

Intervention Targets for Depressive and Cognitive Symptoms in Older Adults: A Simulation-Based Network Analysis

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Abstract

BACKGROUND

The co-occurrence of depressive and cognitive symptoms in older adults is a major driver of functional decline and reduced quality of life. However, conventional management strategies typically address these conditions as distinct entities, potentially overlooking the dynamic symptom-level interactions that sustain them. This study applied a simulation-based network intervention to identify key targets within the depressive - cognitive symptom network.

METHODS

Data were obtained from 10,157 adults aged ≥ 60 years in the 2020 China Health and Retirement Longitudinal Study (CHARLS). Depressive symptoms were assessed using the 10-item CES-D, and cognitive function was evaluated using tests of orientation, memory, calculation, and visuospatial ability. Network analysis based on the Ising model was used to identify central and bridge symptoms. Simulation-based interventions were performed using the NodeIdentifyR algorithm to examine the effects of symptom alleviation and aggravation on the overall network severity.

RESULTS

The estimated network revealed two interconnected symptom clusters. "Calculation" and "Orientation" functioned as bridge symptoms linking cognitive and depressive domains. "Depressed mood," "Exhaust," and "Cannot continue" showed the highest centrality. NIRA simulations indicated that alleviating "Mind distraction" produced the greatest reduction in symptom burden (the mean total score decreased from 6.92 to 5.35). In contrast, aggravating "Fear" and "Cannot continue" resulted in the largest increases in network severity.

CONCLUSIONS

This simulation-based network analysis identifies "Mind distraction" as a key therapeutic target and "Fear" and "Cannot continue" as priority targets for preventive intervention in older adults with co-occurring depressive and cognitive symptoms, supporting a shift toward symptom-focused management strategies.

1. Background

The co-occurrence of depressive symptoms and cognitive impairment in older adults has become increasingly prevalent. Epidemiological data indicate that approximately 17.9% of older adults experience both conditions, and among community-dwelling Chinese older adults the comorbidity rate

reaches roughly 10.5% [1, 2]. Cognitive decline and depressive symptoms share several common features and regularly co-occur among older adults[3]. Research indicates that depression in early life is a risk factor for dementia[4], while depression in later life can be considered a prodrome of dementia[5]. Conversely, this relationship is strictly bidirectional. As older adults experience progressive cognitive deficits, the psychological distress and perceived loss of autonomy associated with this decline frequently precipitate or exacerbate depressive symptoms, thereby fueling a synergistic deterioration of both mental and cognitive health[6, 7]. A recent 16-year longitudinal analysis of 8,268 English adults aged 50 years or older provides empirical support for this bidirectional relationship: greater depressive symptoms were associated with poorer memory at baseline and steeper memory decline over time, while a gradual linear change in depressive symptoms contributed to accelerated memory loss and vice versa, suggesting that psychological mood and memory performance are intrinsically associated[8]. This comorbidity is not a simple additive combination but a mutually reinforcing dynamic: cognitive decline erodes the cognitive resources required for emotion regulation, while depressive symptoms continuously drain the capacity needed for daily cognitive functioning. As a direct consequence, older adults with both conditions face a 1.5- to 2-fold greater risk of functional disability—including limitations in activities of daily living, instrumental activities, and mobility—compared to those with either condition alone [9]. However, current clinical management often addresses depression and cognitive impairment as separate diagnostic entities. This disease-centered approach may overlook the persistent symptom-level interactions that sustain comorbidity, which could partly explain the suboptimal outcomes sometimes observed with conventional interventions[10–12].

The network approach to psychopathology offers a framework for addressing this limitation[13]. In this perspective, mental disorders are not driven by a common latent cause but emerge from systems of mutually causal, activating relationships among symptoms[14]. Within such a network, different symptoms serve distinct functional roles: central symptoms drive overall network activation, whereas bridge symptoms transmit dysfunction from one symptom cluster to another, reinforcing the comorbid state[15]. Within this framework, centrality and regulatory influence are conceptually distinguishable: centrality reflects a symptom's topological position, while regulatory influence reflects the degree to which the whole system responds when that symptom is perturbed[16]. This leads to a testable prediction: the symptoms most capable of "cooling down" the network may not coincide with those identified as most central through conventional static indices.

Several studies have applied network analysis to the comorbidity of depressive and cognitive symptoms in older adults. Sun et al. [1]found that "depressed mood," "exhaustion", and "cannot continue" had the highest expected influence, functioning as core symptoms, while "calculation" and "orientation" showed high bridge expected influence, linking the two clusters. Another studyreported that "fatigue," "cannot continue," and "calculation" played important roles in maintaining network density. These cross-sectional studies provide valuable descriptive insights into the relational architecture between depressive and cognitive symptoms [17]. However, they capture only contemporaneous associations at a single time point and cannot answer the clinically pivotal question: when a symptom is deliberately alleviated or aggravated, what direction and magnitude of cascading change occur across the remaining symptoms?

More critically, cross-sectional centrality cannot distinguish whether a symptom actively drives other symptoms or is passively elevated by them. This interpretive ambiguity means that selecting intervention targets solely on the basis of static centrality risks directing resources toward symptoms that are themselves highly reactive and whose improvement fails to trigger meaningful cascading benefits. This is the core research gap that the current study addresses.

