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Comparing ADHD Symptom Network Structure in Chinese Youth with ADHD:

ADHD Alone vs. ADHD with Comorbidities

Jie Luo¹, Wen Shao², Yuanzhen Wu¹, Huanhuan Huang¹, Gaoyang Xu¹, Yanjie Qi¹,

Paul Overton², Yi Zheng^{1*}, Fan He^{1*}

¹ The National Clinical Research Center for Mental Disorders & Beijing Key
Laboratory of Mental Disorders, Beijing Anding Hospital & the Advanced Innovation
Center for Human Brain Protection, Capital Medical University, Beijing, China.

²Department of Psychology, University of Sheffield, Cathedral Court, 1 Vicar Lane,
Sheffield, S1 2LT, UK.

Correspondence

Yi Zheng, De Sheng Men Wai An Kang Hu Tong 5 Hao, Xi Cheng Qu, Beijing
100088, China; Email: yizheng@ccmu.edu.cn

Fan He, De Sheng Men Wai An Kang Hu Tong 5 Hao, Xi Cheng Qu, Beijing 100088,
China; Email: hf981207@163.com

Abstract

Background: ADHD is often comorbid with other psychiatric conditions, but it remains unclear whether comorbidities influence the structure of ADHD symptoms. This study examines ADHD symptom profiles in individuals with ADHD and comorbidities (AC) and individuals with ADHD only (AP).

Methods: The study used a secondary dataset obtained from a mental health survey of 1,906 Chinese youth with ADHD, comprising AP ($n = 897$) and AC ($n = 1,009$). Symptoms were assessed using the caregiver-rated CBCL DSM-oriented ADHD scale (CBCL-ADH). Partial correlation networks were estimated using the EBICglasso algorithm. Network Comparison Tests (NCTs) were conducted to compare the structure of CBCL-ADH items between the AP and AC groups, as well as across subgroups (e.g., gender and age groups).

Results: The network of CBCL-ADH items remained largely invariant across groups, despite higher ADHD severity observed in the AC group ($p < 0.001$). Subgroup analyses were generally consistent, with some exceptions: a marginal difference in network structure between age groups ($p = .07$) and an elevated risk of developing comorbidities among older participants (12–16 years) and female participants.

Conclusion: While comorbid conditions appear unrelated to the structure of caregiver-informed ADHD symptoms, further investigation is warranted to examine ADHD structure using multi-informant assessments, as well as the complex interactions between ADHD and comorbid symptom profiles. Variations in subgroups highlight the need for developmental and sociocultural considerations in managing comorbidities.

These findings suggest that targeting central ADHD symptoms (e.g., restless/hyperactive) may offer benefits for ADHD improvement across comorbid presentations, though further validation is needed.

Keywords: Attention-Deficit/Hyperactivity Disorder (ADHD); Comorbidity; network analysis; Developmental trajectories.

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a prevalent neurodevelopmental disorder characterized by persistent patterns of inattention, hyperactivity, and impulsivity that interfere with daily functioning and development (Posner et al., 2020). Globally, ADHD affects approximately 5-7% of children and adolescents (Nader et al., 2023), with epidemiological variations across regions. For instance, prevalence rates in the United States are estimated at 6-16% (Melissa L et al., 2022), while lower rates of 6.4% are reported in China (Li et al., 2022). The disorder often leads to academic underachievement, impaired social relationships, and familial conflict (Shaw et al., 2012). Longitudinal studies further indicate that untreated ADHD in childhood is associated with adverse long-term outcomes, including increased risks of low self-esteem, substance abuse, and occupational instability (Faraone et al., 2021).

In addition, psychiatric comorbidities are common in Children and adolescents with ADHD , with about 60% of them being diagnosed with at least one co-occurring disorder prior to adulthood (Laura et al., 2017). Recent meta-analytic evidence suggests that oppositional defiant disorder (ODD; 34.7%) and other disruptive behavior disorders (30.7%) are among the most common comorbid diagnoses in youth with ADHD, with anxiety disorders also frequent (18.4%) (Njardvik et al., 2025). Comorbidity is clinically important because it is associated with greater impairment, more complex assessment, and poorer treatment persistence or the need for multimodal intervention (Roberto et al., 2022). However, beyond increasing overall clinical burden, comorbidity may also be associated with differences in how ADHD symptoms relate to

one another. In other words, comorbid presentations may not simply involve “more” ADHD symptoms, but potentially a different pattern of interrelations among ADHD symptoms themselves (Krakowski et al., 2020; Luo et al., 2019). Investigating this question is important because it may help distinguish whether comorbidity primarily reflects greater severity within a broadly similar ADHD symptom architecture, or a partially different phenotypic organization with altered symptom coupling and potentially different clinical priorities (Bringmann et al., 2022).

One useful way to examine this question is through a symptom-network perspective. Some researchers have proposed a latent general psychopathology (“p factor”) contributing to multiple psychiatric conditions (Avshalom et al., 2014). Complementing this view, network models conceptualize disorders as systems of interacting symptoms rather than manifestations of a single latent construct (Laura F & Markus I, 2018). From a network perspective, ADHD comprises interrelated symptoms whose interaction patterns may vary across individuals and may be influenced by comorbid conditions (An et al., 2019), even when comorbid symptoms themselves are not directly modelled. Studying these patterns may help contextualize heterogeneity and inform hypothesis generation.

Prior network analyses of ADHD in children typically recover clusters that align with inattention and hyperactivity/impulsivity, with stronger connections within domains than between them. Across developmental samples, attention-related symptoms - especially being easily distracted and difficulty sustaining attention - often emerge as highly central (i.e., strongly connected) features of ADHD networks (Silk et

111 al., 2019). Some studies also highlight motoric hyperactivity (e.g., fidgeting) and
112 interrupting as tightly interconnected and, in some cases, bridge-like symptoms linking
113 inattentive and hyperactive/impulsive clusters (Liu et al., 2022). These findings provide
114 a symptom-network context for our hypotheses about comorbidity-related differences
115 in overall connectivity and symptom centrality.

