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Centrality of symptoms in networks of depression and comorbid psychopathology among adults aged 50 years and older: A systematic review and examination of influencing factors

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Abstract

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Background Network analysis has increasingly been adopted over the past decade to study the structure of depressive symptoms in middle-aged to older populations, with ‘central’ symptoms often taken to suggest potential intervention targets. However, questions have been raised regarding the stability and clinical significance of centrality metrics. This systematic review aimed to examine the centrality of depressive symptoms among adults aged 50 years and older across network studies, with a focus on identifying its influencing factors.

Methods We conducted systematic searches via PubMed, PsycINFO, and Web of Science from database inception to February 15, 2025, with one additional study identified during peer review. The National Institutes of Health Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies was adapted to assess the quality of included studies. Symptoms were ranked according to their centrality. Factors associated with the presence of any DSM-based depressive symptom, non-DSM depressive symptom, and comorbid symptom/associated factor as among the top three central nodes, were examined using Firth penalised multivariable logistic regression.

Results Fifty-seven studies comprising 176 networks were reviewed, with 17 different depression scales adopted. Six of them were ‘strong’ in terms of their quality. ‘*Depressed mood*’ was the most central DSM-based depressive symptom, although its rate of being among the most central nodes dropped from 80.6% in depressive symptoms-only networks to 59% in comorbidity and extended networks. ‘*Lack of happiness*’ was among the most central non-DSM depressive symptoms. Meanwhile, comorbid symptoms and risk/protective factors were among the most central nodes in comorbidity and extended networks, with anxiety symptoms of ‘*uncontrollable worry*’, ‘*trouble relaxing*’, and ‘*nervousness*’ showing the highest node and bridge centrality. ‘*Tired in the morning*’, ‘*lack companionship*’, ‘*alone*’, and ‘*isolated*’ were other commonly identified bridge symptoms. Networks constructed during the COVID-19 pandemic was associated with a lower odds of DSM-based depressive symptoms (aOR=0.22, CI = 0.07–0.64) and a higher odds of comorbid symptoms/associated factors (aOR=3.41, CI = 1.31–9.45) being most central, while networks of clinical/subclinical samples had lower odds of comorbid symptoms/associated factors being most central (aOR=0.22, CI = 0.08–0.56). Those adopting DSM-based depression scales showed a trend of DSM-based depressive symptoms being most central across networks (aOR=3.10, CI = 0.97–11.20).

Conclusion Substantial heterogeneity was observed in the centrality of symptoms in networks of depressive symptoms, highlighting the influences of network types, environmental changes during time of data collection, and the choice of depression scales. Clinical implications and future directions in the context of these findings are further discussed.

Background

Depression is among the most prevalent mental health problems worldwide and has significant implications for functioning and quality of life [1]. Aside from its high rates of comorbidity with other mental health symptoms, depression also confers an elevated risk of morbidity and mortality, particularly in later life [2, 3]. Despite efforts to advance treatment efficacy, up to 50% of people with major depressive disorder (MDD) may experience at least one future recurrent depressive episode [4, 5]. Notably, while young people are often at heightened risk for the onset of MDD [6], middle-aged and older adults tend to experience a more recurring and chronic course of depression [7]. With global trends of rapidly ageing populations, identifying more robust clinical intervention targets to not only prevent the onset but also recurrence of depression is imperative.

The network theory of psychopathology has been advanced as an alternative to the traditional common cause disease model for conceptualising mental disorders [8]. Accordingly, mental disorders are conceived to emerge from the direct causal connections and interactions between individual components of complex systems (e.g., mental health symptoms), with their activation being triggered and maintained by a multitude of neurobiological, cognitive, psychological, socio-cultural, and external factors [8]. For instance, in the case of MDD, changes in the external environment (e.g., loss of a significant other) might precipitate the experience of ‘*depressed mood*’ and, in turn, ‘*sleep-onset difficulties*’, ‘*fatigue*’, and ‘*concentration problems*’. Importantly, these symptom-symptom relations are not bounded by conventional diagnostic systems, thereby offering a novel perspective to understanding the

emergence and maintenance of depression, as well as its comorbidity. A common example is that *depressed mood* (a cardinal symptom of MDD) could trigger *uncontrollable worry* and *restlessness* (cardinal symptoms of generalised anxiety disorder [GAD]) [9, 10]. In such comorbid networks, bridge symptoms are theorised to play a crucial role in connecting and maintaining different clusters of more symptoms [9].

Insofar as only mental health symptoms are included in a psychopathology network, non-symptom factors (e.g., genetics and neurobiological processes, stressors) are also assumed to play a role by constituting a symptom, the symptom-symptom connection, or by acting as influencing factors in the external field [8]. Building on this assumption, more recent studies have called for expanding networks of symptoms to incorporate environmental factors and risk/protective factors to elucidate the mechanistic pathways underlying mental disorders [11, 12]. A suite of open-access analytic tools is also available to test these patterns of associations and network structures, leading to its increasing adoption in recent decades [13, 14].

The perspective that individual symptoms are active agents within a complex network which cannot be interchanged is in line with observations that the aetiology and manifestation of depression are highly heterogeneous [15]. While collaborative clinical formulation has long been recognised as a core skill in the practice of psychology and psychiatry [16], the decision of whether a psychiatric disorder is present, particularly in research, has often been reduced to the number of presenting symptoms on the diagnostic criteria or ‘sum-scores’ on rating scales [17, 18]. Nevertheless, studies have shown that over 1,000 unique symptom profiles of depression can be identified using the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [19]. Aside from the nine symptoms of MDD listed on the DSM, the clinical relevance of non-DSM depressive symptoms in clinical populations (e.g., sympathetic arousal, mood reactivity, interpersonal sensitivity) has also been reported in previous work [20, 21]. Considering a wider range of symptoms beyond diagnostic systems may thus offer a more nuanced understanding of the phenomenological experiences of depression in the population.

Within network analysis, central symptoms have been postulated to play influential roles in sustaining symptom activation [20]. Node centrality, which reflects the degree to which a symptom is interconnected with other symptoms in a network, has thus been one of the most frequently reported metrics, with the overarching aim of informing early prevention and treatment targets [22, 23]. However, while symptoms with higher centrality have indeed been found to be associated with relapse outcomes [24], patterns of centrality rankings and their associated outcomes have not been consistent across studies [25, 26]. A simulation study has also suggested that the centrality of a symptom does not reflect its causal influences on other symptoms within a network [27]. The clinical significance of node centrality, particularly in cross-sectional and between-subject networks, thus remains largely debated in the literature [14, 28].

To establish the clinical relevance of centrality metrics, demonstrating their replicability across studies is arguably fundamental for findings to be generalised and applied to inform treatment design. To date, 10 systematic reviews of psychopathology network studies have been published and synthesised centrality findings [29–38] (see summary in Supplementary Table S1). Notably, two of these reviews innovatively explored study-level factors (e.g., sample size, sample type, study design [cross-sectional vs longitudinal]) as potential confounders of centrality rankings [31, 35]. One of them reviewed MDD symptom networks and identified ‘*fatigue*’ and ‘*depressed mood*’ as most central, with no study-level factors found to be associated with centrality rankings [31]. Meanwhile, in the review of depression-anxiety networks, ‘*depressed mood*’, ‘*uncontrollable worry*’, and ‘*excessive worry*’ were identified as most central, in which ‘*depressed mood*’ was found to be associated with a higher odds of being most central in clinical samples (vs community-dwelling samples) [35]. These findings offered preliminary evidence to suggest that several study-level factors, such as sample characteristics and the type of network analysed (i.e., depression-only vs depressive with comorbid symptoms), could play a role in the replicability of centrality findings across studies.

Several important gaps in the understanding of centrality findings, nevertheless, remain. First, as existing reviews of depression networks predominantly focused on DSM-based depressive symptoms, it is unclear how the inclusion of non-DSM symptoms in a network might reveal a different pattern of centrality rankings [20, 21]. Further, whether DSM-based depressive symptoms, non-DSM depressive symptoms, or comorbid symptoms and risk/protective factors are most central across networks, and how their centrality rankings might differ by network types, study-level factors, and the choice of depression measure, remains to be further explored. As the phenomenological experiences of depression and its associated factors are noted to differ between young and older adults (e.g., more agitation,

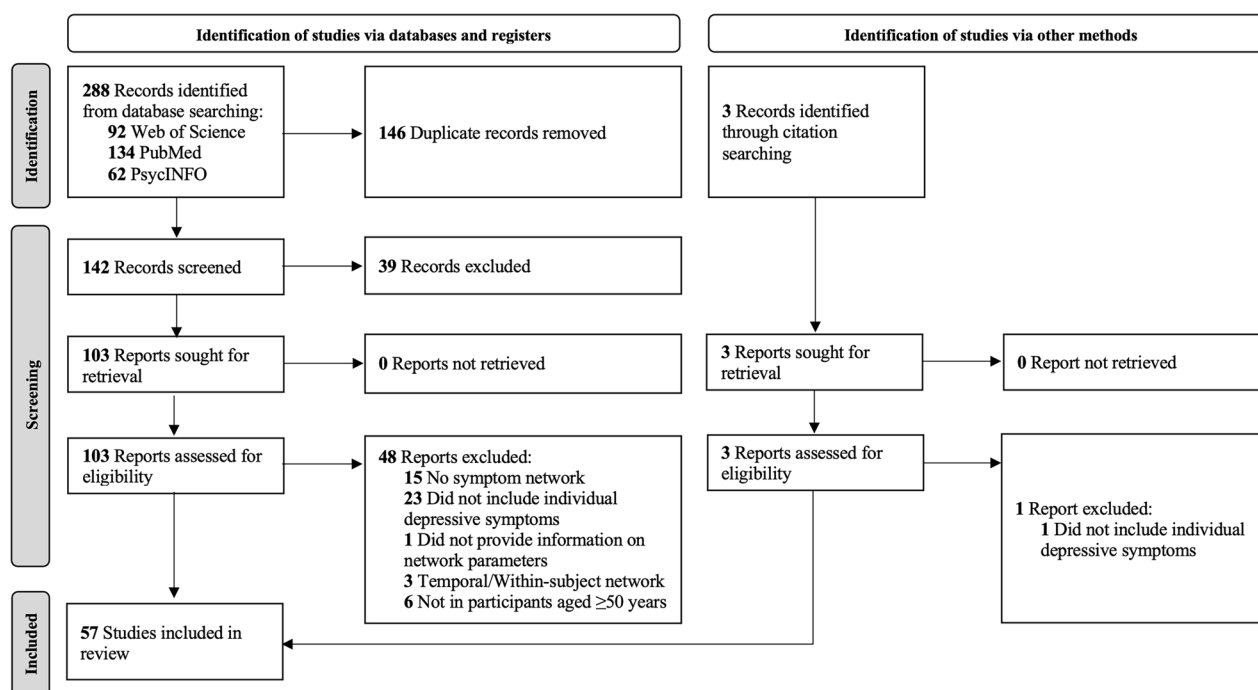


Fig. 1 PRISMA flow diagram of included studies.

general and gastrointestinal somatic symptoms, and less guilt in the latter) [39], a more focused review of depression networks among those aged 50 years and older appears crucial to inform more age-appropriate theoretical developments and clinical work.