To bridge this gap, Lunansky et al.[18] introduced the NodeIdentifyR (NIRA) algorithm. NIRA, built on the Ising model, simulates symptom alleviation or aggravation by adjusting each symptom's threshold and computes the resulting change in overall network activation. The method operates under the assumption that the estimated cross-sectional network structure adequately approximates the conditional dependence relationships among symptoms, thereby supporting inference about cascading effects. While it cannot replace causal evidence from real time-series data, it is capable of generating differential predictions about which nodes have the greatest propagation potential under a given model. The symptom whose relief yields the largest reduction in network burden is considered a potential treatment target, while the symptom whose worsening produces the greatest increase in network burden is considered a potential prevention target. This method has been preliminarily applied to depression–anxiety networks in adolescents and healthcare workers, and those initial applications revealed an important dissociation: the most effective targets identified through simulation were not fully concordant with those indicated by conventional centrality or bridge indices [19, 20]. This empirical dissociation supports the claim that dynamic simulation provides incremental information beyond static topological description.

Guided by this theoretical and methodological background, the present study applies the NIRA simulation framework to a large, nationally representative sample of Chinese older adults and derives the following testable predictions for the depression–cognition comorbidity network: (1) alleviating a specific symptom will produce the greatest reduction in overall network activation, thereby identifying a priority treatment target; (2) aggravating a specific symptom will produce the greatest increase in overall network activation, thereby identifying a priority prevention target. Further, we directly compare simulation-identified targets with those derived from traditional centrality and bridge indices to determine whether dynamic simulation makes a non-redundant contribution to target identification[18]. Because NIRA relies on cross-sectional associative data and uses a preset threshold adjustment (two standard deviations), its outputs reflect cascading effects under model assumptions, not direct quantification of real-world intervention effects[18, 21]. Accordingly, the findings should be regarded as testable predictions to inform the design of future clinical trials, rather than as immediate prescriptions for practice.

2. Methods

2.1. Participants and procedure

This study utilized data from the 2020 China Health and Retirement Longitudinal Study (CHARLS)[22]. CHARLS is a nationally representative, open-source database containing information on the basic demographics, mental health, and physical health of individuals aged 45 years and older in China. The baseline CHARLS survey encompassed 28 provinces, 150 counties or districts, and 450 villages or urban communities across the country, and involved 17,708 individuals from 10,257 households. The inclusion criteria for this study were: (1) age ≥ 60 years and (2) complete information on key variables. Participants were excluded if they had missing data for more than 50% of the items in the main variables, such as depression and cognition measures[23]. After these exclusions, 10,157 participants were included in the final analysis.

2.2. Measures

2.2.1. Depressive Symptoms

Depressive symptoms in older adults were measured using the 10-item Center for Epidemiologic Studies Depression Scale (CESD-10)[24]. The scale includes ten items that describe emotional and behavioral experiences during the past week. Each item is treated as a depressive symptom node in the network. Examples include “I was bothered by things that usually do not bother me”, “I felt lonely”, and “I felt depressed”. Each item was scored from 0 to 3. The total score ranged from 0 to 30. Higher scores indicate more severe depressive symptoms.

The NodeIdentifyR Algorithm (NIRA) uses only the categorical variables. Therefore, each CESD-10 item was converted into a binary variable. A score of 0 was defined as symptom absence (0). A score of 1 or higher was considered a symptom (1). This method has been used in many symptom network studies to determine whether a symptom is present[20].

2.2.2. Cognitive Function

Cognitive function was measured using items from the U.S. Health and Retirement Study (HRS). Cognitive function comprises two dimensions: memory function and mental State. Memory Function has a score range of 0–10 and is assessed using a word recall test, including both immediate and delayed recall. Higher scores indicate better memory ability. Mental State has a score range of 0–11 and is evaluated using the TICS-10 and a figure drawing test, primarily measuring orientation, information processing ability, and visuospatial skills. The total cognitive function score ranges from 0 to 21, with higher scores indicating better cognitive function[25].

The NIRA method requires the use of binary variables[18]. For this reason, the scores for each cognitive domain were converted into binary values using the sample median as the cutoff (scores below the median = 1, scores at or above the median = 0). This median-based dichotomization follows the default specification of the `bootnet::binarize` function for Ising model estimation, which uses the median split as the standard preprocessing step when estimating Ising networks from continuous data[26]. Notably, the Ising model is the network estimation framework that underpins both the symptom network construction and the subsequent NIRA simulations ; adopting the default binarization protocol therefore ensures

internal consistency between the network estimation and intervention simulation stages[18]. This approach defines "impairment" as relative cognitive vulnerability within the sample rather than as a clinically validated diagnosis—an operationalization consistent with the study's aim of examining symptom-level interactions in a population-based context.

To evaluate whether binarization affected the estimated network structure, a Gaussian Graphical Model (GGM) using the original continuous variables was fitted as a complementary analysis (see Section 2.3.3). The cross-model comparison confirmed that the core network structure and bridge symptoms were robust to the dichotomization procedure.