116 At a broader etiological level, emerging evidence from genetic epidemiology
117 further underscores the phenotypic (i.e., observable traits) complexity of ADHD
118 through the lens of comorbidity. Twin and genome-wide association studies (GWAS)
119 consistently demonstrate substantial genetic overlap between ADHD and co-occurring
120 neurodevelopmental and behavioral disorders, including ODD, conduct disorder (CD)
121 (Faraone & Larsson, 2019). This shared genetic architecture raises questions about
122 etiological mechanisms underlying comorbidity. Two broad hypotheses have been
123 proposed: (1) pleiotropic genetic effects, in which shared variants confer risk across
124 ADHD and comorbid traits, and (2) distinct subtype accounts, in which comorbid
125 presentations reflect partially separable etiological signatures (Gnanavel et al., 2019).
126 Neurobiological studies have been interpreted as potentially supporting subgroup-
127 specific patterns in some comorbid presentations involving disruptive behavior
128 disorders (e.g., ADHD with ODD/CD), including differences in brain morphology and
129 functional connectivity, although findings remain mixed and require further replication
130 (Vetter et al., 2019). Together, these accounts provide alternative expectations for the
131 present symptom-level analysis. If comorbidity primarily reflects shared vulnerabilities,
132 ADHD only and ADHD with comorbidity groups may show broadly similar ADHD

133 symptom network structures despite differences in overall symptom severity. By
134 contrast, if comorbid presentations reflect partially distinct subtypes, differences in
135 ADHD symptom network structure, connectivity, and/or centrality may be observed.
136 These possibilities motivate the present comparison of ADHD symptom networks
137 across groups, although the underlying mechanisms are not directly tested in this study.

138 Beyond biological liability, developmental accounts emphasize that ADHD
139 symptoms and comorbidity unfold within family and school contexts (Claussen et al.,
140 2024). Parenting practices and family stress are associated with ADHD symptoms,
141 often in bidirectional, transactional ways (Allmann et al., 2022), and school/peer
142 processes (e.g., teacher student relationship quality and peer difficulties) can shape
143 impairment and downstream internalizing/externalizing problems (MacLean et al.,
144 2023). Although these contextual factors are not measured in the present study, they
145 provide an important developmental backdrop for understanding heterogeneity and
146 motivate future work that integrates symptom networks with environmental/relational
147 variables.

148 Importantly, the present study tests these alternative expectations at the symptom-
149 network level by examining whether comorbidity status is associated with differences
150 in the pattern of associations among ADHD symptoms assessed by the seven-item
151 CBCL DSM-oriented ADHD Problems scale. Genetic, large-scale brain network, and
152 general psychopathology (“p factor”) accounts theoretically motivate the present
153 comparison of ADHD symptom networks across groups. However, because these
154 underlying mechanisms are not directly measured here, they are not targets of inference

(Epskamp et al., 2018).

Guided instead by a strictly symptom-level focus, this study applies network analysis and dimensional comparisons to examine whether comorbidity status influences (i) the severity of caregiver-rated ADHD symptoms and (ii) the structure of symptom interactions, including connectivity (overall strength of symptom-to-symptom links) and strength centrality (how strongly a symptom is connected to the rest of the network). Based on prior evidence and the network 'vulnerability hypothesis'(Borsboom, 2017; van Borkulo et al., 2015)– we hypothesized that the ADHD with comorbidities group would show greater ADHD symptom severity than the ADHD-only group. We further expected that, if comorbidity is accompanied by greater developmental and psychosocial burden, the ADHD with comorbidities group may show stronger overall connectivity among ADHD symptoms. This expectation was theory-informed and interpreted cautiously, given that evidence linking denser symptom networks to poorer longitudinal course has been demonstrated in other disorders and direct evidence for this prediction within ADHD-only symptom networks remains limited. Regarding symptom centrality, prior ADHD network studies suggest that core inattentive and hyperactive/impulsive symptoms often occupy central positions, with symptoms such as distractibility, difficulty sustaining attention, restlessness, and excessive talking also appearing as important nodes in transdiagnostic ADHD-related networks. We therefore expected core inattentive and hyperactive/impulsive symptoms to remain among the most central nodes across groups, while treating specific item-level centrality differences as exploratory.

Method

Participants

Participants were recruited from a large cohort national epidemiological survey for mental disorders in Chinese youth. Current study included a total of 1,906 children with a confirmed diagnosis of ADHD regardless of the conditions of comorbidities. The diagnosis was made by two trained physicians according to the MINI-KIDS and DSM4 criteria. Details of the sampling procedure for the full cohort were reported elsewhere (Li et al., 2022; Zhongliang et al., 2023). Participants were assigned to two groups: ADHD pure (AP: $n = 897$) and ADHD with comorbidities (AC: $n = 1,009$). Descriptive statistics for the demographic and clinical information can be found in Table 1.

Table 1

General Information of the Included Sample

	AP	AC
<i>n</i>	897	1009
Age	10.49 (2.74)	11.03 (2.88)
Gender		
Female	229 (25.56%)	328 (32.54%)
Male	667 (74.44%)	680 (67.46%)
ADHD types		
Mixed	192 (21.40%)	378 (37.46%)
Attention Deficits	600 (66.89%)	348 (34.49%)
Impulsivity/ hyperactivity	105 (11.71%)	283 (28.05%)
Number of comorbidities		
1		856 (84.84%)
2		132 (13.08%)

3 or more	21 (2.08%)
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Comorbidities

Behavioural conditions	521 (51.63%)
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Mental and neurodevelopmental conditions	364 (36.08%)
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Both types	124 (12.29%)
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CBCL-ADH

Sum score	6.00 (1.25)	5.37 (2.25)
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I4. Fails to finish things he/she starts	4.24%	5.85%
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I8. Can't concentrate, can't pay attention for long	88.28%	90.38%
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I10. Can't sit still, restless, or hyperactive	78.79%	78.67%
--	--------	--------

I41. Impulsive or acts without thinking	59.15%	71.83%
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I78. Inattentive or easily distracted	2.68%	4.17%
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I93. Talks too much	58.04%	70.34%
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I104. Unusually loud	49.44%	68.45%
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190 *Note.* Extreme values were identified for specific items in each group; two participants
 191 were excluded from the analyses involving age, gender, and item-level statistics. The
 192 final sample sizes were: ADHD with comorbidities (AC), $n = 1,008$; ADHD without
 193 comorbidities (AP), $n = 896$. Behavioural conditions refer to Disruptive, Impulse-
 194 Control, and Conduct Disorders. Mental and neurodevelopmental conditions include
 195 Anxiety Disorders, Bipolar Disorders, Depressive Disorders, Eating Disorders,
 196 Trauma- and Stressor-Related Disorders, and Psychotic Disorders. Percentages
 197 represent the proportion of participants endorsing each item, with endorsement defined
 198 as a score ≥ 1 .

