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Original Report

Sad Mood Bridges Depressive Symptoms and Cognitive Performance in Community-Dwelling Older Adults: A Network Approach

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Abstract

Background and Objectives: Depression and cognitive impairment are common and often coexist in older adults. The network theory of mental disorders provides a novel approach to understanding the pathways between depressive symptoms and cognitive domains and the potential "bridge" that links and perpetuates both conditions. This study aimed to identify pathways and bridge symptoms between depressive symptoms and cognitive domains in older adults.

Research Design and Methods: Data were derived from 2,792 older adults aged 60 years and older with mild and more severe depressive symptoms from the community in Hong Kong. Depressive symptoms were assessed using the Patient Health Questionnaire (PHQ-9) and cognition using the Montreal Cognitive Assessment 5-minute protocol (MoCA-5min). Summary descriptive statistics were calculated, followed by network estimation using graphical LASSO, community detection, centrality analysis using bridge expected influence (BEI), and network stability analyses to assess the structure of the PHQ-9 and MoCA-5min items network, the pathways, and the bridge symptoms.

Results: Participants (mean age = 77.3 years, *SD* = 8.5) scored 8.2 (*SD* = 3.4) on PHQ-9 and 20.3 (*SD* = 5.4) on MoCA-5min. Three independent communities were identified in PHQ-9 and MoCA-5min items, suggesting that depression is not a uniform entity (2 communities) and has differential connections with cognition. The network estimation results suggested that the 2 most prominent connections between depressive symptoms and cognitive domains were: (1) *anhedonia* with *executive functions/language* and (2) *sad mood* with *memory*. Among all depressive symptoms, *sad mood* had the highest BEI, bridging depressive symptoms and cognitive domains.

Discussion and Implications: *Sad mood* seems to be the pathway between depression and cognition in this sample of older Chinese. This finding highlights the importance of *sad mood* as a potential mechanism for the co-occurrence of depression and cognitive impairment, implying that intervention targeting sad mood might have rippling effects on cognitive health.

Translational Significance: This study used a novel network analysis approach, explored the network structure of depressive symptoms and cognitive domains, and investigated the bridge symptom(s) between depressive symptoms and cognitive performance in community-dwelling older adults. The results revealed three communities in depressive symptoms and cognitive domains. The sad mood was identified as the bridge symptom, implying the importance and potential rippling effects for cognitive health of targeting sad mood in depression interventions.

Keywords: Cognition, Depressive symptoms, Mental health, Network analysis, Quantitative research methods

Late-life depression is one of the most common mental disorders, and it is associated with poor quality of life and substantial societal cost (Zheng et al., 2018). Late-life depression is estimated to occur in 13.3% of older adults globally (Abdoli et al., 2022) and is reported to usually accompany cognitive impairment (Richard et al., 2013). Abundant evidence supports the associations between depression and cognition (Panza et al., 2010). However, research findings are inconsistent, and the mechanism of their relationship is likely complex. Some studies suggest that depression is a causal factor for subsequent cognitive decline, while others show that depression may be part of the prodromal stage of dementia (John et al., 2019). Cognitive impairment and depression may also be associated with common risk factors and neurological

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substrates (Bennett & Thomas, 2014). Previous studies focusing on diagnoses have an underlying "explanatory reductionism" assumption that mental health disorders are unique nosological entities that are ultimately explainable by specific neurobiological dysfunctions (Borsboom et al., 2019).

However, some researchers argue that this approach to psychopathology has not been fruitful in identifying a common pathogenic pathway in most mental disorders (Borsboom et al., 2019; Gorwood, 2008). A reductionist approach may conceal precise relationships between symptoms (Borsboom, 2017; Borsboom et al., 2019), which may explain the current inconclusive evidence regarding the relationship between depression and cognitive impairment in old age. As an alternative to the reductionist view, symptom network theory that regards mental disorders as a network of causal interactions among symptoms has been proposed. According to the network theory, symptoms are interrelated and mutually reinforcing entities (Manfro et al., 2021); the relationship between symptoms, rather than a hierarchically superordinate common cause, perpetuates the mental disorders (Borsboom & Cramer, 2013). Therefore, if an intervention changes the condition of one symptom, the probability distribution of other symptoms will also change (Borsboom et al., 2019).