Building on prior work, the aims of the present review are four-fold: (1) to investigate the study characteristics of network studies of a broad range of DSM-based and non-DSM depressive symptoms, as well as those incorporating comorbid symptoms and associated factors, in adults aged 50 years and older; (2) to examine the consistency of symptom centrality rankings across network types; (3) to identify bridge symptoms in comorbid and extended networks; and (4) to examine the influences of a wide range of study-level factors on centrality rankings. Given the variability in the assessment of depressive symptoms, we also conducted a systematic mapping of depressive symptoms across scales prior to the data synthesis.

Methods

Search strategy

We conducted the first round of systematic search of relevant studies from database inception to 11 April 2024 to identify empirical studies via PubMed, PsycINFO, and Web of Science, focusing on depressive symptoms among adults aged 50 years and older. A further round of search was conducted to identify more updated studies published up to 15 February 2025. One additional study published within this period was included during peer review. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline was followed in conducting this systematic review, with the study pre-registered in PROSPERO [40]. Details of the full search terms used for each database are in Supplementary Table S2.

We did not exclude non-English studies, did not limit the search to specific clinical populations, and hand-searched reference lists of included studies. After removing duplicates, the titles and abstracts of the retrieved articles were first screened by the first author (SMYW). All articles were read in full by SWMY and reviewed independently by two

researchers (ZLYN and YLW). Discrepancies in study selection were resolved through further discussions among SMYW, ZLYN, and YLW. All extracted data were further reviewed by SWMY to ensure consistency and accuracy. Authors were contacted if their full-text articles could not be located.

Selection criteria

Retrieved articles were considered eligible upon meeting the following inclusion criteria: (i) presented original empirical data; (ii) assessed and incorporated individual depressive symptoms in networks; (iii) conducted on participants aged 50 years and older; and (iv) published in a peer-reviewed journal. Studies that did not present empirical data (e.g., review articles, editorials), applied other network analytic approaches (e.g., brain or neural network, social network, semantic network), adopted a single-subject temporal network design (e.g., case studies), and included a summed score for depressive symptoms were excluded. Figure 1 shows the PRISMA flow diagram.

Study quality assessment

While risk of bias assessment is a core component of systematic reviews, there is no standard procedure for evaluating the quality of network analysis studies to date. We adapted the National Institutes of Health (NIH) Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies [41], a tool recommended for evaluating the quality of both cross-sectional and longitudinal observational studies [42]. The quality of included studies was independently reviewed by YLW and ZLYN, with differences resolved through discussions with SMYW.

The NIH Quality Assessment Tool consists of 14 criteria covering aspects including research questions, study population, recruitment and eligibility criteria, sample size justification, assessment of exposure prior to outcome, sufficiency of timeframe to observe associations between exposure and outcome, different levels of exposure assessed, validity and reliability of exposure and outcome measures, existence of repeated measures, follow-up rate, and adjustment of confounding variables. A rating of ‘yes’, ‘no’, ‘cannot determine’, ‘not reported’, or ‘not applicable’ can be given to each criterion. The final rating was determined by dividing the number of ‘yes’ responses by the number of applicable questions according to the following: poor (<60%), fair (60–69%), good (70–79%), and strong (≥80%) [43]. Details of the quality assessment procedure are provided in Supplementary Material.

Choice of depression and comorbid scales

For each depressive symptom scale, we specified the number of symptoms assessed and whether they were based on the DSM. A mapping procedure was applied to identify similar constructs across scales. Details of the scales used to assess comorbid symptoms and associated factors were also extracted.

Study characteristics

Studies were organised according to one of the following network types: (i) depressive symptom-only; (ii) comorbidity network (depressive symptoms with other mental health-related symptoms, e.g., anxiety symptoms); (iii) extended network (depressive symptoms with other risk/protective factors or outcomes); and (iv) depressive symptom-only and comorbid/extended networks separately reported. Networks that included non-DSM depressive symptoms, as assessed using a non-depression scale (e.g., loneliness), were categorised as comorbidity networks. For each included study, we extracted information on the following domains.

Study and sample characteristics

Study characteristics included study design (cross-sectional, longitudinal, or multi-wave), duration of follow-up (for longitudinal and multi-wave studies), and country/region. Sample characteristics included study population, sample size, participant age, and gender or sex ratio.

External influences

Given the influence of environmental triggers on symptoms and the global implications of the COVID-19 pandemic from 2020 to 2023 [8, 44, 45], we extracted information on whether the study was conducted during the pandemic and whether its effects had been directly tested in the network.

Theory testing

Information was extracted regarding whether the study aimed to test an a priori theory. Simply stating the network theory of psychopathology [8, 9] was considered insufficient unless it directly informed study hypotheses (e.g., higher connectivity of symptoms leads to poorer outcomes).

Reproducibility

Reproducibility was indicated by whether the data were open-source, whether analytic codes were openly available, and whether the article was open-access.

Network characteristics

Network models

In network studies, a symptom or variable is referred to as a ‘node’, while the association between two nodes is referred to as an ‘edge’. For each study, we identified the number of network models constructed, the depression scale used, any additional constructs examined, and the number of nodes included.

Centrality metrics

Two most commonly reported centrality metrics in network studies are *strength* (reflects how often a node is directly connected to other nodes, determined by the sum of edge weights connected to one node from all other nodes) and *expected influence* (EI). Whereas strength centrality only accounts for absolute edge weights, EI additionally retains the positive or negative value of an edge, such that a negatively-valenced edge represents reduced EI [46]. Two other centrality metrics are *betweenness* (reflects how often a node lies on the average paths between two other nodes) and *closeness* (reflects how often a node is indirectly connected to other nodes). Another more recently developed centrality metric is *predictability*. While not interchangeable with EI, *predictability* has also been increasingly adopted in recent studies, which reflects the proportion of variance in a node that can be explained by all other connecting nodes (i.e., how well it can be predicted by other nodes) [47]. For comorbid and extended networks, we also extracted information on *bridge centrality* (i.e., the most central symptoms that connect one symptom dimension to another) [9, 48], if examined.

Network accuracy and stability

We also determined whether the information concerning network accuracy and stability was reported. Edge-weight accuracy is typically determined through a data re-sampling procedure using non-parametric bootstrapping with 95% CIs, wherein a smaller difference between the bootstrapped and sample mean confers greater accuracy [49]. Centrality stability is determined using the correlation stability coefficient (CS-C) through a case-drop procedure, which represents the proportion of samples that can be dropped without substantially affecting the centrality estimates whilst maintaining a correlation of 0.7 or higher between the original and subset centrality indices with 95% probability [49]. A CS-C of ≥ 0.5 is recommended and should not fall below 0.25 [49]. Differences between edge-weights and the centrality of each node are typically evaluated using the bootstrapped difference tests to determine their accuracy and stability.

Group differences

For studies that constructed more than one network, we determined whether differences between networks were statistically examined using the Network Comparison Test (NCT), which applies permutation-based testing on the resampling of the dataset [50]. If so, information on differences in overall network connectivity (global strength), edge-weights, and node centrality was extracted.

Symptom centrality ranking

The relative centrality of symptoms across the network types was determined by identifying the top three most central nodes in the networks. EI was taken as the primary metric to determine centrality rankings, as it is one of the most reported centrality metrics and considers the direction of associations between nodes [46]. It has also been shown to predict the correlation of changes among nodes over time [25]. *Strength* or *predictability* was used as alternative if EI was not reported. Similar to a previous systematic review of depression networks [31], *betweenness* and *closeness* were not used to determine symptom centrality rankings as their suitability for application to psychological networks has been questioned [49, 51].

Three Firth penalised multivariable logistic regression models [52, 53] were then conducted to examine factors associated with the presence of any DSM-based depressive symptom, non-DSM depressive symptom, and comorbid mental health or associated factor, respectively, as among the top three most central nodes. Sample size, study design (cross-sectional vs longitudinal/multi-wave), nature of depression scale (non-DSM vs DSM-based), study population (community vs clinical [including depressive or non-depressive conditions]), and whether the study was conducted during the COVID-19 pandemic (no/yes) were included as plausible associated factors. Sample size was first log-transformed in these models to account for its non-normal distribution. Adjusted odds ratios (aORs) with their 95% CIs were reported to reflect effect sizes. As preliminary exploration, a separate regression model was applied to examine whether these factors, as well as study quality, would be associated with the stability of node centrality metrics (CS-C). All analyses were conducted using *R*.

