Article

Theory-of-Mind-Related Social Cognition as a Mediator and Bridge Between Neurocognition and Social Functioning in Chinese Patients With Schizophrenia: Latent Variable Modeling and Psychological Network Analysis

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Abstract

Background: Social functioning impairments are a hallmark of schizophrenia and a strong predictor of long-term disability. While neurocognition and social cognition are known to predict functioning, the micro-level topological architecture connecting these domains—and the exact extent of mediation after adjusting for measurement error—remains poorly understood. Because the social-cognitive measures used in the present study primarily assessed theory of mind (ToM), this study specifically examined whether ToM-related social cognition statistically medicated the association between neurocognition and social functioning, and identified bridge nodes that may represent potential intervention targets.

Methods: This cross-sectional study recruited clinically stable outpatients with schizophrenia using a consecutive sampling strategy at the Wuhan Mental Health Center, Wuhan, China. Neurocognition was assessed using 4 indices (Digit Symbol Test, Digit Span Test, Continuous Performance Test, and Wisconsin Card Sorting Test categories completed). Social cognition was operationalized primarily through two ToM-focused tasks, the Hinting Task (HT) and Faux Pas Recognition Test (FPRT). Social functioning was evaluated using the 4 domains of the Personal and Social Performance (PSP) scale. Data were analyzed using latent variable structural equation modeling (SEM) and psychological network analysis.

Results: A total of 291 patients (mean [standard deviation (SD)] age, 33.6 [9.6] years; 162 [55.7%] men) were included. Latent variable SEM showed that ToM-related social cognition exhibited a statistically significant indirect effect in the association between neurocognition and social functioning (B = –0.494; 95% bootstrap confidence interval (CI), –1.103 to –0.166), accounting for 38.3% of the total effect. Network analysis revealed a 3-cluster structure wherein the FPRT exhibited the highest bridge strength (0.597), followed by PSP-disturbing and aggressive behaviors (0.517) and HT (0.481), effectively linking the cognitive and functional communities. PSP-personal and social relationships exhibited the highest overall node strength (1.144).

Conclusions: Among Chinese patients with schizophrenia, ToM-related social cognition showed a statistically significant partial mediation pattern in the association between neurocognition and social functioning. ToM-related capacities, particularly faux pas recognition and hint comprehension, emerged as prominent cross-domain bridge nodes in the observed network. These findings suggest that ToM-related processes may be relevant to the association between cognition and functioning; however, because of the cross-sectional design, the results should be interpreted as evidence of indirect association rather than a causal mechanism. Longitudinal and interventional studies are needed to clarify temporal directionality and clinical significance.

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Keywords

schizophrenia; theory-of-mind-related social cognition; neurocognition; social functioning; network analysis

Introduction

Schizophrenia is a chronic and debilitating psychiatric disorder that affects approximately 1% of the world's population, imposing substantial burdens on patients, families, and healthcare systems [1,2]. Beyond its hallmark positive and negative symptoms, schizophrenia is fundamentally characterized by profound impairments in social functioning, including deficits in interpersonal relationships, vocational performance, independent living, and community participation [3,4]. Critically, these social functional deficits tend to persist even after the remission of psychotic symptoms, and they are widely recognized as the strongest predictors of long-term disability and poor quality of life [1,5,6]. In China, where an estimated 6 million individuals live with schizophrenia, social functioning impairments are especially consequential given limited access to community-based rehabilitation services and the pervasive influence of stigma on social reintegration [7,8]. Therefore, identifying potentially modifiable factors associated with social functioning in this population is clinically important for informing future rehabilitation and intervention research.

A substantial body of research over the past two decades has identified neurocognition and social cognition as two of the most consistent and influential predictors of social functioning in schizophrenia [9–11]. Neurocognition encompasses basic information-processing capacities, including attention, processing speed, working memory, verbal learning and memory, and executive function, all of which are typically impaired in schizophrenia and linked to functional outcomes across the course of illness [12]. Social cognition, in contrast, refers to higher-order mental processes involved in perceiving, interpreting, and responding to social information, including emotion perception, theory of mind (ToM), social perception, and attributional style [13,14]. In a meta-analysis, Fett *et al*. [9] demonstrated that social cognition was more strongly associated with community functioning (r = 0.48) than neurocognition (r = 0.22), a finding subsequently confirmed by Halverson *et al*. [11] (2019), highlighting social cognition as a more proximal determinant of real-world outcomes.

Importantly, these two cognitive domains do not operate independently but are thought to be hierarchically organized. Influential theoretical models posit that neurocognition provides the foundational information-processing substrate upon which social cognitive operations are built; basic capacities such as attention and working memory enable accurate encoding and manipulation of social cues, which in turn support complex social cognitive processes like emotion recognition and mental state inference [15]. Consequently, it has been hypothesized that social cognition serves as a critical mediator through which neurocognition exerts its influence on social functioning. Brekke *et al*. [16] were among the first to empirically demonstrate this mediating pathway, finding that social cognition fully mediated the neurocognition–functioning relationship. Subsequent studies have broadly replicated this mediation pattern across various samples and cultural contexts [11,17,18], lending support to social cognition as a key mechanistic bridge linking basic cognitive capacities to everyday social behavior.

Despite this growing consensus, two notable methodological limitations in the existing literature merit attention and have limited the precision and clinical interpretability of current findings.

