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Stage-dependent patterns of cognitive network connectivity in early psychosis

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XiaoChen Tang[1,2,3,10], YanYan Wei[1,2,10], JiaoYan Pang[4], LiHua Xu[1,2], HuiRu Cui[1,2], MingLiang Ju[1], HaiChun Liu[5], Qiang Hu[6], LingYun Zeng[7], YingYing Tang[1], ZhengHui Yi[1], ChunBo Li[1], JiJun Wang[1,2,8] ✉ & TianHong Zhang[1,2,9] ✉

Cognitive impairments in early psychosis are common, yet prior studies have focused mainly on domain-specific deficits rather than inter-domain relationships. Analyzing cognitive network connectivity may uncover insights into early psychosis mechanisms. Cognitive functions were assessed from 2,518 participants, including 988 first-episode schizophrenia (FES), 767 clinical high-risk (CHR), and 763 healthy controls (HC), using the Chinese version of the Measurement and Treatment Research to Improve Cognition in Schizophrenia Consensus Cognitive Battery (MCCB). Results revealed a stage-dependent "dedifferentiation" pattern: mean inter-domain correlation increased from HC (0.28) to CHR (0.33) to FES (0.40). Confirmatory factor analysis revealed a common "g" factor across groups, with significantly reduced strength in FES compared to CHR and HC. The reduction in the "g" factor was associated with increased connectivity and stronger inter-domain correlations. These findings highlight cognitive network dedifferentiation and "g" factor decline as key features of early psychosis.

Early psychosis, spanning from the prodromal phase to the first episode, is characterized by cognitive impairments that significantly affect disease risk progression[1,2] and functional outcome[3,4]. Although these deficits are well-documented[5,6], most studies have merely measured the degree of cognitive impairment[7,8], with less emphasis on understanding the correlational features of these highly overlapping cognitive tests as an integrated cognitive network in relation to psychosis onset.

In the early stages of psychosis, the landscape of cognitive impairment is highly complex[9,10]. Cognitive deficits are not isolated within distinct domains[11]; rather, different cognitive dimensions interact with one another in intricate ways[12,13]. For example, a decline in memory function might trigger compensatory mechanisms in attention or executive function[14]. When cognitive functions are analyzed in isolation, the mutual influences among them can confound the results, making it difficult to obtain robust and reliable findings[15]. This domain-specific approach may limit our understanding of how cognitive impairments develop and progress as an integrated system during early psychosis. This potential increase in inter-domain correlations can be conceptualized as a pattern of 'cognitive dedifferentiation'[16,17]. The notion of cognitive dedifferentiation asserts that specific cognitive functions, which are usually specialized and function with a degree of independence in healthy individuals, exhibit diminished distinctiveness and increased inter-correlation in pathological contexts. Such a

[1]Shanghai Key Laboratory of Psychotic Disorders, Shanghai Mental Health Center, Shanghai Jiaotong University School of Medicine, Shanghai, China. [2]Neuromodulation Center, Shanghai Mental Health Center, Shanghai Jiaotong University School of Medicine, Shanghai, China. [3]School of Psychology, Shanghai Normal University, Shanghai, China. [4]School of Government, Shanghai University of Political Science and Law, Shanghai, China. [5]Department of Automation, Shanghai Jiao Tong University, Shanghai, China. [6]Department of Psychiatry, ZhenJiang Mental Health Center, Zhenjiang, China. [7]Department of Psychiatric Rehabilitation, Shenzhen Kangning Hospital, ShenZhen, China. [8]Department of Psychiatry, Nantong Fourth People's Hospital & Nantong Brain Hospital, Nantong, China. [9]Shanghai Engineering Research Center of Intelligent Psychological Evaluation and Intervention, Shanghai Mental Health Center, Shanghai Jiaotong University School of Medicine, Shanghai, China. [10]These authors contributed equally: XiaoChen Tang, YanYan Wei.
✉e-mail: jijunwang27@163.com; zhang_tianhong@126.com

pattern is thought to reflect a loss of functional specialization and is a core tenet of frameworks like the Neurocognitive Integration Hypothesis[18,19]. This hypothesis suggests that psychosis may stem from a disturbance in the brain's capacity to efficiently coordinate and integrate information across specialized neural circuits, resulting in a broader cognitive deficit. To address these gaps, we've turned to advanced techniques like k-shell decomposition[20,21]. This method partitions a network into hierarchically organized shells based on the connectivity of nodes. The k-core, which consists of nodes with high connectivity, and k-shell assignment, which reflects the hierarchical position of nodes in the network, can provide unique insights into the characteristics of the cognitive network. Taking a holistic view of the cognitive network, by focusing on the overall correlations among various cognitive functions, offers a more comprehensive understanding. It allows researchers to observe how changes in one cognitive aspect might ripple through the network, affecting the early stage of psychosis.

Against the backdrop of domain-specific cognitive analyses and unexplored network dynamics in early psychosis, this study employs a holistic, network-based approach to characterize how cognitive functions interrelate across healthy controls (HC), clinical high-risk for psychosis (CHR), and first-episode schizophrenia (FES) using data from 2,518 participants. Leveraging comprehensive cognitive assessments and graph-theoretical methods like k-core/k-shell decomposition, we systematically map the hierarchical organization of cognitive networks, quantify the resilience of core cognitive domains (k-core), and evaluate the positional importance of individual cognitive measures (k-shell assignment). Our central hypothesis is that cognitive network connectivity will exhibit a stage-dependent "dedifferentiation" pattern, with inter-domain correlations increasing sequentially from HC to CHR to FES—reflecting progressively stronger integration of impaired cognitive functions as they transition through early psychosis stages.

Results

Demographic and cognitive characteristics of the sample

Table 1 presented the demographic and cognitive characteristics of HC, CHR, and FES groups. Significant differences were found in age ($F = 118.08$, $p < 0.001$), with the FES group (22.51 ± 6.26) being older than the CHR group (18.81 ± 5.14), while the HC group (22.45 ± 4.85) had an intermediate age. Education levels were significantly lower in CHR (10.85 ± 3.10) and FES (11.73 ± 3.07) compared to HC (14.36 ± 3.07). Across cognitive assessments, the FES group consistently demonstrated the poorest performance, followed by the CHR group, while the HC group performed best (all $p < 0.001$). No significant difference in gender distribution was observed among the three groups ($p = 0.063$). In cognitive assessments, all tests showed significant group differences ($p < 0.001$), with FES performing worst, CHR intermediate, and HC best. Pairwise comparisons further demonstrated that CHR scores were significantly lower than HC in all cognitive tests (e.g., TMT: $t = 7.82$, $p < 0.001$), FES scores were lower than HC across all measures (e.g., BACS: $t = -31.71$, $p < 0.001$), and FES scores were also significantly lower than CHR (e.g., HVLT: $t = -11.43$, $p < 0.001$).