Results

Study selection

The initial database search yielded 288 articles. After removing duplicates, 142 articles were screened for titles and abstracts, with 39 records identified as ineligible. Full-text review was conducted for the remaining 103 articles. Of them, 48 studies were excluded for the following reasons: use of sum-scores as nodes for depressive symptom ($n = 23$); symptom network not reported ($n = 15$); not conducted on people aged ≥ 50 years ($n = 6$); temporal/within-

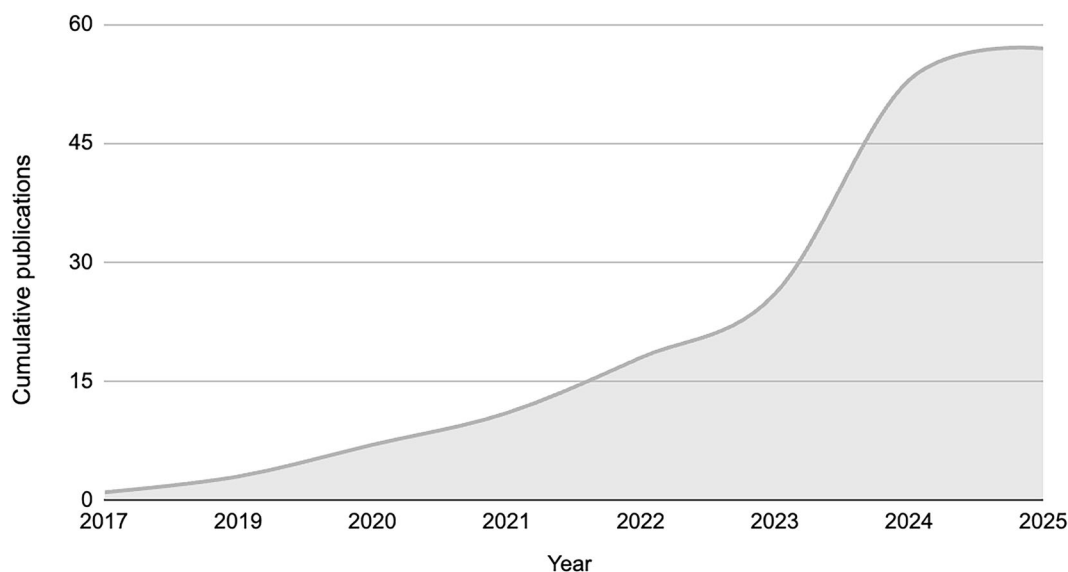


Fig. 2 Overview of network studies published on depressive symptoms among adults aged 50 years and older. *Note.* Articles retrieved from database inception to February 2025. Search terms used to identify network studies are detailed in the Supplementary Material.

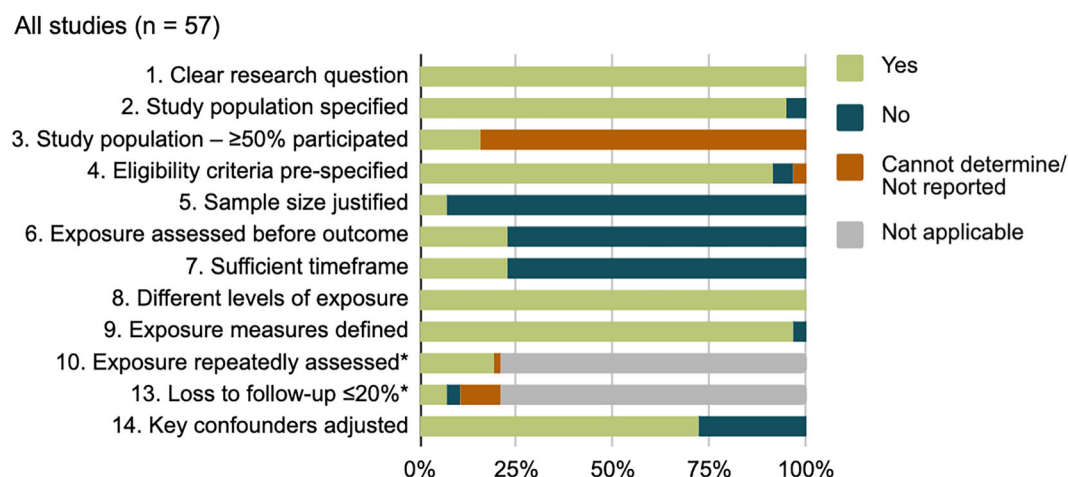


Fig. 3 Quality assessment of included studies. *A rating of ‘not applicable’ was given to criteria 10 and 13 for cross-sectional studies. *Note.* The NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies was used.

subject network ($n = 3$); and network parameters not reported ($n = 1$). Three additional studies were identified as potentially relevant through hand-searching reference lists. One of these did not include individual depressive symptoms and was excluded. A total of 57 studies were retained for synthesis. The first network study conducted in this age group was published in 2017, with 96.4% of studies published from 2020 onwards ($n = 54$) (Fig. 2).

Quality of included studies

Figure 3 summarises the quality of the included studies. A detailed breakdown is provided in Supplementary Table S3 and Figure S1. Six studies were rated as ‘strong’ in terms of their quality [54–59], all of which were longitudinal studies. Most studies were rated as ‘fair’ ($n = 24$); 11 were ‘good’ and 16 were ‘poor’. Among longitudinal studies ($n = 11$), four had <20% loss to follow-up after baseline [54–56, 59] and two had ≥20% loss to

Table 1 Overview of depression scales adopted in the included studies and the number of DSM and non-DSM symptoms assessed.

Scale	# studies adopting the scale	DSM-5 MDD symptom						# DSM symptoms assessed	# non-DSM symptoms assessed
		Depressed mood (subjective or observed)	Worthlessness or guilt	Anhedonia	Concentration problems or indecisiveness	Recurrent suicidal thoughts, ideation, or attempt	Significant appetite or weight change	Insomnia or hypersomnia	Psychomotor agitation or retardation
Fatigue									
BDI	1	✓ 1	✓ 2	✓ 1	✓ 1	✓ 1	✓ 1	✓ 2	✓ 1
CES-D-7^a	2	✓ 1	–	–	–	✓ 1	–	–	–
CES-D-8	4	✓ 2	–	–	–	✓ 1	–	–	–
CES-D-9^b	1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	–	–	–
CES-D-10	15	✓ 1	–	–	–	✓ 1	–	✓ 1	–
CIDI65+ depression module (9 core symptoms)	1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1
CIDI65+ depression module (33 individual symptoms)^c	1	✓ 2	✓ 2	✓ 2	✓ 2	✓ 4	✓ 2	✓ 2	✓ 4
CPRS subset^d	1	✓ 1	–	✓ 1	✓ 1	✓ 1	✓ 2	–	✓ 1
DASS-D	2	✓ 1	✓ 1	–	–	–	–	✓ 1	–
EURO-D	1	✓ 1	✓ 2	✓ 1	✓ 1	✓ 1	–	✓ 1	✓ 1
GDS-15	6	–	✓ 2	–	–	–	–	✓ 1	✓ 1
HAMD^e	1	✓ 1	–	✓ 2	✓ 2	✓ 3	✓ 2	✓ 1	✓ 1
KGDS	1	–	✓ 3	✓ 1	–	–	–	✓ 1	✓ 1
MADRS	1	✓ 2	–	✓ 1	✓ 1	✓ 1	–	✓ 1	✓ 1
PHQ-2	3	✓ 1	✓ 1	–	–	–	–	–	–
PHQ-9	15	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1
WHO-CIDI depression module^f	1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 1	✓ 2	✓ 1	✓ 1

^a Version used in Ramos-Vera et al. (2023).

^b Version used in Cai & Wu (2024).

^c Version used in Belvederi Murri et al. (2022). Note that all items assessed in the study were treated as individual notes and were not compounded.

^d Version used in Triolo et al. (2021).

^e 'Insight' was assessed but removed from the estimated network in Park (2021), as the author noted that the symptom was not connected with any of the other nodes.

^f Version used in de la Torre-Luque et al. (2020).

Note. For DSM-based symptoms, the value in parentheses represents the number of additional items used to assess compound symptoms listed in the DSM (e.g., reduced and increased appetite assessed separately). *BDI* Beck's depression inventory, *CES-D-7* center of epidemiologic studies depression scale, 7-item version, *CES-D-8* center of epidemiologic studies depression scale, 8-item version, *CES-D-10* center of

epidemiologic studies depression scale, 10-item version, *CIDI* composite international diagnostic instrument, *CPRS* comprehensive psychopathological rating scale, *DASS-D* depression subscale of the depression, anxiety and stress scale—21 items, *GDS-15* geriatric depression scale 15-item, *HAMD* Hamilton depression rating scale, *KGDS* Korean form of geriatric depression scale, *MADRS* Montgomery–Åsberg depression rating scale, *MDD* major depressive disorder, *PHQ-2* patient health questionnaire 2-item, *PHQ-9* patient health questionnaire 9-item.

Table 2 Study and sample characteristics of included studies.