2.3. Statistical Analysis

2.3.1. Data Preprocessing and Descriptive Analysis

Descriptive statistical analyses were conducted using SPSS version 26.0. Missing data were handled using the mouse package in R, version 4.5.0. Multiple imputation was performed using a random forest approach to reduce the impact of missing data on the network estimation[27].

The mice procedure generates several imputed values for each missing observation, based on the relationships observed in the data. This process preserves variability in the dataset and avoids bias that may occur with single-imputation methods. During the iterative imputation process, each variable was modeled conditional on the other variables. This helps to account for potential dependencies among variables and ensures that the imputed values remain consistent with the observed data patterns.

2.3.2. Symptom Network Construction

An Ising model was estimated using the estimateNetwork function of the bootnet package[26]. A symptom network with 15 nodes was constructed, including 10 depressive symptoms and five cognitive indicators. In this model, nodes represent symptoms or cognitive functions[13]. The edges represent partial correlations between nodes. Larger edge weights indicate stronger associations between the symptoms.

To identify key symptoms in the network, two centrality indices were calculated. The first is Expected Influence (EI), which reflects the overall impact of a symptom on the network and includes both direct and indirect effects. This index was used to identify core symptoms in the entire network. The second is Bridge Expected Influence (BEI), which reflects the role of a symptom in linking different symptom communities. This index was used to identify bridge symptoms that contributed to symptom spread across clusters[15].

2.3.3 Validation of Network Structure

To evaluate whether binarization distorted the estimated symptom network, a Gaussian Graphical Model (GGM) was estimated using the original continuous variables. The GGM was fitted via the EBICglasso method. Consistency between the Ising and GGM networks was assessed by Spearman correlation of

edge weights, and bridge centrality indices from both models were compared qualitatively to verify the stability of the identified bridge symptoms[28, 29].

2.3.4. Network Stability and Accuracy Assessment

The reliability and accuracy of the estimated network were assessed using two robustness analyses. (1) A non-parametric bootstrap test was performed on the edge weights to generate 95% confidence intervals (CIs). Narrower CIs indicate more precise estimates. (2) The stability of the centrality indices (EI and BEI) was evaluated using centrality stability analysis, which calculates the correlation stability (CS) coefficient. According to established guidelines, a CS coefficient above 0.25 indicates moderate stability, while a coefficient above 0.50 indicates high stability[26].

2.3.5. Simulated Network Interventions using the NodeIdentifyR Algorithm

To investigate the dynamic effects of symptom perturbations on the overall network state and to identify potential targets for clinical intervention, a simulated intervention analysis was conducted using the NodeIdentifyR (NIRA) algorithm from the IsingSampler R package[18]. NIRA simulates hypothetical interventions by adjusting the threshold parameters of individual nodes, and then calculates changes in the overall level of network activation. Two types of simulated intervention were applied to each of the 15 nodes.

(1) Alleviation Intervention: Identifying Treatment Targets The threshold of the target node was lowered by two standard deviations to simulate a moderate level of symptom relief. This reduced the probability that the symptoms would be activated and alter the stable state of the network. The intervention effect was evaluated by comparing total network activation scores before and after the simulation. The nodes associated with the largest decrease in the total score were considered the most relevant treatment targets.

(2) Aggravating Intervention: Identifying Prevention Targets The threshold of the target node was increased by two standard deviations to simulate symptom worsening under stressful conditions. Changes in total network activation scores were compared before and after the simulation. Nodes whose aggravation led to the largest increase in the total score were identified as key prevention targets because their deterioration was most likely to trigger overall network worsening.

2.4. Ethical Considerations

This secondary analysis of the CHARLS dataset relied on publicly available data. The original data collection procedure, which obtained informed consent from all participants, was approved by the Biomedical Ethics Committee of Peking University (Approval No.: IRB00001052-11015).

3. Result

3.1. Descriptive statistics

Among the 10157 eligible individuals, 5243 were female (51.62%). The detailed demographics and characteristics are presented in Table 1. Table 2 presents the abbreviations, scale items, mean scores, standard deviations, expected influence (z-scores), and bridge expected influence of depressive and cognitive symptoms.

Table 1
Demographics and characteristics of the sample(N = 10157)

Characteristics	Values
Age (years), mean (SD)	69.37 (6.53)
Sex, n (%)	
Male	4914 (48.38)
Female	5243 (51.62)
Marital status, n (%)	
Married	8037 (79.13)
Unmarried	58 (0.57)
Divorced	146 (1.44)
Widowed	1916 (18.86)
Education, n (%)	
Unfinished primary school	5188 (51.08)
Elementary School	2104 (20.71)
Middle School	1709 (16.83)
High School and above	1156 (11.38)
Retirement status	
Yes	7733 (76.15)
No	2422 (23.85)

Table 2
Basic information of symptoms and descriptive item statistics.