As shown in Table 1 and visualized in Fig. 1, there is a stepwise cognitive impairment from HC to CHR to FES across multiple domains. Cross-domain correlations among eight cognitive indices (Fig. 1B) showed modest, evenly - distributed associations in HC (mean $r = 0.28$) and CHR (mean $r = 0.33$), while FES exhibited a "dedifferentiation" pattern with an average inter - test correlation of 0.40, indicating domain-performance convergence. This pattern is not driven by statistical artifacts: Supplementary Table S1 shows that FES groups have comparable or larger standard deviations and skewness values within reasonable ranges across all cognitive tests, and Figure S1 further confirms no excessive clustering of scores at the minimum value (i.e., no floor effect) in FES. Differential correlation analysis (Fig. 1C) found 21 of 28 test pairs had significantly stronger correlations in FES than HC (e.g., BACS-TMT: $\Delta r = 0.25$), and a similar but smaller - magnitude pattern in FES vs CHR (e.g., BACS-TMT: $\Delta r = 0.14$). Correlation strengths between CHR and HC were mostly indistinguishable, except VFT-HVLT ($\Delta r = 0.14$). In sum, FES likely has generalized cognitive deterioration, and CHR is an intermediate stage with milder, less correlatively - changed impairments.

Increased connectivity of cognitive network structures from HC to FES

Figure 2 comprehensively examines the cognitive network structures in HC, CHR, and FES groups, highlighting a trend of increasing connectivity from HC to FES. Panel A presents correlation stability curves comparing graph-theory and k-core methods. As the threshold varies, the accuracy of these two methods first rises and then falls, indicating the stability of their correlations under different threshold conditions. In Panel B, box plots of k-max and k-max resilience reveal group differences. The distribution of k-max varies across HC, CHR, and FES, suggesting disparities in the maximum core-related characteristics. Similarly, differences in k-max resilience imply variations in the robustness of the network structures among the three groups. Panel C shows a ROC curve with a threshold of 0.35. This curve evaluates the performance of a binary classifier in distinguishing between groups based on relevant metrics. Panel D depicts the cognitive network structures for each group. The HC group displays a relatively sparse network, with fewer connections among cognitive measures such as WMS, HVLT, and others. The CHR group has a more connected network than HC, but still less dense compared to FES. In contrast, the FES group exhibits a highly interconnected cognitive network, where most cognitive measures are strongly linked.

Identifying the "g" factor through confirmatory factor analysis

In an effort to understand the underlying mechanisms of cognitive network structures and their connectivity patterns, we employed confirmatory factor analysis to explore the factor structure of the MCCB across HC, CHR, and FES groups. The invariance test results and fit for all models (M1–M6) are presented in Table 2. For configural variance, models M1 (HC: g + 3-group factors), M2 (CHR: g + 3-group factors), and M3 (FES: g + 3-group factors) were tested. M1 for HC showed a good fit ($\chi^2 = 13.40$, df=7, $p = 0.063$, CFI = 0.991, TLI = 0.963, RMSEA = 0.042, SRMR = 0.019), indicating that the hypothesized factor structure was appropriate for this group. M2 for CHR and M3 for FES also had acceptable fits, suggesting that the factor structure including the "g" factor and three-group factors was applicable to these groups as well. The model of invariant configurations across groups (M4) was accepted which supported the idea that the overall factor structure was similar across HC, CHR, and FES. Finally, Model M6, with partial invariant factor loadings and invariant intercepts, was accepted ($\chi^2 = 254.52$, $df = 55$, $p < 0.001$, CFI = 0.950, Delta CFI = 0.006, TLI = 0.923, RMSEA = 0.073, SRMR = 0.097). These results suggest that while the overall factor structure is similar across the three groups, the relationships between measures and factors, especially those related to the "g" factor, need to be carefully considered. Supplementary tables present CFA results. Table S2 shows variance explained by factor models. Table S3 lists bifactor model Omega statistics. Table S4 gives fit indexes of partial invariance measurement models. Table S5 offers a model summary with fit measures and parameter estimates. Table S6 contains mediation results, including the Average Causal Mediation Effect (ACME)—a metric quantifying the statistical extent to which the "g" factor relates to inter-domain cognitive correlations—and the Average Direct Effect (ADE)—capturing the direct relationship between group status and cognitive connectivity, independent of the "g" factor.

Table 1 | Demographic and cognitive summary of participants with HC, CHR and FES

Variables	HC (n = 763)	CHR (n = 767)	FES (n = 988)	F/χ^2	p
Age	22.45 (4.85)	18.81 (5.14)	22.51 (6.26)	118.08	9.7E-50
Education	14.36 (3.07)	10.85 (3.10)	11.73 (3.07)	273.17	4.4E-108
Sex (M/F)	359/404	345/422	499/ 489	5.51	0.063
TMT	28.85 (10.49)	33.71 (13.64)	46.42 (26.62)	199.23	4.8E-81
BACS	64.94 (10.10)	57.03 (10.42)	47.98 (12.28)	509.18	2.1E-186
HVLT	26.59 (4.18)	24.28 (5.15)	21.23 (6.03)	229.05	4.1E-92
WMS	16.69 (2.95)	15.37 (3.19)	14.55 (3.47)	94.69	2.2E-40
NAB	19.37 (4.88)	17.35 (6.00)	13.06 (7.01)	246	2.6E-98
BVMT	28.35 (5.34)	26.27 (6.41)	21.77 (7.94)	218.98	2.1E-88
VFT	23.65 (5.67)	20.27 (5.72)	18.33 (5.67)	190.05	1.4E-77
CPT	2.91 (0.65)	2.45 (0.80)	1.93 (0.86)	339.98	2.1E-131
Pair-wise comparisons	**CHR vs HC**			**FES vs HC**			**FES vs CHR**		
	Diff	*t*	*p*	*Diff*	*t*	*p*	*Diff*	*t*	*p*
TMT	4.86	7.82	7.6E-07	17.58	18.94	2.1E-75	12.71	12.98	1.2E-41
BACS	−7.91	−15.08	1.3E-42	−16.97	-31.71	4.5E-186	−9.05	−16.69	3.8E-61
HVLT	−2.31	−9.64	1.4E-17	−5.36	−21.95	1.1E-91	−3.05	−11.43	1.2E-32
WMS	−1.32	−8.41	1.9E-15	−2.14	−13.95	1.8E-41	−0.82	−5.13	1.5E-07
NAB	−2.02	−7.24	1.2E-10	−6.31	−22.19	1.5E-93	−4.29	−13.8	3.3E-46
BVMT	−2.07	−6.88	2.4E-09	−6.58	−20.69	5.9E-84	−4.51	−13.17	5.5E-42
VFT	−3.38	−11.6	1.8E-30	−5.32	−19.49	1.6E-78	−1.95	−7.1	1.5E-12
CPT	−0.46	−12.41	3.4E-30	−0.98	−27.05	1.1E-131	−0.51	−13	3.4E-41

