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Insomnia, depression, and anxiety symptoms interact and individually impact functioning: A network and relative importance analysis in the context of insomnia

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ABSTRACT

Study objectives: Insomnia, depression, and anxiety show high rates of comorbidity and functional impairment. Transdiagnostic symptom interactions may be implicated in this comorbidity. This network analysis sought to assess how symptoms of insomnia, depression, and anxiety may interact and individually predict impairment across several domains for individuals with insomnia.

Methods: Baseline psychometric data from a randomised controlled trial were analysed (N = 1711). A regularized partial correlation network was estimated from the symptom data. Centrality (symptom connectivity), community structure (symptom clustering), and bridging (inter-community connectivity) were assessed. The replicability of the network model was assessed via confirmatory analyses in a holdout sample. Separately, Shapley values were estimated to determine the relative importance of each symptom in predicting functioning (i.e., psychological wellbeing, psychosocial functioning, and physical health impairment).

Results: The most connected nodes were uncontrollable worrying; trouble relaxing; and depressed mood/hopelessness. Five communities were identified with trouble relaxing identified as the bridge symptom between communities. The model showed good fit in the holdout sample. Low energy and depressive affect symptoms (feelings of failure/guilt; depressed mood/hopelessness; anhedonia) were key predictors in the relative importance analysis across multiple domains of impairment.

Conclusion: Trouble relaxing may be of clinical and transdiagnostic significance in the context of insomnia. In terms of how symptoms relate to functioning, it was clear that, while low energy and feelings of failure/guilt were prominent predictors, a range of symptoms are associated with functional impairment. Consideration of both symptoms and functional impairment across domains may be useful in determining targets for treatment.

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1. Introduction

Sleep disturbances are a prime target for transdiagnostic analysis. The far-reaching relevance of sleep in psychopathology is well-established—in fact, it has been proposed that sleep quality should be treated as a fundamental dimension in mental health under the US National Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC) [1,2]. Sleep disturbances are observed across many (possibly most) mental disorders [3,4]. Sleep disturbances, including insomnia, feature in DSM-5 diagnostic criteria for both anxiety and depressive disorders, and an estimated 40% of insomnia patients have a comorbid mental health condition [5]. Insomnia is associated with particularly high rates of comorbidity with depression and anxiety [6] and significantly predicts onset of

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these two disorders (anxiety disorders OR 3.23; depression OR 2.83). This association may be causal where sleep difficulty has been shown to predict the development of mental health conditions, including anxiety and depression [2]. As such, sleep is arguably one of the most critical psychophysiological processes in neural and mental health [1,4].

1.1. The network analytic literature

While disorders assume a common cause, the network approach to psychopathology proposes that symptoms are constitutive (not merely passively reflective) of disorders; disordered states may emerge from causal interaction/reinforcement between symptoms (and relevant external factors) [7]. Symptoms are thus treated as autonomous causal entities: worry may cause trouble sleeping, in turn causing fatigue [8,9]. Then, symptoms are non-interchangeable in that, naturally, different symptom profiles will show different dynamics, and some symptoms will prove more central or interconnected than others, potentially maintaining an episode [10]. Symptoms may cluster into communities [11], and symptoms may act as bridges between these communities identified in a manner statistically agnostic to theoretical constructs (e.g., initial diagnostic categorisation) [12]. Network analysis is thus particularly suited for assessment of transdiagnostic symptom interplay (i.e., in comorbidity). It may provide insight into how comorbidity is maintained through central nodes, pathways, and bridging structures.

Several network studies [13–16] have examined the association between symptoms of depression and anxiety, including sleep problems as a single symptom node. One such analysis suggested that sleep problems may bridge hallmark symptoms of Major Depressive Disorder (MDD) and Generalised Anxiety Disorder (GAD) (alongside attentional control components) [16]. A network outcome analysis, including an outcome (MDD-onset over a 6-year follow-up period) showed that difficulty initiating sleep was particularly related to first-onset MDD [17]. A number of other studies have explored sleep in relation to depression in specific contexts, for instance, low energy and difficulty maintaining sleep/early-morning awakening were identified as bridge symptoms in Wenchuan earthquake survivors at a 10-year follow-up [18]; sleep problems were identified to have the highest clinical relevance in a study in psychiatric healthcare professionals during the COVID-19 pandemic [19]. Insomnia symptoms were most strongly associated with neuroticism and conscientiousness in a network of personality traits [20]. Cognitive therapy (CT) and behavioural therapy (BT) for insomnia show different symptom-specific effects (sleep efficiency; difficulty maintaining sleep; dissatisfaction with sleep for BT, and interference with daily functioning; difficulty initiating sleep; early morning awakenings; and worry about sleep for CT) [21] while, in a separate study, sleep related behaviours were associated with BT, and worry; impaired quality of life (QOL); dysfunctional beliefs; monitoring sleep-related threats for CT [22].

