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Unraveling the insomnia web: a network analysis of insomnia and psychological symptoms in good and poor sleepers among young adults

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ABSTRACT

Introduction: Poor sleep quality and insomnia are major public health concerns, often associated with mental health problems and particularly prevalent among young adults. While the relationship between insomnia and psychological distress is well-documented, the symptom-level interactions underlying these associations remain largely unexplored. Psychometric network analysis, a method for assessing large-scale interactions among sets of variables and identifying influential nodes within symptom networks, was employed in this study to investigate the relationships between insomnia symptoms, depression, anxiety, and stress in a cohort of Italian university students, focusing on differences between good and poor sleepers.
Methods: Participants (n = 1,234, mean age: 23.3 ± 2.4 years) were classified as good sleepers (GS; n = 406) or poor sleepers (PS; n = 808) based on the Pittsburgh Sleep Quality Index. Gaussian Graphical Models were used to estimate network structures for each group, with insomnia symptoms (Insomnia Severity Index items) and subscale scores from the 21-item Depression Anxiety Stress Scale and the 10-item Perceived Stress Scale as nodes.
Results: The PS network showed greater density (26/55 *vs*. 19/55) and more connections linking insomnia and distress symptoms. Expected influence, a measure of node centrality, identified worry about sleep, difficulty maintaining sleep, and stress symptoms as the most central nodes in both groups, while anxiety symptoms were more influential in the PS network, underscoring their role in the interplay between poor sleep and distress.
Conclusion: These findings highlight the transdiagnostic nature of insomnia and support the utility of network analysis in identifying key symptoms that may serve as targets for interventions addressing both sleep and psychological problems.

1. Introduction

With a lifetime prevalence of up to 10 % in the general population, a persistence rate of 40 % over a 5-year interval, and a significant association with long-term physical and mental health outcomes, insomnia is increasingly recognized as chronic, lifespan disorder that impacts individuals' functioning and warrants attention as both a public health and societal issue [1,2]. In this broader context, growing interest has emerged in subclinical sleep complaints, which encompass a diverse range of habits, symptoms, and conditions collectively labelled as "poor sleep" [3]. Although the prevalence of poor sleep is difficult to quantify, the development of prevention strategies along with sleep health promotion programmers is highly recommended and should be informed by a nuanced understanding of the mechanisms leading to poor sleep and, ultimately, insomnia.

Indeed, the association between insomnia and mental health is well-documented, with robust evidence highlighting insomnia as both a risk factor for mental health problems and a condition exhibiting bidirectional influences with acute and chronic stressful experiences and major psychopathologies, such as depression, anxiety disorders, and post-traumatic stress disorder [4–7]. The theoretical framework of allostasis largely supports these connections, suggesting that insomnia and stress-related disorders reflect a cumulative burden on brain and body systems attempting to adapt to internal or external demands [8]. Within

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this model, both chronic sleep and circadian disruptions and perceived stress act as key drivers of allostatic overload, detrimentally impacting physiological regulation and heightening vulnerability to psychological symptoms. Notably, these dynamic associations are particularly salient for young people, who often experience insomnia and irregular sleep patterns [9], and for whom early prevention and treatment may mitigate the subsequent development of mental health issues [10].

However, despite extensive research on the relationship between insomnia and psychological complaints, the underlying dynamics of this interplay remain largely unexplored. Network psychometrics offers a promising approach for investigating the interactions among symptoms within a network and determining the relative importance of individual symptoms in maintaining a clinical condition [11]. By moving beyond static clinical entities, this granular exploration of symptom-symptom interactions has already demonstrated its utility in clarifying the mechanisms underlying psychopathology [12], and may be particularly valuable for disentangling the complex web connecting poor sleep, insomnia, and psychological symptoms [7,13].

Along these lines, this study aimed to examine the symptom networks of insomnia, depression, anxiety, and stress in a large cohort of university students, with a specific focus on differences between habitual good and poor sleepers.

2. Methods

2.1. Participants and procedures

This study used data from a previous cross-sectional cohort study investigating psychological well-being and sleep quality among Italian university students [14]. An online survey was distributed between March 2021 and June 2021 to a convenience sample of students enrolled at Sapienza University of Rome and participants who provided incomplete data or were older than 35 years were excluded (n = 63). No additional exclusion criteria were applied, and the final sample comprised 1234 participants (mean age: 23.3 ± 2.4 years; 87.3 % women, 12.0 % men, 0.7 preferring not to disclose their sex).