Symptoms	Item	Abbreviation	M(SD)	EI	BEI
Depressed mood	I felt depressed	depresl	0.48(0.49)	5.54	0.06
Exhaust	I felt that everything I did was an effort	effortl	0.49(0.49)	5.04	0.36
Sleeplessness	My sleep was restless	sleeprl	0.52(0.49)	3.09	0.00
Unhappy	I was happy	whappyl	0.77(0.42)	0.16	-0.61
Lonely	I felt lonely	flonel	0.31(0.46)	4.54	0.41
Upset	I was bothered by things that usually do not bother me	botherl	0.49(0.49)	3.83	-0.09
Cannot continue	I could not“get going”	goingl	0.25(0.44)	5.02	0.83
Mind distraction	I had trouble keeping my mind on what I was doing	mindtsl	0.50(0.50)	4.39	0.00
Hopeless	I felt hopeful about the future	fhopel	0.63(0.48)	1.82	-0.96
Fear	I felt fearful	fearll	0.22(0.42)	4.67	0.44
Immediate recall	Immediate recall	imrc	0.35(0.48)	2.91	-0.14
Delayed recall	Delayed recall	dlrc	0.49(0.49)	3.28	0.12
Orientation	Orientation	orient	0.37(0.48)	2.69	0.19
Visuospatial ability	Visuospatial ability	draw	0.73(0.44)	1.40	-0.02
Calculation	Calculation	ser7	0.45(0.49)	2.50	0.31
Note. Abbreviations: M = Mean; SD = Standard Deviation; EI = Expected Influence (z-scores); BEI = Bridge Expected Influence.					

3.2. Comorbidity network of depression and cognition

As illustrated in Fig. 1, depressive and cognitive symptoms formed two interconnected clusters. To examine the interaction mechanisms between these domains, we analyzed symptoms with a high Bridge Expected Influence (BEI). The analysis identified calculation and orientation as key bridges linking the

clusters. Calculation was positively associated with the depressive symptom “Cannot continue” ($r = 0.264$) and also significantly correlated with “Fear” ($r = 0.139$). Orientation acted as another crucial bridge, showing a strong negative correlation with “Hopeless” ($r = -0.265$) and a significant positive correlation with “Lonely” ($r = 0.133$). Notably, depressive symptom “Cannot continue” exhibited the highest BEI value (0.83). In turn, it was strongly connected to both “Calculation” ($r = 0.264$) and “Orientation” ($r = 0.211$), thereby further solidifying the interconnection between the clusters. Furthermore, in terms of overall network centrality, “Depressed mood” ($EI = 5.54$), “Exhaust” ($EI = 5.04$), and “Cannot continue” ($EI = 5.02$) were identified as the most central symptoms in the network (Table 1, Fig. 2). These bridge symptoms, particularly calculation, orientation, and “Cannot continue,” play a key role in connecting depressive and cognitive symptom clusters.

Network stability and accuracy analyses indicated that the edge weights were estimated with acceptable precision, as evidenced by their narrow confidence intervals. The Expected Influence (EI) and Bridge Expected Influence (BEI) indices demonstrated high stability, with a correlation stability (CS) coefficient of 0.75. To further evaluate whether binarization affected the network structure, a Gaussian Graphical Model (GGM) was estimated using the original continuous variables. The GGM exhibited a network structure highly consistent with the binary Ising model, with a strong Spearman correlation between their edge weights ($r = 0.607, p < 0.001$). Importantly, both models consistently identified “Calculation” and “Orientation” as the primary bridge symptoms connecting the depressive and cognitive communities. This cross-model convergence supports the robustness of the Ising-based network for the subsequent NIRA analyses. Detailed results of the stability assessments and cross-model validation are provided in Supplementary Materials.

3.3. Simulation of alleviating and aggravating interventions

The alleviation intervention showed that targeting “Mind distraction” significantly reduced the overall symptom level in the network ($t = 28.21, p < 0.001, p.adjust < 0.001$). The mean total score decreased from 6.92 (original threshold) to 5.35, indicating that this symptom may serve as a potential target for reducing overall depressive and cognitive symptoms. Similar effects were observed for alleviation interventions targeting “Exhaust” ($t = 24.50, p < 0.001, p.adjust < 0.001$) and “Depressed mood” ($t = 6.77, p < 0.001, p.adjust < 0.001$), which reduced the mean total score to 5.36.

Conversely, the results of the aggravating intervention revealed that intensifying “Fear” ($t = -27.08, p < 0.001, p.adjust < 0.001$) and “Cannot continue” ($t = -18.02, p < 0.001, p.adjust < 0.001$) led to a significant increase in the overall symptom level, suggesting they are suitable targets for preventing the overall worsening of symptoms within the network.

4. Discussion

This simulation-based network analysis, leveraging a large, nationally representative sample, systematically identified intervention targets within the comorbidity network of depressive and cognitive symptoms in older adults. The estimated network revealed a densely interconnected architecture

between affective and cognitive domains, with "Calculation" and "Orientation" serving as key bridge symptoms linking the two clusters. More importantly, by moving beyond static centrality indices to dynamic in silico perturbations, this study generated differential and testable predictions regarding treatment and prevention targets. Specifically, alleviating "Mind distraction" produced the largest reduction in overall network severity, whereas aggravating "Fear" and "Cannot continue" led to the most pronounced network exacerbation. It is critical to note that these items represent single self-reported indicators rather than formal clinical entities. Thus, our findings should not be interpreted as prescriptions to treat a single symptom in isolation. Instead, they should be viewed as identifying the candidate intervention mechanisms with the greatest cascading potential, thereby providing testable working hypotheses for developing future precision symptom management strategies.