199

200 ***Variables***

201 The DSM-oriented ADHD items of the Child Behaviour Checklist (CBCL-ADH)
 202 was chosen for network analysis. The CBCL is a well-established parent rating scale
 203 used to assess mental and behavioural problems in children, known for its high validity

and reliability in previous studies(K et al., 2002). The CBCL-ADH is a subscale of CBCL that includes items aligned with the DSM criteria for ADHD. It comprises seven items: I4 (Fails to finish things he/she starts), I8 (Can't concentrate, can't pay attention for long), I10 (Can't sit still, restless, or hyperactive), I41 (Impulsive or acts without thinking), I78 (Inattentive or easily distracted), I93 (Talks too much), and I104 (Unusually loud). For readability, we use the following abbreviated labels throughout the remainder of the manuscript: 'Fails to finish things' (I4), 'Can't concentrate' (I8), 'Can't sit still' (I10), 'Impulsive' (I41), 'Inattentive' (I78). No skip structure was applied in the assessment. Missing data were defined at the item level as instances where a caregiver skipped or failed to respond to one or more items on the CBCL-ADH. Because the rate of missing data was negligible ($n = 2$), these incomplete cases were excluded using listwise deletion prior to network estimation.

Network estimation and comparison

We modelled ADHD symptoms as a network in which each item is a node (symptom) and each edge is a unique association between two symptoms after accounting for the others. In this context, connectivity refers to the overall strength of these links, and centrality indicates how strongly a symptom is connected within the network. The networks were estimated by Gaussian Markov random field estimation using graphical LASSO and extended Bayesian information criterion to select optimal regularization parameters (EBICglasso). The tuning parameter was set to default value ($\lambda = 0.50$). The degree of conceptual similarity was low among all included variables(Christensen et al., 2023), as assessed by unique variable analysis. The nonparanormal transformation was

applied due to the skewed distributions(Adela-Maria & Sacha, 2021; Pariya & Ernst C, 2018). The network reliability and stability were assessed by non-parametric bootstrapping based on 3,000 bootstrap samples. The robustness of the network was reflected by the Correlation Stability Coefficient (CS coefficient), and the values ranged between 0 and .75, with higher value indicating the more stable centralities. The edge stability was evaluated by visual check on the confidential intervals, with wider intervals reflecting less stable edge statistics. Network layout was generated by the Fruchterman-Reingold algorithm, and the layouts of different networks were evened to ease the visual inspection. Exploratory graphic analysis (EGA) was conducted to find the network communities. The transformed raw values were used as input. The Walktrap algorithm was used for community searching. A seed of '123' was set to allow repetition. Item stability (IS; the probability that a given node was assigned to a specific community) and structural consistency (SC; the probability that a given community composition was replicated across bootstrapped samples) were estimated based on 1,000 iterations.

Network Comparison Tests (NCT) were utilised to compare between AP and AC groups. To address potential heterogeneity within the comorbidity group, a sensitivity analysis was conducted comparing Children and adolescents with ADHD with different types of comorbidities. Although the internalising - externalising distinction of comorbidities is theoretically meaningful, the sample size for the internalising group was relatively limited ($n = 196$), constraining the reliability of network estimation and sensitivity analyses. Therefore, participants were pragmatically classified as having

either behavioural problems ($n = 521$) or mental health/neurodevelopmental conditions ($n = 364$); those with both were excluded to ensure a clear dichotomy. Results from the Network Comparison Test indicated no statistically significant differences in network structure (maximum edge difference, $M = 0.15$, $p = .35$) or global strength (difference in overall connectivity, $S_o = 0.02$, $p = .96$). In addition, the network structure within the overall comorbid group demonstrated high stability, as indicated by the centrality stability coefficient (CS coefficient = .75). These findings support the decision to analyse comorbid ADHD cases as a single group, given the absence of significant structural variation between comorbidity subtypes. Comparisons were also conducted based on subgroups such as gender (female vs male) and age [younger (6–11 years) vs older (12–16 years)]. For the gender comparison, the sample size was balanced by randomly subsampling male participants. Indices of global connectedness and omnibus tests were used to evaluate group differences in global network density and edge weights (Claudia D et al., 2022). In addition, network density and mean absolute edge weight (MAEW) were reported for descriptive reference. Where omnibus test results were statistically significant, post hoc tests were conducted using the Bonferroni–Holm correction. The NCT was run with 2,000 permutations, and a random seed was set to ‘123’ to ensure reproducibility.

Statistics and Software

All analyses were carried out using R Studio (version 2023.06.0) with R language (version 4.3.1) as the backend. The descriptive analysis offered an overview of group

differences. Network estimation was performed using the *estimateNetwork* function from the *bootnet* package (version 1.5.5). Network comparison testing was conducted using the *NCT* function from the *NetworkComparisonTest* package (version 2.2.2). Visualization of the networks was accomplished using the *qgraph* package (version 1.9.5). Network community detection was executed using the *bootEGA* function from the *EGAnet* package (version 2.0.1).

Results

Comparing AP and AC

The network of AP demonstrated robust stability in node centralities, particularly in the case of node strength (CS coefficient= .67). Other centralities showed lower stabilities: closeness (CS coefficient= .52) and betweenness (CS coefficient= .44). The stability of edge weights was very high (CS coefficient= .75), see Figure 1.