First, prior mediation analyses have predominantly relied on manifest (observed) variable path analysis, in which composite scores or single test indices represent complex, multidimensional constructs such as neurocognition and social cognition [11,17,18]. Manifest variable approaches implicitly assume that the instruments measuring these constructs are free of measurement error; however, clinical and cognitive assessments are inherently susceptible to measurement unreliability [19]. As demonstrated by Cole and Preacher, conducting mediation analyses with uncorrected measurement error can severely bias path coefficients—either inflating or deflating estimated effects—while drastically reducing statistical power and increasing the risk of both type I and type II errors [20]. This methodological limitation likely accounts, at least in part, for the notable inconsistencies across studies: whereas some report full mediation by social cognition [16], others find only partial mediation [17,21], and at least one study found no evidence that affect recognition mediated the association between attentional functioning and social competence [22]. Structural equation modeling (SEM) with latent variables may help address this issue by explicitly modeling measurement error and estimating relationships among latent constructs rather than observed summated scores, potentially providing more robust estimates of indirect associations [23]. However, latent variable mediation models examining the neurocognition–social cognition–social functioning triad remain scarce, particularly in Chinese patient populations.

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Second, traditional mediation frameworks—whether using manifest or latent variables—operate strictly at the macro-level of aggregated constructs, treating complex, multidimensional domains such as neurocognition and social functioning as monolithic entities. This macro-level approach inevitably obscures the micro-level, indicator-by-indicator interactions that may occur between specific cognitive subdomains and particular aspects of functional outcome. Psychological network analysis (PNA) provides an innovative mathematical framework to resolve this limitation [24,25]. In a psychological network, individual neurocognitive domains, social cognitive sub-tasks, and specific social functioning indicators are modeled as nodes, and the conditional associations between them are represented as edges [26,27]. Critically, bridge centrality metrics can identify bridge nodes—variables that sit at the intersection of different communities and show relatively strong cross-community connections [28]. Such bridge nodes may help identify variables positioned at the interface of different communities and can generate hypotheses about potentially important cross-domain processes [29]. To date, although network analysis has been applied to examine symptom–cognition interactions in schizophrenia, its specific application to delineate the fine-grained architecture connecting neurocognition, social cognition, and social functioning—with explicit identification of bridge nodes across these communities—remains limited, and knowledge regarding which specific cognitive indicators may function as bridge indicators across these communities remains limited [30].

The present study aimed to address these two gaps by integrating latent variable SEM with PNA in a cross-sectional sample of Chinese patients with schizophrenia. These two approaches are complementary: SEM provides a construct-level test of a statistical mediation model while accounting for measurement error, whereas PNA offers an indicator-level map of inter-domain associations and identifies bridge nodes that may have translational relevance. Because social cognition in the present study was operationalized primarily through two ToM-focused tasks, our conclusions are intended to apply most directly to the ToM-related component of social cognition rather than to the full social-cognitive domain. Specifically, we hypothesized that (a) ToM-related social cognition would show a statistically significant indirect effect in the association between neurocognition and social functioning within the latent variable SEM framework, and (b) PNA would identify specific social cognition indicators, particularly ToM-related indicators, as nodes with relatively strong bridge characteristics across the neurocognition, social cognition, and social functioning communities, thereby suggesting their potential clinical relevance for future longitudinal or interventional studies.

Materials and Methods

Study Participants

This was a cross-sectional study conducted between March and May 2024 at the outpatient department of Wuhan Mental Health Center, Wuhan, China. Wuhan Mental Health Center is the largest tertiary-level psychiatric specialty hospital in central China, serving over 420,000 outpatient visits and discharging more than 12,000 inpatients annually in 2024. Notably, approximately 30% of its patient population is drawn from regions beyond Wuhan, including other cities within Hubei Province and neighboring provinces, thereby ensuring a relatively broad and diverse catchment area. Participants were recruited using a consecutive sampling strategy.

Patients were eligible for inclusion if they: (a) met the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition diagnostic criteria (DSM-IV) for schizophrenia [31], as confirmed by attending psychiatrists or above; (b) were aged between 18 and 60 years; (c) were in a clinically stable phase, operationally defined as a total score of $\leq$60 on the Positive and Negative Syndrome Scale (PANSS) [32]; (d) were right-handed and had basic familiarity with computer operations; and (e) were judged by the research team, based on an initial clinical interview and mental status examination, to possess adequate capacity to understand and complete the assessment battery.

Patients were excluded if they: (a) had comorbid organic brain disorders, intellectual disability, dementia, schizoaffective disorder, or delusional disorder; (b) had a current or past substance use disorder; (c) had received electroconvulsive therapy within one month prior to assessment; (d) had neurological diseases, severe physical illnesses, or disabilities that would preclude completion of neuropsychological testing; (e) had color blindness, color weakness, or visual impairment that would prevent identification of computer-presented test materials; (f) were pregnant or breastfeeding; (g) had documented serious suicidal risk or impulsive violent behavior; or (h) were currently receiving long-acting injectable antipsychotic treatment (e.g., risperidone or paliperidone palmitate long-acting injections).

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Research Tools and Procedures

All assessments were administered individually in a standardized, one-on-one format by trained graduate students in applied psychology. Assessors underwent systematic training in the administration and scoring of all measures prior to data collection to ensure procedural consistency.

Demographic and clinical information. A structured questionnaire was used to collect demographic data (e.g., age, sex, education, marital status) and clinical characteristics (e.g., family history and illness duration).

Neurocognition was assessed using four well-established measures tapping distinct cognitive domains:

(1) Digit Symbol Test (DSyT). This subtest from the Chinese revision of the Wechsler Adult Intelligence Scale (WAIS-RC) measures processing speed, attention, and visuomotor coordination [33]. Participants were required to fill in symbols corresponding to the digits 1–9 according to a coding key as quickly as possible within 90 seconds. The total number of correct symbols (range: 0–90) served as the performance index, with higher scores indicating better performance [34,35].