Network analysis is a statistical tool for determining how individual symptoms interact with one another within and across mental disorders. Associations between *nodes* are established in network analysis by *edges*. In psychopathological studies, nodes are equivalent to symptoms, operationalized as items from questionnaires that assess mental disorders (Jones et al., 2021). Network analysis may quantify bridge symptoms that link different disorders, that is, as a connector or pathway between different symptom clusters (Jones et al., 2021). For example, both anxiety and depressive symptoms can accelerate sleep problems, which is found to be one "bridge" linking to a higher risk for dementia; in other words, sleep problems might be a bridge between affective disorders and dementia (Beard et al., 2016; Hahn et al., 2014). The ability to discover bridge symptoms across communities is a critical feature of network analysis. Bridge symptoms are conceptualized as the symptoms that connect different clusters of symptoms corresponding to different mental disorders, regardless of any overlap between different symptom clusters. In other words, bridge symptoms can facilitate the discovery of how symptoms from one disorder might "spread" to symptoms of another (Jones et al., 2021). To sum up, network analysis may provide information on the specific symptom connections perpetuating comorbid depressive symptoms and cognitive impairment and the pathways, that is, bridge symptoms, that connect symptoms of one disorder with the other.

To our best knowledge, no previous study has explored the network structure of depressive symptoms and cognitive domains nor investigated the bridge symptom(s) between depressive symptoms and cognitive performance in community-dwelling older adults. This study addressed this research gap by involving community-dwelling older adults with subthreshold depression, that is, with mild or more severe depressive symptoms but no formal diagnosis. Investigation among this population has important clinical implications because selective and indicated prevention for this group may generate better clinical outcomes and alleviate the care burden (Reynolds et al., 2012). Knowledge about the connections within depressive symptoms and their linkage with cognitive performances may guide more person-centered intervention design and maintain cognitive health in the long term.

Method

Participants and Procedures

The sample was derived from a community-based collaborative stepped care with peer support program for preventing late-life depression in Hong Kong, registered with ClinicalTrials.gov (NCT03593889; Liu et al., 2022). The program provides services to older adults, from those at risk of depression to those having subthreshold depression. Because this study aimed to investigate the networks between depressive symptoms, we excluded those at risk of depression, that is, those who scored below 5 on the Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001). The inclusion criteria of this study were: (1) aged 60 years or older; and (2) having mild or more severe symptoms, operationalized as a total score on the PHQ-9 ≥ 5 (Kroenke et al., 2001). The exclusion criteria were: (1) a known history of intellectual disability, autism, bipolar or schizophrenia-spectrum disorder, Parkinson's disease or dementia; and (2) an imminent suicidal risk assessed by a qualified social worker. Trained social workers collected baseline data on participants' depressive symptoms and cognitive function through face-to-face interviews.

Measures

Depressive symptoms

Depressive symptoms were measured by the Chinese version of the PHQ-9, which has a high internal consistency (Cronbach's $\alpha = 0.91$; Yeung et al., 2008). The nine items of the PHQ-9 are congruent with the DSM-5 diagnostic criteria for depression (American Psychiatric Association & Association, 2013). Each item is assessed using a 4-point Likert scale from 0 (*not at all*) to 3 (*nearly every day*), with higher scores representing higher symptom severity. A classification system is commonly used to interpret PHQ-9 score, with 0–4 points indicating no depression, 5–9 as mild, 10–14 as moderate, 15–19 as moderately severe, and 20–27 as having severe depressive symptoms (Kroenke et al., 2001). The PHQ-9 has been used in clinical work with patients with and without dementia (Hancock & Larner, 2009).

Cognitive function

Cognitive function was measured using the Hong Kong version of the Montreal Cognitive Assessment 5-minute protocol (MoCA-5min; Wong et al., 2015). The MoCA-5min protocol has four items that evaluate the following cognitive domains: attention, executive functions (EF)/language, orientation, and memory. The total score of the MoCA-5min ranges from 0 to 30, with a higher score indicating better cognitive function. The MoCA-5min protocol has been validated in differentiating people with and without cognitive impairment (Wong et al., 2015).

Demographic variables

Demographic variables included age, sex, and highest educational attainment. We also assessed participants' economic status by whether they were recipients of means-tested social welfare (i.e., receivers have low economic status), mobility function using a single item ranging between 1 and 5 on the level of assistance needed, and vision with a single item

ranging between 1 and 3 on the level of visual impairment. A score of 1 on mobility and vision indicates no impairment, and higher scores indicate more difficulties and lower functioning.