To our knowledge, only one prior published network analysis has investigated insomnia symptoms discretely in relation to both depression and anxiety symptoms [23]. Bai et al. [23] investigated insomnia, depression, and anxiety symptomatology in the general population in Macau during the COVID-19 pandemic, and symptom-level associations to QOL were also assessed. Within their study, sleep related symptoms were most prominent with maintenance (i.e., difficulty staying asleep) the most central symptom, insomnia/hypersomnia a bridge symptom between communities, and sleep dissatisfaction the strongest predictor of reduced QOL.

1.2. Psychological wellbeing and functional and physical health impairment

Psychosocial wellbeing and functional impairments associated with insomnia, depression, and anxiety appear to be intensified in the case of comorbidity. While levels of impairment in depression and anxiety are roughly comparable, comorbid depression-anxiety is linked with significantly greater impairment than in either disorder alone [24,25]. For those with mood and/or anxiety disorders, comorbid insomnia is associated with significantly higher levels of impairment [3]. The three disorders are linked also to physical health impairment [26–28] and significant decreases in global QOL indices (comparable to or exceeding those observed in congestive heart failure) [29–31].

While functional impairments are inextricable from a diagnosis of insomnia [32], the disorder influences daytime functioning in different ways. Insomnia severity is associated with trouble with work performance, increased work absenteeism, occupational accidents, and utilisation of medical services [33–35]. Subjective poor sleep appraisal consistently predicts decreased life satisfaction (including at 6-year follow-up), while life dissatisfaction does not reliably predict poor sleep [36]. Short sleep duration is negatively associated with psychological wellbeing [37]. Potential effects on wellbeing and functioning are difficult to parse from insomnia's associations with comorbid psychopathology more broadly [36].

This speaks to the utility of transdiagnostic, symptom-level analysis. Treating insomnia as a unitary construct (i.e., solely relying on symptom severity sum scores) may obscure any clinically important symptom-level interactivity and differential impacts on impairment [38]. Associations between symptoms and functioning are complicated, and this is especially the case considering the heterogeneity of symptom and impairment profiles in psychopathology [39–41]. This aligns with a new research framework, *symptomics*, which encourages consideration of symptoms individually, their causal interrelations, and how findings bear on individual patients [42].

1.3. The present study

Considering the overlapping symptomatology and high rates of comorbidity between insomnia, depression, and anxiety [6,13,43], the first arm of this analysis involved estimation and visualisation of a network, mapping associations between symptoms of these three disorders. This aimed to answer the following questions: firstly, in a network of insomnia, depression, and anxiety symptoms, how do symptoms interact? Second, which symptoms are central in this network? Third, what are the recognisable communities (i.e., associative clusters) of symptoms in this network? Fourth, which symptoms bridge (i.e., account for links between) the communities in this network?

The second arm of this study aimed to determine the relative importance of each assessed symptom in their overall prediction of psychological wellbeing, physical health, and various aspects of functioning, as well as job, relationship, and general life satisfaction. Given the nature of the data and analyses, this study was exploratory thus without determinate hypotheses. This study builds on previous work by investigating the associations between insomnia, depression, and anxiety symptoms within UK-, US- and Australia-based insomnia patients. Notably, the data analysed in the present study were gathered prior to onset of the COVID-19 pandemic, so findings can be expected to better generalise to the pre- and post-pandemic periods.

2. Methods

The study is a secondary data analysis using baseline data from a randomised controlled trial evaluating digital CBT for insomnia

compared with sleep hygiene education (ISRCTN 60530898, ethical approval ref: MS-IDREC-C2-2015-024) [44]. Data were analysed at baseline, prior to the receipt of treatment. Research questions and analytic plan were pre-registered via the Open Science Framework (OSF; https://osf.io/p9t3y/).

2.1. Sample

This baseline sample consisted of 1711 UK-, US-, and Australia-based community participants. Eligible participants were adults, who screened positive for probable insomnia disorder based on the 8-item Sleep Condition Indicator [45], reported stable mental and physical health, and were not undergoing professional psychological treatment for insomnia (or expecting this within 6 months). Participants taking medication for any reason were not excluded provided their health was stable. See Espie et al. [46] for a more detailed description of the eligibility criteria.

2.2. Measures

The assessed data were captured pre-treatment, and included baseline symptoms of insomnia, depression, and anxiety (used in both the network analysis and relative importance analysis) as well as psychological wellbeing, life satisfaction, and physical/psychosocial functioning (used in the relative importance analysis only).