For continuous variables, *p*-values were calculated using one-way ANOVA, and for categorical variables (**Gender**), the chi-squared test was applied. "Diff" means group mean difference (CHR–HC, FES–HC, or FES–CHR), negative values indicate poorer performance relative to the comparison group. All *p*-values <0.001 survive Bonferroni correction for 13 measures (α = 0.05/13 ≈ 0.004). *TMT* Trail Making Test, Part A; *BACS* Brief Assessment of Cognition in Schizophrenia, Symbol Coding; *HVLT* Hopkins Verbal Learning Test-Revised, *WMS* Wechsler Memory Scale-Third Edition, Spatial Span; *NAB* Neuropsychological Assessment Battery, Mazes; *BVMT* Brief Visuospatial Memory Test-Revised, *VFT* Category Fluency Test, Animal naming; *CPT* Continuous Performance Test, Identical Pairs version; *HC* health control, *CHR* clinical high risks for psychosis, *FES* first-episode schizophrenia.

Potential link between reduction of "g" factor and increased connectivity

Figure 3 systematically illustrates the relationships between the general cognitive factor ("g") and cognitive connectivity across HC, CHR, and FES groups, with each panel aligning to key reported results:

Panel A (Factor Model): This panel maps the "g" factor to three domain-specific cognitive factors (Attention, Executive Function, Verbal-Visual Memory) and eight individual cognitive measures (e.g., CPT, BACS). The numerical values on the arrows represent standardized factor loadings—a measure of how strongly each cognitive measure or domain factor is linked to the "g" factor. Notably, the loadings are weakest in the FES group (e.g., BACS: ~0.3 in FES vs. ~0.6 in HC) and intermediate in CHR, directly reflecting the progressive reduction in "g" factor influence reported earlier (CHR vs HC: −0.93; FES vs HC: −1.52; FES vs CHR: −0.59).

Panel B (Violin Plots of "g" Factor Scores): The violin plots visualize the distribution of "g" factor scores across groups (HC: teal; CHR: orange; FES: red). The horizontal shift of the violin distribution (from left to right) corresponds to the stepwise decline in "g" factor strength: HC shows the highest central tendency (mean "g" score ~1.2), CHR shifts left (mean ~0.3), and FES shifts further left (mean ~−0.3). This visual shift directly matches the reported numerical differences: the −0.93 reduction (CHR vs HC) is reflected in the ~0.9-unit drop in mean "g" score, while the −1.52 reduction (FES vs HC) aligns with the ~1.5-unit drop. The wider sections of the violins indicate more participants with that score, confirming that the "g" factor decline is a consistent group-level trend, not an outlier-driven effect.

Panel C (Scatterplot: "g" Factor vs. ACME): ACME (Average Causal Mediation Effect) here quantifies the statistical extent to which the "g" factor is related to inter-domain cognitive correlations—serving as an indicator of cognitive connectivity, but not implying a causal mediation relationship. The scatterplot's connecting lines are intended to highlight the cross-group trend (not individual-level connections or causal links): as groups progress from HC to CHR to FES, the cluster of points shifts downward (lower "g" factor) and to the right (higher ACME). This aligns with our key finding that "g" factor decline is *statistically associated* with increased cognitive connectivity: HC (teal points) cluster in the top-left (high "g", low ACME), CHR (orange) in the middle, and FES (red) in the bottom-right (low "g", high ACME). Individual points within each color represent unique participants, and the overall trend confirms the reported association between "g" reduction and stronger connectivity. Importantly, given the study's cross-sectional design, this association does not establish a causal pathway (e.g., that "g" factor decline drives increased connectivity, or vice versa) nor rule out shared underlying factors that may influence both "g" factor variation and cognitive connectivity.

Panel D (ACME-Colored Correlation Matrices): These matrices use color intensity to represent ACME magnitude (darker shades = stronger "g"-mediated connectivity between cognitive tasks). The matrices show a clear stage-dependent pattern: HC has the lightest overall shading (weak "g"-mediated connectivity), CHR is moderately darker, and FES is the darkest. For example, the connection between BACS and TMT (two key executive function measures) is light in HC (ACME ~ 0.1) but dark in FES (ACME ~ 0.3), directly mirroring the reported increase in inter-domain correlations (HC: 0.28; FES: 0.40) and confirming that "g" factor decline drives stronger cognitive connectivity.

Discussion

This study's strengths, including a large sample of 2518 participants from CHR, FES, and HC groups, comprehensively represent the early psychosis continuum. The application of advanced k-core/k-shell decomposition offers an in-depth view of the cognitive network. Our key finding was that cognitive network connectivity followed a stage-dependent "dedifferentiation" pattern. Specifically, the inter-domain correlations increased sequentially from HC to CHR to FES. FES exhibited the highest average inter-test correlation, indicating a