Statistical Analysis

There were four steps to constructing a network analysis, and R version 4.1.2 was used with "qgraph," "igraph," and "bootnet" packages. Details of the analyses are described in the following.

Network estimation

All items were selected because there is no apparent conceptual overlap between PHQ-9 and MoCA-5min items. In the current study's network structure, each node represents one unique item from the PHQ-9 or MoCA-5min measures. The connections, that is, edges, between nodes represent the partial correlation between the two nodes, and the strength of edges is referred to as "weight." The Gaussian Graphical Model (GGM) was applied to estimate the network based on partial correlation coefficients (Epskamp et al., 2018). The GGM was regularized by the graphical Lasso based on an extended Bayesian information criterion (EBICglasso) with a tuning parameter (default: $\gamma = 0.5$; Epskamp et al., 2018). The purpose was to reduce false positives and to build a more parsimonious model. The Fruchterman–Reingold (spring) method, which puts nodes with stronger average relationships toward the center of the network, was utilized to decide on node placement inside the network (Epskamp et al., 2018). Detailed tutorial on network estimation for psychological networks using the statistical programming language R can be found in Epskamp et al. (2018).

Community detection analysis

Community detection, also known as clustering, is a technique in network analysis to identify communities/groups of nodes within a network that are more densely connected with each other than with nodes in other communities (Radicchi et al., 2004). In the current study, a "community" comprises depressive symptoms or cognitive functions densely linked together. The Walktrap algorithm is commonly used to detect communities in large networks via random walks (Pons & Latapy, 2005, 2006). A random walk is a general random process that describes a path that involves a series of random steps in a large network, and the direction of each step to a node is determined probabilistically and uniformly among its neighbors (Xia et al., 2019). Such random walks on a graph/network tend to get "trapped" into densely connected nodes corresponding to communities, and move less frequently between communities (Pons & Latapy, 2005; Smith et al., 2020). The distances between pairs of nodes were calculated using these random walks. Bottom-up hierarchical clustering was then used to arrange nodes into groups with small intracommunity and larger intercommunity distances (Cai et al., 2010). A seed was set to execute the algorithm 1,000 times to boost the reliability of the findings. The nodes that belong to one community tend to have stronger connections with each other while having weaker connections with the nodes in other communities.

Centrality indices calculation

There are several centrality indices, and bridge expected influence (BEI) was used to identify bridge symptom/domain in the current study. BEI indicates a node's sum connectivity with other communities (Jones et al., 2021) and is especially relevant for clinical researchers targeting specific symptoms for therapeutic deactivation (Robinaugh et al., 2016). We used one-step BEI, defined as the sum of all edges extending from a given node (where the sign of each edge is maintained), and calculated as the following:

$$\text{Bridge expected influence} = \sum_{b \in (N(a) - C)} w_{ab}$$

W_{ab} denotes the weight on each edge ab, C is a set of nodes in a community in the network, and $N(a)$ denotes the set of nodes adjacent to a. A positive BEI suggests that increased activation of one node is linked to increased activation of the nodes in another community, and a negative BEI means that increased activation of the node is linked to decreased activation of the nodes in another community. Currently, there is no specific cutoff for empirical studies; a higher absolute value of BEI indicates a higher impact of the node on the overall structure of the network, which may "bridge" between different communities (Jones et al., 2021). Based on previous studies (Elliott et al., 2020; Kaiser et al., 2021) and the meaning of the BEI score, we used the highest BEI score to identify the bridge symptom in this study.

Network stability test

The network stability test assesses a network's robustness by measuring its community structure's stability, and a higher stability score indicates a more robust community structure (Costenbader & Valente, 2003). Case-dropping bootstrap analyses were performed 5,000 times to test the stability of weight and centrality. The stability of the centrality/weight coefficients was assessed by the correlation stability (CS)-coefficient, defined as the maximum proportion of cases that can be dropped (with 95% certainty) to maintain a correlation larger than 0.7 with the original centrality value, represented by CS (cor = 0.7). Thresholds of 0.25 and 0.5 were applied, suggesting modest and strong/high metric stability, respectively (Epskamp et al., 2018). To elaborate, if the value of CS (cor = 0.7) ≥0.25, we can conclude that the results are modestly reliable and robust, and highly robust if CS ≥0.5.