2.2.1. Symptoms

Measures used included the 9-item version of the Sleep Condition Indicator (SCI-9) [45] for insomnia symptoms (reverse-scored for consistency with item 9 [capturing premature early morning awakening] included and items 5, 6, and 7 excluded [to capture only sleep symptoms]); the 9-item Patient Health Questionnaire (PHQ-9) [47] for depression symptoms (with item 3 [insomnia/hypersomnia] excluded); and the 7-item Generalised Anxiety Disorder (GAD-7) [48] scale for anxiety symptoms.

2.2.2. Functioning

Measures used included the 14-item Warwick-Edinburgh Mental Well-Being Scale (WEMWBS) [49] for psychological wellbeing; the Glasgow Sleep Impact Index (GSII) [50] for sleep-related daytime functioning and QOL impairment (with only total score for part 3 used [extent bothered by self-designated effects of poor sleep]); the Work Productivity and Activity Impairment Questionnaire: Specific Health Problem (WPAI:SHP) [51] for sleep-related functional impairment in work and non-work contexts; a single item from the Job Satisfaction Scale (JSS) [52] for overall job satisfaction; the 7-item Relationship Assessment Scale (RAS) [53] for overall relationship satisfaction; the single-item Life Satisfaction Measure [54] for overall life satisfaction; and the Patient-Reported Outcomes Measurement Information System—Global Health Scale (PROMIS-10), Global Physical Health (GPH) subscale [55] for physical health. See Espie et al. [44] for a detailed overview of the measures used.

2.3. Data analyses

Data analyses were conducted using R (version 4.0.2) [56]. The R packages used included *bootnet* [57], *caret* [58], *dplyr* [59], *EGAnet* [60], *vip* [61], *ltm* [62], *networktools* [63], and *qgraph* [64]. The R script is available via OSF at https://osf.io/p9t3y/.

2.3.1. Pre-processing

Espie et al. [44] verified the primary data and calculated composite scores. Missing data were excluded from the present analyses via listwise deletion. Network data were checked for near-zero variance, heteroskedasticity, and asymmetric distributions (factors which violate Gaussian assumptions in network analysis) [65,66]. Topological overlap [65] between node pairs was screened for via the "goldbricker" function (*networktools*) [63].

2.3.2. Train/holdout split and network modelling

Network data were randomly partitioned into a training set and holdout set (60/40%) for later testing of the network model's validity. A Gaussian graphical model (GGM) [67–69] was estimated using the training data. GGMs represent undirected partial correlations (edges) between nodes, i.e., those which persist after controlling for all other nodes [68].

To ensure the network model was *sparse* (i.e., parsimonious, interpretable, and without excess edges), the GGM was selected via *graphical least absolute shrinkage and selection operator* (graphical LASSO, "GLASSO") [64,70] and *Extended Bayesian Information Criterion* (EBIC; $\gamma = 0.5$) [71]. The training network model's goodness-of-fit indices were assessed, including chi square, normed fit index (NFI), parsimonious normed fit index (PNFI), Tucker Lewis index (TLI), non-normed fit index (NNFI), relative fit index (RFI), incremental fit index (IFI), relative noncentrality index (RNI), comparative fit index (CFI), and root mean square error of approximation (RMSEA). For these fit indices (aside from chi square and RMSEA), values increase from 0 to 1 or more with model fit [72]. PNFI values above approximately .75 are generally taken to indicate good, parsimonious fit; for the other indices, values $\geq$ 0.90-0.95 are variably accepted as cut-offs for satisfactory fit [72–74]. RMSEA values decrease with model fit; generally, values $<$ 0.05-0.08 are considered to suggest good fit [72,74]. Chi square results are reported but not interpreted as they are less reliable in large samples [72].

2.3.3. Confirmatory analyses

A confirmatory model was generated by specifying the training network structure to the holdout dataset, and its fit indices were analogously assessed. A network was then generated from the holdout dataset (separate to the confirmatory model), and its fit indices were also calculated. To compare the interrelation between the training and holdout GGMs, the Spearman correlation coefficient between them was obtained [66]. This allowed for testing whether the network estimation was roughly consistent across both data subsets, which would further suggest external validity [75].

2.4. Network inferences

2.4.1. Community estimation

A walktrap algorithm was used to identify communities within the training network [11,60,76]. In this context, these well-connected node clusters with relatively few extra-community edges theoretically represent psychopathological disorder dimensions [11,76].

2.4.2. Node centrality

Expected influence (EI), the summed value of a node's immediately connected edges (both positive and negative), was obtained per node [77,78]. This captured the connectivity of each node in the network.