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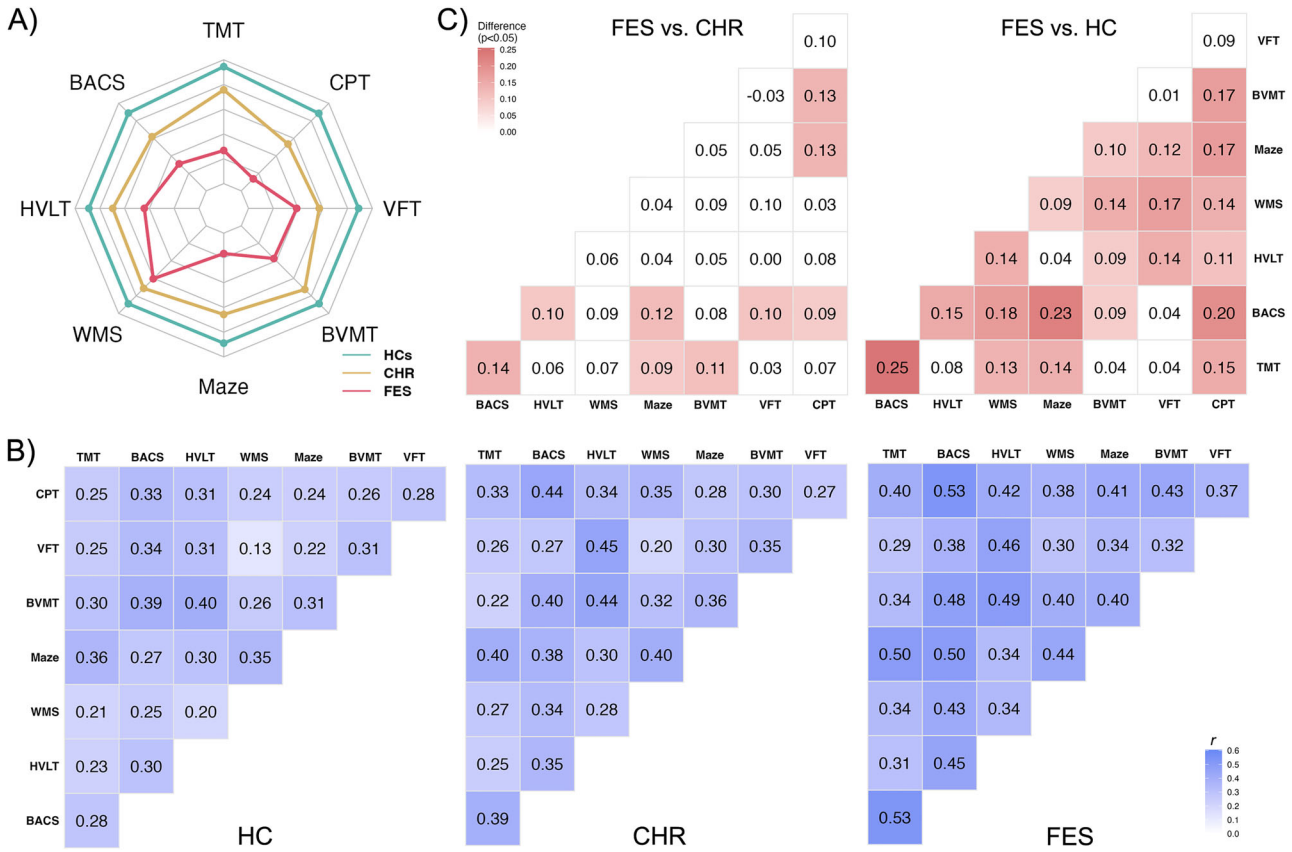

Fig. 1 | Cognitive domain correlations across groups. A Radar plot showing the performance of healthy controls (HCs, teal), clinical high-risk for psychosis (CHR, orange), and first-episode schizophrenia (FES, red) groups across eight cognitive indices: Trail Making Test (TMT), Brief Assessment of Cognition in Schizophrenia - Symbol Coding (BACS), Hopkins Verbal Learning Test - Revised (HVLT), Wechsler Memory Scale - Third Edition, Spatial Span (WMS), Neuropsychological Assessment Battery - Mazes (Maze), Brief Visuospatial Memory Test - Revised (BVMT), Category Fluency Test - Animal naming (VFT), and Continuous Performance Test, Identical Pairs version (CPT). The plot demonstrates a stepwise pattern of cognitive impairment from HC to CHR to FES. All values are z-scores standardized using the HC group as the reference (mean = 0, SD = 1 for HC). The plot demonstrates a stepwise pattern of cognitive impairment from HC to CHR to FES. **B** Within-group Pearson correlation matrices for HC, CHR, and FES, showing the correlations among the eight cognitive indices. The color scale indicates the correlation coefficient (r), with darker shades representing stronger correlations. HC showed modest, evenly distributed associations (mean $r = 0.28$), CHR had slightly stronger correlations (mean $r = 0.33$), and FES displayed a "dedifferentiation" pattern with an average inter-test correlation of 0.40. **C** Difference matrices comparing correlation patterns between FES vs. CHR and FES vs. HC. Pink cells indicate significant differences ($p < 0.05$), highlighting that many test pairs had significantly stronger correlations in FES compared to both CHR and HC. Source data are provided with this paper.

convergence of domain performance. This pattern reflects the progressively stronger integration of impaired cognitive functions during the transition through early psychosis stages. Furthermore, through confirmatory factor analysis, we explored the factor structure of the MCCB across the three groups and found evidence for a common "g" factor. The relationships between measures and factors, especially those related to the "g" factor, varied across groups. Notably, "reduction in 'g' factor" refers to two complementary, consistent metrics: (1) "g" factor scores (derived from CFA, reflecting overall general cognitive resource strength) are stepwise weaker from HC to CHR to FES, with statistically significant group differences; and (2) "g" factor loadings are reduced, meaning cognitive measures are less strongly linked to the "g" factor in psychosis. The degree of this dual reduction (weaker scores + weaker loadings) was associated with increased cognitive network connectivity and stronger inter-domain correlations. For example, FES—with the weakest "g" factor scores and loadings—exhibits the highest mean inter-domain correlation. These findings suggest that diminished "g" factor influence may contribute to the observed changes in cognitive network connectivity, aligning with the dedifferentiation pattern and potentially underpinning cognitive deficits in early psychosis.

The increased inter-domain correlations from HC to CHR to FES suggest a breakdown of the distinctiveness of cognitive domains in early psychosis. In HC, cognitive functions operate relatively independently, which is reflected in the modest correlations among different cognitive tests. However, across the early psychosis continuum, these functions become more intertwined—with CHR and FES groups exhibiting stronger inter-domain correlations than HC. This pattern could be a potential compensatory mechanism of the brain attempting to maintain overall cognitive function amid cognitive vulnerabilities[22,23]. For example, when one cognitive domain, like memory, starts to decline in CHR and FES patients, other domains, such as attention or executive function may become more closely associated with it[24,25]. This increased integration might allow the brain to redistribute cognitive resources, although it ultimately does not fully compensate for the overall cognitive decline. In our previous studies[11,26], we found that in CHR individuals, the correlation between social cognition and neurocognition was significantly stronger compared to HC. Specifically, in the 2016 study, we observed a positive correlation in CHR individuals, which was absent in the HC group. The 2018 study further revealed that the influence of neurocognition on social cognition was significant in CHR but not in HC. These findings suggest that as psychosis progresses, the increased correlation between different cognitive domains may be related to a compensatory mechanism in the brain, aiming to maintain overall cognitive function despite emerging impairments. Moreover, this pattern could