Results

Sample Characteristics

Table 1 summarizes participants' demographic information and the PHQ-9 and MoCA-5min item-level descriptive statistics. A total of 2,792 participants were included in the analysis of this study. Their average age was 77.28 years ($SD = 8.51$), and 78.1% ($n = 2,181$) were female. The majority had a low level of education, with 28.5% received no formal education and 43.2% had primary education. Participants' average mobility difficulty was 1.53 ($SD = 0.67$), and vision impairment was 1.32 ($SD = 0.48$), suggesting normal mobility and vision in general. The average PHQ-9 and MoCA-5min scores were 8.3 ($SD = 3.4$), and sleep disturbance (P3) was the most frequently reported problem. Based on the PHQ-9 cutoff scores, 2,093 (75.0%) of the participants had mild depressive symptoms ($5 \leq$ PHQ-9 ≤ 9), 665 (23.8%) had moderate to moderately severe symptoms ($10 \leq$ PHQ-9 ≤ 19), and 34 (1.2%) had severe depressive symptoms (PHQ-9 ≥20). The average MoCA-5min total score was 20.3 ($SD = 5.4$), based on the education- and age-adjusted cutoff criteria (Wong et al., 2015),

Table 1. Participants' Demographic Characteristics and Scale Item Descriptive Statistics (N = 2,792)

Variable	Short form	Mean (*SD*)	*n* (%)
Demographic characteristics			
Age		77.28 (8.51)	
Female			2,181 (78.1%)
Education level			
No formal education			797 (28.5%)
Primary			1,205 (43.2%)
Secondary		422 (15.1)	
Postsecondary			368 (13.2%)
Mobility difficulty (1–5)		1.53 (0.67)	
Vision impairment (1–3)		1.32 (0.48)	
Scale variables			
Depression (PHQ-9), score range 0–27 (*Individual item score range 0–3*)		8.27 (3.37)	
1. Little interest in doing activities	D1: Anhedonia	1.15 (0.93)	
2. Depressed, feeling down, or hopeless	D2: Sad mood	1.40 (0.86)	
3. Inability to fall or remain asleep, or excessive sleeping	D3: Sleep disturbance	1.68 (0.97)	
4. Feeling tired or having little energy	D4: Fatigue	1.50 (0.88)	
5. Poor appetite or overeating	D5: Appetite	0.36 (0.71)	
6. Feeling bad about self—or feeling a failure or having let down self or family	D6: Guilt	0.69 (0.88)	
7. Trouble concentrating on things, such as reading the newspaper or watching television	D7: Trouble concentrating	0.85 (0.81)	
8. Moving or speaking so slowly that others could have noticed? Conversely, being fidgety or restless and moving around a lot more than usual	D8: Psychomotor agitation or retardation	0.34 (0.63)	
9. Thoughts of being better off dead or hurting self in some way	D9: Suicidal ideation	0.30 (0.60)	
Cognitive performance (MoCA-5min protocol), score range 0–30		20.29 (5.35)	
1. Immediate recall of 5 words, *score range 0–5*	C1: Attention	2.80 (1.52)	
2. 1-min verbal fluency, *score range 0–9*	C2: EF/Language	4.84 (2.59)	
3. 6-item date and geographic orientation, *score range 0–6*	C3: Orientation	4.89 (1.85)	
4. Delayed recall and recognition of 5 words learned in question 1, *score range 0–10*	C4: Memory	5.81 (3.15)	

Note: EF = executive function; MoCA-5min = Montreal Cognitive Assessment 5-minute protocol; PHQ-9 = Patient Health Questionnaire-9 item; *SD* = standard deviation.

422 (15%) of the participants meet the criteria for significant cognitive impairment and need further assessment for neurocognitive disorders.

Network Structure and Communities Detected

With 13 nodes (9 PHQ-9 items and 4 MoCA-5min items), 91 edges were tested, and 57 of them (62.64%) were larger than zero (see Figure 1A). Overall, the nodes were organized more closely with those within the depressive symptoms or cognitive domains (visually two "poles" in Figure 1A) than those from the opposite pole, demonstrating primarily interactions within the depressive symptoms and cognitive domains, respectively. The community detection analysis also revealed three communities in the nodes of depressive symptoms and cognitive domains. All four cognitive items were in one community (C1–C4), *sleep disturbance* (D3) and *fatigue* (D4) were in the second community, and other depressive symptoms were in the third community (D1–D2; D5–D9). In Figure 1A, the partial correlations (pc-GLASSO) suggest that the most robust connections between depression symptoms and cognitive domains were *anhedonia* (D1) with *EF/language* (C2) (pc-GLASSO = −0.04), and *sad mood* (D2) with *memory* (C4) (pc-GLASSO = −0.04). The results of the edges for every pair of two nodes are summarized in Supplementary Table 1.