(2) Digit Span Test (DSpT). This WAIS-RC subtest assesses attention and short-term memory and includes forward and backward conditions [33]. Participants repeated increasingly longer sequences of digits in the same (forward) or reverse (backward) order. The total DSpT score (forward + backward; range: 0–22) was used as the outcome measure, with higher scores reflecting better performance [36].

(3) Continuous Performance Test (CPT). The CPT was administered via computer following the protocol of Liu *et al*. [37], using cognitive assessment software developed by Spaulding *et al*. [38], to measure sustained attention. The task comprised three conditions. In Condition 1, participants pressed the space bar whenever the digit "4" appeared on the screen in a single-digit display. In Condition 2, the same rule applied, but multiple digits were displayed simultaneously. In Condition 3, participants were instructed to ignore "4" and respond only to "7", thus requiring cognitive set shifting. Stimuli were presented for 150 ms with a 550 ms interstimulus interval. In the present implementation, each condition contained 20 scored target trials. One point was awarded for each correctly detected target, with no deduction for incorrect responses. The numbers of correct target detections were summed across the three conditions, yielding a possible raw-score range of range: 0–60, with higher scores indicating better performance. This score range is specific to the trial configuration used in the present study rather than a standardized or universally fixed range for the CPT. Because the CPT is a task paradigm rather than a single uniformly scored instrument, the number of trials—and consequently the possible raw-score range—may be adapted according to factors such as the time available for assessment, the research objectives, and the severity of cognitive impairment in the target population [39,40].

(4) Wisconsin Card Sorting Test (WCST). The modified version of the WCST was administered via computer to assess executive function, including abstract reasoning, cognitive flexibility, and the ability to use feedback [41]. Participants sorted 48 cards according to undisclosed matching rules (color, shape, or number) based solely on feedback of "correct" or "incorrect" after each sort. Key performance indices included perseverative errors (i.e., continued application of a previously incorrect sorting rule) and categories completed. Both indices are widely regarded as sensitive markers of prefrontal executive functioning. In the present study, however, only the number of categories completed of Wisconsin Card Sorting Test (WCST-CC, range: 0–6) was included in the statistical analyses, as it provides a more direct summary of overall concept formation and set-shifting performance. Perseverative errors were not entered in order to reduce indicator redundancy and maintain a more parsimonious neurocognitive measurement model.

Social cognition was operationalized primarily through two ToM-focused tasks:

(1) Hinting Task (HT). The HT, adapted and validated in Chinese by Tuo *et al*. [42], assesses the ability to infer the true communicative intent behind indirect speech [43]. Four short vignettes were read aloud to participants, each ending with one character making an indirect request or hint. If the participant correctly identified the intended meaning, a score of 2 was assigned. If the response was incorrect, a more explicit hint was provided; a correct response at this stage scored 1, whereas an incorrect response scored 0. The total score ranged from 0 to 8, with higher scores indicating better hint comprehension [42].

(2) Faux Pas Recognition Test (FPRT). The adult version of the FPRT, which was also adapted and validated in Chinese by Tuo *et al*. [42], was used to evaluate the ability to detect and understand social faux pas, that is, situations in which a speaker unintentionally says something hurtful or inappropriate [44,45]. The task consisted of 10 faux pas stories, each followed by six distinct questions: one de-

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tection question, one identification question, one comprehension question, one intention (false belief) question, one empathy question, and one control question assessing basic story comprehension. According to the scoring protocol [42], the first five questions are faux pas-related. One point was awarded for each correct answer to these five questions. The sixth question is a control question; if it was answered incorrectly, the data for that story were excluded. Therefore, a maximum of 5 points could be earned for each story, resulting in a total faux pas score ranging from 0 to 50, with higher scores indicating better ToM ability [42].

Because both HT and FPRT primarily assess mental-state inference, the social cognition construct in the present study should be interpreted as reflecting the ToM-related component of social cognition rather than the entire social-cognitive domain. Other major domains, including emotion processing, social perception, and attributional style, were not directly assessed.

Social functioning assessment. Social functioning was assessed using the Personal and Social Performance (PSP) Scale, a clinician-rated instrument specifically designed for evaluating psychosocial functioning in patients with schizophrenia [46]. The PSP provides a global score ranging from 1 to 100 and assesses functioning across four domains: (a) socially useful activities, including work and study (PSP1); (b) personal and social relationships (PSP2); (c) self-care (PSP3); and (d) disturbing and aggressive behaviors (PSP4). Each domain is rated on a six-point severity scale (absent, mild, moderate, marked, severe, very severe), with higher scores indicating greater impairment and poorer psychosocial functioning. In the present study, the four domain ratings were retained as PSP1–PSP4 and used as observed indicators of the latent social functioning construct in the SEM; the derived global PSP score was not used. The Chinese version of the PSP has demonstrated satisfactory reliability and validity in both acute and stable-phase schizophrenia patients [47].

Statistical Analysis

All statistical analyses were conducted in R version 4.6.0 (R Core Team, R Foundation for Statistical Computing, Vienna, Austria). All tests were two-sided, and statistical significance was set at $p < 0.05$. Descriptive statistics were calculated using the psych package. The distribution of continuous variables was assessed using the Shapiro–Wilk test, together with visual inspection of histograms and Q–Q plots. Normally distributed variables were summarized as mean ± standard deviation (SD), whereas skewed variables were summarized as median and interquartile range. For completeness and comparability with previous studies, mean ± SD was also retained as supplementary descriptive information where appropriate. Because most continuous variables included in the correlation analyses showed non-normal or skewed distributions, associations between variables were assessed using Spearman's rank correlation coefficients. Latent variable mediation analysis was conducted using lavaan and semTools and visualized using semPlot. PNA was conducted using qgraph, bootnet, and networktools.