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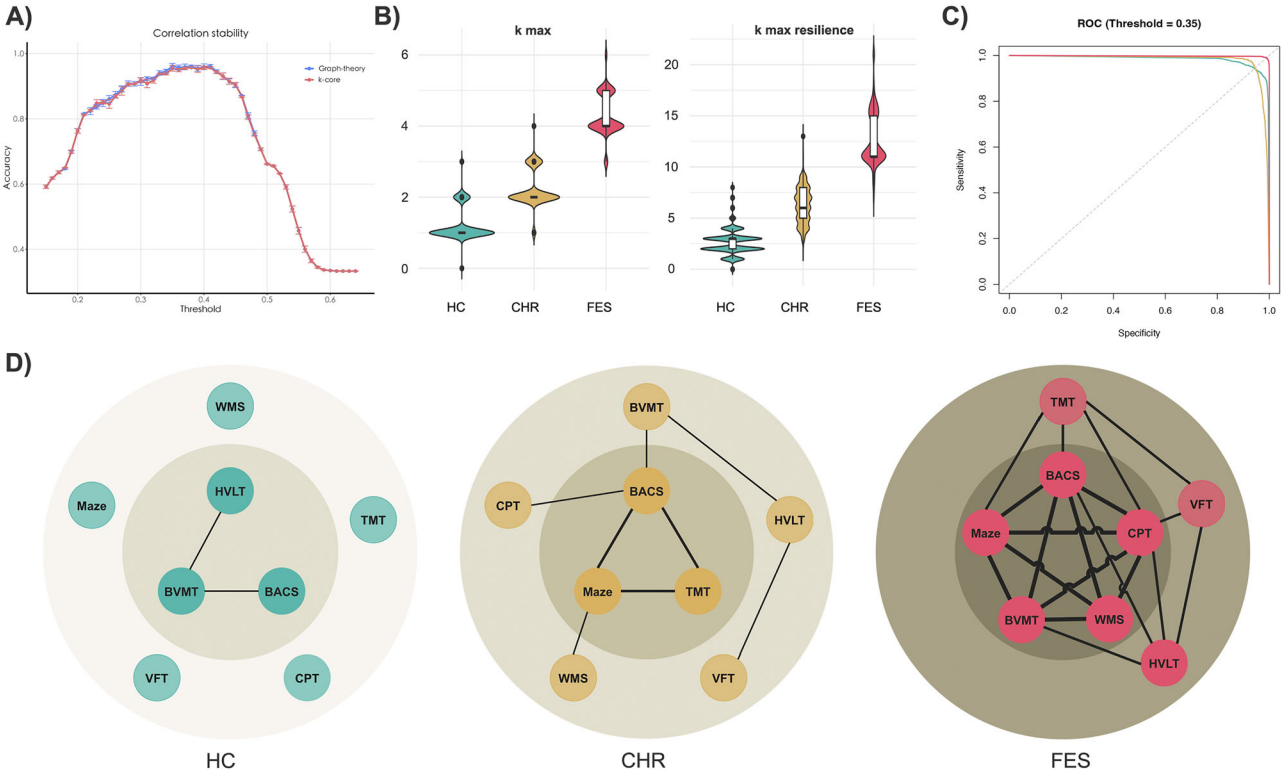

Fig. 2 | Analysis results of network-related metrics and cognitive network structures across different groups. A Correlation stability curves comparing graph-theory and k-core methods across different threshold values. The accuracy of the two methods is plotted against the threshold, showing how stable their correlations are as the threshold changes from 0.15 to 0.65 using random sampling for 1000 times. Error bars as mean ± SD. **B** Box plots of k-max (left) and k-max resilience (right) for healthy controls (HC, $N = 763$), clinical high-risk individuals (CHR, $N = 767$), and first-episode schizophrenia patients (FES, $N = 988$) groups. These plots illustrate the distribution and differences of these two metrics among the three groups a threshold of $r = 0.35$. Box: median line, 25th–75th percentiles; Whiskers=1.5x Interquartile Range; Points=Outliers. **C** Receiver Operating Characteristic (ROC) curve with a threshold of $r = 0.35$, which can be used to evaluate the performance of a binary classifier in distinguishing between different groups based on relevant metrics. **D** K-core Visualizations of cognitive network structures for HC, CHR, and FES groups. Each node represents a cognitive measure (e.g., WMS, HVLT, etc.), and the connections between nodes represent the relationships within the cognitive network, showing differences in network connectivity across groups. WMS Wechsler Memory Scale, HVLT Hopkins Verbal Learning Test, TMT Trail Making Test, BVMT Brief Visuospatial Memory Test, BACS Brief Assessment of Cognition in Schizophrenia, CPT Continuous Performance Test, VFT Category Fluency Test. Source data are provided with this paper.

Table 2 | Fit indexes across models of configural and measurement invariance

Model	*Comparison*	χ^2	*df*	*p*	*CFI*	*ΔCFI*	*TLI*	*RMSEA*	*SRMR*	*Decision*
Configural Variance										
M1. HC: g + 3-group factors	-	13.40	7	0.063	0.991	-	0.963	0.042	0.019	-
M2. CHR: g + 3-group factors	-	31.40	7	<0.001	0.975	-	0.899	0.081	0.026	-
M3. FES: g + 3-group factors	-	26.69	7	<0.001	0.988	-	0.953	0.065	0.019	-
M4. Invariant configurations across groups	-	71.33	21	<0.001	0.985	-	0.940	0.064	0.019	Accept
Measurement Variance										
M5. Invariant factor loadings	M5 Vs M4	320.43	49	<0.001	0.929	0.056	0.879	0.088	0.136	Modify
M5a. Partial invariance: WorkingMemory =~ TMT	M5a Vs M5	218.73	47	<0.001	0.955	0.026	0.920	0.074	0.096	Accept
M5b. Partial invariance: WorkingMemory =~ TMT, g =~ TMT	M5b Vs M5a	214.37	45	<0.001	0.956	0.001	0.917	0.075	0.093	Reject
M6. Partial invariant factor loadings and invariant intercepts	M6 Vs M5a	254.52	55	<0.001	0.950	0.006	0.923	0.073	0.097	Accept

Model invariance is evaluated using differences in the Comparative Fit Index (CFI) rather than the chi-square test. This is because CFI is less influenced by model complexity and sample size, providing a more reliable measure for assessing invariance. A change in CFI (ΔCFI) less than or equal to 0.01 indicates that additional constraints in the model do not negatively impact the fit. In such cases, it supports the hypothesis of invariance, as per the guidelines provided by Cheung & Rensvold (2002). This helps in determining whether the structure and relationships assumed in the models are consistent across different groups. CFI Comparative Fit Index, ranges from 0 to 1, with values closer to 1 indicating a better fit. It compares the fit of the proposed model to a baseline model. ΔCFI Change in the Comparative Fit Index, used to assess the impact of adding constraints to the model on its fit. TLI: Tucker-Lewis Index, also ranges from 0 to 1 and is another measure of model fit. Values closer to 1 suggest a better-fitting model. RMSEA Root Mean Square Error of Approximation, values below 0.05 generally indicate a good fit, while values between 0.05 and 0.08 suggest a reasonable fit. SRMR Standardized Root Mean Square Residual, values less than 0.08 are typically considered indicative of a good-fitting model.