Bridge Symptom

The BEI of each depressive symptom in the cognitive domains is shown in Figure 1B. Among all depressive symptoms, *sad mood* had the highest BEI (absolute value) connecting depressive symptoms and cognitive domains. *Sad mood* was most strongly associated with the cognitive domain of *memory* (C4). In the *sleep disturbance* (D3) and *fatigue* (D4) community, *fatigue* (D4) was more strongly related to the community of cognitive performances. Figure 2 shows the results of case-dropping bootstrap of the centrality indices/edges. The CS-coefficient indicates that the stability of edge weights and BEI met the cutoff for metric stability (edge weights: CS [cor = 0.7] = 0.75; BEI: CS [cor = 0.7] = 0.28), which suggests that the results of edge weights and BEI are reliable.

Discussion

This study is the first to apply a symptom network approach to investigate the relationship between depressive symptoms and cognitive domains in community-dwelling older people.

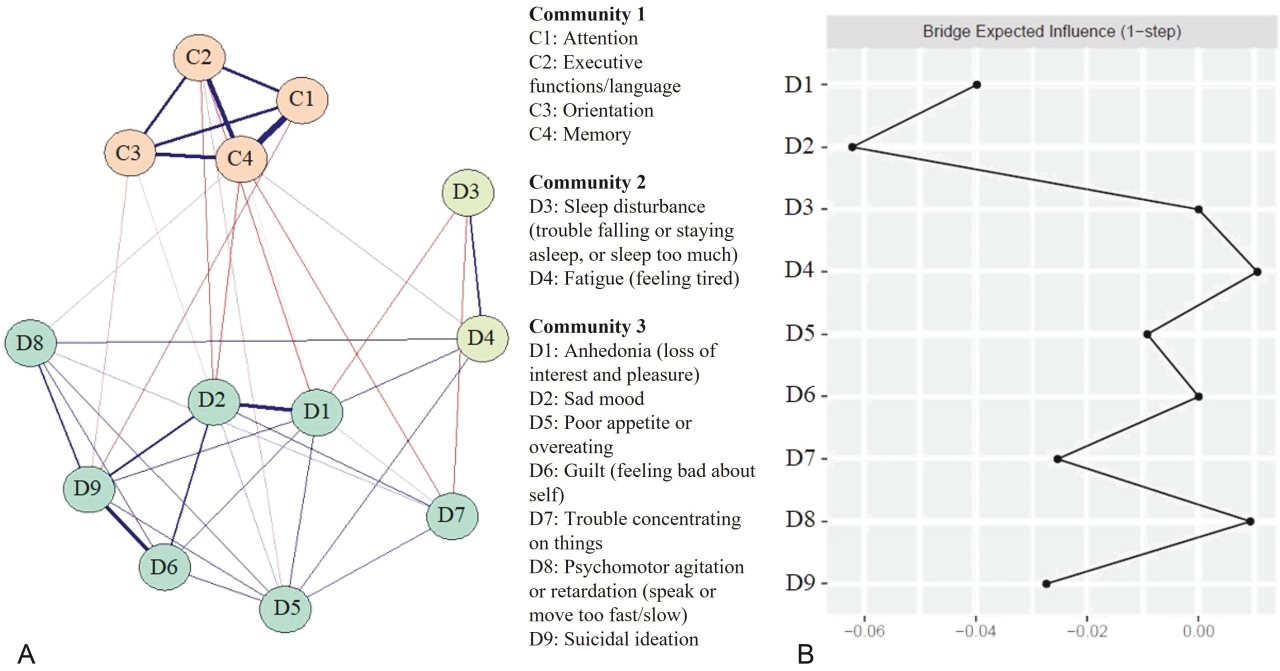

Figure 1. (A) Network of depressive symptoms and cognitive domains, three independent communities were identified; and (B) bridge expected influence value of depressive symptoms to cognitive domains in older adults with depressive symptoms (N = 2,792).