Authors (year)	Study characteristics		Sample characteristics		External factors			Theory testing? _a																																																																																																																																																																																																																																																																																																																																																																																																																																																																				
	Conducted during COVID-19?	Reproducibility	Open source data?	Study design	Fol- low- up period Open access?	Country/ Region	Population	Sample size	Age (years), mean (SD)	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	No	N.S.	Yes	Upon request	

Table 2 continued

Authors (year)	Study characteristics		Sample characteristics		External factors			Theory testing? ^a	Reproducibility				
	Study design	Follow-up period	Country/Region	Population	Sample size	Age (years), mean (SD)	Female %		Conducted during COVID-19?	Effect of COVID-19 mentioned?	Open source data?	Code available?	Open access?
Pyo et al. (2024)	C	–	South Korea	Clinical (AD/MCI)	5353	AD: 72.8 (8.5); MCI: 69.7 (8.1)	AD: 68.7%; MCI: 68%	No	–	No	No	Yes	
Tao et al. (2023)	C	–	Harbin, China	Community	353	≥60	–	Yes	No	No	Upon request	N.S.	Yes
R. Wang et al. (2024)	C	–	China	Community	13993 (5794) ^c	Urban: 62.6 (9.6); Rural: 62.5 (9.3) ^c	Urban: 52.9%; Rural: 49.8% ^c	No	–	No	Yes	N.S.	Yes
X. Wang et al. (2024)	C	–	China	Community	4021	81.5 (10.0)	56.1%	No	–	No	Yes	N.S.	No
Comorbidity network (with other mental health symptoms) (<i>n</i> = 23)													
Caycho-Rodríguez et al. (2024)	C	–	Peru	Community	328	68.4 (7.6)	49.4%	No	–	No	Upon request	N.S.	No
Chan et al. (2024)	C	–	Hong Kong SAR	Community	341	70.9 (9.0)	58.6%	Yes	No	No	Upon request	N.S.	No
Chang et al. (2025)	C	–	China	Community (living alone)	2028	84.3 (9.5)	61.8%	No	–	No	Yes	Yes	Yes
M. Y. Chen et al. (2025)	C	–	China	Community	1365	TCP: 80 (10.5); Non-TCP: 79.6 (10.5)	TCP: 48.8%; Non-TCP: 48.3%	No	–	No	Yes	N.S.	No
P. Chen et al. (2024)	C	–	United States	Community	4100	74.8 (10.4)	56.9%	Yes	No	No	Yes	N.S.	No
Ferreira & França (2023)	C	–	Brazil	Community	384	66.8 (5.6)	66.9%	Yes	No	No	Upon request	N.S.	No
Lam et al. (2024)	C	–	China	Community	3378	HD: 83 (10.7); No HD: 82.7 (10.9)	HD: 57.7%; No HD: 57.7%	No	–	No	Yes	N.S.	No
Liu et al. (2024)	MW	W1 [Pre-COV]: 2016–19 ; W2 [Peri-COV]: 2020–23	Hong Kong SAR	Subclinical (mild depressive symptoms)	W1: 2549; W2: 3506	W1: 73.5 (9.0); W2: 73.7 (8.0)	W1: 78%; W2: 80%	Yes	Yes	No	Yes	N.S.	Yes
Ma et al. (2024)	C	–	China	Clinical (HT)	4993	81.9 (10.2)	56%	No	–	No	Yes	N.S.	Yes
Ramos-Vera et al. (2023)	L	T1 [COV W1]: Jun–Jul 2020; T2 [COV W2]: Nov–Dec 2020	England	Community	T1: 5797; T2: 6512	≥50	T1: 54%; T2: 56%	Yes	Yes	No	Yes	N.S.	Yes
Ramos-Vera et al. (2024)	L	T1 [COV W1]: Jun–Jul 2020; T2 [COV W2]: Nov–Dec 2020	England	Community	T1: 5797; T2: 6512	≥50	T1: 54%; T2: 56%	Yes	Yes	No	Yes	N.S.	Yes
Suari et al. (2020)	L	386.7 days (SD = 48.1)	Washington, United States	Clinical (AD)	3198	77.5 (6.7)	52.8%	No	–	No	Yes	Yes	No
Shang et al. (2023)	C	–	China	Clinical (≥2 chronic diseases)	662	70.4 (6.9)	40.3%	Yes	No	No	Upon request	N.S.	Yes
Sun, Zhang et al. (2024)	L	T1 [Baseline]: 2016–17; T2 [Pre-COV]: 2018–19; T3 [Peri-COV]: Nov–Dec 2020	England	Community	4293	68.0 (7.4)	44.5%	Yes	Yes	No	Yes	N.S.	No
S. S. Wang et al. (2024)	L	6-month follow-up during 2020–2021	China	Community	548	85.2 (8.8)	53.5%	Yes	No	Yes	No	N.S.	Yes

Table 2 continued

Authors (year)	Study characteristics		Sample characteristics		External factors			Theory testing? ^a		Reproducibility			
	Study design	Follow-up period	Country/Region	Population	Sample size	Age (years), mean (SD)	Female %	Conducted during COVID-19?	Effect of COVID-19 mentioned?	Open source data?	Code available?	Open access?	
Xue et al. (2024)	C	–	China	Community	10239	87.6 (9.9)	68.1%	No	–	No	Yes	N.S.	No
Yang et al. (2022)	C	–	Xi'an, China	Subclinical (BI < 100 and elderly care for ≥6 months)	325	75.0 (9.7)	44.6%	Yes	No	No	Upon request	N.S.	Yes
Yao et al. (2024)	C	–	China	Community	469	65–69 yrs	68.7%	Yes	No	No	Yes	N.S.	Yes
C. Zhang et al. (2024)	MW	W1 [Pre-COV]: Sep 2019–Jan 2020; W2 [COV-Strict]: Sep 2020–Jan 2021; W3 [COV-Normal]: Sep 2021	China	Community	W1: 256; W2: 469; W3: 405	W1: 61.7 (5.8); W2: 65.5 (4.6); W3: 65.1 (4.6)	W1: 72.3%; W2: 68.2%; W3: 73.1%	Yes	Yes	No	Upon request	N.S.	Yes
L. Zhang et al. (2022)	C	–	Harbin, China	Community	1302	≥60	68.6%	Yes	No	No	Upon request	N.S.	Yes
P. Zhang et al. (2023)	C	–	China	Subclinical (B-ADL ≥ 7 for disability)	2131	92.9 (9.2)	63.2%	No	–	No	Yes	N.S.	No
W. X. Zhang et al. (2024)	C	–	China	Community	8884	83.2 (11.3)	54.0%	No	–	No	Yes	N.S.	No
Y. J. Zhang et al. (2024)	C	–	China	Clinical (diabetes mellitus)	1685	80.5 (10.0)	55.5%	No	–	No	Yes	N.S.	No
Extended network (with other risk/protective factors or intervention) (n = 14)													
Bai et al. (2023)	C	–	China	Community	9023	80.9 (10.1)	50.7%	No	–	No	Yes	N.S.	No
Bian et al. (2024)	C	–	China	Community	501	71.8 (7.1)	49.9%	No	–	No	Upon request	N.S.	Yes
de la Torre-luque et al. (2023)	L	Biennial, 6 waves (2002–12)	England	Community	3349	58.4 (5.2)	55.2%	No	–	No	Yes	N.S.	No
Jiang et al. (2025)	L	T1: Feb 2023 (lockdown); T2: lockdown lift; T3: 6 months after	China	Community	1230	64.5 (3.8)	46.1%	Yes	No	No	No	N.S.	No
Lee et al. (2023)	C	–	United States	Community	1698	72.9 (n/a)	48%	No	–	No	Yes	N.S.	No
Schönenberg et al. (2024)	C	–	Germany	Clinical (common neurological disorder, excl. dementia)	731	70.2 (8.6)	44.6%	No	–	No	Yes	N.S.	Yes
Sun, Chen et al. (2024)	L	T1: 2016–2017; T2: 2018–2019	England	Community	6433	68.5 (9.0)	56.5%	No	–	No	Yes	N.S.	Yes
van Wanrooij et al. (2019)	L	6.7 years	The Netherlands	Community	3229	74.5 (2.5)	53.9%	No	–	No	N.S.	N.S.	Yes
X. R. Wang (2025)	C	–	China	Community	4253	68.9 (6.3)	51.2%	Yes	No	No	N.S.	N.S.	No
Y. Wang et al. (2024)	C	–	China	Community	3454	82.6 (10.5)	52.3%	No	–	No	Yes	N.S.	No
Wu & Cai (2024)	C	–	China	Community	6061	89.0 (10.0)	68.7%	No	–	No	Yes	N.S.	No
J. H. Yu & Mahendran (2021)	L	Pre-COV: Feb 2018–Jan 2020; Peri-COV: May–Jun 2020	Singapore	Community	419	69.0 (5.5)	65.6%	Yes	Yes	No	Upon request	Yes	Yes
Y. Yu et al. (2024)	C	–	China	Community	12043	43.9% 65–80 yrs	53.1%	No	–	No	Yes	N.S.	Yes

Table 2 continued

Authors (year)	Study characteristics		Sample characteristics			External factors		Theory testing? ^a		Reproducibility			
	Study design	Follow-up period	Country/Region	Population	Sample size	Age (years), mean (SD)	Female %	Conducted during COVID-19?	Effect of COVID-19 mentioned?	Open source data?	Code available?	Open access?	
W. Zhang et al. (2024)	C	–	Hong Kong SAR	Subclinical (PHQ-9 ≥ 5)	2792	77.3 (8.5)	78.1%	No	–	No	Upon request	N.S.	Yes
Depressive symptoms only + separate comorbidity/extended networks (<i>n</i> = 4)													
An et al. (2019)	C	–	Seoul City, South Korea	Subclinical (MADRS ≥ 10)	776	73.9 (8.1)	72.4%	No	–	No	Upon request	N.S.	Yes
Cai & Wu (2024)	C	–	China	Community	1717	66.9 (5.2)	48.5%	No	–	No	Yes	N.S.	No
Semino et al. (2017)	C	–	Austria	Clinical (depressive symptoms and/or ICD-10 cognitive impairment)	264	73.0 (8.2)	70.1%	No	–	No	N.S.	N.S.	No
Triolo et al. (2021)	C	–	Central Stockholm, Sweden	Community	2860	73 (10.4)	63%	No	–	No	Upon request	Upon request	Yes

^a Indicates whether theories informed the study, developed formal theories based on findings, or discussed findings with relevance to prior theories (aside from the network theory).

^b Lee & Hu (2022): sample characteristics after PSM.