To examine the associations among neurocognition, social cognition, and social functioning at the construct level, SEM was performed using a statistical mediation model in which social cognition was specified as an intervening latent construct between neurocognition and social functioning. In the measurement model, neurocognition was indicated by DSyT, DSpT, CPT, and WCST-CC; social cognition by HT and FPRT; and social functioning by the four PSP domain scores (PSP1–PSP4). To maintain consistency with the network analysis, PSP1–PSP4 were retained in their original coding and were not reverse scored. After the measurement model was established, the structural model was tested by specifying paths from neurocognition to social cognition, from social cognition to social functioning, and from neurocognition directly to social functioning. Direct, indirect, and total effects were estimated. Model fit was evaluated using the Chi-square statistic, comparative fit index (CFI), Tucker-Lewis index (TLI), root mean square error of approximation (RMSEA), and standardized root mean square residual (SRMR). CFI and TLI values >0.90 indicated acceptable fit and >0.95 indicated good fit, whereas RMSEA and SRMR values <0.08 indicated acceptable fit and <0.06 indicated good fit [48]. The significance of the mediation effect was assessed using bootstrap resampling (6000 resamples) with 95% confidence intervals (CIs).

The PNA was conducted at the observed-variable level. The network included 10 observed variables: DSyT, DSpT, CPT, WCST-CC, HT, FPRT, and PSP1–PSP4. Because several variables were non-normally distributed, a nonparanormal transformation was applied before estimating a regularized partial correlation network using the EBICglasso method. Node importance was quantified by strength centrality. To examine the positions of nodes connecting different domains, bridge strength was computed after assigning nodes a priori to three communities: neurocognition, social cognition, and social functioning. The stability of strength and bridge strength was evaluated using a case-dropping bootstrap procedure. Stability was quantified by the correlation stability coefficient (CSC), with CSC > 0.25 indicating acceptable stability and CSC > 0.50 indi-

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cating good stability [49,50]. The accuracy of edge weights was further evaluated using nonparametric bootstrap 95% CIs, with narrower intervals indicating greater precision.

Results

Descriptive Statistics

A total of 346 outpatients with schizophrenia were screened for eligibility. Of these, 291 met the inclusion and exclusion criteria, completed the assessments, and were included in the final analyses. The remaining 55 individuals were not enrolled for the following reasons: not meeting the criterion of clinical stability (PANSS total score >60; n = 18); declining to provide written informed consent by the patient and/or family members (n = 10); being judged by the treating psychiatrists to lack adequate capacity to understand and complete the assessment battery (n = 6); not being right-handed and/or lacking basic familiarity with computer operations (n = 5); currently receiving long-acting injectable antipsychotic treatment (n = 5); and other reasons (n = 11), including age outside the 18–60 range, comorbid organic brain disorders or substance use disorders, neurological or severe physical conditions that precluded testing, recent electroconvulsive therapy, documented serious suicidal or impulsive risk, and incomplete assessments.

Of the included patients, 162 (55.7%) were men, with a mean age of 33.6 ± 9.6 years. Other demographic and clinical characteristics are presented in Table 1.

The mean scores of the four PSP subscales, the four neurocognitive indicators, and the two social cognition indicators, as well as their Spearman correlation coefficients, are presented in Table 2.

Latent Variable SEM of the Statistical Mediation Model Involving Social Cognition, Neurocognition, and Social Functioning

The measurement model demonstrated acceptable fit, $\chi^2(32) = 61.42$, $p = 0.001$, CFI = 0.958, TLI = 0.941, RMSEA = 0.057 (90% CI [0.035, 0.078]), and SRMR = 0.037.

At the structural level, neurocognition was significantly associated with better social cognition (unstandardized coefficient [B] = 1.014, $p < 0.001$, standardized coefficient [β] = 0.712). Social cognition was also significantly associated with social functioning (B = –0.487, p = 0.017, β = –0.407). In addition, the direct association between neurocognition and social functioning remained statistically significant in the model (B = –0.797, p = 0.004, β = –0.468).

The bootstrapped indirect effect of neurocognition on social functioning through social cognition was significant (B = –0.494, 95% bootstrap CI [–1.103, –0.166], β = –0.290), indicating a significant mediating role of social cognition. The total effect of neurocognition on social functioning was also significant (B = –1.290, $p <$ 0.001, β = –0.757). The indirect effect via social cognition accounted for approximately 38.3% of the total effect (–0.494/–1.290), whereas the direct effect accounted for approximately 61.8% of the total effect (–0.797/–1.290). Fig. 1 presents the partial mediation model linking neurocognition, social cognition, and social functioning.

Network Analysis of Neurocognition, Social Cognition, and Social Functioning

The network exhibited a three-cluster structure (Fig. 2). Both neurocognitive and social cognitive nodes showed cross-domain edges with social functioning nodes. Among all edges, the PSP2–PSP3 edge (0.315) showed the largest weight, followed by the HT-FPRT (0.284) and PSP1–PSP2 (0.277) edges. Bootstrapped 95% CIs for edge weights are shown in Fig. 3. PSP-personal and social relationships (PSP2) showed the highest observed node strength (1.144), followed by FPRT (0.880) and PSP-disturbing and aggressive behaviors (PSP4; 0.842) (Fig. 4). Regarding bridge nodes, FPRT showed the highest observed bridge strength (0.597), followed by PSP4 (0.517) and HT (0.481) (Fig. 4). As shown in Fig. 5, the CSCs for node strength and bridge strength in the case-dropping procedure were 0.515 and 0.361, respectively.