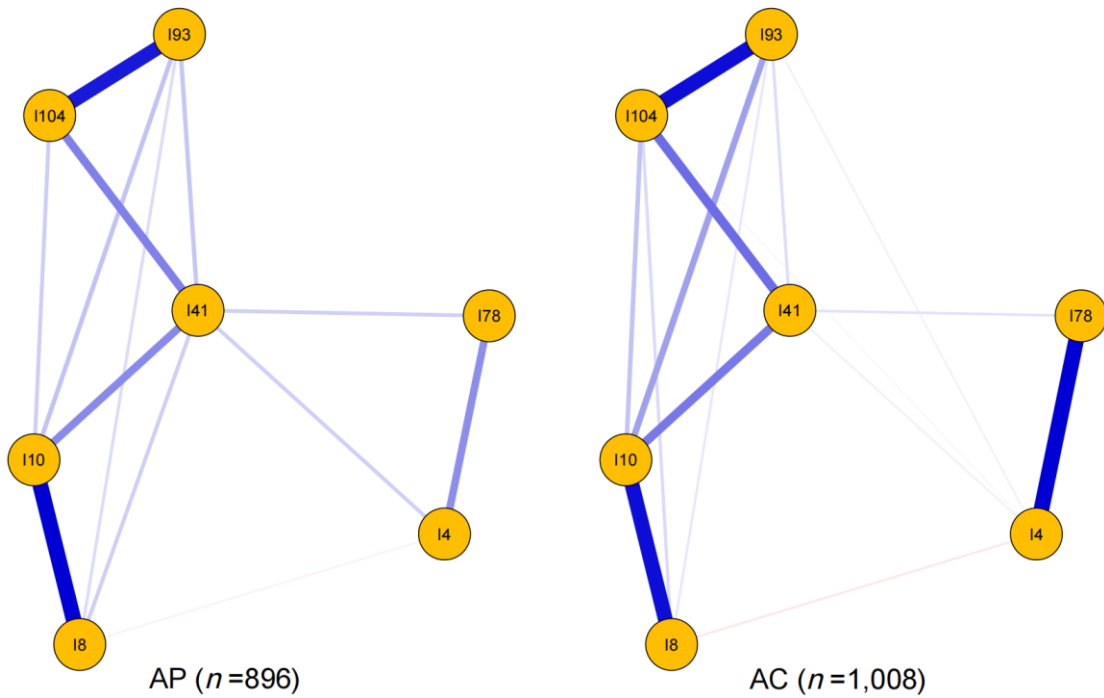


Figure 1

Network structures of the CBCL-ADH symptoms among the two groups

Note. The blue edge represents the positive correlations while the red edge represents the negative correlations. The thickness of the edge reflects the degree of correlation between symptoms. Legends for the nodes: I4 (Fails to finish things he/she starts), I8 (Can't concentrate, can't pay attention for long), I10 (Can't sit still, restless, or hyperactive), I41 (Impulsive or acts without thinking), I78 (Inattentive or easily distracted), I93 (Talks too much), and I104 (Unusually loud).

The network of the CBCL-ADH symptoms in those two groups were visualised on Figure 1. For the network of the AP group, the edge with the highest weight was found between *Can't concentrate* and *Can't sit still* ($w = 0.39$, 95% BCI [0.33, 0.45]), while the lowest weight was found in edge *Fails to finish things* - *Can't concentrate* ($w = -0.02$, 95% BCI [-0.07, 0.03]); please refer to Supplementary Table 1a for further details). The node with highest strength in this network was *Can't sit still*, and *Easily distracted* was the node with lowest degree of strength, see Figure 2 for the detailed centralities.

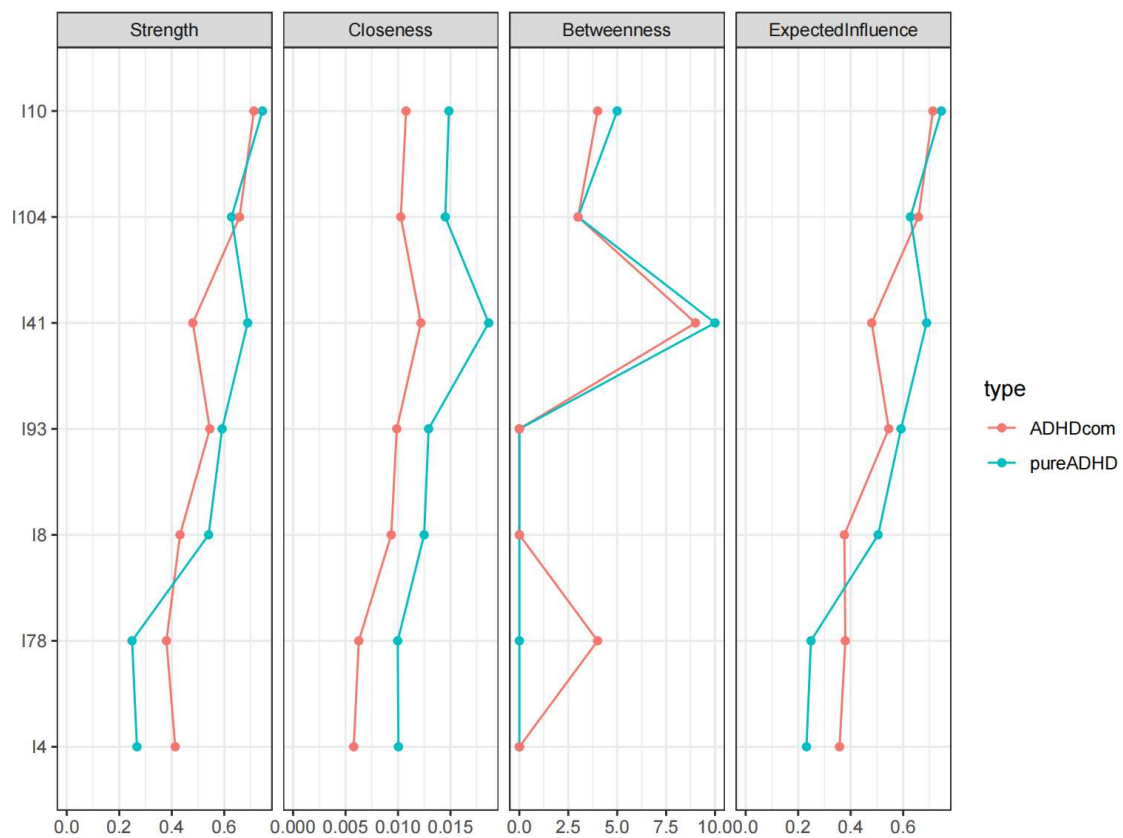


Figure 2

Centralities of the network among the two groups

Note. Raw scale was used to show the degree of strength for each node.