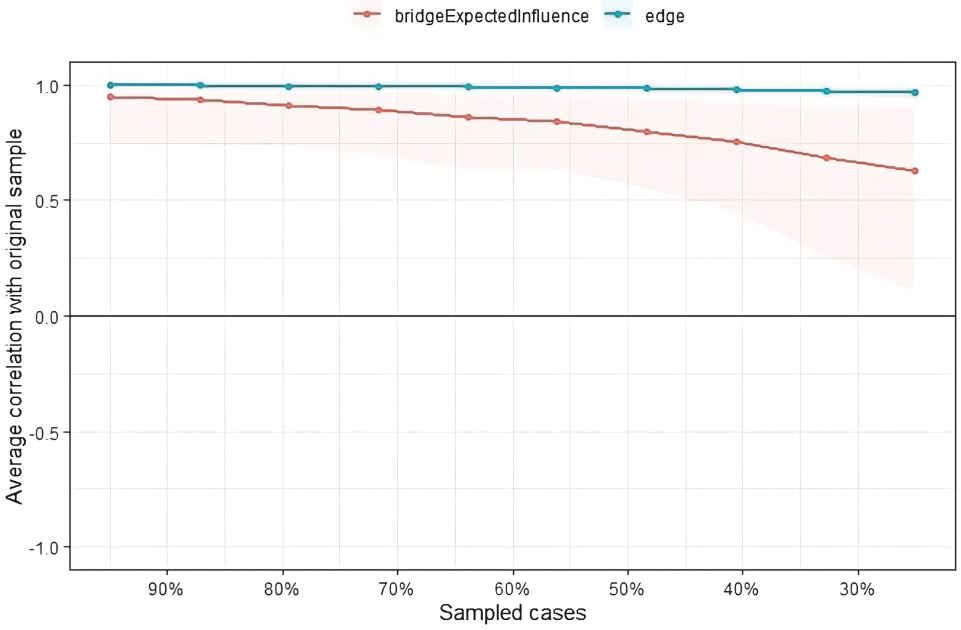

Figure 2. Stability of centrality indices by case-dropping subset bootstrap. *Notes*: The lines show the average correlation between network centrality indices/edge weights of networks in the sample after dropping and the original sample. The range of correlations from the 2.5th to the 97.5th quantile is represented by the shaded regions surrounding the lines.

The results suggest that not every symptom is equal; the structure of the symptoms provides supplementary information to the sum of symptoms, which may have further clinical implications. The same total score on a depression assessment scale may underscore different symptom profiles, leading to differential impacts on cognition.

First, with community detection network analysis, we found three communities in depressive symptoms and cognitive domains measured by the PHQ-9 and MoCA-5min. Four cognitive domains measured in the current study formed one community, suggesting that different cognitive domains are highly connected. *Sleep disturbance* and *fatigue* formed a second community, and the rest of the depressive symptoms formed a third community. The notion of communities of symptoms is similar to symptom clusters, with added value of evaluating how clusters of symptoms interact with each other (Strauss et al., 2019). The traditional confirmatory factor analysis (CFA) has several inherent limitations, for example, being sensitive to sample size and correlation between factors. In contrast, network analysis is

not subject to these limitations and can produce more reliable estimates based on heuristic approaches that complement CFA, and these two analyses may yield different results (Esfahlani et al., 2018).

Previous studies have suggested somatic and non-somatic/affective symptom clusters of depressive symptoms through systematic reviews (Elhai et al., 2012). In the current study, we found two communities of depressive symptoms, consistent with the previous findings that depression is not a uniform entity (Maj, 1998, 2012); *sleep disturbance* and *fatigue* are both somatic symptoms and belong to one community, also consistent with previous research. However, despite being a somatic symptom, *appetite* had a stronger connection with other affective symptoms. This inconsistent finding might reflect how older Chinese adults experience and express depression, presumably due to the cultural belief in the association between food and mood. For example, Traditional Chinese Medicine believes that the consumption of damp-producing foods may result in dampness and phlegm in the body, leading to feelings of depression and further dampening appetite (Ye et al., 2019).