Discussion

The present study examined how neurocognition, social cognition, and real-world social functioning are linked in Chinese outpatients with clinically stable schizophrenia. By combining latent variable SEM with PNA, we evaluated these relationships at both the construct level and the indicator level. At the macro-level, the SEM results showed that ToM-related social cognition partially mediated the association between neurocognition and social functioning, accounting for 38.3% of the total effect. At the micro-level, network analysis further indicated that FPRT, HT, and PSP4 were among the nodes with the highest observed bridge strength in the network. Notably, two of the three bridge nodes with the highest observed bridge strength were social cognition indicators, supporting the relevance of ToM-related processes while also suggesting that bridge central-

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Table 1. Demographic and clinical characteristics of the sample of patients with schizophrenia.

Variable	n/Mean	%/SD
Sex		
Males	162	55.7
Females	129	44.3
Age (years): 18–60 (range)	33.6	9.6
Educational attainment		
Primary school	18	6.2
Junior middle school	98	33.7
Senior middle school or secondary specialized school	84	28.9
Junior college	56	19.2
Bachelor or above	35	12.0
Marital status		
Never married	135	46.4
Married	128	44.0
Re-married	12	4.1
Separated/divorced	16	5.5
Family history of mental disorders		
Positive	82	28.2
Negative	209	71.8
Duration of mental illness (years): 0.1–35.0 (range), median: 5.0, interquartile range: 10	7.3	7.0

SD, standard deviation.

Table 2. Medians, means, and Spearman correlations among observed variables of social functioning, neurocognition, and social cognition.

Variable	Median, interquartile range	Mean ± SD	PSP1	PSP2	PSP3	PSP4	DSyT	DSpT	CPT	WCST-CC	HT	FPRT
PSP1	4, 1	3.44 ± 1.55	1.000	0.592***	0.455***	0.368***	–0.241***	–0.238***	–0.294***	–0.294***	–0.328***	–0.347***
PSP2	3, 1	2.64 ± 1.11		1.000	0.577***	0.448***	–0.246***	–0.338***	–0.347***	–0.189**	–0.348***	–0.337***
PSP3	2, 1	1.90 ± 0.96			1.000	0.330***	–0.211***	–0.276***	–0.226***	–0.179**	–0.272***	–0.288***
PSP4	2, 1	1.99 ± 0.97				1.000	–0.261***	–0.321***	–0.342***	–0.286***	–0.332***	–0.306***
DSyT	40, 16	39.57 ± 10.96					1.000	0.232***	0.229***	0.193***	0.139*	0.278***
DSpT	12, 3	11.49 ± 1.85						1.000	0.331***	0.337***	0.307***	0.329***
CPT	36, 6	35.10 ± 6.10							1.000	0.235***	0.303***	0.188**
WCST-CC	2, 3	1.91 ± 1.62								1.000	0.273***	0.264***
HT	6, 4	5.71 ± 2.09									1.000	0.448***
FPRT	22, 15	23.19 ± 10.56										1.000

SD, standard deviation; PSP1, Personal and Social Performance Scale, socially useful activities; PSP2, Personal and Social Performance Scale, personal and social relationships; PSP3, Personal and Social Performance Scale, self-care; PSP4, Personal and Social Performance Scale, disturbing and aggressive behaviors; DSyT, Digit Symbol Test; DSpT, Digit Span Test; CPT, Continuous Performance Test; WCST-CC, categories completed of Wisconsin Card Sorting Test; HT, Hinting Task; FPRT, Faux Pas Recognition Test. The correspondence between *p*-values and significance symbols: *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

ity was not limited to the social cognition domain. Taken together, these findings suggest that the ToM-related component of social cognition represents an important pathway through which neurocognitive deficits may translate into functional disability, while also indicating that specific functional behaviors such as PSP4 may occupy a prominent bridging position within the broader cognitive-functional network.

These findings should be interpreted within the broader conceptual framework of social cognition in schizophrenia. Current consensus literature defines social cognition as a multidimensional construct that includes emotion processing, social perception, ToM/mental state

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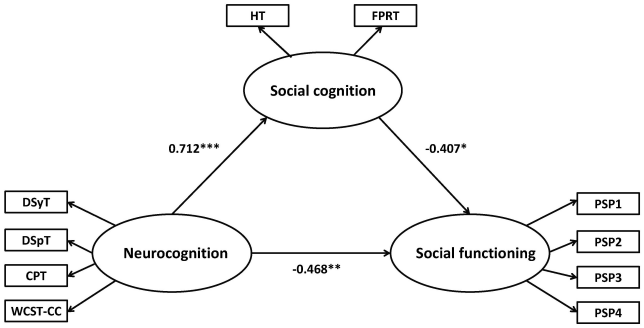


Fig. 1. Latent variable structural equation model of social cognition as a statistical mediator between neurocognition and social functioning. The latent-variable mediation model indicated that better neurocognition was associated with better social cognition and social functioning. Social cognition was also positively associated with social functioning, suggesting that it may mediate the relationship between neurocognition and social functioning. The latent constructs were represented by their respective observed indicators. The arrows indicate the hypothesized direction of the relationships between variables, with the arrowhead pointing from the predictor to the outcome variable. PSP1, Personal and Social Performance Scale, socially useful activities; PSP2, Personal and Social Performance Scale, personal and social relationships; PSP3, Personal and Social Performance Scale, self-care; PSP4, Personal and Social Performance Scale, disturbing and aggressive behaviors; DSyT, Digit Symbol Test; DSpT, Digit Span Test; CPT, Continuous Performance Test; WCST-CC, categories completed of Wisconsin Card Sorting Test; HT, Hinting Task; FPRT, Faux Pas Recognition Test. The correspondence between *p*-values and significance symbols: $*p < 0.05$, $**p < 0.01$, $***p < 0.001$.