4.1. Central and Bridge Symptoms in the Depression-Cognition Network

The identification of "Depressed mood," "Exhaust," and "Cannot continue" as the most central symptoms corroborates findings from multiple prior studies conducted among Chinese older populations[1, 17, 30]. Consistent with the Conservation of Resources theory, the prominence of "Exhaust" suggests that the perceived depletion of psychological resources constitutes a critical nexus from which motivational decline and cognitive impairments emanate [31]. This theoretical account has direct nursing implications: when older patients report extreme fatigue, clinicians should recognize it not as a mere somatic complaint but as a key signal that the patient's ability to engage in restorative activities (e.g., walking, social participation) and cognitive tasks may decline, potentially triggering a self-perpetuating cycle of reduced activity, worsening mood, and further cognitive decline[32].

"Calculation" and "Orientation" were identified as bridge symptoms, a finding that remained robust across cross-model validation, aligning with the work of Li et al. and Zheng et al. [17, 25]. These two cognitive nodes function as critical messengers between the depressive and cognitive symptom clusters. Notably, a negative pathway connected "Orientation" with the depressive symptom "Hopeless." This pathway provides a plausible mechanistic hypothesis: impaired temporal or spatial orientation may distort an individual's perception of their circumstances and future prospects, thereby fueling feelings of hopelessness and helplessness[17]. From a nursing assessment perspective, this suggests that newly emergent difficulties with simple calculation or temporal disorientation should trigger systematic screening for co-occurring motivational deficits and hopelessness. These presentations are unlikely to be independent issues; rather, they are different facets of a single, self-sustaining comorbid syndrome.

4.2. Target Symptoms for the Treatment and Prevention of Depressive and Cognitive Symptoms

A central finding of this study is the divergence between static centrality and dynamic regulatory influence. Although "Mind distraction" was not the most central node in traditional indices, its simulated alleviation yielded the greatest overall network improvement. This finding should not be crudely

interpreted as a mandate to “intervene on a single attention item.” Rather, it generates a more sophisticated hypothesis: the latent cognitive process represented by this item—namely, attentional dyscontrol biased toward internal negative states—may constitute a high-leverage point for disrupting the vicious depression–cognition cycle. This hypothesis is grounded in cognitive neuroscience frameworks, which posit that when attentional resources are rigidly anchored to negative internal thoughts, cognitive flexibility is severely compromised, simultaneously aggravating both cognitive and affective components of the network [33, 34]. For clinical nursing, therefore, the future direction lies not in isolated “attention training,” but in the systematic development and validation of comprehensive nursing modules that target attentional regulation as a core therapeutic process—such as structured mindfulness-based cognitive training or attention bias modification techniques—and in testing whether improving this critical node produces generalized downstream network benefits[35].

The identification of “Fear” as a key prevention target aligns with longitudinal network findings reported by Ma et al.[36]. As an intense emotional response to potential threats, fear may drive network deterioration through multiple pathways: persistent fear activates the hypothalamic–pituitary–adrenal axis, erodes an individual's perceived control and self-efficacy, and leads to social isolation and dissolution of support systems through fear-avoidance behaviors [37, 38]. In geriatric nursing practice, this implies that a new or worsening complaint of “feeling fearful” should not be regarded merely as an ancillary anxiety symptom, but rather as a high-risk early warning signal of impending network-wide decompensation. Preventative nursing interventions targeting fear, such as supportive psychoeducation aimed at enhancing perceived control or problem-solving therapy, may be strategically deployed to buffer this node before it triggers cascading deterioration.

“Cannot continue” was simultaneously identified as a key target by both traditional centrality analysis and NIRA simulations, a cross-method convergence that strongly supports its priority for clinical attention. This item reflects a paralysis of volitional behavior initiation and serves as an objective behavioral marker of motivational deficit. For frontline nurses, when an older patient reports being unable to “get going,” this should be interpreted not as a passive descriptor of fatigue but as an active threat to network stability, warranting urgent intervention. Interventions targeting this node should move beyond simple encouragement toward structured behavioral activation—systematically scheduling and reinforcing meaningful daily activities in a graded, manageable manner to rekindle the patient's behavioral motivation[39, 40]. It must be emphasized that the intervention targets identified through NIRA simulations are generated from a cross-sectionally estimated network model, and the simulated perturbations rely on pre-specified parameters (e.g., threshold adjustment of two standard deviations). The actual effects of real-world clinical interventions may differ in magnitude and pattern. Therefore, these targets should be regarded as high-priority, testable hypotheses requiring validation through prospective cohort studies and pragmatic randomized controlled trials, rather than as immediate prescriptions for clinical practice[18] .

4.3. Implications

This study has several implications for geriatric nursing research and practice. First, by demonstrating the divergence between static centrality and dynamic regulatory influence, our findings caution against the sole reliance on traditional centrality indices for intervention target selection. Symptoms that are not the most central—such as “Mind distraction” in this network—may nonetheless hold the greatest potential for generating cascading therapeutic benefits when their underlying psychological processes are systematically addressed. This insight encourages a paradigm shift in nursing intervention design: from targeting the most prominent symptoms toward targeting the most responsive mechanisms.