Overall sleep quality over the past month was assessed using the Pittsburgh Sleep Quality Index (PSQI), with participants obtaining a total score above 5 classified as poor sleepers, according to the cutoff established in the original validation study [15]. Diurnal and nocturnal insomnia symptoms were evaluated with the 7-item Insomnia Severity Index (ISI) [16], with each item rated on a 5-point scale (0–4). Depression, anxiety (situational anxiety, arousal, and anxious affect), and stress (irritability, agitation, and chronic, generalized anxiety) symptoms were measured using the 21-item Depression Anxiety Stress Scale (DASS-21) [17], while perceived stress in habitual life situations was assessed with the 10-item Perceived Stress Scale (PSS-10) [18].

The study procedures were approved by the Ethics Committee of Sapienza University of Rome (protocol number 0000/2021), and all participants provided informed consent.

2.2. Statistical analysis

All analyses were conducted using jamovi version 2.5.6 and R version 4.4.1 with the *bootnet*, *mgm*, *NetworkComparisonTest*, and *qgraph* packages. Differences in means and proportions between good sleepers (GS) and poor sleepers (PS) were evaluated using Welch's t-test and chi-squared (χ^2) test, with the significance level set at $p < .05$. Cohen's d effect sizes were calculated for the t-tests.

Two network models, each comprising the same 11 nodes (i.e., the ISI items, the subscale scores from the DASS-21, and the PSS-10 total score), were obtained separately for the GS and PS groups. Specifically, Gaussian Graphical Models with edges representing unique (conditioned) associations between pairs of variables were estimated using the model selection procedure implemented in the *bootnet* package [19]. The choice to include individual ISI items as nodes, while using subscale scores and a total score for the DASS-21 and PSS-10, was made to balance statistical power and conceptual informativeness. This strategy reduced the number of estimated parameters while prioritizing symptom-level granularity for insomnia (based on the clinically derived structure of the ISI) in relation to broader, well-established constructs such as depression, anxiety, and perceived stress. A fixed graphical layout was set for facilitating visual comparison.

Network density (i.e., the proportion of estimated edges after regularization), mean predictability (i.e., the amount of a node's variance explained by its neighbors expressed as R^2, averaged over all nodes), and mean edge weight were computed. Additionally, (one-step) expected influence [11,20] was computed for each node to assess its centrality within the network. Expected influence was selected over other centrality indices for its intuitive interpretation as a measure of a node's overall impact on the network and its ability to account for both positive and negative edges [20].

The accuracy of edge weights and the stability of centrality indices were evaluated through non-parametric bootstrap resampling ($n = 1000$ bootstrap samples) following established guidelines [19]. Bootstrapped confidence intervals for edge weights and correlation stability coefficients for expected influence were obtained. Moreover, pairwise bootstrapped difference tests (α = .05) were conducted to evaluate whether differences in edge weights and node expected influence values were statistically significant within each network. Between-network differences in global structure, overall strength, and node-wise centrality were assessed using permutation-based network comparison tests (1000 iterations) implemented via the *NetworkComparisonTest* package [21].

3. Results

3.1. Participants' characteristics and differences between good sleepers and poor sleepers

Demographic characteristics and questionnaire scores for the whole sample, as well as for GS and PS groups are reported in Table 1. A total of 65.5 % of participants (n = 808) scored above 5 on the PSQI and were therefore classified as poor sleepers. Compared to GS, PS showed significantly worse overall sleep quality and greater insomnia severity with large effect sizes, and higher levels of depression, anxiety, and stress symptoms and perceived stress, with medium-to-large effect sizes.

3.2. Network analyses for good sleepers and poor sleepers

Fig. 1 panels A and B present the estimated network structures for GS and PS, along with network densities, mean weights, and mean predictabilities. Continuous blue edges represent positive associations, while dashed red edges indicate negative associations; the thickness of the edges reflects the absolute magnitude of the respective association. Overall, the network for PS exhibited higher density, with more estimated edges linking insomnia symptoms and psychological distress. Although mean predictability was slightly higher for PS, the mean edge weight was identical across the two networks. Detailed edge weights and node predictability values for both networks are provided in Table S1 (**Supplementary Materials**), while bootstrapped confidence intervals for all edge weights are shown in Fig. S1. The PS network exhibited narrower confidence intervals, indicating more stable estimates and less overlap between edges. Consistently, pairwise edge-weight difference tests identified more significant differences between edge pairs in the PS network than in the GS network (Fig. S2). Network comparison tests did not reveal significant differences in overall network structure (M statistic = 0.19, p = .34) or global strength (S statistic = 0.16, p = .62).

Unstandardized expected influence values for each node in the two networks are depicted in Fig. 1 panel C (exact values are provided in Table S2 in **Supplementary Materials**). Correlation stability coefficients for expected influence were above 0.25 for both networks

Table 1
Participants' demographic characteristics and questionnaire scores, with results of statistical comparisons between good sleepers (GS) and poor sleepers (PS).