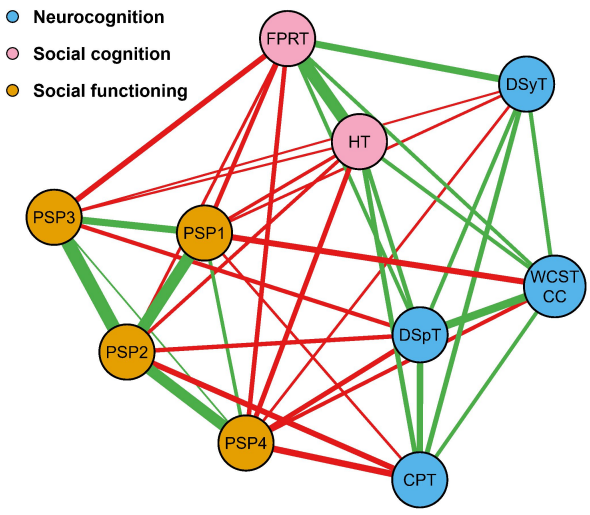


Fig. 2. Network structure of neurocognition, social cognition, and social functioning. The network analysis revealed dense interconnections among neurocognition, social cognition, and social functioning indicators. Each circle (node) represents an individual indicator or measure, and the node colors denote the corresponding domain: blue for neurocognition, pink for social cognition, and orange for social functioning. The lines represent associations between indicators; green lines indicate positive associations, whereas red lines indicate negative associations. Line width reflects the strength of the association, with thicker lines indicating stronger relationships. Overall, the network showed substantial within- and cross-domain relationships, with particularly strong connections among several social functioning indicators. PSP1, Personal and Social Performance Scale, socially useful activities; PSP2, Personal and Social Performance Scale, personal and social relationships; PSP3, Personal and Social Performance Scale, self-care; PSP4, Personal and Social Performance Scale, disturbing and aggressive behaviors; DSyT, Digit Symbol Test; DSpT, Digit Span Test; CPT, Continuous Performance Test; WCST-CC, categories completed of Wisconsin Card Sorting Test; HT, Hinting Task; FPRT, Faux Pas Recognition Test.

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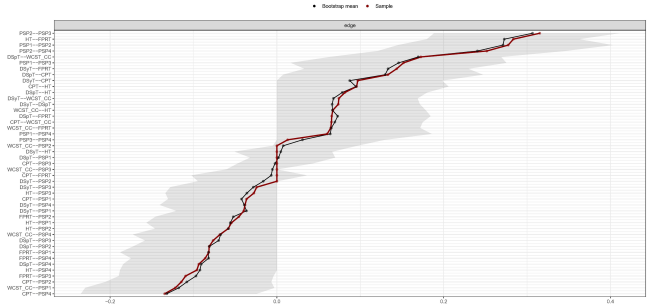


Fig. 3. Bootstrapped 95% CIs for edge weights. This figure shows the estimated edge weights in the sample and their bootstrapped 95% CIs. The red line represents the edge weights from the original sample, whereas the black points represent the bootstrap mean estimates. Narrower CIs indicate more accurate and stable edge-weight estimates, while wider intervals suggest greater uncertainty and should be interpreted with caution. Overall, the strongest edges appear to be estimated more precisely than the weaker edges. PSP1, Personal and Social Performance Scale, socially useful activities; PSP2, Personal and Social Performance Scale, personal and social relationships; PSP3, Personal and Social Performance Scale, self-care; PSP4, Personal and Social Performance Scale, disturbing and aggressive behaviors; DSyT, Digit Symbol Test; DSpT, Digit Span Test; CPT, Continuous Performance Test; WCST-CC, categories completed of Wisconsin Card Sorting Test; HT, Hinting Task; FPRT, Faux Pas Recognition Test.

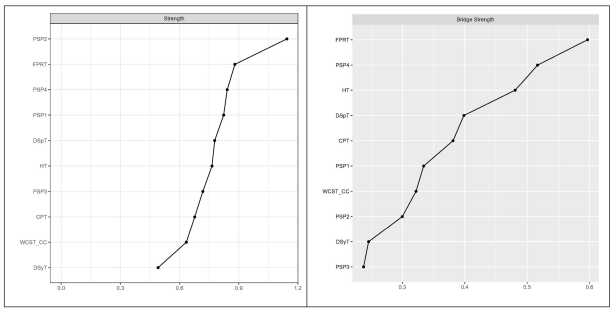


Fig. 4. Node strength and bridge strength ranked in descending order in the network of neurocognition, social cognition, and social functioning. The left panel presents the strength centrality of each node, indicating its overall connectivity within the network. PSP2 showed the highest strength, followed by FPRT and PSP4, whereas DSyT showed the lowest strength. The right panel presents bridge strength, reflecting each node's connectivity across different domains. FPRT had the highest bridge strength, followed by PSP4 and HT, while PSP3 and DSyT had the weakest bridge connections. PSP1, Personal and Social Performance Scale, socially useful activities; PSP2, Personal and Social Performance Scale, personal and social relationships; PSP3, Personal and Social Performance Scale, self-care; PSP4, Personal and Social Performance Scale, disturbing and aggressive behaviors; DSyT, Digit Symbol Test; DSpT, Digit Span Test; CPT, Continuous Performance Test; WCST-CC, categories completed of Wisconsin Card Sorting Test; HT, Hinting Task; FPRT, Faux Pas Recognition Test.

attribution, and attributional style or bias [51–53]. Accordingly, our results should not be interpreted as showing that ToM is equivalent to the full domain of social cognition. Rather, because the HT and FPRT primarily assess mental-state inference, the present study most directly supports the mediating and bridging role of ToM-related social cognition. This distinction is important conceptually and strengthens the precision of our conclusions.