Legends for the nodes: I4 (Fails to finish things he/she starts), I8 (Can't concentrate,

can't pay attention for long), I10 (Can't sit still, restless, or hyperactive), I41 (Impulsive or acts without thinking), I78 (Inattentive or easily distracted), I93 (Talks too much), and I104 (Unusually loud).

The network of AC had similarly high stability on item strength (CS coefficient = .59), but low-level robustness on closeness (CS coefficient = .21) and betweenness (CS coefficient = 0), so please interpret the findings with caution. The edge statistics were very stable (CS coefficient = .75).

The network structures of the AP and the AC did exhibit minor dissimilarities via visual inspection. In the latter network, the edge with the highest weight was *Fails to finish things - Easily distracted* ($w = .34$, 95% BCI [0.21, 0.47]), which is visually thicker than the edge in AP. The edge with the lowest weights was *Fails to finish things - Unusually loud* ($w = .01$, 95% BCI [-0.03, 0.05]). Nonetheless, this edge exhibited a weight of 0 in 57.9% of the bootstrapped samples, suggesting the relationship was unstable. Detailed edge statistics can be found in Supplementary Table 1b. The pattern of node strength was similar to the network in AP, with *Can't sit still* being the node with highest strength and *Easily distracted* with lowest strength, see Figure 2 for detail.

A broadly similar network clustering was observed among AC and AP groups. The EGA identified three communities: community 1 (*Fails to finish things; Easily distracted*), community 2 (*Can't concentrate; Can't sit still*), and community 3 (*Impulsive; Talks too much; Unusually loud*), which suggests that the symptoms within each community are closely connected and exhibit similar relationships with symptoms outside the community, reflecting cohesive patterns of symptom interactions. The

community 1 was very stable across groups of AP (SC = .93) and AC (SC = 1). The community with lowest consistency was community 3 for AP (SC = .60), which the *Impulsive* had unstable placements in the iterated samples. For AC, the community 2 had the lowest stability (SC = .68), see Table 2 for reference.

Table 2

Proportions of the Item Placements in Iterated Samples across Different Groups

CID	AP			AC		
	1	2	3	1	2	3
I4	.93	.02	.05	1	0	.001
I78	.93	.00	.07	1	0	.001
I8	.08	.91	.01	.03	.68	.29
I10	.08	.92	.01	.00	.68	.32
I41	.19	.21	.52	.002	.16	.82
I93	.002	.002	1	.001	0	1
I104	.002	.002	1	0	.001	1
SC	.93	.91	.60	1	.68	.84

Note. CID: Community number, SC: structural consistency; the rate in the box indicates the proportion of the replicated placements to a certain community - the value ranged between 0 and 1, with higher values indicating higher stability. For example, node I8 has been placed to community 2 in 91.2% of the iterated samples of AP.

The network density of AC (.71, 15 out of 21 non-zero edges) was relatively higher than that of AP (.62, 13 out of 21 non-zero edges), and the MAEW was similar between AP (.088) and AC (.086). The NCT revealed that the two networks did not differ between groups in terms of global connectivity ($S_0 = .04$, $p = .85$) or overall network

structure ($M = 0.17$, $p = .10$); therefore, post hoc tests were not conducted.

Subgroup analyses

Descriptive statistics

Four subgroup comparisons were conducted based on AP and AC classifications: (1) FAP (female with AP) vs. FAC (female with AC), (2) MAP (male with AP) vs. MAC (male with AC), (3) YAP (younger participants with AP) vs. YAC (younger participants with AC), and (4) OAP (older participants with AP) vs. OAC (older participants with AC). Overall, participants with AC were generally older than those with AP ($p < .01$). Additionally, the distribution of AC among females and older participants exhibited a distinct pattern. A Chi-Square test of independence was conducted to assess the association between gender, age group, and comorbidity status (AP vs. AC). The test yielded statistically significant results, $\chi^2(3) = 15.83$, $p = .001$, indicating that comorbidity status varied by gender (e.g., female participants) and age (e.g., older participants). The effect size, measured by *Cramér's V* = .06, suggests a weak association.

Additionally, CBCL-ADH severity scores were higher in AC subgroups compared to AP subgroups, with the most pronounced differences observed among males and older participants ($p < .001$). The weakest effect size was found in the comparison between female AP and AC participants ($r = .12$), while the strongest effect size was observed in the comparison between older AP and older AC participants ($r = .29$). Detailed descriptive statistics for these groups are summarised in Table 3, and the distribution of specific items is presented in Supplementary Figure 1.

385

386 **Table 3**387 *Descriptive Information among Subgroups*

	Gender				Age			
	Female		Male		Younger		Older	
	AP	AC	AP	AC	AP	AC	AP	AC
<i>n</i>	229	329	667	680	534	553	362	456
Age, <i>M(SD)</i>	10.45	11.18	10.50	10.97	8.53	8.82	13.38	13.73
	(2.51)	(2.93)	(2.82)	(2.85)	(1.51)	(1.63)	(1.09)	(1.37)
Female, %	100	100	0	0	26.59	29.66	24.03	36.18
CBCL-ADH, %								
Sum score, <i>M(SD)</i>	4.69	5.22	4.43	5.46	4.73	5.38	4.13	5.38
	(2.44)	(2.45)	(2.28)	(2.17)	(2.52)	(2.28)	(2.43)	(2.24)
I4. Fails to finish things he/she starts	5.68	5.17	3.75	6.18	3.93	5.42	4.70	6.36
I8. Can't concentrate, can't pay attention for long	88.21	88.15	88.31	91.47	91.20	89.87	83.98	91.01
I10. Can't sit still, restless, or hyperactive	72.48 ^a	71.12	75.56	82.21	79.77	80.65	67.40	76.10 ^b
I41. Impulsive or acts without thinking	62.01	68.69	58.17	73.24	59.93	68.35	58.01	75.88
I78. Inattentive or easily distracted	3.06	4.26	2.55	4.12	2.06	2.71	3.59	5.92
I93. Talks too much	66.38	73.25	54.87	68.97	60.49	71.43	53.87	69.08

I104. Unusually	55.02	69.91	47.53	67.79	51.87	69.80	45.86	66.89
loud								

Note. The percentage values for each item represent the rate of endorsement, with item scores greater than or equal to 1 indicating the status endorsement.

a. missing in one participant;

b. missing in one participant.