	Total sample (n = 1234)	GS (n = 406)	PS (n = 808)	GS *vs.* PS	
				Statistical test	d
Age	23.3 (2.4)	23.2 (2.5)	23.4 (2.4)	$t_{(833)} = 1.2$	0.1
Sex [n (%) M/W/NR]	1077(87.3)/148(12)/9(0.7)	369(86.6)/56(13.1)/1(0.2)	708(87.6)/92(11.4)/8(1.0)	$\chi^2_{(2)} = 2.9$	–
PSQI	7.2 (3.2)	4.0 (1.1)	8.9 (2.6)	$t_{(1176)} = 47.1^{***}$	2.5
ISI	8.7 (5.2)	4.7 (3.1)	10.8 (4.8)	$t_{(1178)} = 27.3^{***}$	1.5
DASS-21 Depression	10.1 (5.7)	7.8 (5.0)	11.3 (5.6)	$t_{(956)} = 11.4^{***}$	0.7
DASS-21 Anxiety	7.6 (5.2)	5.0 (4.1)	8.9 (5.2)	$t_{(1059)} = 14.4^{***}$	0.8
DASS-21 Stress	13.0 (4.7)	10.9 (4.9)	14.1 (4.3)	$t_{(779)} = 11.2^{***}$	0.7
PSS-10	25.1 (6.3)	22.7 (6.5)	26.4 (5.9)	$t_{(792)} = 9.8^{***}$	0.6

GS: good sleepers; PS: poor sleepers; PSQI: Pittsburgh Sleep Quality Index; M: men; W: women; NR: preferring not to disclose their sex; ISI: Insomnia Severity Index; DASS-21: 21-item Depression Anxiety Stress Scale; PSS-10: 10-item Perceived Stress Scale.
$^{***}p < .001$.

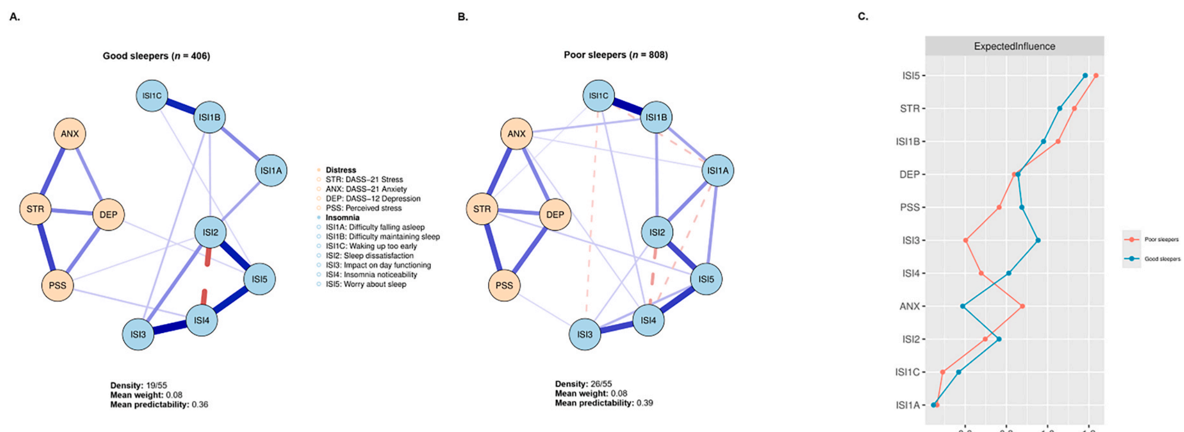

Fig. 1. Panel A and B: Estimated network structures for good sleepers and poor sleepers, including network density, mean edge weight, and mean node predictability. Solid blue lines represent positive associations; dashed red lines represent negative associations. Panel C: Expected influence values for all nodes in the estimated networks for good sleepers and poor sleepers. *Legend.* ISI: Insomnia Severity Index; DASS-21: 21-item Depression Anxiety Stress Scale; PSS-10: 10-item Perceived Stress Scale; ISI1A: difficulty falling asleep; ISI1B: difficulty maintaining sleep; ISI1C: waking up too early; ISI2: sleep dissatisfaction; ISI3: impact on day functioning; ISI4: insomnia noticeability; ISI5: worry about sleep; STR: DASS-21 Stress subscale; ANX: DASS-21 Anxiety subscale; DEP: DASS-12 Depression subscale; PSS: PSS-10 score. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

(0.44 for GS and 0.75 for PS), indicating acceptable stability of these estimates [19]. Notably, worry about sleep (ISI5), stress symptoms (DASS-21 Stress subscale), and difficulty maintaining sleep (ISI1B) emerged as the most central nodes in both networks. In contrast, perceived stress (PSS-10 total score), insomnia's impact on daytime functioning (ISI3), and insomnia noticeability (ISI4) were more influential in GS, whereas anxiety symptoms (DASS-21 Anxiety subscale) showed a higher expected influence in the PS network. Pairwise difference tests for expected influence within each network are illustrated in Fig. S3 (**Supplementary Materials**), again showing a more robust pattern of differences in the PS network. Between-network comparison tests confirmed significantly higher expected influence for the impact on day functioning node (ISI3) in GS (C statistic = 0.35, $p < .01$), and for the anxiety symptoms node (DASS-21 Anxiety subscale) in PS (C statistic = −0.29, $p < .001$).