At the same time, focusing on ToM is theoretically and clinically meaningful. Prior reviews and meta-analyses indicate that ToM impairment is a robust feature of schizophrenia and is observable across illness stages, including first-episode psychosis and high-risk populations [54,55]. More recent evidence also indicates that ToM is meaningfully associated with functional outcomes in schizophrenia and may account for a substantial portion of the relationship between cognition and everyday functioning [9,11,56]. Against this background, our results extend the literature by showing not only a construct-level mediating effect, but also a node-level bridging role for specific ToM indicators.

Our latent variable SEM findings align with and

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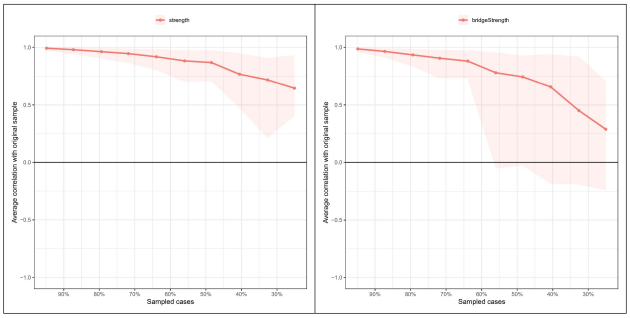


Fig. 5. The stability of strength and bridge strength using case-dropping bootstrap. The x-axis indicates the percentage of cases of the original sample included at each step. The y-axis indicates the average of correlations between the centrality indices from the original network and the centrality indices from the networks that were re-estimated after excluding increasing percentages of cases.

substantially extend the neurodevelopmental cascade hypothesis of schizophrenia [57]. This framework posits that neurocognitive impairments form the foundational information-processing restrictions upon which higher-order social cognitive operations are scaffolded. Basic capacities, such as processing speed (indexed by the DSyT) and attention or working memory (indexed by the DSpT and CPT), determine the "computational bandwidth" of the central nervous system. When this bandwidth is severely constrained by the primary pathophysiology of the disorder, the real-time processing of high-density, dynamic social information becomes compromised, leading to profound deficits in social inference.

Methodologically, the latent variable approach allowed us to estimate the mediation pathway at the construct level while reducing the influence of measurement error [20]. This may help explain why previous studies using manifest indicators have reported variable findings regarding the magnitude, and sometimes even the presence, of indirect effects [16,17]. In our model, ToM-related social cognition accounted for a substantial proportion of the total association between neurocognition and social functioning, but a sizeable direct pathway remained (61.8%). This pattern suggests that although ToM-related processes are important, neurocognition may also affect functioning through non-social routes, such as attentional, executive, or task-management demands that are relevant to independent living and community participation. For instance, impairments in executive function (WCST-CC) or sustained attention (CPT) can directly disrupt independent living skills, workplace task execution, and logistical planning, independent of a patient's interpersonal capacities.

The network findings provide a more fine-grained interpretation of this pathway. Among all nodes, faux pas recognition and hint comprehension ranked among the bridge nodes with the highest observed bridge strength between the cognitive and functional communities, while PSP4 also showed relatively high bridge strength. Although the bridge-centrality findings did not indicate that bridge nodes were restricted to the social cognition domain, two of the three strongest bridge nodes were social cognition indicators (FPRT and HT), which is broadly consistent with our theoretical emphasis on ToM-related processes. Both tasks require individuals to infer others' intentions, beliefs, and implied meanings from socially embedded information. Their central bridging positions are therefore consistent with the view that ToM represents a higher-order social-cognitive process that depends on the integration of contextual, interpersonal, and cognitive information. In everyday life, difficulty understanding indirect communication or recognizing unintended social mistakes may disrupt conversation, increase interpersonal misunderstandings, and weaken relationship maintenance. This may help explain why ToM-related deficits can have downstream relevance for broader social functioning.

An important contribution of this study is that FPRT and HT emerged as prominent bridge nodes across the cognitive and functional communities. Both tasks are gold-standard measures of ToM—the mentalizing capacity to infer others' hidden intentions, beliefs, and emotional states [43,45]. From a cognitive neuroscience perspective, the prominent bridging positions of these ToM indicators can be explained by the profound reliance of the "mentalizing network" (primarily involving the medial prefrontal cortex, superior temporal sulcus, and temporoparietal junction) on foundational neurocognitive integrity [55, 58]. Schizophrenia is characterized by a sparser, poorly integrated mentalizing network connectivity. When a patient encounters complex social scenarios—such as identifying an unintentional social gaffe (FPRT) or deciphering a subtle, indirect request (HT)—the task demands simulta-

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neous maintenance of multiple perspectives, abstract reasoning, and context-dependent cognitive flexibility. Consequently, ToM-related nodes may represent an important structural bottleneck through which underlying neurocognitive resource scarcity is associated with downstream social-cognitive and functional difficulties.

Furthermore, the micro-level architecture provides a potentially informative behavioral pattern linking cognition and functioning. The network revealed direct, negative edges involving the ToM-related bridge nodes and disturbing and aggressive behaviors (PSP4), which itself also emerged as one of the strongest bridge nodes. In the context of impaired mentalizing, patients frequently suffer from hostile attribution biases. When unable to accurately decode an ambiguous social cue or recognize that a hurtful remark was an innocent, accidental faux pas, patients may be more likely to misinterpret the interaction as deliberate or threatening. Such misattributions may contribute to defensive verbal or physical aggression (PSP4), which in turn may interfere with interpersonal relationships and broader community functioning.