2.4.3. Bridge analysis

Bridging items between the communities were also identified via bridge expected influence (BEI) metric: the summed value of edges (positive or negative) between a given node and all other extra-community nodes [63]. Here, BEI theoretically reflects a symptom's transdiagnostic connectivity [77]. A bridging item was

classified as being within the top 80th percentile of BEI values and with attention paid to any significant drop-off in BEI following a bridge node [79]. Bridge analysis results were used to specify groups in a weighted bridge plot of the training network.

2.4.4. Post hoc Stability and accuracy tests

All stability and accuracy tests were performed using bootnet. [57] Specifically, accuracy and stability of edge weights was tested via non-parametric bootstrapping, and that of node EI was tested via case-dropping bootstrapping and calculation of the centrality stability coefficient [57]. Edge weight and centrality difference tests ($\alpha = 0.05$) detected statistical difference (or lack thereof) between individual node pair edge weights and EI estimates [57]. See Supplementary material, Section 1 for details.

2.4.5. Relative importance analysis

Elastic-net regularized generalised linear models were estimated using all symptom variables to predict each functioning variable and tuned using 10-fold cross validation. The models were then used to estimate mean absolute Shapley values [61,80,81] using package *vip* [61].

3. Results

3.1. Descriptive results

See Table 1 for demographic characteristics of the sample. A significant portion (38%) of participants had at some time received a diagnosis for depression or anxiety.

Table 2 below reports applicable descriptive statistics per measure. (See Supplementary material, Section 3 for item-level descriptive results). On average, participants reported moderate depressive symptoms and mild anxiety symptoms (PHQ-9 and GAD-7 mild-moderate threshold: 10[47,48]).

3.2. Network modelling

3.2.1. Data pre-processing and network estimation

PHQ item 9 (regarding suicidality/self-harm) was excluded as it showed zero variance due to the exclusion criteria. No significant topological overlap was detected. Network data were not normally distributed, and many items were heteroskedastic (namely from the SCI-9 and PHQ-9). Thus, partial correlations were estimated following nonparanormal transformation of the data [82,83]. Following listwise deletion of missing data, the training network comprised 1026 cases. Within the training network, 41 of 190 possible edges were included in the selected model (network density = 0.22), and the model displayed excellent fit.

3.2.2. Confirmatory analyses

Results indicated satisfactory to excellent fit across all three models. Correlation between the training and holdout model was high ($r_s = 0.71$). See Supplementary material, Section 4 for goodness-of-fit indices across the training, confirmatory, and holdout network models.

3.2.3. Bootstrapped robustness checks

Results suggested the estimated training network is robust overall, and EI estimates were of good stability and accuracy. The EI centrality stability coefficient was 0.75 (approximately $\leq$75% of cases could be dropped to maintain an average correlation of ≥ 0.70 with the original EI estimates at 95% probability [57]). Section 1 of the Supplementary material reports bootstrapped accuracy/stability test results for edge weights and EI.

3.2.4. Community and bridge analyses

A *walktrap* algorithm detected 5 communities in the training dataset (median = 4, 95% CI [2.83, 5.17]). These reflected (1) *sleep-, eating-, and energy-related symptoms*, (2) *psychomotor disturbance symptoms*, (3) *depressive affect*, (4) *anxiety*, and (5) *difficulty maintaining sleep*. Two nodes, time to sleep and chronicity of sleep problems, were unassigned to any of these communities. Community assignments are detailed in Fig. 1.

Uncontrollable worrying and trouble relaxing were the highest-EI nodes. Notably, apart from poor sleep quality, all SCI items together showed the lowest EI. Trouble relaxing was identified as the bridge node across both community structures. This was the sole bridge node. The BEI of each node is shown in plots included in section 5 of the Supplementary material.

Several nodes were not assigned to the communities corresponding with their origin measures (e.g., some GAD items were not assigned to the anxiety symptom community). Specifically, the psychomotor disturbance symptom community contains two PHQ items (trouble concentrating, psychomotor agitation/retardation) and three GAD items (trouble relaxing, restlessness, irritability). The sleep-, eating-, energy-related symptom community is equally comprised of SCI (sleep problem frequency, poor sleep quality) and PHQ (low energy, poor appetite/overeating) items.

3.3. Relative importance analysis

Table 3 indicates the top 5-ranking symptoms per functioning measure in terms of relative variance explained. Overall, low energy was highly important relative to other symptoms, namely in terms of its prediction of daytime impairment overall (via GSII; 1st-ranked predictor), impairment to physical health (1st-ranked), as well as work and non-work (personal) productivity (1st-ranked for both). Symptoms relating to depressive affect (depressed mood/hopelessness, anhedonia, and feelings of failure/guilt) together showed by far the highest relative importance in predicting both reduced psychological wellbeing and life satisfaction. Of these symptoms, feelings of failure/guilt stood out also vis-à-vis relationship satisfaction (1st-ranked) and job satisfaction (3rd-ranked).