^c R. Wang et al. (2024): sample characteristics after PSM.

Note. AD Alzheimer's disease, B-ADL Bayer activities of daily living scale, BI Barthel index, C cross-sectional study, COV coronavirus disease 2019, HD heart disease, HT hypertension, ICD-10 international statistical classification of diseases and related health problems 10th revision, L longitudinal study, MADRS Montgomery-Åsberg depression rating scale, MCI mild cognitive impairment, MDD major depressive disorder, MW multi-wave study, N.S. not specified, PHQ-9 patient health questionnaire 9-item, PSM propensity-score matching, TCP Tai Chi practitioners.

follow-up [60, 61]; the rest did not report this information [57, 58, 62–64]. Only nine studies had a participation rate of $\geq 50\%$ [58, 65–72] and four justified their sample size [56, 57, 73, 74].

Choice of depression scales

Seventeen different depression scales were used, with the number of depressive symptoms assessed ranging from 2 to 33 (2–24 for DSM-based symptoms, 0–21 for non-DSM symptoms; see Supplementary Table S4). Table 1 provides an overview of the depressive symptoms assessed via each scale. A detailed mapping of each item to DSM and non-DSM-based symptoms is given in Supplementary Tables S5–6.

The Patient Health Questionnaire–9-items (PHQ-9) [75] and the Center of Epidemiologic Studies Depression Scale, 10-item version (CES-D-10) [76] were most adopted ($n = 15$ each), both of which were self-administered. Whereas the PHQ-9 was designed to assess DSM-based depressive symptoms, only four items of the CES-D-10 were based on the DSM criteria, with six additional items assessing non-DSM symptoms (e.g., feeling bothered/irritated, fearful, lonely) [76]. Two interviewer-rated measures that assessed DSM-based depressive symptoms were the WHO–CIDI depression module [77] and the age-adapted CIDI65+ depression module [78]. Of the two studies that adopted the CIDI65+, one incorporated nine nodes representing the presence or absence of the DSM-based MDD symptoms [79], while the other incorporated all 33 items that included non-DSM depressive symptoms [80]. Nine scales assessed specific symptoms that are grouped as compound symptoms in the DSM (e.g., worthlessness and guilt assessed separately on the Beck’s Depression Inventory [BDI] [81]). Notably, the BDI was the only scale that assessed all nine DSM depressive symptoms along with eight non-DSM depressive symptoms.

Study characteristics

Table 2 summarises the study and sample characteristics. Most constructed comorbidity networks ($n = 23$), followed by depressive symptoms-only ($n = 16$) and extended networks ($n = 14$). Four studies presented separate networks of depressive symptoms with extended ($n = 3$) or comorbid ($n = 1$) networks. Further details of the studies that applied propensity score matching ($n = 3$) and examined network structures in youth samples ($n = 3$) are given in Supplementary Table S7–S8.

Study and sample characteristics

Most studies were cross-sectional ($n = 44$), with 11 longitudinal and two multi-wave studies. Of the longitudinal and multi-wave studies, the follow-up duration ranged from eight weeks [62] to 10 years [64]. Most were conducted in Asia ($n = 38$), followed by Europe ($n = 11$), North America ($n = 3$), and South America ($n = 2$). One study was a cross-cultural comparison of network structures in people with MDD in Spain and Nigeria [82], one was conducted in community samples across five western European countries and Israel [80], and one was conducted among people with subclinical depressive symptoms across 19 European countries [83].

Study samples were mainly recruited in the community ($n = 37$ studies). Eight studies were on people with clinical or subclinical levels of depression, while 12 were on people with specific medical or neurological conditions (e.g., hypertension, mild cognitive impairment, Alzheimer’s disease) (Table 2). Sample sizes ranged from 96 (behavioural activation [BA] intervention study [62]) to 13,993 (national survey of older adults [71]), with the average being 3,287. Of those that reported participant age, the cumulative mean sample age (excluding youth participants) was 73.8 years. The proportion of females ranged from 40.3% [74] to 80% [69]. In studies that examined network structures of young people, their cumulative mean age was 19.3 years [56, 66, 84].

Table 3 Network characteristics of included studies.

Authors (year) (model N)	Dep. scale (node N)	Other constructs (node N)	Centrality metrics	Robustness tests		Centrality stability (CS-C) ^a		Network structure difference (global strength)
				Edge	Centrality	Node centrality	Bridge centrality	
Depressive symptoms only (<i>n</i> = 16)								
Belvederi Murri et al. (2020) (5)	EURO-D (12)	–	S.; Bet.; Clo.	95% CIs; Diff. test	Case-drop; Diff. test	0.85 (all; others N.R.)	–	Ø Male–Female; Ø Younger–Older
Belvederi Murri et al. (2022) (7) ^b	CIDI65+ depression module (all symptoms) (33)	–	–	–	–	–	–	–
Castellanos et al. (2020) (4)	CIDI65+ depression module (core symptoms) (9)	–	S.; Bet.; Clo.	–	Case-drop	0.52 (all; others N.R.)	–	No NCT
de la Torre-luque et al. (2020) (2)	WHO–CIDI Depression module (12)	–	S.; Bet.; Clo.	95% CIs	Case-drop	–	–	Ø Spanish–Nigerian
Eli et al. (2022) (1)	CES-D-10 (10)	–	–	95% CIs; Diff. test	Case-drop; Diff. test	0.75	–	–
Hong et al. (2024) (1)	KGDS (30)	–	S.; Bet.; Clo.	95% CIs	Case-drop	0.52	–	–
Janssen et al. (2024) (2)	PHQ-9 (9)	–	–	95% CIs; Diff. test	Case-drop; Diff. test	0.37, 0.52	–	Post > Pre-BA
Jin et al. (2022) (3)	PHQ-9 (9)	–	S.; Bet.; Clo.	95% CIs; Diff. test	Case-drop; Diff. test	0.75 (all; others N.R.)	–	Female > Male
K. M. Kim et al. (2021) (1)	GDS-15 (15)	–	S.; Bet.; Clo.	Diff. test	Diff. test	–	–	–
S. Kim & Lee (2022) (1)	PHQ-9 (9)	–	–	–	–	–	–	–
Lee & Hu (2022) (2)	PHQ-9 (9)	–	S.; Bet.; Clo.	95% CIs; Diff. test	Case-drop; Diff. test	0.75, 0.75	–	Female > Male
Park (2021) (1)	HAMD (17)	–	S.; Bet.; Clo.	–	Case-drop	0.25	–	–
Pyo et al. (2024) (2)	GDS-15 (15)	–	S.; Clo.; Bet.	95% CIs; Diff. test	Case-drop; Diff. test	0.70, 0.70	–	Ø MCL–AD
Tao et al. (2023) (4)	PHQ-9 (9)	–	S.; Predict.	95% CIs; Diff. test	Case-drop; Diff. test	0.28 ^a	–	Older adults > Junior, College;
R. Wang et al. (2024) (4)	CES-D-10 (10)	–	–	95% CIs	Case-drop	0.75, 0.75 [no/with PSM]	–	Senior > Junior, College
X. Wang et al. (2024) (2)	CES-D-10 (10)	–	S.; Clo.; Bet.	95% CIs; Diff. test	Case-drop; Diff. test	0.75, 0.75	–	Ø Urban–Rural [no/with PSM]
Comorbidity network (with other mental health symptoms) (<i>n</i> = 23)								
	PHQ-2 (2)	Loneliness (3)	S.	95% CIs	Case-drop	–	–	Ø Men–Women

Table 3 continued

Authors (year) (model N)	Dep. scale (node N)	Other constructs (node N)	Centrality metrics	Robustness tests		Centrality stability (CS-C) ^a		Network structure difference (global strength)
				Edge	Centrality	Node centrality	Bridge centrality	
Caycho-Rodriguez et al. (2024) (3) (all, men/women)						0.28 (all; others N.R.)		
Chan et al. (2024) (4) (college students/older adults) [dep. only/with hope included]	PHQ-9 (9)	Anxiety (7); Hope (12)	BEI	95% CIs	Case-drop	–	0.36 [dep. only]; 0.67 [with hope] ^a	–
Chang et al. (2025) (3) (all, male/female)	CES-D-10 (10)	Anxiety (7)	S.; Bet.; Clo.; EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.75 (all; others N.R.)	–	Ø Male–Female
M. Y. Chen et al. (2025) (22) (TCP/non-TCP) [male/female; smokers/non-smoker; drinker/ non-drinker; </≥75 yrs; urban/ rural]	CES-D-10 (10)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.67, 0.75 (others N.R.)	0.28, 0.67 (others N.R.)	Ø TCP–Non-TCP
P. Chen et al. (2024) (2) (stroke/no stroke)	CES-D-8 (8)	Insomnia (4)	EI; BEI	95% CIs	Diff. test	0.53, 0.68	–	Ø Stroke–No stroke
Ferreira & França (2023) (1)	PHQ-2 (2)	Loneliness (6)	S.; Bet.; Clo.; Predict.; B. S.	–	Case-drop	0.45	0.45	–
Lam et al. (2024) (4) (HD/no HD) [male/female]	CES-D-10 (10)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.75, 0.67 (subgroup N.R.)	0.75, 0.75 (subgroup N.R.)	Within no HD: Male ≠ Female ^c ; Ø HD–No HD
Liu et al. (2024) (2) (pre-COV/peri-COV)	PHQ-9 (9)	Anxiety (7); Loneliness (3)	EI; S.; Bet. ; Clo.	95% CIs; Diff. test	Case-drop; Diff. test	0.75, 0.75	–	Peri > Pre-COV
Ma et al. (2024) (1)	CES-D-10 (10)	Anxiety (7)	EI; Predict. ; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.75	0.75	–
Ramos-Vera et al. (2023) (3) (COV W1/W2) [contemp., temporal]	CES-D-7 (7)	Anxiety (7); Loneliness (3)	EI; Predict. ; BEI	–	Case-drop	0.75, 0.75	–	Ø W1–W2
Ramos-Vera et al. (2024) (3) (COV W1/W2) [contemp., temporal]	CES-D-7 (7)	Anxiety (7)	EI; BEI	–	Case-drop	0.75, 0.75	–	Ø W1–W2
Saari et al. (2020) (2) (baseline/follow-up)	GDS-15 (15)	Informant-rated affective symptoms (3)	S.	95% CIs; Diff. test	Case-drop; Diff. test	0.67, 0.75	–	Ø Baseline–Follow-up
Shang et al. (2023) (4) (all, non-alexithymia/suspected/ alexithymia)	DASS-D (7)	Anxiety (7); Stress (7); Alexithymia (3)	S.; Bet. ; B. S.	95% CIs; Diff. test	Case-drop; Diff. test	0.75 (all; others N.R.)	–	AT > Non-AT ; Ø Suspected–AT, non-AT
Sun, Zhang, et al. (2024) (3) (baseline/pre-COV/peri-COV)	CES-D-8 (8)	Loneliness (3)	EI	95% CIs	Case-drop	0.75, 0.67, 0.75	–	Ø Baseline–Pre-COV; Ø Pre–Peri-COV
	DASS-D (7)	Anxiety (7); Stress (7)	Out EI ; In EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.44 ^a	0.44 ^a	<i>No NCT</i>