Second, the identification of bridge symptoms linking depressive and cognitive domains—particularly “Calculation” and “Orientation”—underscores the necessity of integrated care models. When an older adult presents with cognitive complaints, nursing assessment should routinely include screening for depressive symptoms such as hopelessness and motivational deficits, as these may be functionally interconnected rather than truly independent. Conversely, older adults receiving care for depression should be monitored for subtle cognitive changes, as early detection of cognitive decline within this intertwined network may open a window for timely intervention.

Third, the computational simulation approach employed in this study offers a cost-efficient framework for generating precise, mechanism-informed hypotheses before committing resources to large-scale clinical trials. Future nursing research should build upon these findings by designing and testing multi-component intervention protocols that explicitly target the identified processes—such as attentional regulation training combined with behavioral activation—and examining whether the symptom network architecture undergoes structural reorganization in response to such tailored strategies.

4.4. Limitations

Several limitations of this study should be noted. First, the cross-sectional design precludes causal inference. Although NIRA simulations were applied to model cascading effects under specific assumptions, the estimated network captures contemporaneous associations at a single time point. Future research should employ longitudinal designs, such as cross-lagged panel network analysis, to examine the temporal directionality and stability of the identified symptom relationships and intervention targets .

Second, both the measurement tools and the data preprocessing steps may have influenced the estimated network. Depressive and cognitive symptoms were assessed through brief self-report and performance-based measures; although widely validated in large epidemiological surveys, these instruments may not capture the full clinical nuance of late-life depression or the fine-grained dimensions of cognitive impairment. Furthermore, the NIRA algorithm requires binary input, which necessitated dichotomizing continuous symptom scores. While cross-model validation with a Gaussian Graphical Model confirmed the robustness of the core network structure, this transformation may have reduced sensitivity to symptom severity. Future studies should incorporate clinician-administered diagnostic interviews and comprehensive neuropsychological batteries to enhance measurement precision.

Third, the sample was drawn from the 2020 CHARLS cohort. Although CHARLS is nationally representative, the findings may not generalize to older adults in other cultural contexts or time periods, particularly given the evolving post-pandemic mental health landscape. More importantly, the intervention targets identified through NIRA are computational hypotheses generated from cross-sectional data; their clinical utility requires validation through pragmatic randomized controlled trials before informing real-world nursing practice [41].

5. Conclusions

Using a large, nationally representative sample of community-dwelling older adults, this study employed simulation-based network analysis to identify dynamic intervention targets within the depressive-cognitive symptom network. Our findings reveal that alleviating “Mind distraction” produces the greatest reduction in overall symptom burden, while aggravating “Fear” and “Cannot continue” leads to the most pronounced network deterioration. Notably, these simulation-identified targets are not fully concordant with traditional centrality indices, underscoring the importance of distinguishing between static prominence and dynamic regulatory influence.

These results support a shift from disease-centered to symptom-focused, mechanism-driven care strategies in geriatric nursing. Specifically, attentional regulation, fear reduction, and behavioral activation should be prioritized as core therapeutic processes in integrated depression-cognition management for older adults. In the future, this simulation-based network approach can be extended to other vulnerable subgroups—such as older adults with multimorbidity, those living in long-term care facilities, or populations in different cultural and socioeconomic contexts—to generate precise, testable hypotheses for tailored nursing interventions.

Declarations

Ethics approval and consent to participate

This study was a secondary analysis of the China Health and Retirement Longitudinal Study (CHARLS), an open-access publicly available dataset. The original data collection was approved by the Biomedical Ethics Committee of Peking University (Approval No.: IRB00001052-11015). Informed consent was obtained from all participants in the original survey. As this study involved only the analysis of de-identified secondary data, no additional ethical approval was required.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no affiliations with or involvement in any organization or entity with any financial interest in the subject matter or materials discussed in this manuscript.

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Author Contribution

JX, ZZ, and YL contributed equally to this work and are the co-first authors. JX, ZZ, and YL were responsible for the study conception and design, data analysis, and drafting of the manuscript. HQ and YG contributed to the data curation, methodological support, and manuscript revision. QW and LZ served as co-corresponding authors, provided overall supervision, contributed to the study design and interpretation of findings, and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Not applicable.

Data Availability

The datasets generated and/or analysed during the current study are available in the China Health and Retirement Longitudinal Study (CHARLS) repository, <https://charls.charlsdata.com/index/zh-cn.html>