4. Discussion

This study employs network analysis to explore the symptom-level associations between insomnia, depression, and anxiety and assess these disorders' symptom-level associations with psychosocial functioning in patients with insomnia. The estimated network model was supported in a confirmatory analysis and shown to be robust, reinforcing the network model's internal validity and generalizability. The most central nodes included uncontrollable worrying; trouble relaxing; and depressed mood/hopelessness. Five communities were identified from the data, grouping symptoms related to sleep, energy, and eating; psychomotor agitation; depressive affect; anxiety; and difficulty maintaining sleep. Trouble relaxing acted as a bridge symptom between these communities. In the relative importance analysis, low energy and depressive affect (i.e., feelings of failure/guilt; depressed mood/hopelessness; anhedonia) were particularly important predictors across several domains of impairment.

Trouble relaxing was identified as a central symptom and, as the identified bridge symptom, between insomnia, depression, and anxiety symptoms. The clinical importance of trouble relaxing in insomnia and in psychopathology broadly is well-substantiated [84]. It is worth noting that trouble relaxing clustered within the psychomotor community and related in the network most strongly to restlessness (of the type which makes it difficult to sit still). This corroborates Bai et al. [23], in which trouble relaxing showed the

Table 1
Demographics of the sample (N = 1711).

Characteristic	*n* (%)	Mean (*SD*)	Median [Min, Max]
Age		48.1 (13.7)	49 [18, 89]
Gender			
- Male	382 (22.3)		
- Female	1329 (77.7)		
Ethnicity			
- Asian	45 (2.6)		
- Black	19 (1.1)		
- Mixed	36 (2.1)		
- Other	35 (2.0)		
- White	1558 (91.1)		
- Do not wish to state	17 (1.0)		
Employment Status			
- Employed full-time	804 (47.0)		
- Employed part-time	108 (6.3)		
- Unemployed	79 (4.6)		
- Retired	348 (20.3)		
- Full-time student	291 (17.0)		
- Full-time homemaker/carer	74 (4.3)		
Education (years of continuous, full-time)		16.5 (3.7)	17 [8, 31]
Partnership status			
- Unpartnered	453 (26.5)		
- Partnered, living apart	141 (8.2)		
- Partnered, living together	1113 (65.0)		
Depression/anxiety diagnosis (ever received)	650 (38.0)		

See Espie et al. (2019) for detailed physical health characteristics of the sample.

Table 2
Descriptive statistics by measure (N = 1711).

Measure; Variable	*M* (*SD*)	Median [Min, Max]	Cronbach α
SCI-9[a] (insomnia symptoms)	7.5 (3.7)	8 [0, 18]	.48
- SCI-8	6.5 (3.2)	8 [0, 18]	.50
PHQ-9[a] (depression symptoms)	9.7 (4.1)	9 [1, 24]	.74
GAD-7 (anxiety symptoms)	7.4 (4.7)	6 [0, 21]	.75
WEMWBS (psychological wellbeing)	43.2 (7.8)	43 [14, 68]	.90
GSII[b] (sleep-related daytime functioning/QoL)	223.9 (45.2)	228 [40, 300]	
WPAI:SHP			
- Sleep-related absenteeism	7.7 (16.6)	0 [0, 100]	
- Non-sleep-related absenteeism	4.4 (13.5)	0 [0, 100]	
- Sleep-related impairment at work	41.6 (23.6)	40 [0, 100]	
- Sleep-related impairment (non-work)	45.3 (24.7)	47 [0, 100]	
JSS (job satisfaction)	3.6 (2.0)	4 [1, 7]	
RAS (relationship satisfaction)	27.7 (5.8)	29 [7, 35]	.92
Life satisfaction	2.8 (0.7)	3 [1, 4]	
PROMIS-10:GPH (global physical health)	14.4 (2.2)	15 [6, 20]	.57

[a] Prior to any reverse scoring and item exclusions.
[b] Item 3 total.

second-highest EI, and psychomotor symptoms (restlessness; irritability; and agitation/retardation) showed the highest BEI after sleep problems. No causal inferences can be drawn, as the findings reflect associations across persons at a single timepoint, and just because a symptom is central does not mean it is clinically relevant or a good target for intervention [85]. However, several well-researched insomnia treatments specifically target trouble relaxing, namely relaxation-based interventions [86], psychoeducation [87], and biofeedback tools [88]. Relaxation training is also a component of CBT-I [89] and has been productively combined with pharmacotherapy [90].