Network comparison test

Network stability was assessed to evaluate the reliability of network comparisons. The stability of edge weights was generally acceptable (CS coefficient $> .50$), except for subgroups with smaller sample sizes, such as the female subgroup (CS coefficient $= .28$). A similar pattern was observed for the stability of node strength. Given these limitations, corresponding results should be interpreted with caution. Interested readers can refer to Supplementary Figure 2 for the distribution of node strength across various AP and AC subgroups.

The NCT indicated no significant differences in network connectivity among AP and AC subgroups ($p > .05$). Additionally, network structures were generally comparable across subgroups, except for age groups, where the difference approached significance ($M = 0.25, p = .07$).

Discussion

The current study examined differences in network structures of caregiver rated ADHD symptoms among Children and adolescents with ADHD with (AC) and without (AP) comorbidities, as well as across relevant subpopulations. The network structure appeared broadly similar between the AC and AP groups, although dimensional analyses indicated higher ADHD symptom severity in the AC group. Similar patterns were observed in symptom clustering across the two groups, with only minor variations noted across age and gender subgroups. Comorbidity risk also varied by age and gender, with a higher proportion of AC observed among older participants (12–16 years) and females.

A broadly similar structure of the ADHD symptoms may suggest a certain degree of stability among the symptom interrelationships. This finding diverged from our initial hypothesis regarding increased connectivity in the presence of comorbidity and possible coupling with psychosocial stressors(Hartman, Rommelse, van der Klugt, Wanders, & Timmerman, 2019). Instead, our results are consistent with psychometric evidence indicating that the internal structure of ADHD dimensions remains remarkably stable across diverse clinical populations (Kate et al., 2020; Rapson, 2013). This suggests a structural robustness in the ADHD symptom architecture that persists even when accounting for the added complexity of co-occurring conditions(Aneta D et al., 2022), and shows longitudinal consistency across multiple informants in recent network analyses(Jonathan, G Leonard, Stephen P, & Mateu, 2020; Michelle M, Patrick K, Christine A, Sarah L, & Joel T, 2021). Consistent with prior research, children with

ADHD and comorbidities conditions exhibited higher symptom severity(Gair et al., 2021; Murray et al., 2022), and our hypotheses are partially supported. However, our network analyses did not reveal meaningful differences in ADHD symptom structure across groups.

This stability is mirrored in genetic studies suggesting that ADHD-comorbidity overlap largely reflects pleiotropic (shared) liability rather than distinct pathogenic pathways(Consortium, 2013). At the behavioral level, comorbidity may exacerbate executive-function difficulties, but current evidence does not consistently support qualitatively new executive-function impairments beyond the additive burden of the component disorders(Willcutt et al., 2005). Together, these findings suggest that comorbidities heighten dysfunction within preexisting circuits rather than creating distinct phenotypic configurations.

Several factors may contribute to the observed structural similarity of ADHD-only networks across comorbidity groups. First, the network was estimated from a brief, seven-item caregiver-rated scale; node selection and measurement can materially shape network structure and reduce sensitivity to subgroup differences(Fried & Cramer, 2017). Second, comorbidity may manifest primarily as higher overall ADHD severity rather than a reorganisation of conditional associations among ADHD symptoms in cross-sectional data. More generally, cross-sectional symptom networks are typically undirected and should be interpreted as patterns of conditional association rather than causal or neurobiological pathways; centrality indices can be unstable and do not, by themselves, justify intervention targets without prospective or interventional

validation(Bringmann et al., 2019; Epskamp et al., 2018).

Exploratory subgroup comparisons suggested only small differences in density/community detection, and NCT results were non-significant ($p > .05$), with the age-group comparison approaching but not reaching significance ($p = .07$). This pattern suggests that the structure of caregiver-rated ADHD symptoms is largely robust across age and sex in this sample, supporting our primary AC-AP comparison. Any minor descriptive variation may reflect developmental changes in symptom expression across childhood/adolescence as well as sampling and measurement factors that can influence subgroup network estimation(Fried & Cramer, 2017). Therefore, these subgroup findings should be interpreted with caution, and further investigation is warranted.

For example, impulsivity showed modest differences in community assignment between males and females. Meta-analytic and clinical reviews suggest that, compared with boys, girls with ADHD are often rated as having less overt hyperactivity/impulsivity and may be under-referred/underdiagnosed, which can shape observed symptom patterns in clinical datasets(Rucklidge, 2010). Given the cross-sectional observational design, we interpret these patterns as sex/gender-related variation in symptom presentation rather than evidence for distinct mechanisms; causal processes cannot be inferred from the present networks(Fried & Cramer, 2017).

Building on these findings, we outline provisional, hypothesis-generating implications restricted to the modelled ADHD symptom set, rather than immediate practice recommendations. The similarity of ADHD-only networks suggests that comorbidity may be associated with higher overall ADHD severity without marked

reorganisation of caregiver-rated ADHD symptom interrelations in this sample. Centrality estimates therefore highlight candidate ADHD symptoms within this seven-item network that warrant prospective and interventional testing, but they cannot determine transdiagnostic centrality or cross-disorder symptom effects. Addressing these questions will require transdiagnostic networks that include comorbid symptom nodes (Imperiale et al., 2021) and, ideally, longitudinal network approaches to test symptom dynamics over time (Chavez-Baldini et al., 2022).