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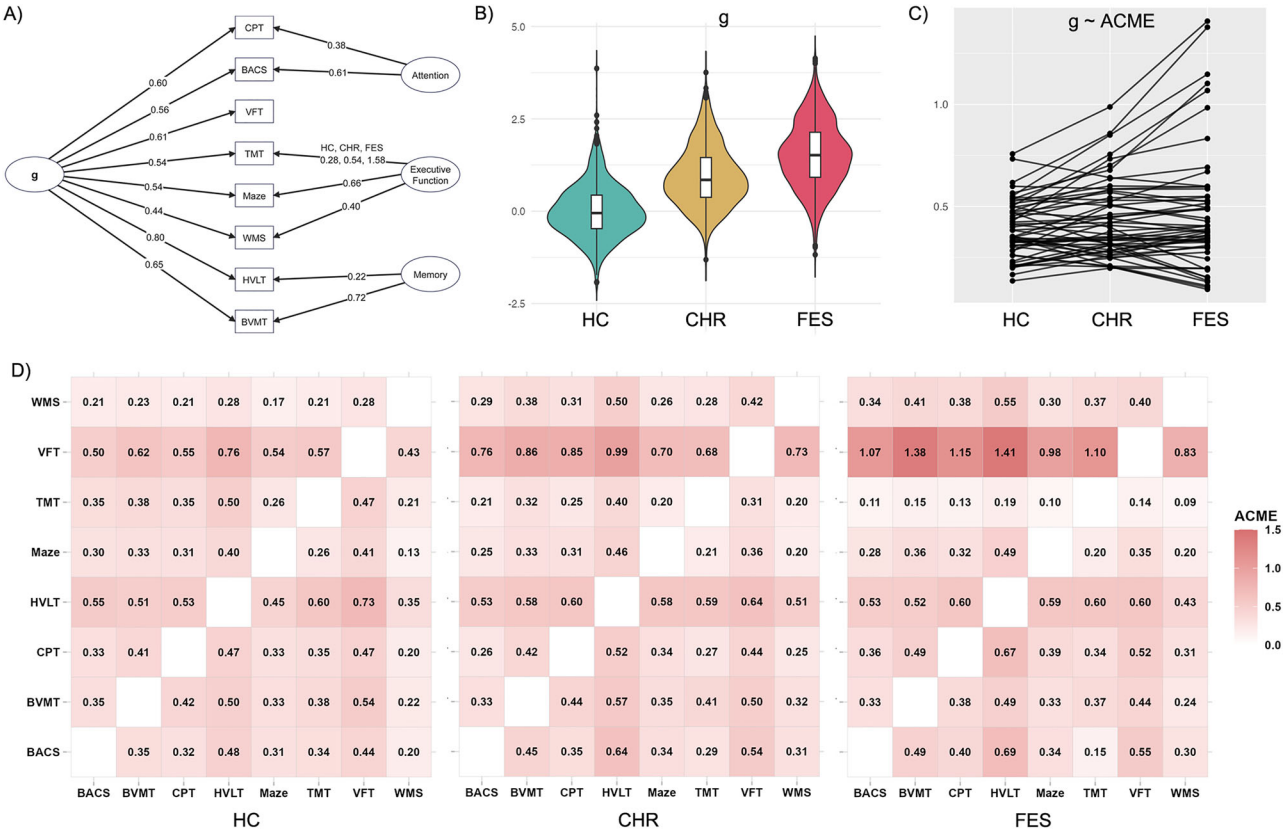

Fig. 3 | Relationships between the general cognitive factor (g) and cognitive connectivity across early psychosis. A Path diagram linking the "g" factor (general cognitive resource) to three domain-specific factors (Attention, Executive Function, Memory) and eight cognitive measures (CPT, BACS, TMT, VFT, Maze, WMS, HVLT, BVMT). Numbers on arrows = standardized factor loadings (weaker loadings in FES/CHR reflect reduced "g" influence, matching reported differences: CHR vs HC −0.93, FES vs HC −1.52, FES vs CHR −0.59). **B** Violin plots of "g" factor scores (HC: teal, $n = 763$; CHR: orange, $n = 767$; FES: red, $n = 988$). Violin width indicates participant density (wider = more participants with that score). The upward shift of FES/CHR violins relative to HC visually represents the stepwise "g" factor decline. Box: median line, 25th–75th percentiles; Whiskers=1.5x Interquartile Range; Points=Outliers. **C** Scatterplot of ACME (Average Causal Mediation Effect, a measure of "g"-mediated cognitive connectivity). Connecting lines highlight the cross-group trend (not individual connections) of cognitive connectivity between tasks (e.g., VFT - HVLT, the topmost line). **D** ACME-colored correlation matrices for each group. Darker shades = stronger "g"-mediated connectivity between cognitive tasks. FES shows the darkest shading, CHR intermediate, and HC lightest—mirroring the stage-dependent "dedifferentiation" pattern (inter-domain correlations: HC 0.28, CHR 0.33, FES 0.40). CPT Continuous Performance Test, BACS Brief Assessment of Cognition in Schizophrenia, VFT Category Fluency Test, TMT Trail Making Test, Maze Neuropsychological Assessment Battery - Mazes, WMS Wechsler Memory Scale, HVLT Hopkins Verbal Learning Test - Revised, BVMT Brief Visuospatial Memory Test - Revised. Source data are provided with this paper.

also imply a shared underlying pathophysiological process affecting multiple cognitive domains simultaneously[27].

The presence of the "g" factor across the HC, CHR, and FES groups indicates a common underlying factor influencing cognitive performance. In HC, the "g" factor seems to play a relatively stable and well-defined role in coordinating different cognitive functions. However, in CHR and particularly in FES, the reduction of the "g" factor is remarkable. Notably, the "g" factor—our primary metric of interest—demonstrated relative stability across age in our sample, with minimal influence from age-related variability (Supplementary Fig. S4). This aligns with the observation that general cognitive factors are less sensitive to age effects compared to domain-specific cognitive scores, supporting the robustness of our findings to covariate influences. The "g" factor is often considered a measure of general intelligence or a common cognitive resource that supports various cognitive tasks[28,29]. Its decline in early psychosis[30,31] could suggest a fundamental disruption in the brain's ability to efficiently allocate cognitive resources across different domains. This reduction might be due to inflammatory processes[32,33], abnormal neural connectivity[34,35], or dysregulation of neurotransmitter systems[36] in the brain regions associated with general cognitive processing. For instance, areas like the prefrontal cortex, which are crucial for high-level cognitive functions and are also thought to be related to the "g" factor, may be affected in early psychosis. As a result, the integrity of the "g" factor is compromised, leading to a decline in overall cognitive performance and potentially contributing to the specific cognitive profiles observed in CHR and FES patients.