The strongest edges in the network (i.e., uncontrollable worrying—excessive worrying; anhedonia—depressed mood/hopelessness) corroborate empirical literature on symptom interaction in depression and anxiety [13,15,23,91–93]. This analysis suggests that such findings largely generalise to insomnia patients, including those with subclinical and/or undiagnosed depression/anxiety.

Results diverged in certain respects from Bai et al. [23], in which sleep maintenance (difficulty staying asleep) was identified as the highest-EI node. In the present study, difficulty maintaining sleep in the night and early morning ranked 3rd- and 4th-lowest in EI, respectively. The high EI of sleep maintenance in Bai et al. [23] largely reflects the node's very strong edge with severity of sleep onset (difficulty initiating sleep), the strongest edge in their network, as such this may be a statistical artefact reflecting topological overlap between the two nodes in their study. This association was also not evident in the present analysis where there was a negative association between similar items: night-time awakenings and time to sleep. As Bai et al. [23] discuss, this divergence may reflect the fact that their study was conducted during the COVID-19 pandemic. They also included nodes which were excluded in the present analysis, such as PHQ items 3 (insomnia/hypersomnia) and 9 (suicidality) as well as sleep problems' interference with daytime functioning.

In the community analysis, data-derived symptom groups corresponded to (1) sleep-, energy-, and eating-related; (2) psychomotor disturbance; (3) depressive affect; and (4) anxiety symptoms. While communities (3) and (4) reflected standard

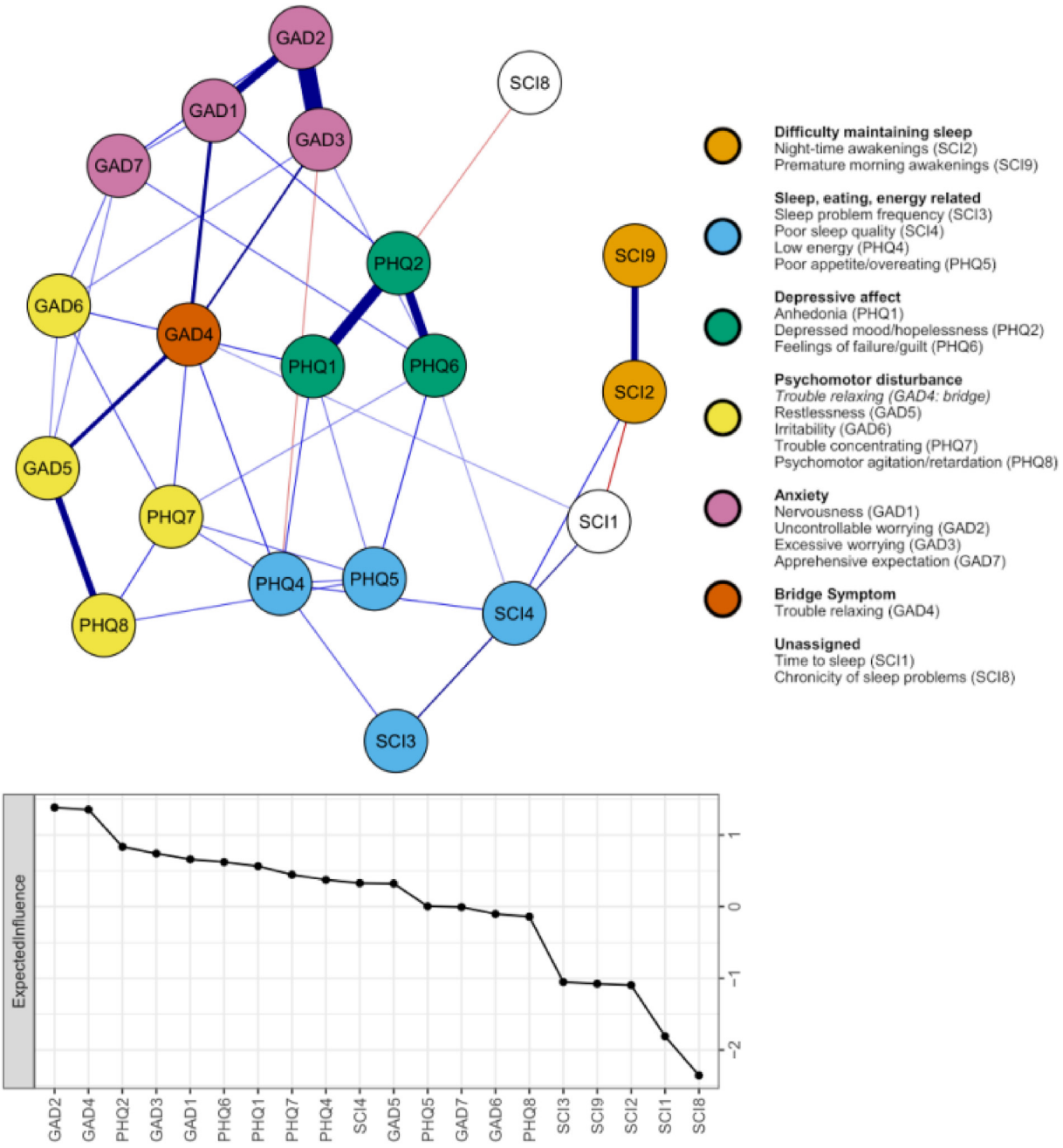
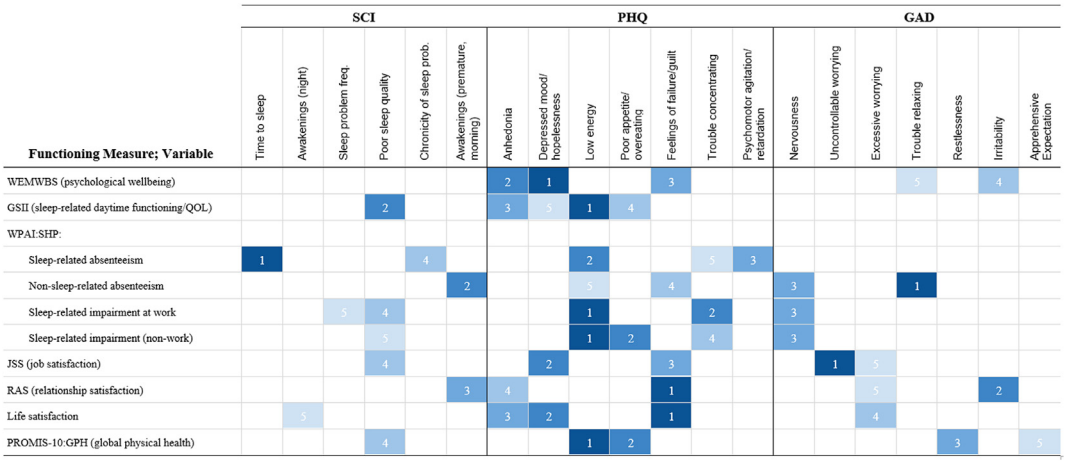