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Figures

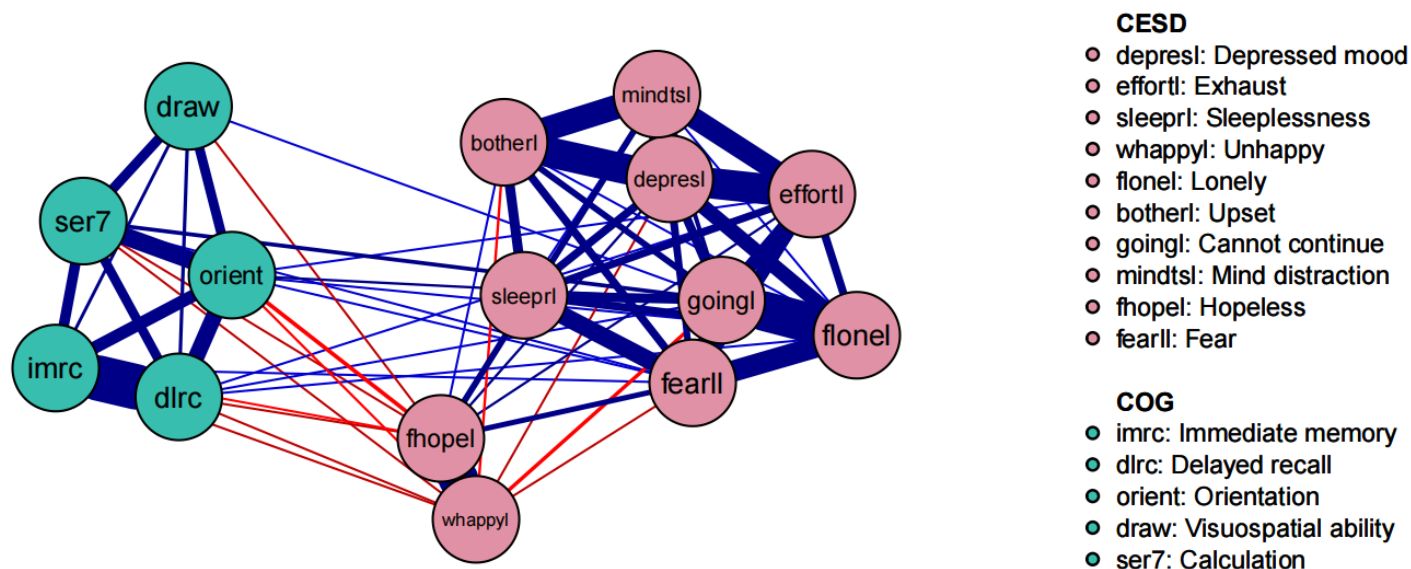
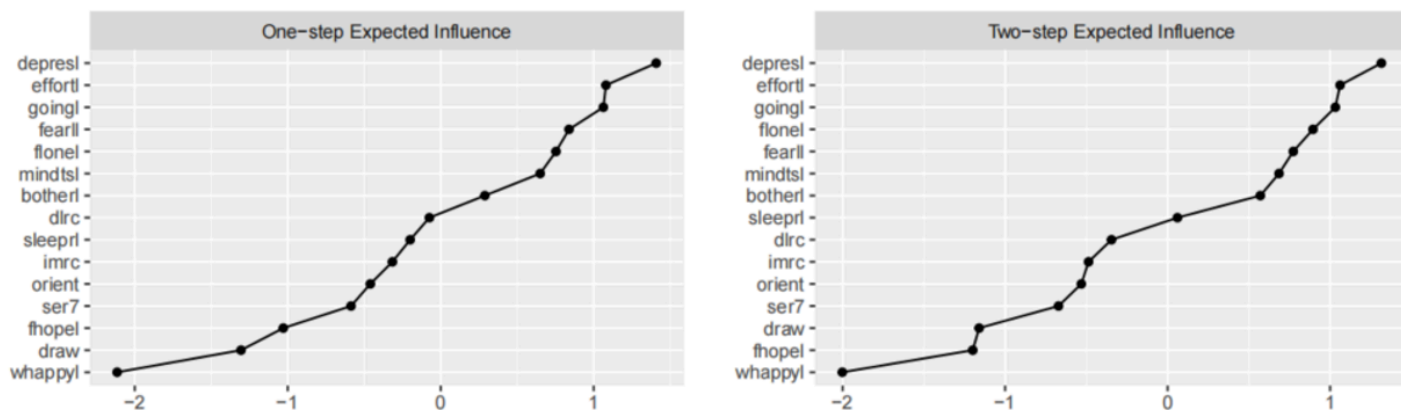


Figure 1

Ising Network of Depressive and Cognition Symptoms. *Note.* Edges with weights greater than 0.1 are displayed in the graph.