The association between group-level differences in "g" factor strength and increased cognitive network connectivity is a complex but crucial finding. As the "g" factor weakens from HC to CHR to FES, the inter-domain correlations strengthen—consistent with a non-causal statistical relationship between reduced general cognitive resource strength and enhanced cognitive domain coupling. While this pattern aligns with the hypothesis that "g" factor variation may be linked to changes in cognitive network organization, the cross-sectional design prevents us from inferring causal direction (e.g., whether "g" decline drives connectivity changes, or vice versa) or ruling out shared underlying factors that influence both. This pattern is consistent with a non-causal statistical relationship: as "g" factor strength weakens across groups (HC → CHR → FES), cognitive domains show stronger inter-correlations—potentially reflecting greater reliance on cross-domain coupling to support cognitive function. For example, FES (the group with the weakest "g" factor strength) exhibits the highest cognitive network connectivity—suggesting that stronger

domain coupling may be a response to reduced general cognitive resource strength, rather than a longitudinal re-organization of cognitive processes. However, this re-organization also leads to the "dedifferentiation" of cognitive domains, as seen by the higher inter-test correlations. It is also possible that the underlying neural mechanisms causing the "g" factor decline are simultaneously promoting the increased connectivity between different cognitive-related brain regions. Future research is needed to disentangle the causal relationships between the "g" factor decline, increased connectivity, and cognitive deficits in early psychosis, which could potentially lead to more targeted interventions for patients at risk or in the early stages of psychosis.

The discovery of the stage-dependent "dedifferentiation" pattern—with inter-domain correlations increasing from HC to CHR to FES—represents a key contribution. This pattern reflects abnormal cognitive integration: from modular, independent function in health to excessive coupling in psychosis. Importantly, this phenomenon is not isolated from clinical or neural phenotypes. Prior work links similar cognitive integration anomalies to prodromal symptom severity in CHR individuals[8], and it aligns with neuroimaging findings of abnormal cortico-cognitive connectivity in early psychosis—including hyper-connectivity in fronto-temporal networks and thalamic-prefrontal pathways[37–39]—providing cognitive-level evidence for shared mechanisms. Additionally, identifying a common "g" factor and its association with increased connectivity offers mechanistic insights: progressive "g" decline (a marker of general cognitive resource loss) may drive abnormal domain coupling, explaining reduced efficiency despite stronger inter-test correlations. These findings complement neuroimaging work, forming a multi-level framework to understand cognitive impairment in early psychosis.

This study has several limitations that should be acknowledged. First, the cross-sectional design limits our ability to infer temporal or causal relationships—including those related to the mediation analysis. While we observed a statistical association between "g" factor variation, group status, and cognitive connectivity, we cannot confirm whether "g" factor decline contributes to increased correlations, or if both are driven by shared unmeasured factors (e.g., pre-existing neural vulnerabilities). Conclusions about "mediation" should therefore be interpreted as reflecting statistical associations, not causal mechanisms. While we controlled for key confounders (age, education) via statistical adjustments and strict inclusion criteria (e.g., excluding substance abuse or prior treatment), longitudinal studies with repeated cognitive assessments are critical to validate the directionality of these changes and their predictive value for psychosis onset. Second, although efforts were made to match demographic characteristics, the significant age differences among the CHR, FES, and HC groups may confound the results. Age-related cognitive changes could influence the observed cognitive network connectivity and factor structures[40], making it difficult to isolate the effects of psychosis risk status. Third, while our multi-center design includes diverse clinical sites across China, all participants are from a single geographic region, which may limit generalizability to other populations. Demographic, socioeconomic, and cultural factors—such as educational systems, linguistic backgrounds, and stigma-related help-seeking behaviors—could influence cognitive test performance and psychosis trajectories, warranting replication in ethnically and geographically diverse samples. Fourth, the exclusion of individuals with substance abuse or dependence and those with prior psychiatric treatment may limit the generalizability of our findings. These excluded populations may have unique cognitive profiles and network patterns, and their absence could lead to an incomplete understanding of cognitive impairments in early psychosis. Fifth, the cognitive assessments used in this study, while comprehensive, may not fully capture the complexity of real-world cognitive functions. Some subtle cognitive deficits in areas such as social cognition[41,42] may have been overlooked, which could impact the interpretation of cognitive network relationships and their association with psychosis. Sixth, while we mitigated overfitting risk by validating network properties across a range of correlation thresholds ($r = 0.15–0.65$), the fixed threshold of $r = 0.35$ for Fig. 2 was derived from the same dataset used to analyze group differences—introducing potential bias. Future studies could adopt model-based network estimation approaches (e.g., EBICglasso) to generate data-driven weighted networks, which may reduce threshold subjectivity and further enhance robustness, particularly for integration with weighted graph-theoretical metrics. To strengthen clinical and pathophysiological relevance, future analyses should integrate cognitive network metrics with: (1) psychopathology data to clarify links with prodromal symptom trajectories; (2) structural/functional MRI data to map cognitive dedifferentiation onto neural connectivity patterns; and (3) longitudinal outcomes to validate its predictive value for psychosis conversion, building on prior risk prediction models.

This research identifies a stage-dependent "dedifferentiation" pattern in cognitive networks across early psychosis, with inter-domain correlations strengthening sequentially from HC to CHR to FES. This pattern, coupled with stepwise group differences in "g" factor strength (weaker in FES than CHR, and weaker in CHR than HC), provides a insightful framework for understanding cognitive deficits. The findings highlight the potential of cognitive network metrics as markers of psychosis progression, laying groundwork for targeted early interventions.

Methods

Participants

This study utilized data from two complementary research programs to characterize cognitive profiles across the early psychosis continuum: the National Key R&D Program of China (2016YFC1306800) and the Shanghai At Risk for Psychosis-Extended (SHARP-Extended). Both programs were leaded by the Shanghai Mental Health Center (SMHC), with other five tertiary psychiatric hospitals in China contributed to these programs, which aimed to identify early-stage cognitive and biological markers of psychosis. A total of 988 FES patients, 767 CHR individuals, and 763 HC were enrolled between January 2016 and December 2024. Crucially, all participants were free of prior psychiatric treatment, including psychotropic medications, and met strict exclusion criteria for substance abuse or dependence, as confirmed by clinical interviews and standardized screening tools. This design isolated primary psychotic disorders, avoiding confounding effects of substance-induced psychosis, which often presents with relatively preserved cognition. HC were recruited from community and school settings, matched for age and socioeconomic status to FES participants. Both programs were approved by the Research Ethics Committees at SMHC (IRB2016-009, 2017-24R1) and participating hospitals, adhering to the Declaration of Helsinki. Written informed consent was obtained from all participants; for those under 18 years, parental consent was supplemented by assent from the participants themselves.