To ensure that these micro-level topological inferences were methodologically rigorous and suitable for clinical translation, we subjected the network to an exhaustive case-dropping bootstrap procedure [24]. The resulting CSCs were 0.515 for node strength and 0.361 for bridge strength, both exceeding 0.25 and indicating acceptable-to-good stability. These findings suggest that the prominent bridging roles of faux pas recognition and hint comprehension are relatively stable features of the network architecture rather than artifacts of sampling variability. Nevertheless, these findings should be interpreted cautiously, as bridge centrality does not by itself indicate that intervening on these nodes will improve connected domains. Instead, these nodes may be considered candidate targets for future longitudinal and interventional research.

From a clinical perspective, the bridge findings may help prioritize ToM-related processes for further assessment and hypothesis-driven intervention development, while also highlighting the relevance of specific behavioral-functional nodes such as PSP4 within the broader network architecture. However, bridge centrality in a cross-sectional network reflects topological position rather than causal leverage [29]. Therefore, the present results should not be interpreted as demonstrating that directly modifying faux pas recognition or hint comprehension will necessarily improve functioning. Instead, these nodes should be viewed as candidate targets for future longitudinal and interventional studies.

Rather than adopting generalized training, clinical programs should prioritize targeted social-cognitive interventions that explicitly train mentalizing and perspective-taking capacities, such as Social Cognition and Interaction Training [59,60]. By specifically reinforcing a patient's capacity to recognize social faux pas and interpret indirect communication, clinicians can efficiently uncouple underlying, unmodifiable neurocognitive deficits from real-world functional deterioration. This streamlined approach could maximize the therapeutic yield per intervention hour, substantially reducing interpersonal conflicts and aggressive incidents in community care settings. Nevertheless, because our assessment emphasized ToM rather than the entire social-cognitive domain, the present findings should be taken as supporting targeted mentalizing interventions in particular, not all forms of social-cognitive training equally.

Several limitations of this study warrant consideration.

First, the cross-sectional design precludes conclusions about temporal precedence or causal directionality among the identified nodes. Although the neurodevelopmental cascade model provides a theoretical basis for the proposed pathways, longitudinal or ecological momentary assessment studies are needed to determine whether these relationships unfold dynamically over time.

Second, the eligibility criteria may have limited generalizability and introduced selection bias. Participants were clinically stable, right-handed outpatients aged 18–60 years with adequate capacity, basic computer literacy, and the ability to complete demanding computerized cognitive assessments; those receiving long-acting injectable treatment were excluded. These criteria may have favored a relatively stable, higher-functioning subgroup and reduced clinical heterogeneity. The findings should therefore be generalized cautiously to acutely ill, older, more severely impaired, or otherwise clinically complex patients, in whom network topology may differ.

Third, social cognition was assessed primarily using two ToM tasks. Other domains, including emotion processing, social perception, and attributional style or bias, were not directly examined. Thus, the findings apply mainly to ToM-related processes rather than to social cognition more broadly.

Fourth, several potentially relevant confounders were not fully addressed. Education and illness duration were reported descriptively but were not included as covariates in the SEM or network models, because additional adjustment or subgroup analyses in this modest sample could

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have reduced power and model stability. Within this framework, such factors may also exert distributed effects across multiple cognitive and functional indicators. PANSS subscale scores, chlorpromazine-equivalent dose, anticholinergic burden, and other medication-related factors were not systematically available and could not be examined directly. These variables may have influenced the observed mediation and bridge-centrality patterns.

Fifth, the bridge-strength stability coefficient was 0.361, indicating acceptable but limited stability. The bridge-centrality results, particularly the exact ranking of bridge nodes, should therefore be interpreted cautiously. Moreover, because the network was cross-sectional, bridge centrality should not be taken as evidence that intervening on a given node would causally improve connected domains.

Sixth, schizophrenia was diagnosed using DSM-IV criteria during eligibility assessment, without a structured or semi-structured diagnostic interview because of feasibility constraints in the real-world outpatient setting. Future studies should use updated diagnostic criteria and standardized diagnostic procedures.

Finally, larger longitudinal studies with broader multidomain social-cognitive batteries and more comprehensive clinical and medication characterization are needed to test whether the present mediation and bridge-node findings generalize beyond ToM-related processes.

Conclusions

In conclusion, the ToM-related component of social cognition appears to represent an important pathway linking neurocognitive deficits and impaired social functioning in schizophrenia. Using latent variable SEM and PNA, this study identified faux pas recognition and indirect hint comprehension as two of the most prominent bridge nodes in the observed cognitive-functional network, while PSP4 also emerged as a strong bridge node. Although these findings highlight the potential relevance of ToM-related processes, they should not be interpreted as evidence that intervening on these nodes will necessarily improve functional outcomes. Moreover, the present results apply most directly to the ToM-related component of social cognition rather than the full social-cognitive domain. Future longitudinal, interventional, and broader multidomain studies are needed to clarify the causal and generalizable significance of these pathways.

Availability of Data and Materials

De-identified datasets supporting the current study are available from the corresponding author upon reasonable request, with proper ethical approval and a data use agreement.

Author Contributions

YZ and XBL: data acquisition, analysis, and manuscript drafting; YZ and BLZ: study design, manuscript revision, and data interpretation. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study protocol was approved by the Ethics Committee of Wuhan Mental Health Center prior to data collection (Approval number: KY2024.0227.01). The study was conducted in accordance with the ethical principles outlined in the 2024 Declaration of Helsinki. All participants and their guardians were informed about the purpose of the study, assured of confidentiality, and provided written consent prior to participation. Participation was voluntary, and respondents could withdraw at any time without consequence.

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Conflict of Interest

The authors declare that they have no competing interests.

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