Fig. 1. Bridge Plot of the Training Network and Centrality (Expected Influence) Plot. Coloured nodes reflect communities. Edges (lines connecting nodes) represent partial correlations with blue indicating positive and red negative associations. The centrality plot at the bottom shows z-scores expected influence. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

disorder constructs, (1) and (2) contained symptoms from multiple measures (i.e., items which crossed from their corresponding measured constructs). Items in the SCI showed low internal consistency, and within the network, were split across communities or unassigned. This highlights the need for the SCI to be used as a diagnostic tool [45], as it was developed, and not using the sum score as a measure of unitary construct (i.e., insomnia). Conceptualising disorders as unitary and discreet constructs may obscure influential transdiagnostic factors and limit construct validity, even in those with a single psychological diagnosis [84].

In terms of relative contribution to functional and physical health impairment, low energy was a key predictor, especially in occupational domains. It was top ranked in predicting overall daytime impairment as well as reduced work and non-work productivity and physical health. It also ranked second in sleep-related absenteeism. These findings corroborate a prior symptom-level analysis [94] of functional impairment across psychiatric profiles, in which penalised regression identified low energy's explanation of reduced functioning as the second greatest among the 51 assessed DSM-5 symptoms (and the first of the depression symptoms). Bai et al. [23] found relatedly that, of insomnia, depression, and anxiety symptoms, fatigue was associated directly and relatively strongly with QOL. Low energy is also the most prevalent motivating factor in treatment-seeking for insomnia [95]. Notably,

Table 3
Relative importance results.



The top 5-ranking symptoms (in terms of individual relative contribution to total variance explained) per functioning measure are shown. Numbers indicate ranking. Cell shading darkens with increasing relative importance.

within the network analysis, low energy shared an edge with the bridge symptom, trouble relaxing. The reciprocal relationship between stress and low energy is well-established [96]. Findings suggest transdiagnostic associations between insomnia, depression, and anxiety symptoms may manifest as functional and physical health impairment in the insomnia patient largely via low energy.