Table 3 continued

Authors (year) (model N)	Dep. scale (node N)	Other constructs (node N)	Centrality metrics	Robustness tests		Centrality stability (CS-C) ^a		Network structure difference (global strength)
				Edge	Centrality	Node centrality	Bridge centrality	
S. S. Wang et al. (2024) (3) (adolescents/college students/ older adults)								
Xue et al. (2024) (4) (non-widowed/widowed, widowed [low/high comor.])	CES-D-10 (10)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.75, 0.75, 0.67, 0.75	0.75, 0.67, 0.36, 0.36	Ø Non-Widowed
Yang et al. (2022) (1)	PHQ-9 (9)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.60	0.52	–
Yao et al. (2024) (7) (all, male/female, pri./sec. or above edu., with/no spouse)	PHQ-9 (9)	Anxiety (7)	EI; S.; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.60 (all; others N.R.)	0.52 (all; others N.R.)	Ø Male-Female; Ø Pri.-Sec. or above; Ø Spouse-No spouse
C. Zhang et al. (2024) (3) (pre-COV/COV-Strict/COV- Normal)	PHQ-9 (9)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.44, 0.67, 0.52	0.29, 0.52, 0.44	<i>No NCT</i>
L. Zhang et al. (2022) (3) (all, male/female)	PHQ-2 (2)	Anxiety (2); Insomnia (3)	S.; B. S.	95% CIs; Diff. test	Case-drop; Diff. test	–	–	Ø Male-Female
P. Zhang et al. (2023) (3) (all, male/female)	CES-D-10 (10)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.75 (all; others N.R.)	0.36 (all; others N.R.)	Ø Male-Female
W. X. Zhang et al. (2024) (2) (exercise/non-exercise)	CES-D-10 (10)	Anxiety (7)	EI; S.; Bet.; Clo.; BEI	95% CIs	Case-drop	–	–	Ø Non-exercise-Exercise
Y. J. Zhang et al. (2024) (1)	CES-D-10 (10)	Anxiety (7)	EI; BEI	95% CIs; Diff. test	Case-drop; Diff. test	0.75	0.75	–
Extended network (with other risk/protective factors or outcome) (n = 14)								
Bai et al. (2023) (1)	CES-D-10 (10)	Cognition (6)	S.; B. S.	95% CIs; Diff. test	Case-drop; Diff. test	0.75	0.67	–
Bian et al. (2024) (7) (all, male/female, urban/rural, with/no som. chronic comor.)	PHQ-9 (9)	Anxiety (7); Personal mastery (7)	S.; Predict.; B. S.	95% CIs; Diff. test	Case-drop; Diff. test	0.67 (all; others N.R.)	–	Ø Male-Female; Ø Urban-Rural; Ø Chronic somatic comorbidity-No comorbidity Low > Mod., High; Mod.-High
de la Torre-luque et al. (2023) (3) (low/mod./high symptom trajectory)	CES-D-8 (8)	Inflammatory (3); Metabolic (6); Covariates (4)	EI; S.	95% CIs	Case-drop	0.67, 0.59, 0.51	–	–
Jiang et al. (2025) (1)	PHQ-9 (9)	Social connections (6)	EI; S.; Bet.; Clo.,	95% CIs; Diff. test	Case-drop	–	–	–
Lee et al. (2023) (2) (dep. + CRP, dep. + CRP + cov.)	PHQ-9 (9)	CRP (1); Covariates (11)	Predict.	95% CIs	–	–	–	<i>No NCT</i>
Schönenberg et al. (2024) (4) (dep. + medication adherence)	BDI (21)	Med. non-adherence (1)	–	–	Case-drop	0.60, 0.60, 0.60, 0.52	–	–

Table 3 continued

Authors (year) (model N)	Dep. scale (node N)	Other constructs (node N)	Centrality metrics	Robustness tests		Centrality stability (CS-C) ^a		Network structure difference (global strength)
				Edge	Centrality	Node centrality	Bridge centrality	
[SAMS total], dep. + SAMS subscales [31]								
Sun, Chen et al. (2024) (2) (contemp., temporal)	CES-D-8 (8)	Cognition (3)	Out EI; In EI; BEI; Predict.	95% CIs; Diff. test	Case-drop; Diff. test	>0.50 (temporal)	>0.75 (contemp.)	<i>No NCT</i>
van Wanrooij et al. (2019) (1)	GDS-15 (15)	Functioning (1); Dementia at follow-up (1)	S.; Bet.; Clo.	95% CIs; Diff. test	Case-drop; Diff. test	0.75	–	–
X. R. Wang (2025) (3) (all, non-frail/pre-frail)	PHQ-9 (9)	Frailty (5); Anxiety (7); Loneliness (3); Covariates (6)	EI; BEI	95% CIs	–	–	–	<i>No NCT</i>
Y. Wang, et al. (2024) (4) (CP low comor./CV low comor./CV high comor./resp. low comor.)	CES-D-10 (10)	Anxiety (7); BADL (6); IADL (8)	EI; BEI	–	Case-drop	0.75, 0.75, 0.60, 0.75	–	CP low comor. > Resp. low comor.; Ø Other comparisons
Wu & Cai (2024) (3) (all, male/female)	CES-D-10 (10)	Anxiety (7); Quality of life (1)	S.; B. S.	95% CIs	Case-drop	0.75	0.75	Ø Male-Female
J. H. Yu & Mahendran (2021) (2) (pre-COV/peri-COV)	GDS-15 (15)	Anxiety (20); Social isolation (6)	S.; B. S.	–	Case-drop	0.44	–	Ø Pre-Peri-COV
Y. Yu et al. (2024) (9) (all, male/female, 65–80/81–105 yrs, yes/not living alone, urban/rural)	CES-D-10 (10)	Social participation (10)	S.; B. S.	95% CIs; Diff. test	Case-drop; Diff. test	0.75 (all; others N.R.)	0.75 (all; others N.R.)	Female > Male; Older > Younger; Yes > Not living alone; Ø Urban–Rural
W. Zhang et al. (2024) (1) Depressive symptoms only + separate comorbidity/extended networks (n = 4)	PHQ-9 (9)	Cognition (4)	BEI	–	Case-drop	–	0.28	–
An et al. (2019) (6) (dep., dep. + anxiety, dep. [low/high anx.]) [no/with PSM]	MADRS (10)	Anxiety (1)	S.; Bet.; Clo.	95% CIs	Case-drop	–	–	Low ≠ High anxiety [no PSM] ^c ; Ø Low ≠ High anxiety [with PSM]
Cai & Wu (2024) (4) (dep., dep. + social support) [internet use/non-use]	CES-D-9 (9)	Family support (3); Peer support (3); Community support (2)	EI; S.	95% CIs	Case-drop	0.75, 0.75 [internet use], 0.67, 0.75 [non-use]	–	<i>No NCT</i>
Semino et al. (2017) (3) (dep., dep. + cog. [low/high cog. impair.])	GDS-15 (15)	Cognition (10)	S.; Bet.; Clo.	95% CIs	Case-drop	–	–	Ø Low–High cognitive impairment
Triolo et al. (2021) (2) (dep., dep. + som. disease)	CPRS subset (16)	Som. diseases (8)	EI; BEI	–	Case-drop	>0.90, >0.90	–	–

^a CS-C presented only for the older adult network.

^b Belvederi Murri et al. (2022) conducted 1 network model using the Gaussian graphical model (GGM) and 6 latent network models (LNMs). Specifically, 3 LNMs were conducted based on the GGM network model using different algorithms to identify symptom complexes (Walktrap, Spinglass, Multilevel), while 3 other LNMs were conducted according to different approaches to defining the symptom loadings (results of exploratory factor analysis; content of items; random assignment into 7 complexes).

^c Direction for differences in global strength not specified in Lam et al. (2024) and An et al. (2019).

Note. *BA* Behavioural activation, *BADL* basic activities of daily living, *BEI* bridge expected influence, *Bet.* betweenness, *B. S.* bridge strength, *Clo.* closeness, *Contemp.* contemporaneous, *CS-C* centrality stability coefficient, *DBP* diastolic blood pressure, *EI* expected influence, *GGM* Gaussian graphical model, *HD* heart disease, *IADL* instrumental activities of daily living, *N. R.* not reported, *PSM* propensity score matching, *SBP* systolic blood pressure, *WBC* white blood cell, *S.* strength.