A



B

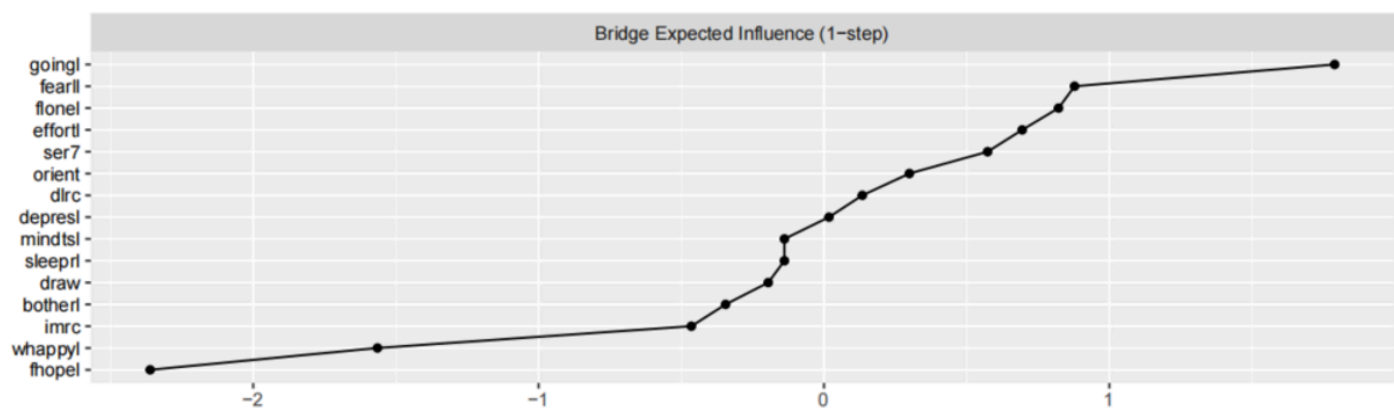


Figure 2

Node Expected Influence and Bridge Expected Influence of Depressive and Cognition Symptoms. Note. Fig. 2A displays the expected influence of each symptom and Fig. 2B displays the bridge expected influence of each symptom. EI and BEI indices are depicted using standardized z scores.

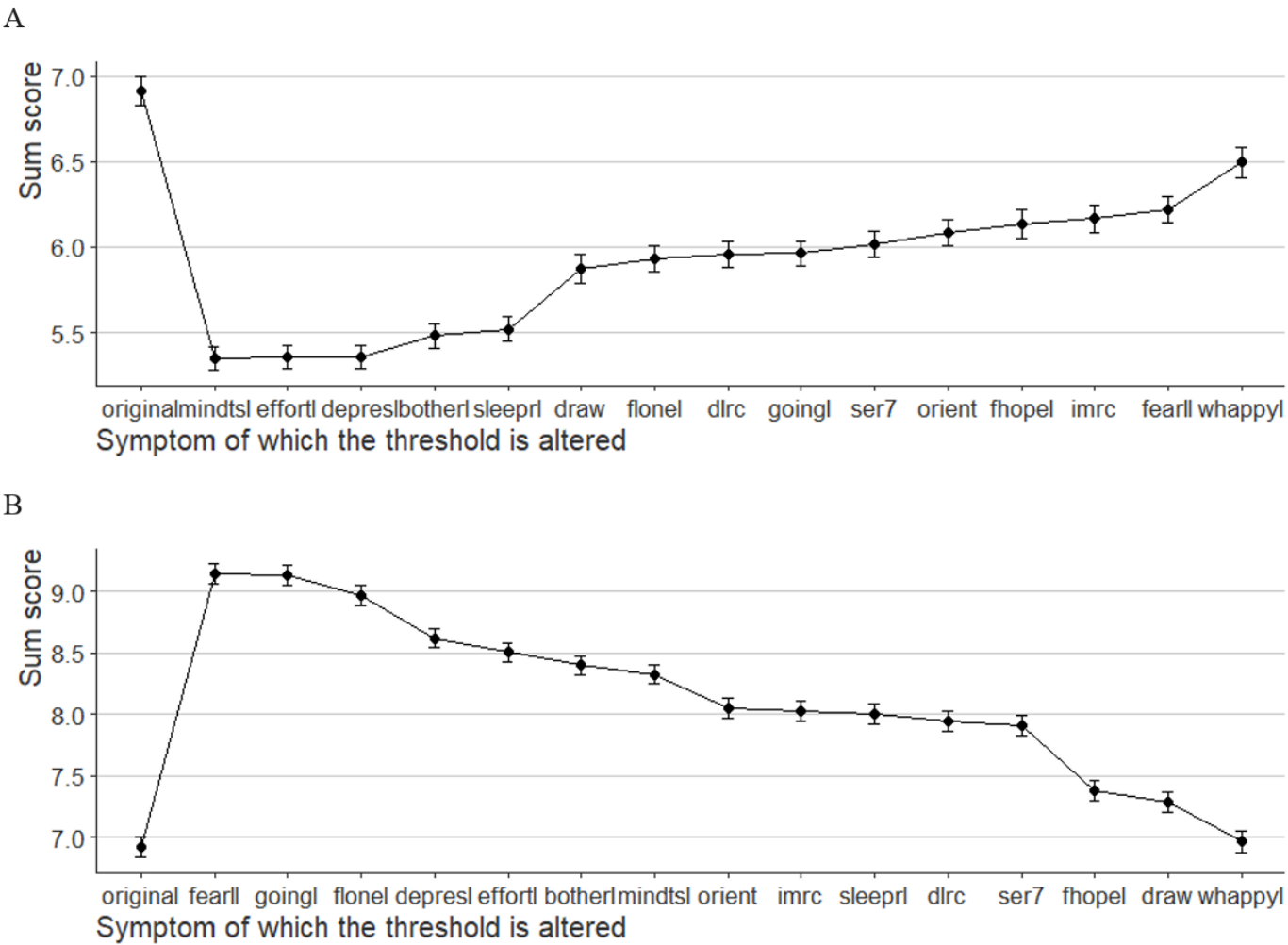


Figure 3

Results of Simulating Intervention on the Ising Model. Note. Fig. 3A displays the results of alleviating intervention and Fig. 3B displays the results of aggravating intervention. Black dots represent the network sum scores, while lines represent the 95 % confidence interval. The symptoms are listed based on the size of the intervention's effects, along with the original total score before the intervention.

Supplementary Files

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