleep, mood, and PLEs, future studies are recommended to include such measures.

Conclusions

In conclusion, our findings showed that mood disturbances partially mediated the relationship between insomnia and PLEs. Although the nature of PLEs remains to a large extent still unknown, our results may deserve some clinical attention. Up to now, interventions derived from several psychological approaches have been applied to the prevention of psychotic symptoms [33–34] (e.g., based on cognitive-behavioural principles, psychodynamic theories). This is mostly due to the fact that a multitude of factors have been involved in the aetiology of psychotic symptoms. The results of this study suggest the usefulness of the management of insomnia and mood problems in the general population as a potential preventive strategy to limit the onset of more severe psychotic symptoms. It is well-know, in fact, that having one or more PLEs is considered a risk factor for future onset of clinical psychotic symptoms [35]. Thus, impacting the risk factors associated with the onset of PLEs may indirectly reduce the incidence of more severe forms of psychotic symptoms. For example, cognitive behavioural therapy for insomnia [8] (CBT-I) is shown as effective in improving insomnia and objective sleep [36] and mood symptoms [37–40]. Moreover, preliminary evidence from a large randomised controlled trial conducted in university students with insomnia [34] showed that CBT-I was efficacious in reducing symptoms of paranoia and hallucinations with significant yet small effect sizes. Initial findings from our study suggest the need of future longitudinal research to advance the field and to translate empirical research into preventive interventions.

Declaration of Competing Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.sleepe.2021.100019.

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