4. Discussion

In a large cohort of Italian university students, we observed a high prevalence of poor sleep quality (65.5 %), as assessed using the PSQI. While poor sleepers exhibited a general worsening in psychological distress, network analysis offered a more nuanced, symptom-level understanding of the dynamics linking insomnia symptoms and psychological complaints. Specifically, the network for poor sleepers showed a denser network structure, with more associations connecting insomnia and distress nodes, as well as insomnia nodes among themselves, pointing to a more pronounced interdependence between the sleep and distress domains. Within the framework of network psychopathology [12], this suggests that in individuals reporting poorer sleep quality, changes in sleep-related perceptions and experiences (i.e., the symptom patterns associated with insomnia as measured by the ISI) are more likely to reverberate across psychological functioning and vice versa. These results support the importance of preventive interventions targeting both subclinical distress and sleep problems [3] to counteract the co-occurrence of full-blown insomnia and mental health conditions, and are largely consistent with the above mentioned theory of allostasis [8], highlighting how the intertwined co-occurrence of sleep disruption and other psychological stressors may jointly impair the overall capacity for adaptive regulation, ultimately increasing the likelihood of chronic symptoms.

Poorer sleep quality was associated with greater network centrality of anxious affect and situational, arousal-related anxiety symptoms, alongside reduced centrality of diurnal insomnia aspects. Nevertheless, worry about sleep problems, stress/generalized anxiety, and difficulty maintaining sleep emerged as the most central and influential nodes across both groups, with the two largely comparable network topologies highlighting their potential as treatment targets for jointly addressing insomnia and distress-related complaints.

Taken together, these findings underscore the transdiagnostic nature of insomnia [7] and demonstrate the feasibility of network analysis for identifying granular, symptom-level mechanisms underlying cross-domain pathways to comorbidity [22]. This aligns with growing evidence on the efficacy of insomnia-focused treatments, such as cognitive behavioral therapy for insomnia (CBT-I), in mitigating co-occurring mental health symptoms [23]. These interventions may act on central processes within the web of insomnia and psychological complaints, addressing specific aspects that perpetuate distress within the domain of anxiety and worry. In line with recent conceptualizations of CBT-I as a set of modular, targeted treatment strategies (i.e., "therapeutics") addressing specific disruptions [24], tailored case formulations may guide the selection of techniques suited to worry-related mechanisms of insomnia (e.g., cognitive de-arousal, or behavioral experiments) potentially enhanced by personalized network-based symptom maps [25].

However, several limitations of this study should be considered when interpreting these preliminary results. First, network structures were estimated from a convenience sample recruited at a single university and predominantly composed of women participants with a high prevalence of poor sleep quality. This likely introduces selection bias and may limit external validity, as potential sex-related differences in symptom expression and network topology were not examined. Future large-scale studies should aim to replicate these findings in more representative and gender-balanced student cohorts. Second, clinical information (e.g., physical and mental health conditions, sleep-related problems, and current or previous treatments) was not available, precluding adjustment for potential confounding variables. Covariate-adjusted network models should be implemented in future studies to clarify how clinical diagnoses and treatment history may shape the relationship between sleep and psychological symptoms. Finally, the cross-sectional design precludes causal inferences about the observed associations and limits hypotheses regarding their temporal trajectories, while the use of still-developing statistical approaches for testing within- and between-network differences [19,21] did not allow for strict control of inferential errors (e.g., multiple comparison corrections), rendering the present findings essentially exploratory.

Future research could also extend these observations by incorporating additional variables relevant to insomnia-related dynamics – such as daytime sleepiness, circadian preferences, and psychological traits – or more specific, fine-grained mental health symptoms (e.g., low mood, hopelessness, somatization, fatigue, or rumination) into network analyses. In addition, clinically-driven applications of this approach, such as characterizing insomnia phenotypes, longitudinally exploring comorbidities, and evaluating responses to different treatment modalities, are warranted to advance our understanding of insomnia and its complexities.

CRediT authorship contribution statement

Matteo Carpi: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Daniel Ruivo Marques:** Writing – review & editing, Visualization, Methodology. **Claudio Liguori:** Writing – review & editing, Visualization, Supervision.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Declaration of competing interest

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.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2025.106590.

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