Limitations

Several limitations should be acknowledged: 1) Some centrality indices showed limited stability; larger samples are needed to improve the precision of centrality and edge estimates. 2) The DSM-oriented CBCL ADHD scale is well validated, but parent report may introduce observer bias and the seven-item content limits network granularity; replication using multi-informant and/or performance-based measures is warranted. 3) The present analysis modeled ADHD symptoms only. Because network structure and global connectivity depend strictly on the included nodes, these findings have limited generalizability to cross-disorder symptom structures or the potential coupling effects between ADHD symptoms and psychosocial stressors. These complex dynamics - including how environmental risks might act as bridge variables - remain beyond the reach of this study. Future investigations should utilize transdiagnostic approaches that incorporate symptoms from common comorbid domains (e.g., internalizing symptoms) and longitudinal network models capable of detecting symptom-level causality. Such research would be vital for assessing how

comorbid symptoms and potential environmental interactions influence the underlying mechanisms and long-term persistence of ADHD manifestations. (4) Although the inclusion of various comorbidities into a single group may lack strong theoretical grounding, sensitivity analyses suggested limited heterogeneity; further work should test structural invariance across specific comorbidity profiles.

Conclusion

The network of caregiver-rated ADHD symptoms was broadly similar between ADHD with and without comorbidities, whereas comorbid conditions were associated with elevated ADHD symptom severity. These results support the robustness of the AC–AP comparison within an ADHD-only symptom set, but they do not establish centrality or cross-disorder symptom effects in a broader psychopathology network. Accordingly, any clinical implications of ADHD symptom centrality should be treated as hypotheses requiring prospective and interventional testing, and future transdiagnostic networks that include comorbid symptom nodes are needed.

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Authors' contributions

All authors were involved in data collection. J.L. and W.S. performed the data analysis and wrote the manuscript. P.O. made revisions to the manuscript. YZ.W, HH.H, GY.X, and YJ.Q were involved throughout the data collection process. F.H. made practical contributions to the conduct and advancement of the trial. Y.Z. led the entire team and directed and supervised the whole trial. The authors have declared no competing or potential conflicts of interest.

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Availability of data and materials

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Declarations

Ethical Consideration and consent to participate

The project was approved by the Ethics Committee of Beijing Anding Hospital in 2017 (Approval number: 201743FS-2) and performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All subjects and their parents signed an informed consent form before joining the trial.

572

573 Consent for publication

574 Participants gave consent for data collected to be published in anonymous form in
575 academic journals.

576

577 Competing financial and/or non-financial interests

578 The authors declare that they have no competing interests, or other interests that might
579 be perceived to influence the results and/or discussion reported in this paper.

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581 Clinical trial number: not applicable.

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Supplementary Table 1a

Edge weight stability estimates for the ADHD-only group (AP)

Edge	Weight	95% BCI lower	95%BCI upper	P ₀
I8--I10	0.39	0.33	0.45	0.00
I93--I104	0.36	0.29	0.42	0.00
I41--I104	0.20	0.13	0.27	0.00
I10--I41	0.18	0.12	0.24	0.00
I4--I78	0.18	0.03	0.32	0.01
I10--I93	0.09	0.03	0.16	0.00
I41--I93	0.09	0.02	0.15	0.01
I10--I104	0.08	0.02	0.14	0.01
I8--I41	0.08	0.01	0.14	0.02
I4--I41	0.07	0.00	0.15	0.05
I41--I78	0.07	0.01	0.14	0.02
I8--I93	0.05	0.00	0.11	0.05
I78--I104	0.00	-0.05	0.05	0.61
I78--I93	0.00	-0.04	0.04	0.70
I10--I78	0.00	-0.04	0.04	0.73
I4--I104	0.00	-0.03	0.03	0.71
I4--I10	0.00	-0.03	0.03	0.77
I8--I104	0.00	-0.03	0.03	0.62
I4--I93	0.00	-0.03	0.03	0.72
I8--I78	0.00	-0.04	0.04	0.68
I4--I8	-0.02	-0.07	0.03	0.44

Note. AP = ADHD-only group. The bootstrapped confidence interval (BCI) was computed based on 3,000 bootstrapped samples. P_0 represents the proportion of bootstrap samples in which the edge was estimated to be zero. Rows are bolded where $P_0 > .70$, indicating unstable or weakly estimated edges.