Overall, the relative importance analysis showed that insomnia, depression, and anxiety symptoms vary considerably in their relative prediction of reduced wellbeing and impaired psychosocial functioning, corroborating prior research on depression [40] and comorbid depression-anxiety [66]. Although the majority of participants had never received a diagnosis of depression, some of the hallmark symptoms of depression (i.e., depressed mood/hopelessness; anhedonia; feelings of failure/guilt) were found to bear most significantly on psychological wellbeing, life satisfaction, and relationship satisfaction. Correspondingly, these symptoms were also strongly clustered in the network, forming an identifiable community. This corroborates research on wellbeing and life satisfaction generally [23,97–99], hypothetically extending such findings to insomnia patients. In the context of insomnia, worry about and dissatisfaction with sleep has been found to strongly predict feelings of failure and depressed mood [100] and relate to reduced quality of life [23]. Findings are consistent with views that psychopathological comorbidity could be treated as normative [66], where even subclinical levels of depression may relate to impairment in insomnia above and beyond directly sleep-related symptom consequences.

Of the assessed depressive affect symptoms, feelings of failure/guilt stood out also in its associations with relationship and job satisfaction. This is consistent with prior findings which show negative affect moderates insomnia's associations with reduced job satisfaction [101]. Severe insomnia is also associated with the feeling that one is a burden on others and a reduced desire for social support following an experience of social exclusion [102]. This is theoretically consistent with the high relative importance of feelings of failure/guilt in predicting relationship satisfaction. Given the key role of symptoms of depression in reducing psychological wellbeing and life satisfaction in those with insomnia, empirically investigating the potential clinical value of routinely screening for and addressing mood/anxiety symptomatology in insomnia patients may be warranted, regardless of diagnostic thresholds of symptom severity.

Although the present findings cannot lend themselves directly to clinical heuristics, there are clinical implications that can be considered. The network approach offers insight into interactions between symptoms and diagnoses. This can be extended further through the use of idiographic assessment of symptoms and functioning which could inform the personalization of insomnia interventions (e.g., in terms of target symptoms). This would require repeated assessment, this could help the clinician and patient better understand their insomnia and how changes in one area may drive changes in others. The ability to capture these data is becoming more accessible to clinicians [103]. In line with this, assessment should be transdiagnostic, as symptoms cross diagnosis and will interact and influence one another.

4.1. Strengths and limitations

This study employed robust methodology employing train/holdout data partitioning as well as bootstrapped tests, which helped ensure the internal and external validity of the model and the centrality results. This approach to confirmatory testing is new to the network analytic literature and improved credibility of the results overall. While the sample was relatively large and multinational, there were large gender and ethnicity skews (78% women; 91% white), and it only represented English speaking, western nations. All participants qualified for insomnia diagnosis, ensuring generalizability to insomnia patients, however, clinical status for depression/anxiety was not considered for inclusion/exclusion (barring suicidality or a prognosis of hospitalisation). Thus, external validity may be limited vis-à-vis insomnia patients who qualify for diagnosis of depression and/or anxiety.

Given the data were cross-sectional, inferences on causality and directionality of effect are hypothetical. Further, while psychopathological processes are within-person, analyses operated at the group level, potentially limiting construct validity and further restricting direct clinical utility [104]. Use of, idiographic network analysis [105], causal search algorithms,[106] and network intervention or outcome analytic approaches [17,91] could elucidate causal and intra-individual dynamics involved in the interplay between insomnia, depression, and anxiety symptoms. Such analyses could inform new directions in personalised, transdiagnostic treatments for patients experiencing these symptoms.

5. Conclusion

In the context of insomnia, symptoms of insomnia, depression, and anxiety arranged themselves along distinctly transdiagnostic dimensions (such as those relating to psychomotor disturbance). Individual symptoms were highly variable in terms of their centrality, inter-community influence (bridging), and relative importance vis-à-vis functional impairment and wellbeing/life satisfaction levels. Overall, findings further substantiate the value of the burgeoning symptomic approach in psychopathology. Sole reliance on sum scores and the assumption of symptom interchangeability in diagnosing and treating these disorders may be problematic. Assessment of transdiagnostic symptom interplay alongside consideration of how symptoms variably relate to impairment and QOL could help inform targets for clinical intervention. This secondary data analysis also bolsters calls for the investigation of sleep and psychomotor symptoms under the RDoC, i.e., as basic dimensions of psychopathology.[1,2,107]

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Data availability

The data are not publicly available for data protection purposes and may be available upon request from Big Health Ltd. Restrictions apply.

CRediT authorship contribution statement

H. Ariel Bard: Writing – original draft, Formal analysis. **Ciarán O'Driscoll:** Conceptualization, Methodology, Writing – review & editing. **Christopher B. Miller:** Conceptualization, Writing – review & editing. **Alasdair L. Henry:** Writing – review & editing. **John Cape:** Writing – review & editing. **Colin A. Espie:** Supervision, Funding acquisition.

Declaration of competing interest

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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.2022.12.005.

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