Second, the two core features and criterion items toward the diagnosis of major depressive episodes, *sad mood* and *anhedonia*, were found to have strong associations with *memory* and *EF/language* domains of cognition, respectively. The association between *anhedonia* and cognitive impairment is well documented (McIntyre et al., 2016), and our findings add to the literature that the association might be most prominent between *anhedonia* and *executive function/language* domain. Anhedonia refers to the diminished ability to experience pleasure, and a recent study on the neurobiological mechanisms of anhedonia suggested that the severity of anhedonia is associated with a deficit of activity of the ventral striatum and an excess of activity in the ventral region of the prefrontal cortex with a pivotal role of dopamine (Gorwood, 2008). The ventral region of the prefrontal cortex plays a role in inhibition, response selection, and monitoring (Jones & Graff-Radford, 2021), all belong to the EF skills; and dopamine also plays an essential role in the frontal cortex in mediating EF (Hosenbocus & Chahal, 2012). The strong connection between *sad mood* and *memory* (delayed word recall in this study) may be attributed to the priming effects of mood on learning and recall of schema-congruent material, precluding any mood state-dependent effects (Ellis & Moore, 1999).

Third, *sad mood* bridges depressive symptoms and cognition, while the *sleep disturbance/fatigue* community had no significant association with the cognitive community, inconsistent with existing literature on the strong association between sleep and cognition (Yaffe et al., 2014). The core role of *sad mood* as a bridge symptom merits further exploration. There has been evidence that emotion regulation is a resource-demanding process that interrupts tasks performed concurrently or afterwards (Robinson et al., 2013). Negative emotions can hinder cognitive performance in a wide range of tasks, from simple discrimination to complex reasoning tasks in older adults (Roquet et al., 2022). The link between *sad mood* and *memory* might be a potential pathway that activates the whole symptom network, which can be tested in future studies designed for hypothesis testing. On the other hand, the absence of a connection between *sleep disturbance/fatigue* might be due to the partial correlation used in the present study; the impact of sleep disturbance on the cognition of older adults with depressive symptoms was adjusted by other symptoms, such as *sad mood*.

Implications

The findings of the present study may have two clinical implications. First, symptom communities may provide complementary information to diagnostic criteria, allowing early identification of depressed individuals with comorbid cognitive impairment. We found two communities of depressive symptoms, and *sleep disturbance/fatigue* had a weak connection with cognition, suggesting that clients exhibiting mainly symptoms from this community have a lower risk for cognitive impairment than those with depressive symptoms from the other community. Second, the bridge symptoms can be considered transdiagnostic features, and interventions targeting them are more likely to benefit patients with depression and cognitive impairment. There is evidence that the change in central and bridge symptoms connects to changes in other symptoms (Elliott et al., 2020). These findings suggest that interventions focusing on *sad mood*, such as emotion-focused therapy, might also benefit clients' cognitive health. However, it is still hard to delineate the effects of interventions on a single symptom. Alternatively, measures of the relevant change process might be considered to assess the symptom-specific change mechanisms insinuated by the interventions.

Limitations

This study has several limitations. First, as a network analysis with cross-sectional data, the edges between measured symptoms were undirected, and we cannot distinguish the relationship between sad mood and cognitive impairment. *Sad mood* may be a bridge, consequence, or byproduct of cognitive impairment. Hence, caution should be exercised in generating clinical interventions for older adults with mood and cognitive issues. Future studies might consider using longitudinal data to explore this relationship further. Second, this study used a community older Chinese sample with depressive symptoms, and the results may not be generalized to clinical samples. Future studies may recruit a clinical sample with DSM-5 diagnoses to validate or compare with our findings. Third, this study used the MoCA-5min protocol to assess cognitive functioning. Although this measurement has been validated as a screening tool for cognitive impairment (Wong et al., 2015), it is more commonly used to assess general cognition rather than separate cognitive domains. Other, more specific cognitive domains, such as cognitive flexibility, might associate with depressive symptoms and need to be assessed with more specific tools.

Conclusion

In summary, network analyses among a large community-dwelling sample of older adults with depressive symptoms revealed three interrelated communities of depressive symptoms and cognitive domains. *Sad mood* was identified as the bridge symptom between depression and cognition, implying the importance and potential rippling effects of targeting *sad mood* in interventions. Future research might consider validating these findings in clinical populations and through longitudinal studies.

Supplementary Material

Supplementary data are available at *Innovation in Aging* online.

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Conflicts of Interest

None.

Data Availability

Data used in these analyses are available upon reasonable request from the corresponding author. The study reported in the manuscript was preregistered with ClinicalTrials.gov (NCT03593889).

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Ethics Approval

This study was approved by the Human Research Ethics Committee of the University of Hong Kong (reference number: EA1709021). The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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