Inclusion and exclusion criteria

CHR individuals were identified using the validated Chinese version of the Structured Interview for Prodromal Syndromes/Scale of Prodromal Symptoms (SIPS/SOPS)[43,44], adapted through prior research[45–47]. This instrument assesses three risk categories: attenuated psychotic symptoms (e.g., unusual thought content, perceptual abnormalities), brief limited intermittent psychotic episodes, and genetic/functional risk indicators (e.g., first-degree family history of psychosis, significant social/academic decline). The Chinese SIPS demonstrates strong inter-rater reliability (intraclass correlation coefficient for total score: $r = 0.96$, $p < 0.01$) and predictive validity, with a 26.4% conversion rate to clinical psychosis within two years. All assessments were conducted by clinicians trained to SIPS standards.

FES diagnosis followed DSM-IV-TR criteria, requiring symptom duration ≤3 years and no history of systematic psychiatric treatment (≤2 weeks of treatment, including medications, psychological therapies, and neuromodulation techniques, prior to recruitment). This strict inclusion criterion targeted treatment-naïve individuals to isolate untreated disease mechanisms, excluding those with prior interventions that might confound cognitive or clinical profiles.

Regarding age, participants were required to be between 14 and 45 years old. Those with a mental disability (IQ lower than 70) or organic mental disorders were excluded. HC had the same inclusion and exclusion criteria as the CHR and FES groups, with the additional requirement that they had no history of mental illness as screened by the Mini International Neuropsychiatric Interview (MINI 5.0)[48] and no first-degree relatives with a history of psychosis. When recruiting HC, efforts were made to match their demographic characteristics to those of the FES group as closely as possible.

Cognitive assessments

Neurocognitive functioning was evaluated through the Chinese version of the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) Consensus Cognitive Battery (MCCB)[49], with all assessments carried out following the standardized procedures described in the MCCB test manual. Notably, the original English MCCB includes a digit span task for verbal working memory, but this subtest was excluded during Chinese adaptation due to significant variability in Chinese participants' familiarity with English letters, which would introduce confounding bias unrelated to cognitive ability. Similar to prior research[31,40,50], this study utilized eight subtests including Trail Making Test, Part A (TMT) for assessing visual-motor tracking and attention, Symbol Coding from the Brief Assessment of Cognition in Schizophrenia (BACS) which measures processing speed and symbol-number association ability, Category Fluency Test (VFT) for evaluating verbal fluency and semantic memory, Continuous Performance Test, Identical Pairs version (CPT) focusing on sustained attention and response inhibition, Spatial Span from the Wechsler Memory Scale-III (WMS) to examine visuospatial working memory, Hopkins Verbal Learning Test-Revised (HVLT) for testing verbal learning and memory, Brief Visuospatial Memory Test-Revised (BVMT) assessing visuospatial memory, and Mazes from the Neuropsychological Assessment Battery (NAB) for measuring problem-solving and planning skills. The inter-rater reliability of the MCCB, determined by trained evaluators' ratings, ranged from 0.82 to 0.95.

Statistical analysis

All data analyses were carried out using R (v. 4.4.2) and Python (v. 3.10.12). Demographic and cognitive features were analyzed in multiple ways, with rigorous correction for multiple comparisons applied across key analyses. Continuous variables like age and education years were compared across groups through ANOVA in the rstatix package (v. 0.7.2), with post-hoc pairwise comparisons adjusted by the emmeans package (v. 1.9.0) to control for family-wise error rate (FWER). Categorical variables such as sex were evaluated via chi-square tests with the base R stats module. Pearson correlation matrices for the 8 cognitive sub-tests were generated for each diagnostic group using psych (v 2.4.0). Partial correlations, controlling for age, sex, and years of education, were computed when constructing networks and performing factor analyses to isolate intrinsic cognitive relationships. Detailed results of these covariate-adjusted analyses are reported in Supplementary Figs. S2, 3 and Table S7. Between-group differences in individual correlation coefficients were tested with Steiger's z tests in the cocor package (v1.1-4), with p values corrected by FDR within each group-wise comparison.

To characterize network properties of cognitive test correlations, adjacency matrices (r> = 0.15) were constructed and analyzed in Python using NetworkX (v. 3.1). Graph-theoretical metrics (e.g., global efficiency, modularity) and k-core decomposition indices (e.g., k-max, k-max resilience) were extracted used a fixed threshold across a range of values (thresholds = np.arange (0.15, 0.65, 0.01)) to systematically explore how network properties vary with sparsity and mitigate overfitting risk. Key findings (e.g., stage-dependent connectivity) were validated as consistent across thresholds r = 0.25–0.40. For the ROC analysis and k-shell visualizations in Fig. 2a, threshold of r = 0.35 was selected for optimal group discrimination; this value was chosen within the 'robustness window' (r = 0.25–0.40) to ensure stability. For classification tasks, R's randomForest package (v. 4.7.1.1) was used to train models, and related metrics were derived with the caret (v.6.0.94) and pROC (v.1.18.0) packages. To uncover latent structure, exploratory factor analysis was first applied with psych::fa, followed by a confirmatory bifactor model in lavaan (v 0.6-17), and scalar-invariant factor scores were compared across groups. Mediation analyses via the mediation package (v. 4.5.0) were conducted to study the role of general cognitive ability in mediating test correlations, and repeated-measures ANOVA was used to assess group differences in average causal mediation effects (ACME) magnitudes. Finally, the hypothesis that the general factor of intelligence mediates group-related amplification of inter-test correlations was tested, with ACME estimated for each test pair in each group, and repeated-measures ANOVA and FDR correction applied. All tests were two-tailed with $\alpha = 0.05$.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The data generated in this study have been deposited in the OSF database [https://osf.io/f5ehn/files]. Source data are provided with this paper.

Code availability

The statistical code for analysis may be made available based on email request to zhang_tianhong@126.com, using a code availability agreement.

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Author contributions

Dr. X.C.T., Y.Y.W., T.H.Z., and J.J.W. conceptualized the study, wrote the first draft of manuscript, and conducted the statistical analyses. L.H.X., J.Y.P., M.L.J., H.R.C., M.L.J., and Y.Y.W. interviewed participants and collected and organized the primary data. Z.H.Y., C.B.L., L.Y.Z., H.C.L., Q.H., and Y.Y.T. managed the literature searches, statistical analyses, and edited the manuscript. T.H.Z. and J.J.W. designed the study and provided supervision in the implementation of the study. All authors have approved the final manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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Correspondence and requests for materials should be addressed to JiJun Wang or TianHong Zhang.

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