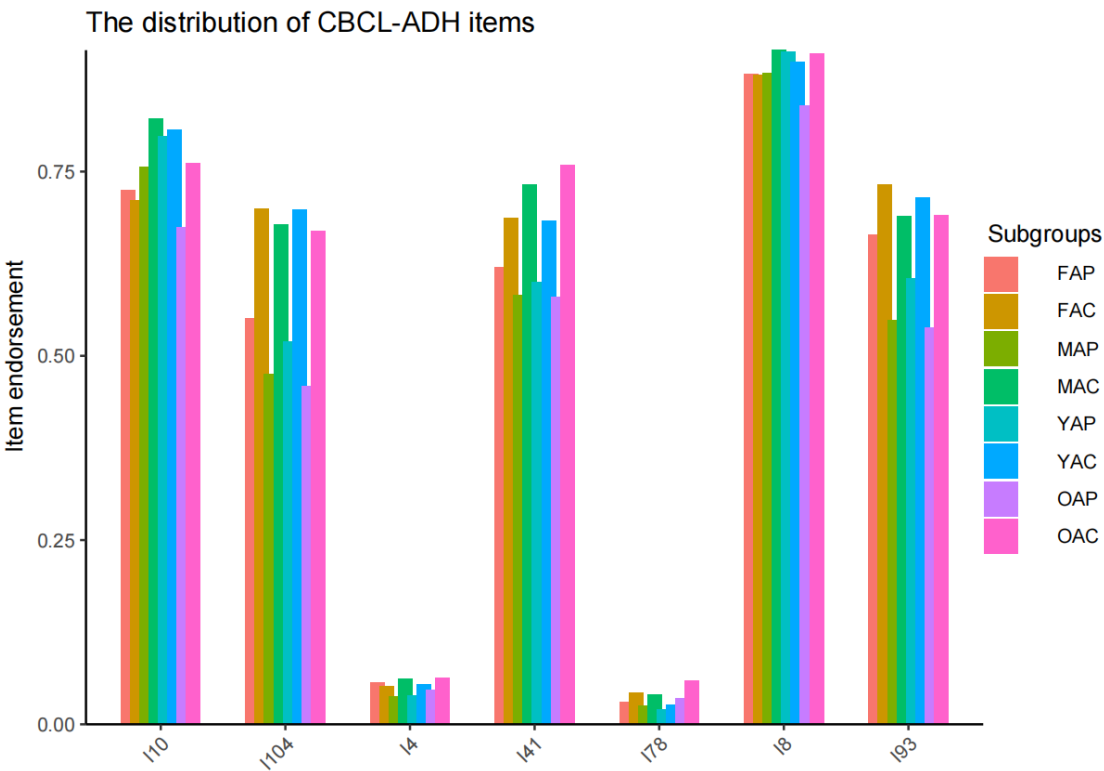
Supplementary Table 1b

Edge weight stability estimates for the ADHD + comorbidity group (AC)

Edge	Weight	95% BCI lower	95%BCI upper	P ₀
I4--I78	0.34	0.21	0.47	0.00
I93--I104	0.32	0.26	0.39	0.00
I8--I10	0.32	0.26	0.39	0.00
I41--I104	0.20	0.14	0.26	0.00
I10--I41	0.18	0.12	0.24	0.00
I10--I93	0.13	0.07	0.19	0.00
I10--I104	0.08	0.02	0.14	0.00
I8--I104	0.05	-0.01	0.10	0.07
I41--I93	0.05	-0.01	0.10	0.08
I41--I78	0.04	-0.02	0.09	0.23
I8--I93	0.03	-0.02	0.08	0.18
I4--I41	0.02	-0.03	0.07	0.45
I4--I93	0.02	-0.03	0.06	0.46
I4--I104	0.01	-0.03	0.05	0.58
I4--I10	0.00	-0.03	0.03	0.76
I10--I78	0.00	-0.03	0.03	0.78
I78--I104	0.00	-0.03	0.03	0.76
I8--I41	0.00	-0.03	0.03	0.75
I78--I93	0.00	-0.03	0.03	0.78
I8--I78	0.00	-0.04	0.04	0.71
I4--I8	-0.03	-0.09	0.03	0.42

Note. AC = ADHD with comorbidities. The bootstrapped confidence interval (BCI) was computed based on 3,000 bootstrapped samples. P_0 represents the proportion of

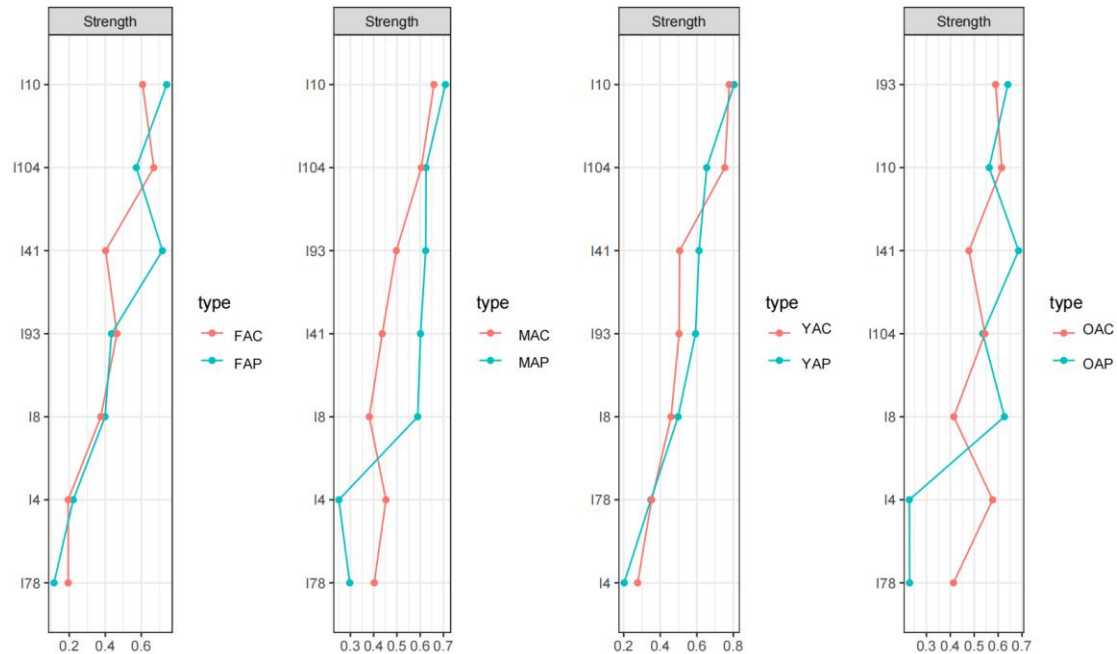
bootstrap samples in which the edge was estimated to be zero. Rows are bolded where $P_0 > .70$, indicating unstable or weakly estimated edges.



Supplementary figure 1

The distribution of ADHD items in various subgroups with AP or AC.

Note. F: Female, M: Male, Y: Younger participants, O: Older participants, A: ADHD, P: without comorbidities, C: with comorbidities; I4 (Fails to finish things he/she starts), I8 (Can't concentrate, can't pay attention for long), I10 (Can't sit still, restless, or hyperactive), I41 (Impulsive or acts without thinking), I78 (Inattentive or easily distracted), I93 (Talks too much), and I104 (Unusually loud).



Supplementary Figure 2

The Node strength in various subgroups with AP or AC.

Note. F: Female, M: Male, Y: Younger participants, O: Older participants, A: ADHD, P: without comorbidities, C: with comorbidities; I4 (Fails to finish things he/she starts), I8 (Can't concentrate, can't pay attention for long), I10 (Can't sit still, restless, or hyperactive), I41 (Impulsive or acts without thinking), I78 (Inattentive or easily distracted), I93 (Talks too much), and I104 (Unusually loud).