External influences

Except for one study that did not specify the time of data collection [85], 20 studies were conducted during the COVID-19 pandemic. Eight of them adopted a longitudinal or multi-wave design: three were conducted across two phases of the pandemic [54–56]; four captured periods before and during the COVID-19 pandemic [56, 61, 69, 86]; and one captured periods during and after pandemic lockdowns [57]. Two of the longitudinal studies were from the same dataset (ELSA Covid-19 Substudy) [54, 55]. Six of them explicitly explored the influences of the pandemic on symptom networks [54, 55, 61, 63, 69, 86], although none of them directly included pandemic-related stress or the number of pandemic-related stressful events as a node within depression networks.

Theory testing

Only one of included studies was explicitly informed by prior theoretical models [56]. Specifically, the study tested assumptions of the tripartite model, which postulates the existence of both shared and distinct symptoms of depression and anxiety (e.g., sadness and worry as shared symptoms, physiological hyperarousal as specific to anxiety, and anhedonia as specific to depression) [87]. The study found that physiological symptoms (mouth dryness and breathing difficulty) had the highest inward-EI in a depression-anxiety network (i.e., sum of values of incoming edges), thereby challenging the specificity hypothesis in the tripartite model.

Six studies discussed their findings with reference to prior theories: the cognitive avoidance theory of worry to discuss uncontrollable worry as the most central symptom [70]; the escape theory of suicide to discuss suicidal ideation as the second most central symptom among senior high school students [84]; theories of BA and the ‘positive spiral’ to discuss higher symptom connectivity post-BA vs pre-BA [62]; the diathesis-stress model, which was not supported by the finding of a lack of structural difference between urban and rural samples [88]; the Hierarchical Taxonomy of Psychopathology [80]; and theories linking depression with inflammation to discuss the association between C-reactive protein and appetite changes [89].

Reproducibility

Twenty-eight studies had their dataset made publicly available or utilised open-source data, while 18 indicated that their dataset could be made available upon request. Only five studies had their analytic codes made openly available. Thirty-one studies were open access.

Network characteristics and structures

Network models

Table 3 summarises network characteristics. Overall, 176 networks were constructed (50 depressive symptoms-only networks, 77 comorbidity networks, 49 extended networks); seven networks were conducted among youths.

Of the comorbid/extended networks in the adult population, the vast majority included anxiety symptoms (85 of 126 networks; 2–20 nodes). The DSM-based Generalized Anxiety Disorder Scale–7-items (GAD-7) was the most adopted scale for assessing anxiety symptoms (Supplementary Table S9). Symptoms of loneliness (14 networks; 3–6 nodes) and cognition (7 networks; 3–10 nodes) were also commonly assessed. Other constructs examined included other comorbid mental health symptoms (stress, insomnia, alexithymia, informant-rated affective symptoms), psychological factors (hope, personal mastery), social factors (social connections, social isolation, social participation, community/family/peer support), physical health conditions (dementia, somatic diseases, frailty), inflammatory and metabolic markers, well-being (functioning, instrumental and basic activities of everyday living, quality of life), and sociodemographic covariates.

Fig. 4 Top three central nodes ranked according to network type. *Note.* Proportions are computed after excluding networks that did not incorporate a specific symptom.

Centrality metrics were reported in most studies (96.5%), except for two [80, 90]. One of them explicitly stated that centrality findings could not address their main research question (i.e., associations between individual depressive symptoms and medication adherence) [90]; the other aimed at identifying depressive symptom complexes and examining their model fit [80].

Of those that reported centrality metrics, EI and strength were most frequently reported (30 and 34 studies, respectively); 10 studies reported both metrics [57, 64, 69, 88, 91–96]. Bridge EI was the most reported metric for bridge centrality ($n = 21$), followed by bridge strength ($n = 8$).

Network accuracy and stability

Regarding edge accuracy, bootstrapped 95% confidence intervals for edge-weights were estimated in 44 studies. Twelve studies did not report information on edge accuracy [54, 55, 61, 68, 72, 79, 80, 90, 92, 97–99]. For the accuracy of centrality metrics, the bootstrapped case-dropping procedure was applied in 51 studies. Four studies did not report this information [80, 89, 92, 100]. Bootstrapped different tests for both edges and centrality metrics were applied in 30 studies. Of the 44 studies that assessed node centrality stability, all networks had a CS-C value above the minimum threshold of 0.25, with an average of 0.67 (range=0.25–0.90). Among comorbid/extended networks, the stability of bridge centrality was reported in only 17 networks, with the average being 0.57 (range=0.28–0.75).

Group differences

Forty-four studies estimated more than one network (median=3; range=1–22). Thirty-nine studies had networks conducted among subgroups, of them 32 applied NCT to compare network structures (Table 3). Details of edge and node centrality differences across networks are in Supplementary Table S10.

Regarding gender differences in network connectivity, three studies found higher global strength among females compared to males [101–103], while nine studies did not observe significant gender differences [71, 73, 83, 93, 94, 104–107]. Higher network global strength was reported among older samples in two studies (old-olds [81–105 years] vs young-olds [65–80 years] [103] and older adults vs junior and college students [84]), although another study did not find differences between those aged 65–73 and ≥ 74 years [83]. Of the four studies that compared networks in samples living in urban vs rural areas, only one observed higher global strength among urban-living samples [108]. One study found higher global strength in those living alone [83].

Considering the influences of symptom severity and health conditions, higher global strength was reported in those with lower depressive symptom trajectory (vs moderate and high trajectory; [64]), those with alexithymia (vs non-alexithymia; [74]), and those with cardiopulmonary diseases and low comorbidity (vs respiratory diseases and low comorbidity; [98]). Marital status, education level, ethnicity, cognitive status, other health conditions (heart disease, stroke), and physical activity did not appear to influence network structures.

Among longitudinal and multi-wave studies, participants showed higher connectivity following an 8-week BA intervention [62]. Five studies examined differences in network structure across the various phases of the COVID-19 pandemic. Only one of these that adopted a multi-wave design with different participants found significantly greater network connectivity during the pandemic (vs pre-COVID; [69]); the other four that adopted a longitudinal design following up on the same sample did not observe significant network structure differences [54, 55, 61, 63].

Ranking of symptom centrality

Node centrality

Node centrality metrics were reported for 109 of 176 networks. Figure 4 visualises the rate of a node being identified as the top three most central across the three network types. A detailed visualisation of their rankings is provided in Supplementary Figure S2 for older adults and Supplementary Figure S3 for youth samples.

Among networks with centrality metrics reported, the proportion of each depressive symptom identified as the top three most central nodes ranged from 0% to 66.7% (DSM-based) and 0% to 27.6% (non-DSM-based) (Fig. 4). For DSM-based depressive symptoms, the proportion ranged from 0% ('*appetite changes*') to 80.6% ('*depressed mood*') in depressive symptoms-only networks, 0% ('*appetite changes*', '*psychomotor changes*') to 59.6% ('*depressed*

Table 4 Logistic regression models showing factors associated with the top three central nodes.

	Top three central nodes					
	DSM-based depressive symptoms		Non-DSM-based depressive symptoms		Comorbid symptoms/Risk/Protective factors	
	aOR (95% CIs)	<i>p</i>	aOR (95% CIs)	<i>p</i>	aOR (95% CIs)	<i>p</i>
Sample size	1.02 (0.39–2.66)	0.96	0.58 (0.21–1.49)	0.26	0.72 (0.30–1.66)	0.44
Study design						
Cross-sectional	1 (Ref)		1 (Ref)		1 (Ref)	
Longitudinal/Multi-wave	0.68 (0.23–2.18)	0.50	3.11 (0.87–12.21)	0.081	0.76 (0.25–2.23)	0.63
Study population						
Community	1 (Ref)		1 (Ref)		1 (Ref)	
Clinical/Subclinical	1.24 (0.43–3.88)	0.70	1.30 (0.47–3.67)	0.61	0.22 (0.08–0.56)	0.001
DSM-based depression scale						
No	1 (Ref)		1 (Ref)		1 (Ref)	
Yes	3.10 (0.97–11.20)	0.056	0.01 (0.0001–0.0912)	<0.001	0.51 (0.18–1.41)	0.20
External influences						
Not during COVID-19	1 (Ref)		1 (Ref)		1 (Ref)	
During COVID-19	0.22 (0.07–0.64)	0.005	2.14 (0.62–7.80)	0.23	3.41 (1.31–9.45)	0.012

Note. Only networks of adults aged 50 years and older with node centrality reported are included ($k = 109$).

mood”) in comorbidity networks, and 0% (‘appetite changes’, ‘psychomotor changes’, ‘sleep problems’, ‘concentration problems’) to 59.3% (‘depressed mood’) in extended networks. For non-DSM-based depressive symptoms, ‘lack of happiness’ was the most frequently identified central node, which presented as the top three central nodes in 17.2% of comorbidity networks, 28.6% of depressive symptoms-only networks, and 46.7% of extended networks.

Of the comorbidity/extended networks with centrality reported, the proportions of comorbid symptoms and risk/protective factors identified as the top three central nodes were 66.2% and 47.4%, respectively. Generalised anxiety symptoms were most frequently identified as the top three central nodes, specifically uncontrollable worry (22 networks), trouble relaxing (18 networks), and nervousness (17 networks) (Supplementary Table S11).

Bridge centrality

Bridge centrality was reported in 47 networks (Supplementary Figure S4). Comorbid symptoms and risk/protective factors were most frequently identified as the top three central bridge symptoms (75% and 50%, respectively) (Supplementary Table S12). Specifically, anxiety symptoms of ‘nervousness’ (19 networks), ‘uncontrollable worry’ (5 networks), ‘excessive worry’ (5 networks), and ‘trouble relaxing’ (5 networks) were the most commonly identified bridge symptoms (Supplementary Table S13). Other associated factors that have been identified in two or more networks as the top three most central bridge symptoms included insomnia (‘tired in the morning’), loneliness (‘lack companionship’), social isolation (‘alone’, ‘isolated’), and independent living abilities (‘bathing’, ‘dressing’, ‘transfers’, ‘toilet use’),

Among depressive symptoms, ‘irritability’ (non-DSM-based depressive symptom and a GAD symptom), alongside ‘depressed mood’ and ‘fatigue’ (DSM-based), were identified as the top three central bridge symptoms in 45.5%, 24.4%, and 20% of the networks, respectively.

Factors influencing node centrality ranking and stability indices

Multivariable penalised logistic regression models showed that networks estimated in studies during the COVID-19 pandemic had a higher odds of comorbid symptoms or associated factors (aOR=3.41, CI = 1.31–9.45, $p = 0.012$)

and a lower odds of DSM-based depressive symptoms ($aOR=0.22$, $CI=0.07-0.64$, $p=0.005$) being the most central nodes across the networks (Table 4). Meanwhile, networks conducted in clinical/subclinical samples were associated with a lower odds of comorbid symptoms or associated factors being the most central nodes ($aOR=0.22$, $CI=0.08-0.56$, $p=0.001$). Those that utilised DSM-based measures (PHQ-9, WHO-CIDI depression module, and CIDI65+ depression module [9 core items]) showed a trend of higher odds of DSM-based depressive symptoms being the most central nodes ($aOR=3.10$, $CI=0.97-11.20$, $p=0.056$).

Regarding the stability of node centrality indices (CS-C), preliminary findings suggest that networks examined with larger sample sizes were associated with higher stability ($\beta=0.18$, $SE=0.02$, $p<0.001$), while those examined during the COVID-19 pandemic showed a trend of lower stability ($\beta=-0.058$, $SE=0.03$, $p=0.040$) (Supplementary Table S14).

Discussion

In the present review, we aimed to systematically examine the centrality rankings of symptoms in depressive symptom networks, as well as their influencing factors, in adults aged 50 years and older. This was among the first to have comprehensively synthesised findings of network studies of depressive symptoms beyond those under the DSM framework. This was also the first attempt to identify symptoms with the highest node and bridge centrality across depressive symptom network types. The finding that the centrality of symptoms differed depending on network composition, study population, choice of depression scale, and the occurrence of major external stressors at time of data collection highlights the need to account for each of these factors when interpreting centrality findings.

A major research aim of network studies has been to inform clinical practice by identifying which symptoms may serve as targets for intervention. One of our most prominent observations, nevertheless, was the relative instability of centrality statistics. Indeed, we found ‘*depressed mood*’ to be among the most central symptoms, which is in line with previous systematic reviews of depression networks [31, 35] and its conceptualisation as a cardinal symptom of MDD [109]. However, we also found that its centrality substantially dropped once comorbid symptoms and associated factors were included in the network. Whereas a previous systematic review of MDD networks in clinical populations identified ‘*fatigue*’ as another highly central symptom of depression [31], we found support for this only in networks restricted to depressive symptoms (i.e., top three central symptoms in 50% depressive symptoms-only networks and 2.7% in comorbid networks).

By systematically mapping symptoms assessed across 17 depression scales, we found two non-DSM symptoms, namely ‘*lack of happiness*’ and ‘*a sense of emptiness*’, to be generally more central across network types and populations. Notably, their proportions of being the top three central nodes were higher than several DSM-based MDD symptoms. While ‘*depressed mood*’ and ‘*lack of happiness*’ are conceptually closely related, previous work has shown partial differences in the neurobiological mechanisms underlying the dimensions of negative and positive affect [110, 111]. Whether two strongly associated but partially distinct symptoms should be aggregated or modelled as separate nodes remains debated in current network studies [112]. These findings, nevertheless, highlight the relevance of additionally accounting for symptoms not listed in the DSM for a more elaborate understanding of the lived experience of depression across individuals.

Through a series of logistic regression models, we identified two key factors that affected centrality rankings. First, we found comorbid symptoms or risk/protective factors to be more central in non-clinical than in clinical samples. In line with the network theory of psychopathology [8], a possible explanation is that the feedback relations among depressive symptoms are sufficiently strong in clinical samples, such that their activation and maintenance are no longer contingent on other factors. However, as networks constructed on non-clinical samples tended to include more nodes with comorbid symptoms or associated risk/protective factors, it is also possible that this finding could have been an artefact of varied network composition. Second, we found that networks constructed amid the COVID-19 pandemic were associated with a higher odds of comorbid symptoms or associated factors—and not DSM-based depressive symptoms—being most central. It is possible that an elevated sense of uncertainty (e.g., about health and the situation), loneliness (e.g., due to lockdowns), and heightened reactivity to environmental stress during such

turbulent times could have played more prominent roles in reinforcing the connectivity of symptom networks during such times [113]. Nevertheless, only slightly over half of the studies conducted during the pandemic directly tested its effects on network structures using a longitudinal or multi-wave design, with none having incorporated individual experiences of COVID-19 stressors into their networks. The use of a multilayer network approach may help clarify the interactions between depressive symptoms and non-symptom factors, such as pandemic-related stressors and other cognitive processes, while accommodating both the topological and mechanistic explanations of psychiatric disorders [11, 12].

Notably, several bridge symptoms appear more consistently observed across depressive symptom networks, specifically ‘*uncontrollable worry*’, ‘*excessive worry*’, ‘*nervousness*’, and ‘*trouble relaxing*’, as well as ‘*insomnia*’, ‘*social isolation*’, and ‘*loneliness*’, all of which have been identified as core transdiagnostic processes that underlie depressive symptoms [114, 115]. For instance, *worry* has been closely linked to *rumination*, both of which are known as *repetitive negative thinking* characterised by heightened self-referential processing of information and impaired attentional control [114, 116, 117]. In line with the ruminative response styles theory, a recent study of older adults has found rumination to be a key bridge symptom that links stressful life events with depressive and anxiety symptoms [118]. Further, social isolation and loneliness have been associated with poorer cognitive control, heightened sensitivity, and bias to social threats [119, 120], as well as depressive and anxiety symptoms [121]; both of which have also been increasingly recognised as a public health threat to older adults [122, 123]. The present findings suggest that these phenomena might perpetuate depressive symptomatology and may warrant greater clinical attention in future work. How they might interact with one another and be causally related over time, nevertheless, remain areas for future investigation.

Aside from the heterogeneity in centrality rankings observed in the present work, some researchers have, in fact, argued that key assumptions of centrality measures might not be met in psychopathological networks [12, 124], which calls for a revisit of the interpretation of network findings. Despite the appeal of *directed acyclic graphs* to inform causal relations among variables, their replicability and validity have also been questioned [12, 125]. *Latent network modelling* has been proposed as a possible alternative for identifying more reliable nodes derived from multiple measures of a symptom to reduce measurement errors and may be considered [51, 80, 125, 126]. Further, as most included studies relied on between-subjects analyses, our findings might at best offer insights into the network structures of depressive symptoms in middle-aged and older adults and inform broad hypotheses and theoretical developments, but may not be directly applicable to explaining within-person variations in the experience of depressive symptoms [127]. Building on the present work, longitudinal network analysis using intensive time-series data (e.g., the experience sampling method) or large panel data is recommended to fully capture the individualised dynamic interactions among symptoms and associated factors over time [128]. Ultimately, with only one of 57 studies being explicitly theory-driven and only half making their datasets available, we concur with some researchers in emphasising that a priori, more clearly defined theoretical frameworks are needed to guide the interpretation of network analysis findings in future studies [14, 129].

Strengths and limitations

This study had several strengths. This was among the first studies that systematically examined networks of depressive symptoms, comorbid mental health symptoms, and both risk and protective factors in adults aged 50 years and older. While previous systematic reviews have restricted their examination of findings to specific populations (e.g., clinical MDD [31]) or scales (e.g., PHQ and GAD scales [35]) to reduce heterogeneity, our inclusion of different study populations and depression scales offered insights into how these factors also play critical roles in the interpretation of network findings.

We also note some limitations. While we retrieved all available data on centrality statistics of the included studies, our synthesis could be performed only on the values reported and not with original data. Similar to previous systematic reviews of network studies [31, 35], we therefore cannot account for the relative distance between nodes. Given the vast amount of data synthesised, centrality rankings were aggregated for synthesis. While our findings may

have shed light on the patterns of centrality rankings observed across studies and their plausibly associated factors, caution is warranted when inferring them to individual studies. The Meta-analytic Gaussian Network Aggregation is a promising technique for comparing networks across studies that estimates a new Gaussian graphical model from the original correlation matrices across datasets [130]. Its current version, nevertheless, could not yet handle a large number of nodes in networks. Further developments of this approach would be an important direction to verify our present observations, with particular consideration of the robustness of centrality statistics across studies. Moreover, as only three of the studies comprised youth samples, how their network structures and symptom dynamics might differ from older age groups remain an important research question in future work.

Conclusions

The present review offered a comprehensive synthesis of the latest evidence in network analysis studies of depression in middle-aged and older adults. To capture a wider range of psychological experiences, we examined depressive symptoms beyond the DSM criteria, along with comorbid symptoms and both risk and protective factors. '*Depressed mood*' and '*lack of happiness*' were among the most frequently reported central symptoms, while comorbid symptoms of anxiety, namely '*excessive*' and '*uncontrollable worry*', '*nervousness*', and '*trouble relaxing*', as well as symptoms of '*insomnia*', '*loneliness*', and '*social isolation*', were among most frequently reported bridge symptoms. Whether targeting these phenomena in intervention would reduce the spread and maintenance of depressive symptomatology requires additional consideration of their within-person dynamic interactions over time. Given the influences of the study populations, external influences, and choice of depression scale on centrality statistics, caution may be needed not to over-generalise findings from a single network analysis study. Testing theory-informed psychometric networks in future work is encouraged.

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